



NADONA

**INFECTION
PREVENTIONIST
BOARD CERTIFICATION
REVIEW COURSE
RESOURCE TOOLKIT**

Updated February 2017

Acute Gastroenteritis / Norovirus Case Report Worksheet

Reporting facility: _____ Contact Name/Phone Number: _____ Estimated number of exposed patients during outbreak

Street Address: _____ Outbreak Identification Number (Health Dept. assigned) _____ Estimated number of exposed staff during outbreak

Unit: _____

Patient/Staff Demographics					Case Location	Symptoms					Outcome		Diagnostics				
Name	Unique ID (optional)	Patient (P) Staff (S)	Age	Sex (M/F)	Patients only: Room/Bed	Symptom onset date (mm/dd/yy)	Vomiting (Y/N)	Diarrhea (Y/N)	Bloody stools (Y/N)	Fever (Y/N)	Abdominal cramps (Y/N)	First symptom-free date (mm/dd/yy)	Died (Y/N/Unk)	Specimen(s) collected for diagnostics (Y/N/Unk)	Date of specimen collection (mm/dd/yy)	Lab Results	Location of stool specimen testing (H=HCF lab, C=contracted lab, S=state lab, CD=CDC lab)
1.																	
2.																	
3.																	
4.																	
5.																	
6.																	
7.																	
8.																	
9.																	
10.																	
11.																	
12.																	
13.																	
14.																	
15.																	
16.																	
17.																	
18.																	
19.																	
20.																	

If required, REDACT Name column prior to faxing; FAX to local/state health department upon completion

NOROVIRUS

What healthcare providers should know

What is norovirus?

A virus that can cause severe and sudden gastroenteritis (i.e., inflammation of the lining of the stomach and intestines). Both healthy and compromised persons can be affected.

What are the symptoms?

Nausea, vomiting, diarrhea, and some stomach cramping

Is it contagious?

Norovirus is very easily transmitted through contaminated hands, equipment/surfaces, or food/water

What can I do to prevent norovirus?

Always perform appropriate hand hygiene, particularly after contact with fecal material or after contact with anyone suspected /confirmed with norovirus. Wear gloves when caring for symptomatic patients.

If you have symptoms consistent with norovirus infection, stay home for a *minimum* of 48 hrs after symptom resolution

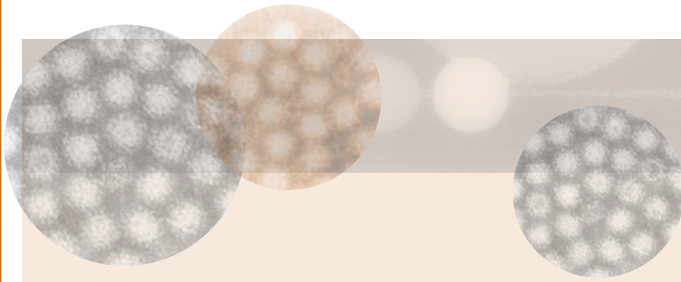
If an outbreak is suspected contact Infection Prevention and Control

► For more information, visit www.cdc.gov



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

Norovirus in Healthcare Facilities Fact Sheet



General Information

Virology

Noroviruses (genus *Norovirus*, family *Caliciviridae*) are a group of related, single-stranded RNA, non-enveloped viruses that cause acute gastroenteritis in humans. Norovirus is the official genus name for the group of viruses provisionally described as “Norwalk-like viruses”. Currently, human noroviruses belong to one of three norovirus genogroups (GI, GII, or GIV), which are further divided into >25 genetic clusters. Over 75% of confirmed human norovirus infections are associated with genotype GII.

Clinical manifestations

The average incubation period for norovirus-associated gastroenteritis is 12 to 48 hours, with a median period of approximately 33 hours. Illness is characterized by nausea, acute-onset vomiting, and watery, non-bloody diarrhea with abdominal cramps. In addition, myalgia, malaise, and headache are commonly reported. Low-grade fever is present in about half of cases. Dehydration is the most common complication and may require intravenous replacement fluids. Symptoms usually last 24 to 60 hours. Up to 30% of infections may be asymptomatic.

Epidemiology of transmission

Noroviruses are highly contagious, with as few as 18 virus particles thought to be sufficient to cause infection. This pathogen is estimated to be the causative agent in over 21 million gastroenteritis cases every year in the United States, representing approximately 60% of all acute gastroenteritis cases from known pathogens. Noroviruses are transmitted primarily through the fecal-oral route, either by direct person-to-person spread or fecally contaminated food or water. Noroviruses can also spread via a droplet route from vomitus. These viruses are relatively stable in the environment and can survive freezing and heating to 60°C (140°F). In healthcare facilities, transmission can also occur through

hand transfer of the virus to the oral mucosa via contact with materials, fomites, and environmental surfaces that have been contaminated with either feces or vomitus.

Norovirus infections are seen in all age groups, although severe outcomes and longer durations of illness are most likely to be reported among the elderly. Among hospitalized persons who are immunocompromised or have significant medical comorbidities, norovirus infection can directly result in prolonged hospital stays, additional medical complications, and, rarely, death. There is currently no vaccine available for norovirus and, generally, no specific medical treatment is offered for norovirus infection apart from oral or intravenous repletion of volume.

The ease of its transmission, a very low infectious dose, a short incubation period, environmental persistence, and lack of durable immunity following infection enables norovirus to spread rapidly through confined populations. Healthcare facilities and other institutional settings (e.g., daycare centers, schools, etc.) are particularly at-risk for outbreaks because of increased person-to-person contact. Healthcare facilities managing outbreaks of norovirus gastroenteritis may experience significant costs relating to isolation precautions and personal protective equipment, ward closures, supplemental environmental cleaning, staff cohorting or replacement, and sick time.

Diagnosis of norovirus infection

Diagnosis of norovirus infection relies on the detection of viral RNA in the stools of affected persons, by use of reverse transcription-polymerase chain reaction (RT-PCR) assays. This technology is available at CDC and most state public health laboratories and should be considered in the event of outbreaks of gastroenteritis in healthcare facilities. Enzyme immune-assays may also be used for identification of norovirus outbreak but are not recommended for diagnosis of individuals. Identification of the virus can be best made from stool specimens taken within 48 to 72 hours after onset of symptoms, although positive results can be obtained by using RT-PCR on samples taken as long as 7 days after symptom onset. Because of the limited availability of timely and routine laboratory diagnostic methods, a clinical diagnosis of norovirus infection is often used, especially when other agents of gastroenteritis have been ruled out.



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention



Measures to Limit Transmission*

Patient Cohorting and Isolation Precautions

Avoid exposure to vomitus or diarrhea. Place patients on Contact Precautions in a single occupancy room if they present with symptoms consistent with norovirus gastroenteritis.

Hand Hygiene

During outbreaks, use soap and water for hand hygiene after providing care or having contact with patients suspected or confirmed with norovirus gastroenteritis.

Patient Transfer and Ward Closure

Consider limiting transfers to those for which the receiving facility is able to maintain Contact Precautions; otherwise, it may be prudent to postpone transfers until patients no longer require Contact Precautions. During outbreaks, medically suitable individuals recovering from norovirus gastroenteritis can be discharged to their place of residence.

Diagnostics

In the absence of clinical laboratory diagnostics or in the case of delay in obtaining laboratory results, use Kaplan's clinical and epidemiologic criteria to identify a norovirus gastroenteritis outbreak.

Kaplan's Criteria

1. Vomiting in more than half of symptomatic cases and,
2. Mean (or median) incubation period of 24 to 48 hours and,
3. Mean (or median) duration of illness of 12 to 60 hours and,
4. No bacterial pathogen isolated in stool culture

Environmental Cleaning

Increase the frequency of cleaning and disinfection of patient care areas and frequently touched surfaces during outbreaks of norovirus gastroenteritis (e.g., increase ward/unit level cleaning to twice daily to maintain cleanliness, with frequently touched surfaces cleaned and disinfected three times daily using the US Environmental Protection Agency's list of approved products for healthcare settings (<http://www.epa.gov/oppad001/chemregindex.htm>).

Staff Leave and Policy

Develop and adhere to sick leave policies for healthcare personnel who have symptoms consistent with norovirus infection.

Exclude ill personnel from work for a minimum of 48 hours after the resolution of symptoms. Once personnel return to work, the importance of performing frequent hand hygiene should be reinforced, especially before and after each patient contact.

Establish protocols for staff cohorting in the event of an outbreak of norovirus gastroenteritis. Ensure staff care for one patient cohort on their ward and do not move between patient cohorts (e.g., patient cohorts may include symptomatic, asymptomatic exposed, or asymptomatic unexposed patient groups).

Communication and Notification

As with all outbreaks, notify appropriate local and state health departments, as required by state and local public health regulations, if an outbreak of norovirus gastroenteritis is suspected.

*Prevention and control recommendations taken from priority recommendations in the CDC HICPAC Guideline for the Prevention and Control of Norovirus Gastroenteritis Outbreaks in Healthcare Settings (<http://www.cdc.gov/hicpac/pdf/norovirus/Norovirus-Guideline-2011.pdf>)

Date last modified: September 6, 2011

Content source: Division of Healthcare Quality Promotion (DHQP), National Center for Preparedness, Detection, and Control of Infectious Diseases (NCEZID)

Contact Us: Centers for Disease Control and Prevention
1600 Clifton Road, Atlanta, GA 30333, USA

1-800-CDC-INFO (1-800-232-4636)

TTY:888-232-6348,

24 hours/everyday at cdcinfo@cdc.gov (TTY)



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

A Norovirus Outbreak Control Resource Toolkit for Healthcare Settings

Norovirus is the most common cause of sporadic gastroenteritis as well as gastroenteritis outbreaks. Because of high levels of contact and vulnerable patient populations, healthcare settings can be particularly susceptible to outbreaks of norovirus. To help address the challenges of managing and controlling norovirus gastroenteritis outbreaks in healthcare settings, the Centers for Disease Control and Prevention (CDC) is offering a toolkit for healthcare professionals including up-to-date information, recommended infection control measures, and tools for outbreak response coordination and reporting.

The toolkit serves as a complementary resource to the CDC HICPAC Guideline for the Prevention and Control of Norovirus Gastroenteritis Outbreaks in Healthcare Settings, 2011 (<http://www.cdc.gov/hicpac/norovirus/pubs.html>). These resources were jointly developed by CDC's Division of Healthcare Quality Promotion and Division of Viral Diseases and in consultation with infection preventionists around the country.

For healthcare professionals, the toolkit contains a variety of materials to support outbreak response as well as staff and patient education efforts including:

- ▶ A presentation on general norovirus epidemiology, infection control measures, and outbreak reporting guidance
- ▶ A norovirus fact sheet with general information and measures to limit transmission
- ▶ A poster for healthcare providers highlighting signs and symptoms of norovirus gastroenteritis and preventive infection control measures
- ▶ Key infection control recommendations based on the CDC HICPAC Guideline for the Prevention and Control of Norovirus Gastroenteritis Outbreaks in Healthcare Settings
- ▶ A sample line list for tracking and reporting norovirus cases among patients and healthcare personnel
- ▶ Sample worksheets to coordinate efforts to support
 - Laboratory confirmation of norovirus from stool (or vomitus) specimens
 - Internal and external communications for outbreak management

We encourage you to share these materials with your colleagues to help inform them about outbreaks of norovirus in healthcare settings and the recommended strategies for prevention and control.



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

Key Infection Control Recommendations

for the Control of Norovirus Outbreaks in Healthcare Settings

Patient Cohorting and Isolation Precautions

Place patients with norovirus gastroenteritis on Contact Precautions for a minimum of 48 hours after the resolution of symptoms

When symptomatic patients cannot be accommodated in single occupancy rooms, efforts should be made to separate them from asymptomatic patients. These efforts may include placing patients in multi-occupancy rooms, or designating patient care areas or contiguous sections within a facility for patient cohorts.

- ▶ Staff who have recovered from recent suspected norovirus infection associated with an outbreak may be best suited to care for symptomatic patients until the outbreak resolves.

Consider the following precautions:

- ▶ Minimize patient movements within a ward or unit during norovirus outbreaks
- ▶ Restrict symptomatic and recovering patients from leaving the patient-care area unless it is for essential care or treatment
- ▶ Suspend group activities (e.g., dining events) for the duration of a norovirus outbreak.

Hand Hygiene

- ▶ Actively promote adherence to hand hygiene among healthcare personnel, patients, and visitors in patient care areas affected by outbreaks of norovirus gastroenteritis
- ▶ During outbreaks, use soap and water for hand hygiene after providing care or having contact with patients suspected or confirmed with norovirus gastroenteritis.

*For all other hand hygiene indications refer to the 2002 HICPAC Guideline for Hand Hygiene in Health-Care Settings (<http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf>).



Personal Protective Equipment (PPE)

- ▶ If norovirus infection is suspected, adherence to PPE use according to Contact and Standard Precautions is recommended for individuals entering the patient care area (i.e., gowns and gloves upon entry).



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

Patient Transfer and Ward Closure

- ▶ Consider the closure of wards to new admissions or transfers as a measure to attenuate the magnitude of a norovirus outbreak.
- ▶ Consider limiting transfers to those for which the receiving facility is able to maintain Contact Precautions; otherwise, it may be prudent to postpone transfers until patients no longer require Contact Precautions. During outbreaks, medically suitable individuals recovering from norovirus gastroenteritis can be discharged to their place of residence.

Diagnostics

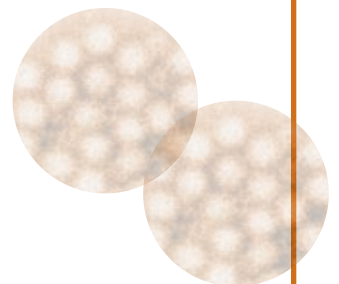
- ▶ In the absence of clinical laboratory diagnostics or in the case of delay in obtaining laboratory results, use Kaplan's clinical and epidemiologic criteria to identify a norovirus gastroenteritis outbreak.


Kaplan's Criteria:

1. Vomiting in more than half of symptomatic cases, and
 2. Mean (or median) incubation period of 24 to 48 hours, and
 3. Mean (or median) duration of illness of 12 to 60 hours, and
 4. No bacterial pathogen isolated from stool culture
- ▶ Consider submitting stool specimens as early as possible during a suspected norovirus gastroenteritis outbreak and ideally from individuals during the acute phase of illness (within 2-3 days of onset).
 - ▶ Specimens obtained from vomitus may be submitted for laboratory identification of norovirus when fecal specimens are unavailable (consult with your lab). Testing of vomitus as compared to fecal specimens may be less sensitive due to lower detectable viral concentrations.
 - ▶ Routine collecting and processing of environmental swabs during a norovirus outbreak is not required.

Environmental Cleaning

- ▶ Perform routine cleaning and disinfection of frequently touched environmental surfaces and equipment in isolation and cohorted areas, as well as high traffic clinical areas. Frequently touched surfaces include, but are not limited to, commodes, toilets, faucets, hand/bedrailing, telephones, door handles, computer equipment, and kitchen preparation surfaces.
- ▶ Increase the frequency of cleaning and disinfection of patient care areas and frequently touched surfaces during outbreaks of norovirus gastroenteritis (e.g., increase ward/unit level cleaning twice daily to maintain cleanliness, with frequently touched surfaces cleaned and disinfected three times daily using EPA-approved products for healthcare settings).



- 
- ▶ Clean and disinfect surfaces starting from the areas with a lower likelihood of norovirus contamination (e.g., tray tables, counter tops) to areas with highly contaminated surfaces (e.g., toilets, bathroom fixtures). Change mop heads when new solutions are prepared, or after cleaning large spills of emesis or fecal material.
 - ▶ No additional provisions for using disposable patient service items such as utensils or dishware are suggested for patients with symptoms of norovirus infection. Silverware and dishware may undergo normal processing and cleaning using standard procedures.
 - ▶ Use Standard Precautions for handling soiled patient-service items or linens, which includes the appropriate use of PPE.
 - ▶ Consider changing privacy curtains routinely and upon patient discharge or transfer.

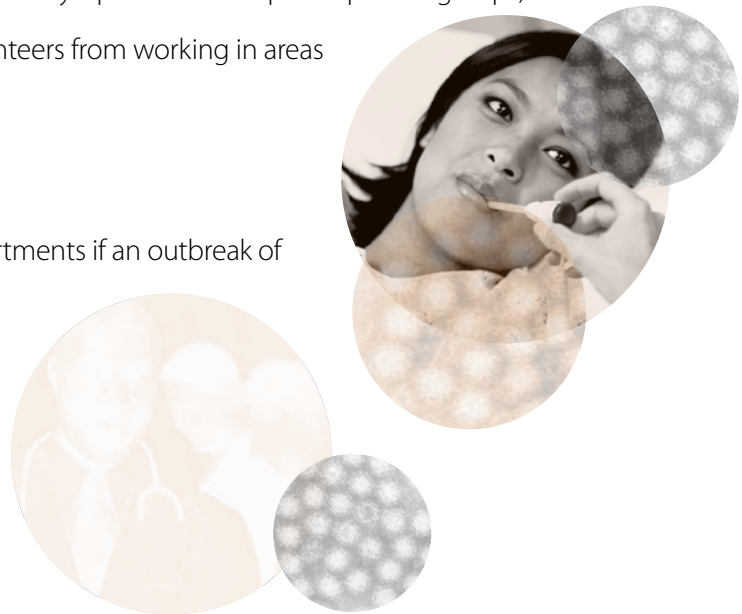


Staff Leave and Policy

- ▶ Exclude ill personnel from work for a minimum of 48 hours after the resolution of symptoms. Once personnel return to work, the importance of performing frequent hand hygiene should be reinforced.
- ▶ Establish protocols for staff cohorting in the event of an outbreak of norovirus. Ensure staff care for one patient cohort on their ward and do not move between patient cohorts (e.g., patient cohorts may include symptomatic, asymptomatic exposed, or asymptomatic unexposed patient groups).
- ▶ Exclude non-essential staff, students, and volunteers from working in areas experiencing outbreaks of norovirus.

Communication and Notification

- ▶ Notify appropriate local and state health departments if an outbreak of norovirus gastroenteritis is suspected.



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention



Guidelines for the Prevention of Intravascular Catheter-Related Infections, 2011

Naomi P. O'Grady, M.D.¹, Mary Alexander, R.N.², Lillian A. Burns, M.T., M.P.H., C.I.C.³, E. Patchen Dellinger, M.D.⁴, Jeffery Garland, M.D., S.M.⁵, Stephen O. Heard, M.D.⁶, Pamela A. Lipsett, M.D.⁷, Henry Masur, M.D.¹, Leonard A. Mermel, D.O., Sc.M.⁸, Michele L. Pearson, M.D.⁹, Issam I. Raad, M.D.¹⁰, Adrienne Randolph, M.D., M.Sc.¹¹, Mark E. Rupp, M.D.¹², Sanjay Saint, M.D., M.P.H.¹³ and the Healthcare Infection Control Practices Advisory Committee (HICPAC)¹⁴.

1National Institutes of Health, Bethesda, Maryland

2Infusion Nurses Society, Norwood, Massachusetts

3Greenich Hospital, Greenwich, Connecticut

4University of Washington, Seattle, Washington

5Wheaton Franciscan Healthcare-St. Joseph, Milwaukee, Wisconsin

6 University of Massachusetts Medical School, Worcester, Massachusetts

7Johns Hopkins University School of Medicine, Baltimore, Maryland

8Warren Alpert Medical School of Brown University and Rhode Island Hospital, Providence, Rhode Island

9Office of Infectious Diseases, CDC, Atlanta, Georgia

10MD Anderson Cancer Center, Houston, Texas

11The Children's Hospital, Boston, Massachusetts

12University of Nebraska Medical Center, Omaha, Nebraska

13Ann Arbor VA Medical Center and University of Michigan, Ann Arbor, Michigan

¹⁴ Healthcare Infection Control Practices Advisory Committee

CHAIRMAN

BRENNAN, Patrick J., MD
Chief Medical Officer
Division of Infectious Diseases
University of Pennsylvania Health System

MEMBERSHIP

BRATZLER, Dale, DO, MPH
President and CEO
Oklahoma Foundation for Medical Quality

BURNS, Lillian A., MT, MPH
Infection Control Coordinator
Greenwich Hospital, Infectious Diseases
Department

ELWARD, Alexis, MD
Assistant Professor, Pediatrics Infectious
Diseases
Washington University School of Medicine
Department of Pediatrics
Division of Infectious Diseases

HUANG, Susan, MD, MPH
Assistant Professor
Medical Director, Epidemiology and Infection
Prevention
Division of Infectious Diseases
UC Irvine School of Medicine

LUNDSTROM, Tammy, MD, JD
Chief Medical Officer
Providence Hospital

MCCARTER, Yvette S., PhD
Director, Clinical Microbiology
Laboratory
Department of Pathology
University of Florida Health Science
Center

MURPHY, Denise M. RN, MPH, CIC
Vice President, Quality and Patient Safety
Main Line Health System

EXECUTIVE SECRETARY

BELL, Michael R., MD
Deputy Director
Division of Healthcare Quality Promotion
Centers for Disease Control and Prevention

OSTROFF, Stephen, MD
Director, Bureau of Epidemiology
Pennsylvania Department of Health

OLMSTED, Russell N., MPH, CIC
Epidemiologist
Infection Control Services
St. Joseph Mercy Health System

PEGUES, David Alexander, MD
Professor of Medicine, Hospital Epidemiologist
David Geffen School of Medicine at
UCLA

PRONOVOST, Peter J., MD, PhD, FCCM
Director, Johns Hopkins Quality and Safety Research
Group
Johns Hopkins Quality and Safety Research Group

SOULE, Barbara M., RN, MPA, CIC
Practice Leader
Infection Prevention and Control Services
Joint Commission Resources/Joint
Commission International

SCHECTER, William, P., MD
Professor of Surgery
Department of Surgery
San Francisco General Hospital

Guidelines for the Prevention of Intravascular Catheter-Related Infections

EX-OFFICIO MEMBERS

Agency for Healthcare Research and Quality (AHRQ)

BAINE, William B., MD
Senior Medical Advisor
Center for Outcomes and Evidence

Center for Medicare & Medicaid Services (CMS)

MILLER, Jeannie, RN, MPH
Deputy Director, Clinical Standards Group

Food and Drug Administration (FDA)

MURPHEY, Sheila A., MD
Division of Anesthesiology, General Hospital Infection
Control Dental Devices
Center for Devices and Radiology Health

LIAISONS

Advisory Council for the Elimination of Tuberculosis (ACET)

STRICOF, Rachel L., MPH

American College of Occupational and Environmental Medicine

RUSSI, Mark, MD, MPH
Professor of Medicine
Yale University School of Medicine
Director, Occupational Health
Yale-New Haven Hospital

American Health Care Assn (AHCA)

FITZLER, Sandra L., RN
Senior Director of Clinical Services

American Hospital Association (AHA)

SCHULMAN, Roslyne, MHA, MBA
Director, Policy Development

Association of Professionals of Infection Control and Epidemiology, Inc. (APIC)

DeBAUN, Barbara, MSN, RN, CIC

Association of periOperative Registered Nurses (AORN)

BLANCHARD, Joan C., RN, BSN

Council of State and Territorial Epidemiologists (CSTE)

KAINER, Marion MD, MPH
Director, Hospital Infections and Antimicrobial Resistance
Program
Tennessee Department Health

National Institute of Health (NIH)

HENDERSON, David, MD
Deputy Director for Clinical Care
Associate Director for Hospital Epidemiology and Quality
Improvement
NIH Clinical Center

Department of Veterans Affairs (VA)

ROSELLE, Gary A., MD
National Program Director, Infectious Diseases
VA Central Office
Cincinnati VA Medical Center

Consumers Union

MCGIFFERT, Lisa
Senior Policy Analyst on Health Issues
Project Director Stop Hospital Infections Organization

Infectious Disease Society of America (IDSA)

HUSKINS, W. Charles MD, MSc
Division of Pediatric Infectious Diseases
Assistant Professor of Pediatrics
Mayo Clinic

Public Health Agency of Canada

PATON, Shirley, RN, MN
Senior Advisor Healthcare Acquired Infections
Center for Communicable Diseases and Infection Control

Society for Healthcare Epidemiology of America (SHEA)

MARAGAKIS, Lisa, MD
Assistant Professor of Medicine
John Hopkins Medical Institutions

Society of Hospital Medicine

SAINT, Sanjay, MD, MPH
Director, Ann Arbor VA Medical Center/University of
Michigan Patient Safety Enhancement Program

The Joint Commission

WISE, Robert A., MD
Vice President
Division of Standards & Survey Methods

Use of trade names and commercial sources is for identification only and does not imply endorsement by the U.S. Department of Health and Human Services.

Acknowledgments

We wish to acknowledge Ingi Lee, MD, MSCE and Craig A. Umscheid, MD, MSCE from Center for Evidence-based Practice, University of Pennsylvania Health System Philadelphia, PA who performed a systematic review for issues raised the during the guideline process.

Disclosure of potential conflicts of interests. N.P.O.'G. served as a board member for the ABIM Subspecialty Board for Critical Care Medicine. M.A. is an employee of the Infusion Nurses Society, Honoraria from 3M, Becton Dickinson, Smiths Medical. L.A.B. is a consultant for Institute of Healthcare Improvement, Board membership for Theradoc, Medline. Honoraria from APIC, Clorox. E.P.D. consulting from Merck, Baxter, Ortho-McNeil, Targanta, Schering-Plough, Optimer, Cadence, Cardinal, BDGeneOhm, WebEx, Cerebrio, and Tyco. Grant support through the NIH. Payment for lecture from Merck. Payment for development of educational presentation from Medscape. Travel and meeting expenses paid for by ASHP, IDSA, ASM, American College of Surgeons, NQF, SHEA/CDC, HHS, Trauma Shock Inflammation and Sepsis Meeting (Munich), University of Minnesota. J.G. Honoria from Ethicon. S.O.H. provides research support from Angiotech; Honoraria from Angiotech, Merck. L.A.M provides research support from Astellas, Theravance, Pfizer; Consulting for Ash Access, Cadence, CorMedix, Catheter Connections, Carefusion, Sage, Bard, Teleflex; Payment for manuscript preparation from Catheter Connections. I.I.R. provides research support from Cubist, Enzon, and Basilea; Consulting for Clorox; Stock Equity or Options in Great Lakes Pharmaceuticals and Inventive Protocol; Speakers Bureau for Cook, Inc.; Royalty income (patents owned by MD Anderson on which Dr. Raad is an inventor: American Medical Systems, Cook, Inc., Cook urological, Teleflex, TyRx, Medtronic, Biomet, Great Lakes Pharmaceuticals. A.R. consulting income from Eisai Pharmaceuticals, Discovery Laboratories. M.E.R. provides research support from Molnlycke, Cardinal Healthcare Foundation, Sanofi-Pasteur, 3M, and Cubist; Consulting from Semprus; Honorarium for lectures from 3M, Carefusion, Baxter and Becton Dickinson. Previously served on Board of Directors for Society for Healthcare Epidemiology of America. All other authors: no conflicts.

Table of Contents

NOTICE TO READERS: 7

Introduction 8

Summary Of Recommendations 9

 Education, Training and Staffing 9

 Selection of Catheters and Sites 10

 Peripheral Catheters and Midline Catheters 10

 Central Venous Catheters 11

 Hand Hygiene and Aseptic Technique 12

 Maximal Sterile Barrier Precautions 12

 Skin Preparation 13

 Catheter Site Dressing Regimens 13

 Patient Cleansing 15

 Catheter Securement Devices 15

 Antimicrobial/Antiseptic Impregnated Catheters and Cuffs 15

 Systemic Antibiotic Prophylaxis 15

 Antibiotic/Antiseptic Ointments 15

 Antibiotic Lock Prophylaxis, Antimicrobial Catheter Flush and Catheter Lock Prophylaxis 16

 Anticoagulants 16

 Replacement of Peripheral and Midline Catheters 16

 Replacement of CVCs, Including PICCs and Hemodialysis Catheters 16

 Umbilical Catheters 17

 Peripheral Arterial Catheters and Pressure Monitoring Devices for Adult and Pediatric Patients 18

 Replacement of Administration Sets 19

 Needleless Intravascular Catheter Systems 19

 Performance Improvement 20

Background Information 20

 Terminology and Estimates of Risk 20

 Epidemiology and Microbiology in Adult and Pediatric Patients 23

 Pathogenesis 23

 Strategies for Prevention of Catheter-Related Infections in Adult and Pediatric Patients 25

 Education, Training and Staffing 25

 Selection of Catheters and Sites 26

 Peripheral and Midline Catheter Recommendations 26

 Central Venous Catheters Recommendations 27

 Hand Hygiene and Aseptic Technique 29

 Maximal Sterile Barrier Precautions 30

 Skin Preparation 31

 Catheter Site Dressing Regimens 32

 Patient Cleansing 35

 Catheter Securement Devices 36

Antimicrobial/Antiseptic Impregnated Catheters and Cuffs	36
Systemic Antibiotic Prophylaxis	39
Antibiotic/Antiseptic Ointments	40
Antibiotic Lock Prophylaxis, Antimicrobial Catheter Flush and Catheter Lock Prophylaxis	41
Anticoagulants	43
Replacement of Peripheral and Midline Catheters	45
Replacement of CVCs, Including PICCs and Hemodialysis Catheters	46
Umbilical Catheters	49
Peripheral Arterial Catheters and Pressure Monitoring Devices for Adult and Pediatric Patients	51
Replacement of Administration Sets	53
Needleless Intravascular Catheter Systems	54
Performance Improvement	57
References	59

NOTICE TO READERS:

In 2009, the Centers for Disease Control and Prevention (CDC) and Healthcare Infection Control Practices Advisory Committee (HICPAC) integrated current advances in guideline production and implementation into its development process (<http://www.cdc.gov/hicpac/guidelineMethod/guidelineMethod.html>). The new methodology enables CDC and HICPAC to improve the validity and usability of its guidelines while also addressing emerging challenges in guideline development in the area of infection prevention and control. However, the *Guidelines for the Prevention of Intravascular Catheter-Related Infections* were initiated before the methodology was revised. Therefore, this guideline reflects the development methods that were used for guidelines produced prior to 2009. Future revisions will be performed using the updated methodology.

These guidelines have been developed for healthcare personnel who insert intravascular catheters and for persons responsible for surveillance and control of infections in hospital, outpatient, and home healthcare settings. This report was prepared by a working group comprising members from professional organizations representing the disciplines of critical care medicine, infectious diseases, healthcare infection control, surgery, anesthesiology, interventional radiology, pulmonary medicine, pediatric medicine, and nursing. The working group was led by the Society of Critical Care Medicine (SCCM), in collaboration with the Infectious Diseases Society of America (IDSA), Society for Healthcare Epidemiology of America (SHEA), Surgical Infection Society (SIS), American College of Chest Physicians (ACCP), American Thoracic Society (ATS), American Society of Critical Care Anesthesiologists (ASCCA), Association for Professionals in Infection Control and Epidemiology (APIC), Infusion Nurses Society (INS), Oncology Nursing Society (ONS), American Society for Parenteral and Enteral Nutrition (ASPEN), Society of Interventional Radiology (SIR), American Academy of Pediatrics (AAP), Pediatric Infectious Diseases Society (PIDS), and the Healthcare Infection Control Practices Advisory Committee (HICPAC) of the Centers for Disease Control and Prevention (CDC) and is intended to replace the Guideline for Prevention of Intravascular Catheter-Related Infections published in

2002. These guidelines are intended to provide evidence-based recommendations for preventing intravascular catheter-related infections. Major areas of emphasis include 1) educating and training healthcare personnel who insert and maintain catheters; 2) using maximal sterile barrier precautions during central venous catheter insertion; 3) using a > 0.5% chlorhexidine skin preparation with alcohol for antisepsis; 4) avoiding routine replacement of central venous catheters as a strategy to prevent infection; and 5) using antiseptic/antibiotic impregnated short-term central venous catheters and chlorhexidine impregnated sponge dressings if the rate of infection is not decreasing despite adherence to other strategies (i.e., education and training, maximal sterile barrier precautions, and >0.5% chlorhexidine preparations with alcohol for skin antisepsis). These guidelines also emphasize performance improvement by implementing bundled strategies, and documenting and reporting rates of compliance with all components of the bundle as benchmarks for quality assurance and performance improvement.

As in previous guidelines issued by CDC and HICPAC, each recommendation is categorized on the basis of existing scientific data, theoretical rationale, applicability, and economic impact. The system for categorizing recommendations in this guideline is as follows:

Category IA. Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiologic studies.

Category IB. Strongly recommended for implementation and supported by some experimental, clinical, or epidemiologic studies and a strong theoretical rationale; or an accepted practice (e.g., aseptic technique) supported by limited evidence.

Category IC. Required by state or federal regulations, rules, or standards.

Category II. Suggested for implementation and supported by suggestive clinical or epidemiologic studies or a theoretical rationale.

Unresolved issue. Represents an unresolved issue for which evidence is insufficient or no consensus regarding efficacy exists.

Introduction

In the United States, 15 million central vascular catheter (CVC) days (i.e., the total number of days of exposure to CVCs among all patients in the selected population during the

selected time period) occur in intensive care units (ICUs) each year [1]. Studies have variously addressed catheter-related bloodstream infections (CRBSI). These infections independently increase hospital costs and length of stay [2-5], but have not generally been shown to independently increase mortality. While 80,000 CRBSIs occur in ICUs each year [1], a total of 250,000 cases of BSIs have been estimated to occur annually, if entire hospitals are assessed [6]. By several analyses, the cost of these infections is substantial, both in terms of morbidity and financial resources expended. To improve patient outcome and to reduce healthcare costs, there is considerable interest by healthcare providers, insurers, regulators, and patient advocates in reducing the incidence of these infections. This effort should be multidisciplinary, involving healthcare professionals who order the insertion and removal of CVCs, those personnel who insert and maintain intravascular catheters, infection control personnel, healthcare managers including the chief executive officer (CEO) and those who allocate resources, and patients who are capable of assisting in the care of their catheters.

The goal of an effective prevention program should be the elimination of CRBSI from all patient-care areas. Although this is challenging, programs have demonstrated success, but sustained elimination requires continued effort. The goal of the measures discussed in this document is to reduce the rate to as low as feasible given the specific patient population being served, the universal presence of microorganisms in the human environment, and the limitations of current strategies and technologies.

Summary of Recommendations

Education, Training and Staffing

1. Educate healthcare personnel regarding the indications for intravascular catheter use, proper procedures for the insertion and maintenance of intravascular catheters, and appropriate infection control measures to prevent intravascular catheter-related infections [7–15]. Category IA
2. Periodically assess knowledge of and adherence to guidelines for all personnel involved in the insertion and maintenance of intravascular catheters [7–15]. Category IA

3. Designate only trained personnel who demonstrate competence for the insertion and maintenance of peripheral and central intravascular catheters. [14–28]. Category IA
4. Ensure appropriate nursing staff levels in ICUs. Observational studies suggest that a higher proportion of "pool nurses" or an elevated patient–to-nurse ratio is associated with CRBSI in ICUs where nurses are managing patients with CVCs [29–31]. Category IB

Selection of Catheters and Sites

Peripheral Catheters and Midline Catheters

1. In adults, use an upper-extremity site for catheter insertion. Replace a catheter inserted in a lower extremity site to an upper extremity site as soon as possible. Category II
2. In pediatric patients, the upper or lower extremities or the scalp (in neonates or young infants) can be used as the catheter insertion site [32, 33]. Category II
3. Select catheters on the basis of the intended purpose and duration of use, known infectious and non-infectious complications (e.g., phlebitis and infiltration), and experience of individual catheter operators [33–35]. Category IB
4. Avoid the use of steel needles for the administration of fluids and medication that might cause tissue necrosis if extravasation occurs [33, 34]. Category IA
5. Use a midline catheter or peripherally inserted central catheter (PICC), instead of a short peripheral catheter, when the duration of IV therapy will likely exceed six days. Category II
6. Evaluate the catheter insertion site daily by palpation through the dressing to discern tenderness and by inspection if a transparent dressing is in use. Gauze and opaque dressings should not be removed if the patient has no clinical signs of infection. If the patient has local tenderness or other signs of possible CRBSI, an opaque dressing should be removed and the site inspected visually. Category II
7. Remove peripheral venous catheters if the patients develops signs of phlebitis (warmth, tenderness, erythema or palpable venous cord), infection, or a malfunctioning catheter [36]. Category IB

Central Venous Catheters

1. Weigh the risks and benefits of placing a central venous device at a recommended site to reduce infectious complications against the risk for mechanical complications (e.g., pneumothorax, subclavian artery puncture, subclavian vein laceration, subclavian vein stenosis, hemothorax, thrombosis, air embolism, and catheter misplacement) [37–53].
Category IA
2. Avoid using the femoral vein for central venous access in adult patients [38, 50, 51, 54].
Category 1A
3. Use a subclavian site, rather than a jugular or a femoral site, in adult patients to minimize infection risk for nontunneled CVC placement [50–52]. Category IB
4. No recommendation can be made for a preferred site of insertion to minimize infection risk for a tunneled CVC. Unresolved issue
5. Avoid the subclavian site in hemodialysis patients and patients with advanced kidney disease, to avoid subclavian vein stenosis [53,55–58]. Category IA
6. Use a fistula or graft in patients with chronic renal failure instead of a CVC for permanent access for dialysis [59]. Category 1A
7. Use ultrasound guidance to place central venous catheters (if this technology is available) to reduce the number of cannulation attempts and mechanical complications. Ultrasound guidance should only be used by those fully trained in its technique. [60–64].
Category 1B
8. Use a CVC with the minimum number of ports or lumens essential for the management of the patient [65–68]. Category IB
9. No recommendation can be made regarding the use of a designated lumen for parenteral nutrition. Unresolved issue
10. Promptly remove any intravascular catheter that is no longer essential [69–72].
Category IA
11. When adherence to aseptic technique cannot be ensured (i.e catheters inserted during a medical emergency), replace the catheter as soon as possible, i.e, within 48 hours [37,73–76]. Category IB

Hand Hygiene and Aseptic Technique

1. Perform hand hygiene procedures, either by washing hands with conventional soap and water or with alcohol-based hand rubs (ABHR). Hand hygiene should be performed before and after palpating catheter insertion sites as well as before and after inserting, replacing, accessing, repairing, or dressing an intravascular catheter. Palpation of the insertion site should not be performed after the application of antiseptic, unless aseptic technique is maintained [12, 77–79]. Category IB
2. Maintain aseptic technique for the insertion and care of intravascular catheters [37, 73, 74, 76]. Category IB
3. Wear clean gloves, rather than sterile gloves, for the insertion of peripheral intravascular catheters, if the access site is not touched after the application of skin antiseptics. Category IC
4. Sterile gloves should be worn for the insertion of arterial, central, and midline catheters [37, 73, 74, 76]. Category IA
5. Use new sterile gloves before handling the new catheter when guidewire exchanges are performed. Category II
6. Wear either clean or sterile gloves when changing the dressing on intravascular catheters. Category IC

Maximal Sterile Barrier Precautions

1. Use maximal sterile barrier precautions, including the use of a cap, mask, sterile gown, sterile gloves, and a sterile full body drape, for the insertion of CVCs, PICCs, or guidewire exchange [14, 75, 76, 80]. Category IB
2. Use a sterile sleeve to protect pulmonary artery catheters during insertion [81]. Category IB

Skin Preparation

1. Prepare clean skin with an antiseptic (70% alcohol, tincture of iodine, or alcoholic chlorhexidine gluconate solution) before peripheral venous catheter insertion [82].
Category IB
2. Prepare clean skin with a >0.5% chlorhexidine preparation with alcohol before central venous catheter and peripheral arterial catheter insertion and during dressing changes. If there is a contraindication to chlorhexidine, tincture of iodine, an iodophor, or 70% alcohol can be used as alternatives [82, 83]. Category IA
3. No comparison has been made between using chlorhexidine preparations with alcohol and povidone-iodine in alcohol to prepare clean skin. Unresolved issue.
4. No recommendation can be made for the safety or efficacy of chlorhexidine in infants aged <2 months. Unresolved issue
5. Antiseptics should be allowed to dry according to the manufacturer's recommendation prior to placing the catheter [82, 83]. Category IB

Catheter Site Dressing Regimens

1. Use either sterile gauze or sterile, transparent, semipermeable dressing to cover the catheter site [84–87]. Category IA
2. If the patient is diaphoretic or if the site is bleeding or oozing, use a gauze dressing until this is resolved [84–87]. Category II
3. Replace catheter site dressing if the dressing becomes damp, loosened, or visibly soiled [84, 85]. Category IB
4. Do not use topical antibiotic ointment or creams on insertion sites, except for dialysis catheters, because of their potential to promote fungal infections and antimicrobial resistance [88, 89]. Category IB
5. Do not submerge the catheter or catheter site in water. Showering should be permitted if precautions can be taken to reduce the likelihood of introducing organisms into the

- catheter (e.g., if the catheter and connecting device are protected with an impermeable cover during the shower) [90–92]. Category IB
6. Replace dressings used on short-term CVC sites every 2 days for gauze dressings.
Category II
 7. Replace dressings used on short-term CVC sites at least every 7 days for transparent dressings, except in those pediatric patients in which the risk for dislodging the catheter may outweigh the benefit of changing the dressing [87, 93]. Category IB
 8. Replace transparent dressings used on tunneled or implanted CVC sites no more than once per week (unless the dressing is soiled or loose), until the insertion site has healed.
Category II
 9. No recommendation can be made regarding the necessity for any dressing on well-healed exit sites of long-term cuffed and tunneled CVCs. Unresolved issue
 10. Ensure that catheter site care is compatible with the catheter material [94, 95].
Category IB
 11. Use a sterile sleeve for all pulmonary artery catheters [81]. Category IB
 12. Use a chlorhexidine-impregnated sponge dressing for temporary short-term catheters in patients older than 2 months of age if the CLABSI rate is not decreasing despite adherence to basic prevention measures, including education and training, appropriate use of chlorhexidine for skin antisepsis, and MSB [93, 96–98]. Category 1B
 13. No recommendation is made for other types of chlorhexidine dressings. Unresolved issue
 14. Monitor the catheter sites visually when changing the dressing or by palpation through an intact dressing on a regular basis, depending on the clinical situation of the individual patient. If patients have tenderness at the insertion site, fever without obvious source, or other manifestations suggesting local or bloodstream infection, the dressing should be removed to allow thorough examination of the site [99–101]. Category IB
 15. Encourage patients to report any changes in their catheter site or any new discomfort to their provider. Category II

Patient Cleansing

Use a 2% chlorhexidine wash for daily skin cleansing to reduce CRBSI [102–104].

Category II

Catheter Securement Devices

Use a sutureless securement device to reduce the risk of infection for intravascular catheters [105]. Category II

Antimicrobial/Antiseptic Impregnated Catheters and Cuffs

Use a chlorhexidine/silver sulfadiazine or minocycline/rifampin -impregnated CVC in patients whose catheter is expected to remain in place >5 days if, after successful implementation of a comprehensive strategy to reduce rates of CLABSI, the CLABSI rate is not decreasing. The comprehensive strategy should include at least the following three components: educating persons who insert and maintain catheters, use of maximal sterile barrier precautions, and a >0.5% chlorhexidine preparation with alcohol for skin antisepsis during CVC insertion [106–113]. Category IA

Systemic Antibiotic Prophylaxis

Do not administer systemic antimicrobial prophylaxis routinely before insertion or during use of an intravascular catheter to prevent catheter colonization or CRBSI [114].

Category IB

Antibiotic/Antiseptic Ointments

Use povidone iodine antiseptic ointment or bacitracin/gramicidin/ polymyxin B ointment at the hemodialysis catheter exit site after catheter insertion and at the end of each dialysis session only if this ointment does not interact with the material of the hemodialysis catheter per manufacturer's recommendation [59, 115–119]. Category IB

Antibiotic Lock Prophylaxis, Antimicrobial Catheter Flush and Catheter Lock Prophylaxis

Use prophylactic antimicrobial lock solution in patients with long term catheters who have a history of multiple CRBSI despite optimal maximal adherence to aseptic technique [120– 138]. Category II

Anticoagulants

Do not routinely use anticoagulant therapy to reduce the risk of catheter-related infection in general patient populations [139]. Category II

Replacement of Peripheral and Midline Catheters

1. There is no need to replace peripheral catheters more frequently than every 72-96 hours to reduce risk of infection and phlebitis in adults [36, 140, 141]. Category 1B
2. No recommendation is made regarding replacement of peripheral catheters in adults only when clinically indicated [142–144]. Unresolved issue
3. Replace peripheral catheters in children only when clinically indicated [32, 33]. Category 1B
4. Replace midline catheters only when there is a specific indication. Category II

Replacement of CVCs, Including PICCs and Hemodialysis Catheters

1. Do not routinely replace CVCs, PICCs, hemodialysis catheters, or pulmonary artery catheters to prevent catheter-related infections. Category IB
2. Do not remove CVCs or PICCs on the basis of fever alone. Use clinical judgment regarding the appropriateness of removing the catheter if infection is evidenced elsewhere or if a noninfectious cause of fever is suspected. Category II
3. Do not use guidewire exchanges routinely for non-tunneled catheters to prevent infection. Category IB
4. Do not use guidewire exchanges to replace a non-tunneled catheter suspected of infection. Category IB

5. Use a guidewire exchange to replace a malfunctioning non-tunneled catheter if no evidence of infection is present. Category IB
6. Use new sterile gloves before handling the new catheter when guidewire exchanges are performed. Category II

Umbilical Catheters

1. Remove and do not replace umbilical artery catheters if any signs of CRBSI, vascular insufficiency in the lower extremities, or thrombosis are present [145]. Category II
2. Remove and do not replace umbilical venous catheters if any signs of CRBSI or thrombosis are present [145]. Category II
3. No recommendation can be made regarding attempts to salvage an umbilical catheter by administering antibiotic treatment through the catheter. Unresolved issue
4. Cleanse the umbilical insertion site with an antiseptic before catheter insertion. Avoid tincture of iodine because of the potential effect on the neonatal thyroid. Other iodine-containing products (e.g., povidone iodine) can be used [146– 150]. Category IB
5. Do not use topical antibiotic ointment or creams on umbilical catheter insertion sites because of the potential to promote fungal infections and antimicrobial resistance [88, 89]. Category IA
6. Add low-doses of heparin (0.25—1.0 U/ml) to the fluid infused through umbilical arterial catheters [151–153]. Category IB
7. Remove umbilical catheters as soon as possible when no longer needed or when any sign of vascular insufficiency to the lower extremities is observed. Optimally, umbilical artery catheters should not be left in place >5 days [145, 154]. Category II
8. Umbilical venous catheters should be removed as soon as possible when no longer needed, but can be used up to 14 days if managed aseptically [155, 156]. Category II
9. An umbilical catheter may be replaced if it is malfunctioning, and there is no other indication for catheter removal, and the total duration of catheterization has not exceeded 5 days for an umbilical artery catheter or 14 days for an umbilical vein catheter. Category II

Peripheral Arterial Catheters and Pressure Monitoring Devices for Adult and Pediatric

Patients

1. In adults, use of the radial, brachial or dorsalis pedis sites is preferred over the femoral or axillary sites of insertion to reduce the risk of infection [46, 47, 157, 158]. Category IB
2. In children, the brachial site should not be used. The radial, dorsalis pedis, and posterior tibial sites are preferred over the femoral or axillary sites of insertion [46]. Category II
3. A minimum of a cap, mask, sterile gloves and a small sterile fenestrated drape should be used during peripheral arterial catheter insertion [47, 158, 159]. Category IB
4. During axillary or femoral artery catheter insertion, maximal sterile barriers precautions should be used. Category II
5. Replace arterial catheters only when there is a clinical indication. Category II
6. Remove the arterial catheter as soon as it is no longer needed. Category II
7. Use disposable, rather than reusable, transducer assemblies when possible [160–164]. Category IB
8. Do not routinely replace arterial catheters to prevent catheter-related infections [165, 166, 167, 168]. Category II
9. Replace disposable or reusable transducers at 96-hour intervals. Replace other components of the system (including the tubing, continuous-flush device, and flush solution) at the time the transducer is replaced [37, 161]. Category IB
10. Keep all components of the pressure monitoring system (including calibration devices and flush solution) sterile [160, 169–171]. Category IA
11. Minimize the number of manipulations of and entries into the pressure monitoring system. Use a closed flush system (i.e, continuous flush), rather than an open system (i.e, one that requires a syringe and stopcock), to maintain the patency of the pressure monitoring catheters [163, 172]. Category II
12. When the pressure monitoring system is accessed through a diaphragm, rather than a stopcock, scrub the diaphragm with an appropriate antiseptic before accessing the system [163]. Category IA

13. Do not administer dextrose-containing solutions or parenteral nutrition fluids through the pressure monitoring circuit [163, 173, 174]. Category IA
14. Sterilize reusable transducers according to the manufacturers' instructions if the use of disposable transducers is not feasible [163, 173–176]. Category IA

Replacement of Administration Sets

1. In patients not receiving blood, blood products or fat emulsions, replace administration sets that are continuously used, including secondary sets and add-on devices, no more frequently than at 96-hour intervals, [177] but at least every 7 days [178–181]. Category IA
2. No recommendation can be made regarding the frequency for replacing intermittently used administration sets. Unresolved issue
3. No recommendation can be made regarding the frequency for replacing needles to access implantable ports. Unresolved issue
4. Replace tubing used to administer blood, blood products, or fat emulsions (those combined with amino acids and glucose in a 3-in-1 admixture or infused separately) within 24 hours of initiating the infusion [182–185]. Category IB
5. Replace tubing used to administer propofol infusions every 6 or 12 hours, when the vial is changed, per the manufacturer's recommendation (FDA website Medwatch) [186]. Category IA
6. No recommendation can be made regarding the length of time a needle used to access implanted ports can remain in place. Unresolved issue

Needleless Intravascular Catheter Systems

1. Change the needleless components at least as frequently as the administration set. There is no benefit to changing these more frequently than every 72 hours. [39, 187–193]. Category II

2. Change needleless connectors no more frequently than every 72 hours or according to manufacturers' recommendations for the purpose of reducing infection rates [187, 189, 192, 193]. Category II
3. Ensure that all components of the system are compatible to minimize leaks and breaks in the system [194]. Category II
4. Minimize contamination risk by scrubbing the access port with an appropriate antiseptic (chlorhexidine, povidone iodine, an iodophor, or 70% alcohol) and accessing the port only with sterile devices [189, 192, 194–196]. Category IA
5. Use a needleless system to access IV tubing. Category IC
6. When needleless systems are used, a split septum valve may be preferred over some mechanical valves due to increased risk of infection with the mechanical valves [197–200]. Category II

Performance Improvement

Use hospital-specific or collaborative-based performance improvement initiatives in which multifaceted strategies are "bundled" together to improve compliance with evidence-based recommended practices [15, 69, 70, 201–205]. Category IB

Background Information

Terminology and Estimates of Risk

The terminology used to identify different types of catheters is confusing, because many clinicians and researchers use different aspects of the catheter for informal reference. A catheter can be designated by the type of vessel it occupies (e.g., peripheral venous, central venous, or arterial); its intended life span (e.g., temporary or short-term versus permanent or long-term); its site of insertion (e.g., subclavian, femoral, internal jugular, peripheral, and peripherally inserted central catheter [PICC]); its pathway from skin to vessel (e.g., tunneled versus nontunneled); its physical length (e.g., long versus short); or some special characteristic of the catheter (e.g., presence or absence of a cuff, impregnation with heparin, antibiotics or antiseptics, and the number of lumens). To accurately define a specific type of catheter, all of these aspects should be described (Table 1).

Likewise the terms used to describe intravascular catheter-related infections can also be confusing because catheter-related bloodstream infection (CRBSI) and central line–associated bloodstream infection (CLABSI) are often used interchangeably even though the meanings differ.

CRBSI is a clinical definition, used when diagnosing and treating patients, that requires specific laboratory testing that more thoroughly identifies the catheter as the source of the BSI. It is not typically used for surveillance purposes. It is often problematic to precisely establish if a BSI is a CRBSI due to the clinical needs of the patient (the catheter is not always pulled), limited availability of microbiologic methods (many labs do not use quantitative blood cultures or differential time to positivity), and procedural compliance by direct care personnel (labeling must be accurate). Simpler definitions are often used for surveillance purposes. For example, CLABSI is a term used by CDC’s National Healthcare Safety Network (NHSN) (visit NHSN CLABSI information) [206]. A CLABSI is a primary BSI in a patient that had a central line within the 48-hour period before the development of the BSI and is not bloodstream related to an infection at another site. However, since some BSIs are secondary to other sources other than the central line (e.g., pancreatitis, mucositis) that may not be easily recognized, the CLABSI surveillance definition may overestimate the true incidence of CRBSI.

Table 1. Catheters used for venous and arterial access.

Catheter type	Entry Site	Length	Comments
Peripheral venous catheters	Usually inserted in veins of forearm or hand	<3 inches	Phlebitis with prolonged use; rarely associated with bloodstream infection
Peripheral arterial catheters	Usually inserted in radial artery; can be placed in femoral, axillary, brachial, posterior tibial arteries	<3 inches	Low infection risk; rarely associated with bloodstream infection
Midline catheters	Inserted via the antecubital fossa into the proximal basilic or cephalic veins; does not enter central veins, peripheral catheters	3 to 8 inches	Anaphylactoid reactions have been reported with catheters made of elastomeric hydrogel; lower rates of phlebitis than short peripheral catheters
Nontunneled central venous catheters	Percutaneously inserted into central veins (subclavian, internal jugular, or femoral)	≥8 cm depending on patient size	Account for majority of CRBSI
Pulmonary artery catheters	Inserted through a Teflon® introducer in a central vein (subclavian, internal jugular, or femoral)	≥30 cm depending on patient size	Usually heparin bonded; similar rates of bloodstream infection as CVCs; subclavian site preferred to reduce infection risk
Peripherally inserted central venous catheters (PICC)	Inserted into basilic, cephalic, or brachial veins and enter the superior vena cava	≥20 cm depending on patient size	Lower rate of infection than nontunneled CVCs
Tunneled central venous catheters	Implanted into subclavian, internal jugular, or femoral veins	≥8 cm depending on patient size	Cuff inhibits migration of organisms into catheter tract; lower rate of infection than nontunneled CVC
Totally implantable	Tunneled beneath skin and have subcutaneous port accessed with a needle; implanted in subclavian or internal jugular vein	≥8 cm depending on patient size	Lowest risk for CRBSI; improved patient self-image; no need for local catheter-site care; surgery required for catheter removal
Umbilical catheters	Inserted into either umbilical vein or umbilical artery	≤6 cm depending on patient size	Risk for CRBSI similar with catheters placed in umbilical vein versus artery

Epidemiology and Microbiology in Adult and Pediatric Patients

National estimates of CLABSI rates are available through CDC's NHSN, a surveillance system for healthcare-associated infections, and are available on CDC's website. A recent report highlights data from 1,545 hospitals in 48 States and the District of Columbia that monitor infections in one or more ICUs and/or non-ICUs (e.g., patient care areas, wards) [207]. Because BSI rates are influenced by patient-related factors, such as severity of illness and type of illness (e.g., third-degree burns versus post-cardiac surgery), by catheter-related factors, (such as the condition under which the catheter was placed and catheter type), and by institutional factors (e.g., bed-size, academic affiliation), these aggregate, risk-adjusted rates can be used as benchmarks against which hospitals can make intra-and inter-facility comparisons.

The most commonly reported causative pathogens remain coagulase-negative staphylococci, *Staphylococcus aureus*, enterococci, and *Candida* spp [208]. Gram negative bacilli accounted for 19% and 21% of CLABSIs reported to CDC [209] and the Surveillance and Control of Pathogens of Epidemiological Importance (SCOPE) database, respectively [208].

For all common pathogens causing CLABSIs, antimicrobial resistance is a problem, particularly in ICUs. Although methicillin-resistant *Staphylococcus aureus* (MRSA) now account for more than 50% of all *Staphylococcus aureus* isolates obtained in ICUs, the incidence of MRSA CLABSIs has decreased in recent years, perhaps as a result of prevention efforts [210]. For gram negative rods, antimicrobial resistance to third generation cephalosporins among *Klebsiella pneumoniae* and *E. coli* has increased significantly as has imipenem and ceftazidime resistance among *Pseudomonas aeruginosa* [209]. *Candida* spp. are increasingly noted to be fluconazole resistant.

Pathogenesis

There are four recognized routes for contamination of catheters: 1) migration of skin organisms at the insertion site into the cutaneous catheter tract and along the surface of the catheter with colonization of the catheter tip; this is the most common route of infection for short-term catheters [37, 211, 212]; 2) direct contamination of the catheter or catheter hub by

contact with hands or contaminated fluids or devices [213, 214]; 3) less commonly, catheters might become hematogenously seeded from another focus of infection [215]; and 4) rarely, infusate contamination might lead to CRBSI [216].

Important pathogenic determinants of CRBSI are 1) the material of which the device is made; 2) the host factors consisting of protein adhesions, such as fibrin and fibronectin, that form a sheath around the catheter [217]; and 3) the intrinsic virulence factors of the infecting organism, including the extracellular polymeric substance (EPS) produced by the adherent organisms [218]. Some catheter materials also have surface irregularities that enhance the microbial adherence of certain species (e.g., *S. epidermidis* and *C. albicans*) [219, 220]. Catheters made of these materials are especially vulnerable to microbial colonization and subsequent infection. Due to the formation of the fibrin sheath, silastic catheters are associated with higher risk of catheter infections than polyurethane catheters [217]. On the other hand, biofilm formation by *C. albicans* occurs more readily on silicone elastomer catheter surfaces than polyurethane catheters [219]. Modification of the biomaterial surface properties has been shown to influence the ability of *C. albicans* to form biofilm [220]. Additionally, certain catheter materials are more thrombogenic than others, a characteristic that also might predispose to catheter colonization and infection [221, 222]. This association has led to emphasis on preventing catheter-related thrombus as an additional mechanism for reducing CRBSI [223, 224].

The adherence properties of a given microorganism in relationship to host factors are also important in the pathogenesis of CRBSI. For example, *S. aureus* can adhere to host proteins (e.g., fibrinogen, fibronectin) commonly present on catheters by expressing clumping factors (ClfA and ClfB) that bind to the protein adhesins [217, 222, 225, 226]. Furthermore, adherence is enhanced through the production by microbial organisms, such as coagulase negative staphylococci [227, 228], *S. aureus* [229], *Pseudomonas aeruginosa* [230], and *Candida* species [231] of an extracellular polymeric substance (EPS) consisting mostly of an exopolysaccharide that forms a microbial biofilm layer [218, 232]. This biofilm matrix is enriched by divalent metallic cations, such as calcium, magnesium and iron, which make it a solid enclave in which microbial organisms can embed themselves [233–235]. Such a biofilm

potentiates the pathogenicity of various microbes by allowing them to withstand host defense mechanisms (e.g., acting as a barrier to engulfment and killing by polymorphonuclear leukocytes) or by making them less susceptible to antimicrobial agents (e.g., forming a matrix that binds antimicrobials before their contact with the organism cell wall or providing for a population of metabolically quiescent, antimicrobial tolerant "persister" cells) [228, 236, 237]. Some *Candida* spp., in the presence of dextrose-containing fluids, produce slime similar to that of their bacterial counterparts, potentially explaining the increased proportion of BSIs caused by fungal pathogens among patients receiving parenteral nutrition fluids [238].

Strategies for Prevention of Catheter-Related Infections in Adult and Pediatric Patients

Education, Training and Staffing

Recommendations

1. Educate healthcare personnel regarding the indications for intravascular catheter use, proper procedures for the insertion and maintenance of intravascular catheters, and appropriate infection control measures to prevent intravascular catheter-related infections [7–15]. Category IA
2. Periodically assess knowledge of and adherence to guidelines for all personnel involved in the insertion and maintenance of intravascular catheters [7–15]. Category IA
3. Designate only trained personnel who demonstrate competence for the insertion and maintenance of peripheral and central intravascular catheters. [14–28]. Category IA
4. Ensure appropriate nursing staff levels in ICUs. Observational studies suggest that a higher proportion of "pool nurses" or an elevated patient-to-nurse ratio is associated with CRBSI in ICUs where nurses are managing patients with CVCs [29–31]. Category IB

Background

Well-organized programs that enable healthcare providers to become educated and to provide, monitor, and evaluate care are critical to the success of this effort. Reports spanning

the past four decades have consistently demonstrated that risk for infection declines following standardization of aseptic care [7, 12, 14, 15, 239–241] and that insertion and maintenance of intravascular catheters by inexperienced staff might increase the risk for catheter colonization and CRBSI [15, 242]. Specialized "IV teams" have shown unequivocal effectiveness in reducing the incidence of CRBSI, associated complications, and costs [16–26]. Additionally, infection risk increases with nursing staff reductions below a critical level [30].

Selection of Catheters and Sites

Peripheral and Midline Catheter Recommendations

1. In adults, use an upper-extremity site for catheter insertion. Replace a catheter inserted in a lower extremity site to an upper extremity site as soon as possible. Category II
2. In pediatric patients, the upper or lower extremities or the scalp (in neonates or young infants) can be used as the catheter insertion site [32, 33]. Category II
3. Select catheters on the basis of the intended purpose and duration of use, known infectious and non-infectious complications (e.g., phlebitis and infiltration), and experience of individual catheter operators [33–35]. Category IB
4. Avoid the use of steel needles for the administration of fluids and medication that might cause tissue necrosis if extravasation occurs [33, 34]. Category IA
5. Use a midline catheter or peripherally inserted central catheter (PICC), instead of a short peripheral catheter, when the duration of IV therapy will likely exceed six days. Category II
6. Evaluate the catheter insertion site daily by palpation through the dressing to discern tenderness and by inspection if a transparent dressing is in use. Gauze and opaque dressings should not be removed if the patient has no clinical signs of infection. If the patient has local tenderness or other signs of possible CRBSI, an opaque dressing should be removed and the site inspected visually. Category II
7. Remove peripheral venous catheters if the patients develops signs of phlebitis (warmth, tenderness, erythema or palpable venous cord), infection, or a malfunctioning catheter [36]. Category IB

Central Venous Catheters Recommendations

1. Weigh the risks and benefits of placing a central venous device at a recommended site to reduce infectious complications against the risk for mechanical complications (e.g., pneumothorax, subclavian artery puncture, subclavian vein laceration, subclavian vein stenosis, hemothorax, thrombosis, air embolism, and catheter misplacement) [37–53].
Category IA
2. Avoid using the femoral vein for central venous access in adult patients [38, 50, 51, 54].
Category 1A
3. Use a subclavian site, rather than a jugular or a femoral site, in adult patients to minimize infection risk for nontunneled CVC placement [50–52]. Category IB
4. No recommendation can be made for a preferred site of insertion to minimize infection risk for a tunneled CVC. Unresolved issue
5. Avoid the subclavian site in hemodialysis patients and patients with advanced kidney disease, to avoid subclavian vein stenosis [53, 55–58]. Category IA
6. Use a fistula or graft in patients with chronic renal failure instead of a CVC for permanent access for dialysis [59]. Category 1A
7. Use ultrasound guidance to place central venous catheters (if this technology is available) to reduce the number of cannulation attempts and mechanical complications. Ultrasound guidance should only be used by those fully trained in its technique. [60–64].
Category 1B
8. Use a CVC with the minimum number of ports or lumens essential for the management of the patient [65–68]. Category IB
9. No recommendation can be made regarding the use of a designated lumen for parenteral nutrition. Unresolved issue
10. Promptly remove any intravascular catheter that is no longer essential [69–72].
Category IA

11. When adherence to aseptic technique cannot be ensured (i.e catheters inserted during a medical emergency), replace the catheter as soon as possible, i.e, within 48 hours [37, 73–76]. Category IB

Background

The site at which a catheter is placed influences the subsequent risk for catheter-related infection and phlebitis. The influence of site on the risk for catheter infections is related in part to the risk for thrombophlebitis and density of local skin flora.

As in adults, the use of peripheral venous catheters in pediatric patients might be complicated by phlebitis, infusion extravasation, and catheter infection [243]. Catheter location, infusion of parenteral nutritional fluids with continuous IV fat emulsions, and length of ICU stay before catheter insertion, have all increased pediatric patients' risk for phlebitis. However, contrary to the risk in adults, the risk for phlebitis in children has not increased with the duration of catheterization [243, 244].

The density of skin flora at the catheter insertion site is a major risk factor for CRBSI. No single trial has satisfactorily compared infection rates for catheters placed in jugular, subclavian, and femoral veins. In retrospective observational studies, catheters inserted into an internal jugular vein have usually been associated with higher risk for colonization and/or CRBSI than those inserted into a subclavian [37–47]. Similar findings were noted in neonates in a single retrospective study [245]. Femoral catheters have been demonstrated to have high colonization rates compared with subclavian and internal jugular sites when used in adults and, in some studies, higher rates of CLABSIs [40, 45–47, 50, 51, 246]. Femoral catheters should also be avoided, when possible, because they are associated with a higher risk for deep venous thrombosis than are internal jugular or subclavian catheters [48–50, 53, 247]. One study [38] found that the risk of infection associated with catheters placed in the femoral vein is accentuated in obese patients. In contrast to adults, studies in pediatric patients have demonstrated that femoral catheters have a low incidence of mechanical complications and might have an equivalent infection rate to that of non-femoral catheters [248–251]. Thus, in adult patients, a subclavian site is preferred for infection control purposes, although other

factors (e.g., the potential for mechanical complications, risk for subclavian vein stenosis, and catheter-operator skill) should be considered when deciding where to place the catheter.

In two meta-analyses, the use of real-time two-dimensional ultrasound for the placement of CVCs substantially decreased mechanical complications and reduced the number of attempts at required cannulation and failed attempts at cannulation compared with the standard landmark placement [60, 61]. Evidence favors the use of two-dimensional ultrasound guidance over Doppler ultrasound guidance [60]. Site selection should be guided by patient comfort, ability to secure the catheter, and maintenance of asepsis as well as patient-specific factors (e.g., preexisting catheters, anatomic deformity, and bleeding diathesis), relative risk of mechanical complications (e.g., bleeding and pneumothorax), the availability of bedside ultrasound, the experience of the person inserting the catheter, and the risk for infection.

Catheters should be inserted as great a distance as possible from open wounds. In one study, catheters inserted close to open burn wounds (i.e, 25 cm² overlapped a wound) were 1.79 times more likely to be colonized and 5.12 times more likely to be associated with bacteremia than catheters inserted farther from the wounds [252].

Type of Catheter Material. Polytetrafluoroethylene (Teflon[®]) or polyurethane catheters have been associated with fewer infectious complications than catheters made of polyvinyl chloride or polyethylene [36, 253, 254]. Steel needles used as an alternative to catheters for peripheral venous access have the same rate of infectious complications as do Teflon[®] catheters [33, 34]. However, the use of steel needles frequently is complicated by infiltration of intravenous (IV) fluids into the subcutaneous tissues, a potentially serious complication if the infused fluid is a vesicant [34].

Hand Hygiene and Aseptic Technique

Recommendations

1. Perform hand hygiene procedures, either by washing hands with conventional soap and water or with alcohol-based hand rubs (ABHR). Hand hygiene should be performed before and after palpating catheter insertion sites as well as before and after inserting, replacing, accessing, repairing, or dressing an intravascular catheter. Palpation of the

insertion site should not be performed after the application of antiseptic, unless aseptic technique is maintained [12, 77–79]. Category IB

2. Maintain aseptic technique for the insertion and care of intravascular catheters [37, 73, 74, 76]. Category IB
3. Wear clean gloves, rather than sterile gloves, for the insertion of peripheral intravascular catheters, if the access site is not touched after the application of skin antiseptics. Category IC
4. Sterile gloves should be worn for the insertion of arterial, central, and midline catheters [37, 73, 74, 76]. Category IA
5. Use new sterile gloves before handling the new catheter when guidewire exchanges are performed. Category II
6. Wear either clean or sterile gloves when changing the dressing on intravascular catheters. Category IC

Background

Hand hygiene before catheter insertion or maintenance, combined with proper aseptic technique during catheter manipulation, provides protection against infection [12]. Proper hand hygiene can be achieved through the use of either an alcohol-based product [255] or with soap and water with adequate rinsing [77]. Appropriate aseptic technique does not necessarily require sterile gloves for insertion of peripheral catheters; a new pair of disposable nonsterile gloves can be used in conjunction with a "no-touch" technique for the insertion of peripheral venous catheters. Sterile gloves must be worn for placement of central catheters since a "no-touch" technique is not possible.

Maximal Sterile Barrier Precautions

Recommendations

1. Use maximal sterile barrier precautions, including the use of a cap, mask, sterile gown, sterile gloves, and a sterile full body drape, for the insertion of CVCs, PICCs, or guidewire exchange [14, 75, 76, 80]. Category IB
2. Use a sterile sleeve to protect pulmonary artery catheters during insertion [81]. Category IB

Background

Maximum sterile barrier (MSB) precautions are defined as wearing a sterile gown, sterile gloves, and cap and using a full body drape (similar to the drapes used in the operating room) during the placement of CVC. Maximal sterile barrier precautions during insertion of CVC were compared with sterile gloves and a small drape in a randomized controlled trial. The MSB group had fewer episodes of both catheter colonization (RR = .32, 95% CI, .10–.96, P = .04) and CR-BSI (RR = .16, 95% CI, .02–1.30, P = .06). In addition, the group using MSB precautions had infections that occurred much later and contained gram negative, rather than gram positive, organisms [76]. A study of pulmonary artery catheters also secondarily demonstrated that use of MSB precautions lowered risk of infection [37]. Another study evaluated an educational program directed at improving infection control practices, especially MSB precautions. In this study, MSB precautions use increased and CRBSI decreased [14]. A small trial demonstrated a reduced risk of skin colonization at the insertion site when MSB precautions were used [OR 3.40, 95%CI 1.32 to 3.67] [80].

Skin Preparation

Recommendations

1. Prepare clean skin with an antiseptic (70% alcohol, tincture of iodine, an iodophor or chlorhexidine gluconate) before peripheral venous catheter insertion [82]. Category IB
2. Prepare clean skin with a >0.5% chlorhexidine preparation with alcohol before central venous catheter and peripheral arterial catheter insertion and during dressing changes. If there is a contraindication to chlorhexidine, tincture of iodine, an iodophor, or 70% alcohol can be used as alternatives [82, 83]. Category IA
3. No comparison has been made between using chlorhexidine preparations with alcohol and povidone-iodine in alcohol to prepare clean skin. Unresolved issue.
4. No recommendation can be made for the safety or efficacy of chlorhexidine in infants aged <2 months. Unresolved issue
5. Antiseptics should be allowed to dry according to the manufacturer's recommendation prior to placing the catheter [82, 83]. Category IB

Background

Two well-designed studies evaluating the chlorhexidine-containing cutaneous antiseptic regimen in comparison with either povidone iodine or alcohol for the care of an intravascular catheter insertion site have shown lower rates of catheter colonization or CRBSI associated with the chlorhexidine preparation [82, 83]. (The comparison of chlorhexidine gluconate alcohol to povidone iodine alcohol has not been done.) When 0.5% tincture of chlorhexidine was compared with 10% povidone iodine, no differences were seen in central venous catheter (CVC) colonization or in CRBSI [256]. In a three-armed study (2% aqueous chlorhexidine gluconate vs 10% povidone-iodine vs 70% alcohol), 2% aqueous chlorhexidine gluconate tended to decrease CRBSI compared with 10% povidone iodine or 70% alcohol [82]. A meta-analysis of 4,143 catheters suggested that chlorhexidine preparation reduced the risk of catheter related infection by 49% (95% CI .28 to .88) relative to povidone iodine [257]. An economic decision analysis based on available evidence suggested that the use of chlorhexidine, rather than povidone iodine, for CVC care would result in a 1.6% decrease in the incidence of CRBSI, a 0.23% decrease in the incidence of death, and a savings of \$113 per catheter used [258]. While chlorhexidine has become a standard antiseptic for skin preparation for the insertion of both central and peripheral venous catheters, 5% povidone iodine solution in 70% ethanol was associated with a substantial reduction of CVC-related colonization and infection compared with 10% aqueous povidone iodine [259].

Catheter Site Dressing Regimens

Recommendations

1. Use either sterile gauze or sterile, transparent, semipermeable dressing to cover the catheter site [84–87]. Category IA
2. If the patient is diaphoretic or if the site is bleeding or oozing, use gauze dressing until this is resolved [84–87]. Category II
3. Replace catheter site dressing if the dressing becomes damp, loosened, or visibly soiled [84, 85]. Category IB

4. Do not use topical antibiotic ointment or creams on insertion sites, except for dialysis catheters, because of their potential to promote fungal infections and antimicrobial resistance [88, 89]. Category IB
5. Do not submerge the catheter or catheter site in water. Showering should be permitted if precautions can be taken to reduce the likelihood of introducing organisms into the catheter (e.g., if the catheter and connecting device are protected with an impermeable cover during the shower) [90–92]. Category IB
6. Replace dressings used on short-term CVC sites every 2 days for gauze dressings. Category II
7. Replace dressings used on short-term CVC sites at least every 7 days for transparent dressings, except in those pediatric patients in which the risk for dislodging the catheter may outweigh the benefit of changing the dressing [87, 93]. Category IB
8. Replace transparent dressings used on tunneled or implanted CVC sites no more than once per week (unless the dressing is soiled or loose), until the insertion site has healed. Category II
9. No recommendation can be made regarding the necessity for any dressing on well-healed exit sites of long-term cuffed and tunneled CVCs. Unresolved issue
10. Ensure that catheter site care is compatible with the catheter material [94, 95]. Category IB
11. Use a sterile sleeve for all pulmonary artery catheters [80]. Category IB
12. Use a chlorhexidine-impregnated sponge dressing for temporary short-term catheters in patients older than 2 months of age if the CLABSI rate is not decreasing despite adherence to basic prevention measures, including education and training, appropriate use of chlorhexidine for skin antisepsis, and MSB [93, 96–98]. Category 1B
13. No recommendation is made for other types of chlorhexidine dressings. Unresolved issue
14. Monitor the catheter sites visually when changing the dressing or by palpation through an intact dressing on a regular basis, depending on the clinical situation of the individual patient. If patients have tenderness at the insertion site, fever without obvious source,

or other manifestations suggesting local or bloodstream infection, the dressing should be removed to allow thorough examination of the site [99–101]. Category IB

15. Encourage patients to report any changes in their catheter site or any new discomfort to their provider. Category II

Background

Transparent, semi-permeable polyurethane dressings permit continuous visual inspection of the catheter site and require less frequent changes than do standard gauze and tape dressings. In the largest controlled trial of dressing regimens on peripheral catheters, the infectious morbidity associated with the use of transparent dressings on approximately 2,000 peripheral catheters was examined [254]. Data from this study suggest that the rate of colonization among catheters dressed with transparent dressings (5.7%) is comparable to that of those dressed with gauze (4.6%) and that no clinically substantial differences exist in the incidence of either catheter site colonization or phlebitis. Furthermore, these data suggest that transparent dressings can be safely left on peripheral venous catheters for the duration of catheter insertion without increasing the risk for thrombophlebitis [254].

A meta-analysis has assessed studies that compared the risk for CRBSIs using transparent dressings versus using gauze dressing [260]. The risk for CRBSIs did not differ between the groups. The choice of dressing can be a matter of preference. If blood is oozing from the catheter insertion site, gauze dressing is preferred. Another systemic review of randomized controlled trials comparing gauze and tape to transparent dressings found no significant differences between dressing types in CRBSIs, catheter tip colonization, or skin colonization [261].

Chlorhexidine impregnated dressings have been used to reduce the risk of CRBSI. In the largest multicenter randomized controlled trial published to date comparing chlorhexidine impregnated sponge dressings vs standard dressings in ICU patients, rates of CRBSIs were reduced even when background rates of infection were low. In this study, 1636 patients (3778 catheters, 28 931 catheter-days) were evaluated. The chlorhexidine- impregnated sponge dressings decreased the rates of major CRBSIs (10/1953 [0.5%], 0.6 per 1000 catheter-days vs 19/1825 [1.1%], 1.4 per 1000 catheter-days; hazard ratio [HR], 0.39 [95% confidence interval

{CI}, .17–.93]; $P = .03$) and CRBSIs (6/1953 catheters, 0.40 per 1000 catheter-days vs 17/1825 catheters, 1.3 per 1000 catheter-days; HR, 0.24 [95% CI, .09–.65]) [93]. A randomized controlled study of polyurethane or a chlorhexidine impregnated sponge dressing in 140 children showed no statistical difference in BSIs; however, the chlorhexidine group had lower rates of CVC colonization [98]. In 601 cancer patients receiving chemotherapy, the incidence of CRBSI was reduced in patients receiving the chlorhexidine impregnated sponge dressing compared with standard dressings ($P = .016$, relative risk 0.54; confidence interval 0.31–.94) [262]. A meta-analysis that included eight randomized controlled trials demonstrated that chlorhexidine impregnated sponge dressings are associated with a reduction of vascular and epidural catheter exit site colonization but no significant reduction in CRBSI (2.2% versus 3.8%, OR 0.58, 95% CI: .29–1.14, $p = .11$) [97].

Although data regarding the use of a chlorhexidine impregnated sponge dressing in children are limited, one randomized, controlled study involving 705 neonates reported a substantial decrease in colonized catheters in infants in the chlorhexidine impregnated sponge dressing group compared with the group that had standard dressings (15% versus 24%; RR = .6; 95% CI 0.5–.9), but no difference in the rates of CRBSI or BSI without a source. Chlorhexidine impregnated sponge dressings were associated with localized contact dermatitis in infants of very low birth weight. In 98 neonates with very low birth weight, 15 (15%) developed localized contact dermatitis; four (1.5%) of 237 neonates weighing >1,000 g developed this reaction ($P < .0001$). Infants with gestational age <26 weeks who had CVCs placed at age <8 days were at increased risk for having localized contact dermatitis, whereas no infants in the control group developed this local reaction [96].

Patient Cleansing

Recommendation

Use a 2% chlorhexidine wash for daily skin cleansing to reduce CRBSI [102–104].

Category II

Background

Daily cleansing of ICU patients with a 2% chlorhexidine impregnated washcloth may be a simple, effective strategy to decrease the rate of primary BSIs. In a single center study of 836 ICU patients, patients receiving the chlorhexidine intervention were significantly less likely to acquire a primary BSI (4.1 vs 10.4 infections per 1000 patient days; incidence difference, 6.3 [95% confidence interval, 1.2–11.0]) than those bathed with soap and water [102].

Catheter Securement Devices

Recommendation

Use a sutureless securement device to reduce the risk of infection for intravascular catheters [105]. Category II

Background

Catheter stabilization is recognized as an intervention to decrease the risk for phlebitis, catheter migration and dislodgement, and may be advantageous in preventing CRBSIs. Pathogenesis of CRBSI occurs via migration of skin flora through the percutaneous entry site. Sutureless securement devices avoid disruption around the catheter entry site and may decrease the degree of bacterial colonization. [105]. Using a sutureless securement device also mitigates the risk of sharps injury to the healthcare provider from inadvertent needlestick injury.

Antimicrobial/Antiseptic Impregnated Catheters and Cuffs

Recommendation

Use a chlorhexidine/silver sulfadiazine or minocycline/ rifampin -impregnated CVC in patients whose catheter is expected to remain in place >5 days if, after successful implementation of a comprehensive strategy to reduce rates of CLABSI, the CLABSI rate is not decreasing. The comprehensive strategy should include at least the following three components: educating persons who insert and maintain catheters, use of maximal sterile barrier precautions, and a >0.5% chlorhexidine preparation with alcohol for skin antisepsis during CVC insertion [106–113]. Category IA

Background

Certain catheters and cuffs that are coated or impregnated with antimicrobial or antiseptic agents can decrease the risk for CRBSI and potentially decrease hospital costs associated with treating CRBSIs, despite the additional acquisition cost of an antimicrobial/antiseptic impregnated catheter [110]. Nearly all of the studies involving antimicrobial/antiseptic-impregnated catheters have been conducted using triple-lumen, uncuffed catheters in adult patients whose catheters remained in place <30 days. While most of the studies have been conducted in adults, these catheters have been approved by FDA for use in patients weighing >3 kg. Two non-randomized studies [112, 113] in pediatric ICU patients suggest that these catheters might reduce risk of catheter-associated infection. No antiseptic or antimicrobial impregnated catheters currently are available for use in infants weighing <3kg.

Chlorhexidine/Silver Sulfadiazine Catheters coated with chlorhexidine/silver sulfadiazine only on the external luminal surface have been studied as a means to reduce CRBSI. Two meta-analyses of first-generation catheters [1, 263] demonstrated that such catheters reduced the risk for CRBSI compared with standard non-coated catheters. The duration of catheter placement in one study ranged from 5.1 to 11.2 days [264]. A second-generation catheter is now available with chlorhexidine coating the internal surface extending into the extension set and hubs while the external luminal surface is coated with chlorhexidine and silver sulfadiazine. The external surface has three times the amount of chlorhexidine and extended release of the surface bound antiseptics than that in the first generation catheters. All three prospective, randomized studies of second-generation catheters demonstrated a significant reduction in catheter colonization, but they were underpowered to show a difference in CRBSI [106–108]. Prolonged anti-infective activity provides improved efficacy in preventing infections [265]. Although rare, anaphylaxis with the use of these chlorhexidine/silver sulfadiazine catheters has been observed [266–270].

Chlorhexidine/silver sulfadiazine catheters are more expensive than standard catheters. However, one analysis has suggested that the use of chlorhexidine/silver sulfadiazine catheters should lead to a cost savings of \$68 to \$391 per catheter [271] in settings in which the risk for CRBSI is high, despite adherence to other preventive strategies (e.g., maximal barrier precautions and aseptic techniques). Use of these catheters might be cost effective in ICU

patients, burn patients, neutropenic patients, and other patient populations in which the rate of infection exceeds 3.3 per 1,000 catheter days [264].

Minocycline/Rifampin In a multicenter randomized trial, CVCs impregnated on both the external and internal surfaces with minocycline/rifampin were associated with lower rates of CRBSI when compared with the first generation chlorhexidine/ silver sulfadiazine impregnated catheters [109]. The beneficial effect began after day 6 of catheterization. Silicone minocycline/ rifampin impregnated CVCs with an average dwell time of over 60 days have been shown to be effective in reducing CRBSI [111]. No minocycline/rifampin-resistant organisms were reported in these studies. Two trials demonstrated that use of these catheters significantly reduced CRBSI compared with uncoated catheters [110, 111]. No comparative studies have been published using the second-generation chlorhexidine/silver sulfadiazine catheter. Although there have been concerns related to the potential for development of resistance, several prospective clinical studies have shown that the risk is low [272, 273]. Further, no resistance to minocycline or rifampin related to the use of the catheter has been documented in the clinical setting. Two studies using decision model analysis revealed these catheters were associated with superior cost savings compared with first generation chlorhexidine/ silver sulfadiazine catheters [274, 275]. Such analysis needs to be done compared with the second-generation catheters. However, as baseline rates of infection decrease and the cost of catheters decrease, the cost-benefit ratio will likely change.

The decision to use chlorhexidine/silver sulfadiazine or minocycline/rifampin impregnated catheters should be based on the need to enhance prevention of CRBSI after bundled standard procedures have been implemented (e.g., educating personnel, using maximal sterile barrier precautions, and using >0.5% chlorhexidine preparation with alcohol for skin antiseptics) and then balanced against the concern for emergence of resistant pathogens and the cost of implementing this strategy.

Platinum/Silver A combination platinum/silver impregnated catheter (i.e., a silver iontophoretic catheter) is available for use in the United States. Several prospective, randomized studies have been published comparing these catheters to uncoated catheters [276–279]. One study showed a reduction in the incidence density of catheter colonization and

CRBSI [278], but the other studies found no difference in catheter colonization or CRBSI between the impregnated catheter and a non-impregnated catheter [39, 276, 277]. In light of this, a firm recommendation for or against the use of these catheters cannot be made.

Systemic Antibiotic Prophylaxis

Recommendation

Do not administer systemic antimicrobial prophylaxis routinely before insertion or during use of an intravascular catheter to prevent catheter colonization or CRBSI [114].

Category IB

Background

Several studies have examined the role of systemic antibiotic prophylaxis in prevention of catheter-related infection. A recent meta-analysis reviewed these studies in oncology patients [114]. Four studies used a prophylactic glycopeptide prior to catheter insertion. However, heterogeneity in these studies precludes making any conclusion regarding efficacy.

In a study examining the effect of ongoing oral prophylaxis with rifampin and novobiocin on catheter-related infection in cancer patients treated with interleukin-2 [280], a reduction in CRBSI was observed, even though 9 of 26 subjects (35%) discontinued the prophylactic antibiotics due to side effects or toxicity. In non-oncology patients, no benefit was associated with vancomycin administration prior to catheter insertion in 55 patients undergoing catheterization for parenteral nutrition [281]. Similarly, extending perioperative prophylactic antibiotics in cardiovascular surgery patients did not reduce central venous catheter colonization [282]. A recent Cochrane review of prophylactic antibiotics in neonates with umbilical venous catheters concluded that there is insufficient evidence from randomized trials to support or refute the use of prophylactic antibiotics [283].

Late onset neonatal sepsis is often due to coagulase negative staphylococci and is thought to frequently stem from infected central venous catheters. Five trials involved a total of 371 neonates comparing vancomycin by continuous infusion via parenteral nutrition or intermittent dosing, and placebo. The infants treated with vancomycin experienced less sepsis (RR .11; 95% CI .05-.24) and less sepsis due to coagulase negative staphylococci (RR .33; 95% CI

.19–.59) [284]. However, mortality and length of stay were not significantly different between the two groups. There were insufficient data to evaluate the risk of selection for vancomycin resistant organisms.

Antibiotic/Antiseptic Ointments

Recommendation

Use povidone iodine antiseptic ointment or bacitracin/ gramicidin/polymyxin B ointment at the hemodialysis catheter exit site after catheter insertion and at the end of each dialysis session only if this ointment does not interact with the material of the hemodialysis catheter per manufacturer’s recommendation [59, 115–119]. Category IB

Background

A variety of topical antibiotic or antiseptic ointments have been utilized in attempts to lower the antimicrobial burden at the catheter insertion site and thus prevent infection. A number of older studies, examining primarily peripheral venous catheters, yielded varying conclusions [82, 285, 286]. In addition, the use of antibiotic ointments that have limited antifungal activity may serve to increase colonization and/or infection due to *Candida* species [89].

More recent studies have examined this approach in high-risk patients, particularly those undergoing hemodialysis [116–119]. Three randomized, controlled trials have evaluated the use of 10% povidone iodine [117–119]. A significant decrease in colonization, exit-site infection, or bloodstream infection was observed. The beneficial effect was most prominent in subjects with nasal colonization by *Staphylococcus aureus* [117–119].

Nasal carriers of *S. aureus* are more likely to experience a CRBSI than non-colonized persons [287–289]. This has prompted investigators to assess the utility of topical mupirocin, a potent anti-staphylococcal agent. Several studies have demonstrated a reduced risk of CRBSI when mupirocin ointment was applied at the catheter insertion site [117, 290–292]. Others have shown similar benefits when mupirocin was applied nasally [288, 289, 293]. However, enthusiasm for this measure has been dampened by the rapid emergence of mupirocin

resistance observed at some centers [88, 294, 295], and the potential degrading effect that mupirocin has on polyurethane catheters [94, 95].

In the only study demonstrating a significant effect on mortality, the application of bacitracin/gramicidin/polymyxin B ointment at the catheter insertion site was compared with placebo in 169 hemodialysis patients [296]. Infections were observed in more patients in the placebo group than in the bacitracin/gramicidin/polymyxin B group (34 versus 12%; relative risk, 0.35; 95% CI, .18 to .68). The number of infections per 1,000 catheter days (4.10 versus 1.02; $P < .0001$) and the number of bacteremias per 1,000 catheter days (2.48 versus .63; $P = .0004$) were also greater in the placebo group. Within the 6-month study period, there were 13 deaths in the placebo group as compared with three deaths in the bacitracin/gramicidin/polymyxin B group ($P = .004$). Thus, there is evidence from one study in hemodialysis patients that bacitracin/gramicidin/polymyxin B ointment can improve outcome, but no similar data exist for use in other patient populations [296]. It should be noted that the gramicidin-containing ointment is not currently available in the United States.

Antibiotic Lock Prophylaxis, Antimicrobial Catheter Flush and Catheter Lock Prophylaxis

Recommendation

Use prophylactic antimicrobial lock solution in patients with long term catheters who have a history of multiple CRBSI despite optimal maximal adherence to aseptic technique [120– 138]. Category II

Background

To prevent CRBSI, a wide variety of antibiotic and antiseptic solutions have been used to flush or lock catheter lumens [120– 138]. Catheter lock is a technique by which an antimicrobial solution is used to fill a catheter lumen and then allowed to dwell for a period of time while the catheter is idle. Antibiotics of various concentrations that have been used either alone (when directed at a specific organism) or in combination (to achieve broad empiric coverage) to prophylactically flush or lock central venous catheters include vancomycin, gentamicin, ciprofloxacin, minocycline, amikacin, cefazolin, cefotaxime, and ceftazidime; while antiseptics have included alcohol, taurolidine, trisodium citrate. (Taurolidine and trisodium citrate are not

approved for this use in the United States). These agents are usually combined with a compound acting as an anticoagulant, such as heparin or EDTA. Most of these studies have been conducted in relatively small numbers of high-risk patients, such as hemodialysis patients, neonates, or neutropenic oncology patients. Although most studies indicate a beneficial effect of the antimicrobial flush or lock solution in terms of prevention of catheter-related infection, this must be balanced by the potential for side effects, toxicity, allergic reactions, or emergence of resistance associated with the antimicrobial agent. The wide variety of compounds used, the heterogeneity of the patient populations studied, and limitations in the size or design of studies preclude a general recommendation for use. In addition, there are no FDA approved formulations approved for marketing, and most formulations have been prepared in hospital pharmacies. A brief overview of some of the studies follows.

At least 10 studies regarding catheter flush or lock solutions have been performed in hemodialysis patients [128, 129, 131– 138]. Three meta-analyses have all demonstrated that catheter lock solutions reduce risk of CRBSI in hemodialysis patients [297–299]. In the largest of these studies, 291 subjects were enrolled in a prospective randomized comparison of 30% trisodium citrate versus heparin [133]. The rate of CRBSI was significantly lower in the group whose catheters were locked with trisodium citrate (4.1 BSI/1,000 CVC days vs. 1.1 BSI/1,000 CVC days, $P < .001$), and no significant difference in thrombosis or occlusion of the catheter was noted. However, if infused rapidly, concentrated citrate can result in serious hypocalcaemia, cardiac dysrhythmia, and death. The second largest study in hemodialysis subjects examined the effect of a catheter lock solution containing cefazolin, gentamicin, and heparin compared with control patients receiving only heparin [135]. In 120 subjects, the rate of CRBSI was significantly lower in those receiving the antibiotic lock solution (0.44 BSI/1,000 CVC days vs. 3.12 BSI/1,000 CVC days, $P = .03$) [135]. Other trials in hemodialysis patients have studied minocycline, gentamicin, EDTA, heparin, taurolidine, vancomycin, and cefotaxime.

At least five studies have been conducted in pediatric oncology patients [120, 121, 124, 126, 127]. In the largest trial, 126 subjects were enrolled in a prospective, randomized, double blind study comparing vancomycin/ciprofloxacin/heparin (VCH) to vancomycin/heparin (VH) to heparin (H) alone [124]. The time to CVC-related infection was significantly longer in the VCH or

VH arms of the study compared with heparin, and the rate of infection was significantly lower with either of the antibiotic containing solutions compared with heparin alone (1.72/1,000 CVC days [H] vs. 0.55/1,000 CVC days [VCH] vs. 0.37/1,000 CVC days [VH]).

In a meta-analysis of seven randomized, controlled trials examining the utility of vancomycin-containing lock or flush solutions compared with heparin alone, the risk ratio for vancomycin/heparin solutions was 0.49 (95% CI .26–.95, P = .03) [300]. Use of the catheter lock technique appeared to have greater benefit than simply flushing vancomycin through the catheter.

Recently, a prospective, double blind, randomized trial compared the utility of 70% ethanol lock versus heparinized saline for the prevention of primary CRBSI in oncology patients. Patients receiving the ethanol lock preventive therapy were significantly less likely to experience a primary CRBSI (0.60/ 1,000 CVC days vs. 3.11/1,000 CVC days; OR 0.18, 95% CI .05-.65, P5 .008) [301].

Anticoagulants

Recommendation

Do not routinely use anticoagulant therapy to reduce the risk of catheter-related infection in general patient populations [139]. Category II

Background

Shortly after insertion, intravascular catheters are coated with a conditioning film, consisting of fibrin, plasma proteins, and cellular elements, such as platelets and red blood cells [213, 302]. Microbes interact with the conditioning film, resulting in colonization of the catheter [303]. There is a close association between thrombosis of central venous catheters and infection [221, 304, 305]. Therefore, anticoagulants have been used to prevent catheter thrombosis and presumably reduce the risk of infection.

In a meta-analysis evaluating the benefit of heparin prophylaxis (3 units/mL in parenteral nutrition, 5,000 units every 6 or 12 hours flush or 2,500 units low molecular weight heparin subcutaneously) in patients with short-term CVCs, the risk for catheter-related central venous thrombosis was reduced with the use of prophylactic heparin [139]. However, no

substantial difference in the rate of CRBSI was observed. In a more recent prospective, randomized trial, 204 patients with non-tunneled catheters were assigned to receive a continuous infusion of heparin (100 units/kg/ d) or saline (50 mL/d) [306]. The rate of CRBSI was significantly decreased in the group receiving heparin (2.5 BSI/1,000 CVC days vs. 6.4 BSI/1,000 CVC days). Because the majority of heparin solutions contain preservatives with antimicrobial activity, whether any decrease in the rate of CRBSI is a result of the reduced thrombus formation, the preservative, or both is unclear. The majority of pulmonary artery, umbilical, and central venous catheters are available as heparin-bonded devices. The majority of catheters are heparin bonded with benzalkonium, which provides the catheters with antimicrobial activity [307] and provides an anti-thrombotic effect [308]. However, some catheters have heparin bound directly to the catheter without benzalkonium [309]. Studies have shown that heparin-bonded catheters reduce risk of thrombosis and risk of CRBSI [306, 308– 310], but are less effective at reducing catheter colonization than catheters impregnated with chlorhexidine/silver sulfadiazine [311]. Unfortunately, heparin-induced thrombocytopenia can occur and has prompted many clinicians to avoid heparin [312]. Trisodium citrate has been recommended as a catheter lock solution because it possesses both anticoagulant and antimicrobial properties [133]. In a prospective, randomized, double blind study in hemodialysis patients, use of interdialytic heparin (5,000 U/mL) was associated with a significantly greater rate of CRBSIs compared with use of 30% trisodium citrate (4.1 BSI/ 1,000 CVC days vs. 1.1BSI/1,000 CVC days [313].

Warfarin has been evaluated as a means to reduce CVC thrombus formation and, hence, infection [314–318]. In patients with long-term CVCs, low dose warfarin (i.e., 1 mg/day) reduced the incidence of catheter thrombus [142, 143]. However, other studies have not confirmed reduced thrombosis and still others have found untoward interactions in patients receiving 5-FU [319, 320]. Data are limited; although low dose warfarin decreases the risk of thrombus formation in cancer patients, it has not been shown to reduce infectious complications. Over 20% of patients in some studies develop prolonged prothrombin times and required dosage adjustment [321]. Other anticoagulants, such as factor Xa inhibitors or direct

thrombin inhibitors, have not been adequately assessed in terms of reducing the risk of catheter-associated infection.

Replacement of Peripheral and Midline Catheters

Recommendations

1. There is no need to replace peripheral catheters more frequently than every 72–96 hours to reduce risk of infection and phlebitis in adults [36, 140, 141]. Category 1B
2. No recommendation is made regarding replacement of peripheral catheters in adults only when clinically indicated [142–144]. Unresolved issue
3. Replace peripheral catheters in children only when clinically indicated [32, 33]. Category 1B
4. Replace midline catheters only when there is a specific indication. Category II

Background

Scheduled replacement of intravascular catheters has been proposed as a method to prevent phlebitis and catheter-related infections. Studies of short peripheral venous catheters indicate that the incidence of thrombophlebitis and bacterial colonization of catheters increases when catheters are left in place >72 hours [258]. However, rates of phlebitis are not substantially different in peripheral catheters left in place 72 hours compared with 96 hours [141]. Because phlebitis and catheter colonization have been associated with an increased risk for catheter-related infection, short peripheral catheter sites commonly are replaced at 72–96 hour intervals to reduce both the risk for infection and patient discomfort associated with phlebitis.

Some studies have suggested that planned removal at 72 hours vs. removing as needed resulted in similar rates of phlebitis and catheter failure [142–144]. However, these studies did not address the issue of CRBSI, and the risk of CRBSIs with this strategy is not well studied.

Midline catheters are associated with lower rates of phlebitis than short peripheral catheters and with lower rates of infection than CVCs [322–324]. In one prospective study of 140 midline catheters, their use was associated with a BSI rate of 0.8 per 1,000 catheter days [324]. No specific risk factors, including duration of catheterization, were associated with

infection. Midline catheters were in place a median of 7 days, but for as long as 49 days. Although the findings of this study suggested that midline catheters could be changed only when there is a specific indication, no prospective, randomized studies have assessed the benefit of routine replacement as a strategy to prevent CRBSI associated with midline catheters.

Replacement of CVCs, Including PICCs and Hemodialysis Catheters

Recommendations

1. Do not routinely replace CVCs, PICCs, hemodialysis catheters, or pulmonary artery catheters to prevent catheter-related infections. Category IB
2. Do not remove CVCs or PICCs on the basis of fever alone. Use clinical judgment regarding the appropriateness of removing the catheter if infection is evidenced elsewhere or if a noninfectious cause of fever is suspected. Category II
3. Do not use guidewire exchanges routinely for non-tunneled catheters to prevent infection. Category IB
4. Do not use guidewire exchanges to replace a non-tunneled catheter suspected of infection. Category IB
5. Use a guidewire exchange to replace a malfunctioning non-tunneled catheter if no evidence of infection is present. Category IB
6. Use new sterile gloves before handling the new catheter when guidewire exchanges are performed. Category II

Background

Catheter replacement at scheduled time intervals as a method to reduce CRBSI has not lowered rates. Two trials have assessed a strategy of changing the catheter every 7 days compared with a strategy of changing catheters as needed [165, 325]. One of these studies involved 112 surgical ICU patients needing CVCs, pulmonary artery catheters, or peripheral arterial catheters [165], whereas the other study involved only subclavian hemodialysis catheters [325]. In both studies, no difference in CRBSI was observed in patients undergoing

scheduled catheter replacement every 7 days compared with patients whose catheters were replaced as needed.

Scheduled guidewire exchange of CVCs is another proposed strategy for preventing CRBSI. The results of a meta-analysis of 12 randomized, controlled trials assessing CVC management failed to demonstrate any reduction of CRBSI rates through routine replacement of CVCs by guidewire exchange compared with catheter replacement on an as needed basis [326]. Thus, routine replacement of CVCs is not necessary for catheters that are functioning and have no evidence of causing local or systemic complications.

Catheter replacement over a guidewire has become an accepted technique for replacing a malfunctioning catheter or exchanging a pulmonary artery catheter for a CVC when invasive monitoring no longer is needed. Catheter insertion over a guidewire is associated with less discomfort and a significantly lower rate of mechanical complications than are those percutaneously inserted at a new site [327]. In addition, this technique provides a means of preserving limited venous access in some patients. Replacement of temporary catheters over a guidewire in the presence of bacteremia is not an acceptable replacement strategy because the source of infection is usually colonization of the skin tract from the insertion site to the vein [37, 327]. However, in selected patients with tunneled hemodialysis catheters and bacteremia, catheter exchange over a guidewire, in combination with antibiotic therapy, is an alternative as a salvage strategy in patients with limited venous access [328–331].

Because of the increased difficulty obtaining vascular access in children, attention should be given to the frequency with which catheters are replaced in these patients. In a study in which survival analysis techniques were used to examine the relation between the duration of central venous catheterization and complications in pediatric ICU patients, all of the patients studied ($n = 397$) remained uninfected for a median of 23.7 days [250]. In addition, no relation was found between duration of catheterization and the daily probability of infection ($r = 0.21$; $P > .1$), suggesting that routine replacement of CVCs likely does not reduce the incidence of catheter-related infection [250].

Vascular access sites can be even more limited among neonates. Four randomized trials ($n = 368$) summarized in a recent Cochrane Database Systemic Review compared the effects of

giving parenteral nutrition through percutaneous central venous catheters vs. peripheral intravenous catheters. Fewer painful procedures (venipunctures) were required in neonates randomized to percutaneously placed CVCs, and there was no evidence for increased risk of BSIs [332].

CVC occlusion due to thrombus formation is one of the most common reasons for CVC removal in neonates. Various methods have been tried to prevent catheter occlusion. Recently, a randomized trial (n = 201) evaluated whether a continuous heparin infusion (0.5 units/kg/hour) could effectively prolong the duration of catheterization when compared with a placebo infusion. The rate of catheter occlusion requiring catheter removal was lower in the heparin group (6% vs. 31%, P = .001; NNT = 4). Rates of CRBSI were similar, although the study was not powered to evaluate CRBSI rate differences. Heparin associated antibody levels were not routinely measured [333].

Hemodialysis Catheters. The use of catheters for hemodialysis is the most common factor contributing to bacteremia in dialysis patients [334, 335]. The relative risk for bacteremia in patients with dialysis catheters is sevenfold the risk for patients with arteriovenous (AV) fistulas [336]. AV fistulas and grafts are preferred over hemodialysis catheters in patients with chronic renal failure, due to their lower associated risk of infection. If temporary access is needed for dialysis, a tunneled cuffed catheter is preferable to a non-cuffed catheter, even in the ICU setting, if the catheter is expected to stay in place for >3weeks [59].

Pulmonary Artery Catheters. Pulmonary artery catheters are inserted through a Teflon® introducer and typically remain in place an average of 3 days. The majority of pulmonary artery catheters are heparin bonded, which reduces not only catheter thrombosis but also microbial adherence to the catheter [307]. Meta-analysis indicates that the CRBSI rate associated with pulmonary artery catheterization is 3.7 per 1,000 catheter days and somewhat higher than the rate observed for unmedicated and non-tunnelled CVCs (2.7 per 1,000 catheter days)[6, 45].

Data from prospective studies indicate that the risk of significant catheter colonization and CRBSI increases the longer the catheter remains in place. In general, the risk of significant catheter colonization increases after 4 days of catheterization [75, 337, 338], whereas the risk of CRBSI increases beyond 5-7 days of catheterization [75, 84, 166]. Efforts must be made to

differentiate between infection related to the introducer and that related to the pulmonary artery catheter. Significant colonization of the introducer occurs earlier than that of the pulmonary artery catheter [337, 339]. However, no studies indicate that catheter replacement at scheduled time intervals is an effective method to reduce risk of CRBSI [165, 327, 339]. In patients who continue to require hemodynamic monitoring, pulmonary artery catheters do not need to be changed more frequently than every 7 days [339]. No specific recommendation can be made regarding routine replacement of catheters that need to be in place for >7 days.

Pulmonary artery catheters are usually packaged with a thin plastic sleeve that prevents touch contamination when placed over the catheter. In a study of 166 catheters, patients who were randomly assigned to have their catheters self-contained within this sleeve had a reduced risk for CRBSI compared with those who had a pulmonary artery catheter placed without the sleeve ($P = .002$) [81].

Umbilical Catheters

Recommendations

1. Remove and do not replace umbilical artery catheters if any signs of CRBSI, vascular insufficiency in the lower extremities, or thrombosis are present [145]. Category II
2. Remove and do not replace umbilical venous catheters if any signs of CRBSI or thrombosis are present [145]. Category II
3. No recommendation can be made regarding attempts to salvage an umbilical catheter by administering antibiotic treatment through the catheter. Unresolved issue
4. Cleanse the umbilical insertion site with an antiseptic before catheter insertion. Avoid tincture of iodine because of the potential effect on the neonatal thyroid. Other iodine-containing products (e.g., povidone iodine) can be used [146– 150]. Category IB
5. Do not use topical antibiotic ointment or creams on umbilical catheter insertion sites because of the potential to promote fungal infections and antimicrobial resistance [88, 89]. Category IA
6. Add low-doses of heparin (0.25–1.0 U/ml) to the fluid infused through umbilical arterial catheters [151–153]. Category IB

7. Remove umbilical catheters as soon as possible when no longer needed or when any sign of vascular insufficiency to the lower extremities is observed. Optimally, umbilical artery catheters should not be left in place >5 days [145, 154]. Category II
8. Umbilical venous catheters should be removed as soon as possible when no longer needed, but can be used up to 14 days if managed aseptically [155, 156]. Category II
9. An umbilical catheter may be replaced if it is malfunctioning, and there is no other indication for catheter removal, and the total duration of catheterization has not exceeded 5 days for an umbilical artery catheter or 14 days for an umbilical vein catheter. Category II

Background

Although the umbilical stump becomes heavily colonized soon after birth, umbilical vessel catheterization often is used for vascular access in newborn infants. Umbilical vessels can be cannulated easily and permit both collection of blood samples and measurement of hemodynamic status. The incidences of catheter colonization and BSI are similar for umbilical vein catheters and umbilical artery catheters. In several studies, an estimated 40%–55% of umbilical artery catheters were colonized and 5% resulted in CRBSI; umbilical vein catheters were associated with colonization in 22%–59% of cases [147, 148, 340] and with CRBSI in 3%–8% of cases [148]. Although CRBSI rates are similar for umbilical catheters in the high position (i.e, above the diaphragm) compared with the low position (i.e, below the diaphragm and above the aortic bifurcation), catheters placed in the high position result in a lower incidence of vascular complications without an increase in adverse sequelae [148].

Risk factors for infection differ for umbilical artery and umbilical vein catheters. In one study, neonates with very low birth weight who also received antibiotics for >10 days were at increased risk for umbilical artery CRBSIs [148]. In comparison, those with higher birth weight and receipt of parenteral nutrition fluids were at increased risk for umbilical vein CRBSI. Duration of catheterization was not an independent risk factor for infection of either type of umbilical catheter.

A recent randomized trial (n = 210) evaluated whether long-term umbilical venous catheterization (up to 28 days) would result in the same or fewer CRBSIs when compared with

neonates who were randomized to short-term umbilical venous catheterization for 7–10 days followed by percutaneous central venous catheterization. CRBSI rate was higher (20%) among long term catheterized neonates when compared with short term catheterized neonates (13%). The difference was not statistically significant ($P = .17$), although the study was underpowered. The study was not powered to evaluate differences in venous thrombosis rates [341].

Peripheral Arterial Catheters and Pressure Monitoring Devices for Adult and Pediatric

Patients

Recommendations

1. In adults, use of the radial, brachial or dorsalis pedis sites is preferred over the femoral or axillary sites of insertion to reduce the risk of infection [46, 47, 157, 158]. Category IB
2. In children, the brachial site should not be used. The radial, dorsalis pedis, and posterior tibial sites are preferred over the femoral or axillary sites of insertion [46]. Category II
3. A minimum of a cap, mask, sterile gloves and a small sterile fenestrated drape should be used during peripheral arterial catheter insertion [47, 158, 159]. Category IB
4. During axillary or femoral artery catheter insertion, maximal sterile barriers precautions should be used. Category II
5. Replace arterial catheters only when there is a clinical indication. Category II
6. Remove the arterial catheter as soon as it is no longer needed. Category II
7. Use disposable, rather than reusable, transducer assemblies when possible [160–164]. Category IB
8. Do not routinely replace arterial catheters to prevent catheter-related infections [165, 166, 167, 168]. Category II
9. Replace disposable or reusable transducers at 96-hour intervals. Replace other components of the system (including the tubing, continuous-flush device, and flush solution) at the time the transducer is replaced [37, 161]. Category IB
10. Keep all components of the pressure monitoring system (including calibration devices and flush solution) sterile [160, 169–171]. Category IA

11. Minimize the number of manipulations of and entries into the pressure monitoring system. Use a closed flush system (i.e, continuous flush), rather than an open system (i.e, one that requires a syringe and stopcock), to maintain the patency of the pressure monitoring catheters [163, 172]. Category II
12. When the pressure monitoring system is accessed through a diaphragm, rather than a stopcock, scrub the diaphragm with an appropriate antiseptic before accessing the system [163]. Category IA
13. Do not administer dextrose-containing solutions or parenteral nutrition fluids through the pressure monitoring circuit [163, 173, 174]. Category IA
14. Sterilize reusable transducers according to the manufacturers' instructions if the use of disposable transducers is not feasible [163, 173–176]. Category IA

Background

Arterial catheters are usually inserted into the radial or femoral artery and permit continuous blood pressure monitoring and blood gas measurements. The risk of CRBSI for arterial catheters is lower than that associated with non-coated, uncuffed, non-tunneled short term CVCs (1.7 versus 2.7 per 1,000 catheter days) [6]. However, risk of CRBSI rates are comparable between arterial catheters and coated, uncuffed, non-tunneled short term CVCs [6]. Unlike CVCs, use of full barrier precautions during arterial cannulation does not appear to reduce the risk of arterial CRBSI [158, 159]. Nonetheless, when arterial catheters are inserted using a protocol which includes maximum barrier precautions, a very low risk of CRBSI (0.41/1,000 catheter days) can be achieved [47]. Although a meta-analysis failed to discern a difference in rates of CRBSI among three sites of insertion (radial, femoral, and axillary) [342], colonization of catheters inserted in the femoral site occurs more often [158]. In addition, a prospective observational study of over 2,900 arterial catheters that were inserted using maximum barrier precautions demonstrated an almost 8-fold increase in the incidence of CRBSI when the femoral site was used compared with the radial site [343]. Furthermore, there is a greater risk of CRBSI caused by gram-negative bacteria when the femoral site is used [343]. The rates of catheter colonization and CRBSI appear similar between the radial and dorsalis pedis sites [157]. The risk of developing a CRBSI increases with the duration of catheterization [166,

344]; however, the routine changing of arterial catheters at scheduled times does not result in a diminution of the risk of CRBSI [165]. Catheters that need to be in place for >5 days should not be routinely changed if no evidence of infection is observed.

Replacement of Administration Sets

Recommendations

1. In patients not receiving blood, blood products or fat emulsions, replace administration sets that are continuously used, including secondary sets and add-on devices, no more frequently than at 96-hour intervals, [177] but at least every 7 days [178–181]. Category IA
2. No recommendation can be made regarding the frequency for replacing intermittently used administration sets. Unresolved issue
3. No recommendation can be made regarding the frequency for replacing needles to access implantable ports. Unresolved issue
4. Replace tubing used to administer blood, blood products, or fat emulsions (those combined with amino acids and glucose in a 3-in-1 admixture or infused separately) within 24 hours of initiating the infusion [182–185]. Category IB
5. Replace tubing used to administer propofol infusions every 6 or 12 hours, when the vial is changed, per the manufacturer's recommendation (FDA website Medwatch) [186]. Category IA
6. No recommendation can be made regarding the length of time a needle used to access implanted ports can remain in place. Unresolved issue

Background

The optimal interval for routine replacement of IV administration sets has been examined in a number of well-controlled studies and meta-analyses. Data from these studies reveal that replacing administration sets no more frequently than 72–96 hours after initiation of use is safe and cost-effective [141, 177, 179–181]. More recent studies suggest that administration sets may be used safely for up to 7 days if used in conjunction with antiseptic catheters or if fluids that enhance microbial growth (e.g., parenteral nutrition or blood) have

not been used [216, 345]. When a fluid that enhances microbial growth is infused (e.g., fat emulsions and blood products), more frequent changes of administration sets are indicated as these products have been identified as independent risk factors for CRBSI [182, 216, 346–350]. Little data exist regarding the length of time a needle used to access implanted ports can remain in place and the risk of CRBSI. While some centers have left them in place for several weeks without CRBSI, [351], this practice has not been adequately studied.

Needleless Intravascular Catheter Systems

Recommendations

1. Change the needleless components at least as frequently as the administration set. There is no benefit to changing these more frequently than every 72 hours. [39, 187–193]. Category II
2. Change needleless connectors no more frequently than every 72 hours or according to manufacturers' recommendations for the purpose of reducing infection rates [187, 189, 192, 193]. Category II
3. Ensure that all components of the system are compatible to minimize leaks and breaks in the system [194]. Category II
4. Minimize contamination risk by scrubbing the access port with an appropriate antiseptic (chlorhexidine, povidone iodine, an iodophor, or 70% alcohol) and accessing the port only with sterile devices [189, 192, 194–196]. Category IA
5. Use a needleless system to access IV tubing. Category IC
6. When needleless systems are used, a split septum valve may be preferred over some mechanical valves due to increased risk of infection with the mechanical valves [197–200]. Category II

Background

Stopcocks used for injection of medications, administration of IV infusions, and collection of blood samples represent a potential portal of entry for microorganisms into vascular access catheters and IV fluids. Whether such contamination is a substantial entry point of microorganisms that cause CRBSI has not been demonstrated. Nonetheless, stopcocks

should be capped when not being used. In general, closed catheter access systems are associated with fewer CRBSIs than open systems and should be used preferentially [352].

"Piggyback" systems (secondary intermittent infusions delivered through a port on a primary infusion set) are used as an alternative to stopcocks. However, they also pose a risk for contamination of the intravascular fluid if the device entering the rubber membrane of an injection port is exposed to air or if it comes into direct contact with nonsterile tape used to fix the needle to the port. Modified piggyback systems have the potential to prevent contamination at these sites [353].

Attempts to reduce the incidence of sharps injuries and the resultant risk for transmission of bloodborne infections to healthcare personnel have led to the introduction and mandating of needleless infusion systems. There are several types of needleless connectors on the market.

The first type of needleless system connectors consisted of a split septum connector, which is accessed with a blunt cannula instead of a needle (external cannulae activated split septums). Because of the large amount of space in the connector to accommodate the cannula, when the cannula is removed it may result in the creation of negative pressure which may cause blood to be aspirated into the distal lumen, possibly increasing the risk of catheter occlusion or thrombosis. A luer-activated device, which incorporates a valve preventing the outflow of fluid through the connector, was designed to eliminate this problem. Some luer devices require a cap to be attached to the valve when not in use, which can be difficult to maintain aseptically, and therefore they may be prone to contamination.

Another type of second-generation needleless system addressed the occlusion issue by incorporating positive or neutral fluid displacement to either flush out aspirated blood or prevent its aspiration into infusion catheters.

Use of needleless connectors or mechanical valves appear to be effective in reducing connector colonization in some [196, 354, 355], but not all studies [356] when compared with stopcocks and caps. In one study [354], the incidence of CRBSI was reduced when the needleless connector was compared with standard stopcocks. Appropriate disinfectants must be used to prevent transmission of microbes through connectors [357]. Some studies have

shown that disinfection of the devices with chlorhexidine/alcohol solutions appears to be most effective in reducing colonization [195, 196]. In addition, the time spent applying the disinfectant may be important. One study found that swiping the luer-activated device with 70% alcohol for only 3 to 5 seconds did not adequately disinfect the septal surface [358]. However, a number of outbreak investigations have reported increases in CRBSIs associated with a switch from external cannulae activated split septum needleless devices to mechanical valve devices [197, 198, 200, 359]. The reasons for these associations are not known and it is also not known if this is a device-specific or class association, particularly as physical and mechanical properties of needleless connectors vary from device to device. In addition, one investigation found CRBSIs increased with the switch from a luer-activated negative displacement mechanical valve to a luer-activated positive fluid displacement mechanical valve [199]. However in an observational study, a switch from a luer-activated negative displacement mechanical valve to a different luer-activated positive displacement mechanical valve as part of a bundled intervention resulted in a significant decrease in CRBSIs [201]. Potential explanations for outbreaks associated with these devices include difficulty encountered in adequate disinfection of the surface of the connector due to physical characteristics of the plastic housing diaphragm interface, fluid flow properties (laminar vs. turbulent), internal surface area, potential fluid dead space, inadequate flushing of the device due to poor visualization of the fluid flow pathway in opaque devices, and the presence of internal corrugations that could harbor organisms, particularly if the catheters are used to withdraw blood [199]. Some studies have shown that the increase in CRBSIs with the change to lueractivated devices may be related to improper cleaning and infection control practices such as infrequently changing the devices [192, 194]. Additionally, silver-coated connector valves have been FDA approved; however, there are no published randomized trials with this device and no recommendation can be made regarding its use. Likewise, an antiseptic-barrier cap for needleless connectors has been studied in a laboratory setting and appears to be effective in preventing the entry of microorganisms [360], but has not yet been studied in a clinical trial.

Performance Improvement

Recommendation

Use hospital-specific or collaborative-based performance improvement initiatives in which multifaceted strategies are "bundled" together to improve compliance with evidence-based recommended practices [15, 69, 70, 201–205]. Category IB

Background

Clinical decision makers, healthcare payers, and patient safety advocates emphasize the importance of translating research findings into everyday practice. Rigorous evaluations of CRBSI preventive practices using study designs with high internal validity and including study populations that optimize external validity remain necessary. Once practices have been determined to be effective and economically efficient, the next step is to implement these evidence-based practices so they become part of routine clinical care. Unfortunately, implementation of evidence-based CRBSI preventive practices in U.S. hospitals has been suboptimal [361, 362]. In a national survey conducted in March 2005 of over 700 U.S. hospitals, approximately one quarter of U.S. hospitals indicated that either maximal sterile barrier precautions during central line insertion or chlorhexidine gluconate as site disinfectant, two practices widely recommended in the guidelines published in 2002 [363], were not being used routinely [364]. Approximately 15% of U.S. hospitals reported routinely changing CVCs to prevent infection despite evidence that this practice should no longer be used [362, 364].

Accordingly, investigators have attempted various approaches to better translate research findings and evidence-based recommendations into clinical practice. Numerous quality improvement studies have been published during the past several years that have used various methods, such as education of healthcare personnel, audit and feedback, organizational change, and clinical reminders [8–11, 69, 70, 202, 365–367]. The educational interventions primarily targeted hand hygiene, use of maximal sterile barriers during insertion, appropriate insertion site selection, proper site care using chlorhexidine gluconate, and prompt removal of unnecessary catheters. While a large number of before-and-after studies with a few using concurrent control groups [15, 70] have been published, no randomized, controlled trial

evaluating a quality improvement strategy to prevent CRBSI has been reported [368]. The vast majority of before-and-after studies reported statistically significant decreases in CRBSI rates after a quality improvement strategy was implemented [368]. Additionally, both controlled trials also found statistically significant reductions of CRBSI in the intervention units compared with control units [15, 70].

Investigators have also employed multifaceted approaches in which several strategies are bundled together to improve compliance with evidence-based guidelines [15, 69, 70]. One such collaborative cohort study [69] of 108 ICUs in Michigan targeted clinicians' use of five evidence-based practices: hand hygiene, maximum barrier precautions, chlorhexidine site disinfection, avoiding the femoral site, and promptly removing unnecessary central venous catheters. In addition to educating clinicians about CRBSI prevention, interventions used included: 1) a central venous catheter cart that contained all the necessary supplies; 2) a checklist to ensure adherence to proper practices; 3) stoppage of procedures in non-emergent situations, if evidence-based practices were not being followed; 4) prompt removal of unnecessary central catheters identified during daily patient rounds; 5) feedback to the clinical teams regarding the number of CRBSI episodes and overall rates; and 6) buy-in from the chief executive officers of the participating hospitals that chlorhexidine gluconate products/solutions would be stocked prior to study initiation. Using an interrupted time series analysis and multivariable regression, the investigators reported a statistically significant 66% decrease in CRBSI rates approximately 18 months after the intervention began [69] and sustained reductions over time [369]. Specific process and outcome measures for tracking and feedback (i.e rate of central line infections, proportion of central lines placed with all or individual bundle elements performed AND documented) should be identified in individual institutions based on areas that have been identified for performance improvement.

Finally, emphasis on the care and maintenance of catheters once they are in place should be a focus of performance improvement and quality assurance in all programs. A study to assess practice and staff knowledge of CVC post-insertion care and identify aspects of CVC care with potential for improvement revealed several areas of opportunity to improve post-insertion care [370]. Data were recorded on 151 CVCs in 106 patients giving a total of 721

catheter days. In all, 323 breaches in care were identified giving a failure rate of 44.8%, with significant differences between intensive care unit (ICU) and non-ICU wards. Dressings (not intact) and caps (incorrectly placed) were identified as the major lapses in CVC care with 158 and 156 breaches per 1000 catheter days, respectively. Interventions to improve reliability of care should focus on making the implementation of best practice easier to achieve.

References

1. Mermel LA. Prevention of intravascular catheter-related infections. (Erratum: Ann Intern Med 133:395, 2000). *Ann Intern Med* 2000; 132:391–402.
2. Dimick JB, Pelz RK, Consunji R, Swoboda SM, Hendrix CW, Lipsett PA. Increased resource use associated with catheter-related bloodstream infection in the surgical intensive care unit. *Arch Surg* 2001; 136:229–34.
3. Warren DK, Quadir WW, Hollenbeak CS, Elward AM, Cox MJ, Fraser VJ. Attributable cost of catheter-associated bloodstream infections among intensive care patients in a nonteaching hospital. *Crit Care Med* 2006; 34:2084–9.
4. Blot SI, Depuydt P, Annemans L, et al. Clinical and economic outcomes in critically ill patients with nosocomial catheter-related bloodstream infections. *Clin Infect Dis* 2005; 41:1591–8.
5. Renaud B, Brun-Buisson C. Outcomes of primary and catheter-related bacteremia. A cohort and case-control study in critically ill patients. *Am J Respir Crit Care Med* 2001; 163:1584–90.
6. Maki DG, Kluger DM, Crnich CJ. The risk of bloodstream infection in adults with different intravascular devices: a systematic review of 200 published prospective studies. *Mayo Clin Proc* 2006; 81:1159–71.
7. Yoo S, Ha M, Choi D, Pai H. Effectiveness of surveillance of central catheter-related bloodstream infection in an ICU in Korea. *Infect Control Hosp Epidemiol* 2001; 22:433–6.
8. Warren DK, Zack JE, Cox MJ, Cohen MM, Fraser VJ. An educational intervention to prevent catheter-associated bloodstream infections in a non-teaching community medical center. *Crit Care Med* 2003; 31:1959–63.
9. Warren DK, Zack JE, Mayfield JL, et al. The effect of an education program on the incidence of central venous catheter-associated bloodstream infection in a medical ICU. *Chest* 2004; 126:1612–8.
10. Warren DK, Cosgrove SE, Diekema DJ, et al. A multicenter intervention to prevent catheter-associated bloodstream infections. *Infect Control Hosp Epidemiol* 2006; 27:662–9.
11. Higuera F, Rosenthal VD, Duarte P, Ruiz J, Franco G, Safdar N. The effect of process control on the incidence of central venous catheter-associated bloodstream infections and mortality in intensive care units in Mexico. *Crit Care Med* 2005; 33:2022–7.
12. Coopersmith CM, Rebmann TL, Zack JE, et al. Effect of an education program on decreasing catheter-related bloodstream infections in the surgical intensive care unit. *Crit Care Med* 2002; 30:59–64.

13. Coopersmith CM, Zack JE, Ward MR, et al. The impact of bedside behavior on catheter-related bacteremia in the intensive care unit. *Arch Surg* 2004; 139:131–6.
14. Sherertz RJ, Ely EW, Westbrook DM, et al. Education of physicians-in-training can decrease the risk for vascular catheter infection. *Ann Intern Med* 2000; 132:641–8.
15. Eggimann P, Harbarth S, Constantin MN, Touveneau S, Chevrollet JC, Pittet D. Impact of a prevention strategy targeted at vascular-access care on incidence of infections acquired in intensive care. *Lancet* 2000; 355:1864–8.
16. Nehme AE. Nutritional support of the hospitalized patient. The team concept. *JAMA* 1980; 243:1906–8.
17. Soifer NE, Borzak S, Edlin BR, Weinstein RA. Prevention of peripheral venous catheter complications with an intravenous therapy team: a randomized controlled trial. *Arch Intern Med* 1998; 158:473–7.
18. Tomford JW, Hershey CO, McLaren CE, Porter DK, Cohen DI. Intravenous therapy team and peripheral venous catheter-associated complications. A prospective controlled study. *Arch Intern Med* 1984; 144:1191–4.
19. Scalley RD, Van CS, Cochran RS. The impact of an i.v. team on the occurrence of intravenous-related phlebitis. A 30-month study. *J Intraven Nurs* 1992; 15:100–9.
20. Palefski SS, Stoddard GJ. The infusion nurse and patient complication rates of peripheral-short catheters. A prospective evaluation. *J Intraven Nurs* 2001; 24:113–23.
21. Miller JM, Goetz AM, Squier C, Muder RR. Reduction in nosocomial intravenous device-related bacteremias after institution of an intravenous therapy team. *J Intraven Nurs* 1996; 19:103–6.
22. Hunter MR. Development of a Vascular Access Team in an acute care setting. *J Infus Nurs* 2003; 26:86–91.
23. Hawes ML. A proactive approach to combating venous depletion in the hospital setting. *J Infus Nurs* 2007; 30:33–44.
24. Brunelle D. Impact of a dedicated infusion therapy team on the reduction of catheter-related nosocomial infections. *J Infus Nurs* 2003; 26:362–6.
25. Bosma TL, Jewesson PJ. An infusion program resource nurse consult service: our experience in a major Canadian teaching hospital. *J Infus Nurs* 2002; 25:310–5.
26. Pierce CA, Baker JJ. A nursing process model: quantifying infusion therapy resource consumption. *J Infus Nurs* 2004; 27:232–44.
27. Tomford JW, Hershey CO. The i.v. therapy team: impact on patient care and costs of hospitalization. *NITA* 1985; 8:387–9.
28. Davis D, O'Brien MA, Freemantle N, Wolf FM, Mazmanian P, Taylor-Vaisey A. Impact of formal continuing medical education: do conferences, workshops, rounds, and other traditional continuing education activities change physician behavior or health care outcomes? *JAMA* 1999; 282:867–74.

29. Alonso-Echanove J, Edwards JR, Richards MJ, et al. Effect of nurse staffing and antimicrobial-impregnated central venous catheters on the risk for bloodstream infections in intensive care units. *Infect Control Hosp Epidemiol* 2003; 24:916–25.
30. Fridkin SK, Pear SM, Williamson TH, Galgiani JN, Jarvis WR. The role of understaffing in central venous catheter-associated bloodstream infections. *Infect Control Hosp Epidemiol* 1996; 17: 150–8.
31. Robert J, Fridkin SK, Blumberg HM, et al. The influence of the composition of the nursing staff on primary bloodstream infection rates in a surgical intensive care unit. *Infect Control Hosp Epidemiol* 2000; 21:12–7.
32. Maki DG, Goldman DA, Rhame FS. Infection control in intravenous therapy. *Ann Intern Med* 1973; 79:867–87.
33. Band JD, Maki DG. Steel needles used for intravenous therapy. Morbidity in patients with hematologic malignancy. *Arch Intern Med* 1980; 140:31–4.
34. Tully JL, Friedland GH, Baldini LM, Goldmann DA. Complications of intravenous therapy with steel needles and Teflon catheters. A comparative study. *Am J Med* 1981; 70:702–6.
35. Ryder MA. Peripheral access options. *Surg Oncol Clin N Am* 1995; 4:395–427.
36. Maki DG, Ringer M. Risk factors for infusion-related phlebitis with small peripheral venous catheters. A randomized controlled trial. *Ann Intern Med* 1991; 114:845–54.
37. Mermel LA, McCormick RD, Springman SR, Maki DG. The pathogenesis and epidemiology of catheter-related infection with pulmonary artery Swan-Ganz catheters: a prospective study utilizing molecular subtyping. *Am J Med* 1991; 91:197S–205.
38. Parienti JJ, Thirion M, Megarbane B, et al. Femoral vs jugular venous catheterization and risk of nosocomial events in adults requiring acute renal replacement therapy: a randomized controlled trial. *JAMA* 2008; 299:2413–22.
39. Moretti EW, Ofstead CL, Kristy RM, Wetzler HP. Impact of central venous catheter type and methods on catheter-related colonization and bacteraemia. *J Hosp Infect* 2005; 61:139–45.
40. Nagashima G, Kikuchi T, Tsuyuzaki H, et al. To reduce catheter-related bloodstream infections: is the subclavian route better than the jugular route for central venous catheterization? *J Infect Chemother* 2006; 12:363–5.
41. Ruesch S, Walder B, Tramer MR. Complications of central venous catheters: internal jugular versus subclavian access—a systematic review. *Crit Care Med* 2002; 30:454–60.
42. Sadoyama G, Gontijo Filho PP. Comparison between the jugular and subclavian vein as insertion site for central venous catheters: microbiological aspects and risk factors for colonization and infection. *Braz J Infect Dis* 2003; 7:142–8.
43. Heard SO, Wagle M, Vijayakumar E, et al. Influence of triple-lumen central venous catheters coated with chlorhexidine and silver sulfadiazine on the incidence of catheter-related bacteremia. *Arch Intern Med* 1998; 158:81–7.
44. Richet H, Hubert B, Nitemberg G, et al. Prospective multicenter study of vascular-catheter-related complications and risk factors for positive central-catheter cultures in intensive care unit patients. *J Clin Microbiol* 1990; 28:2520–5.

45. Safdar N, Kluger DM, Maki DG. A review of risk factors for catheter-related bloodstream infection caused by percutaneously inserted, noncuffed central venous catheters: implications for preventive strategies. *Medicine (Baltimore)* 2002; 81:466–79.
46. Lorente L, Jimenez A, Iribarren JL, Jimenez JJ, Martin MM, Mora ML. The micro-organism responsible for central venous catheter related bloodstream infection depends on catheter site. *Intensive Care Med* 2006; 32:1449–50.
47. Traore O, Liotier J, Souweine B. Prospective study of arterial and central venous catheter colonization and of arterial-and central venous catheter-related bacteremia in intensive care units. *Crit Care Med* 2005; 33:1276–80.
48. Joynt GM, Kew J, Gomersall CD, Leung VY, Liu EK. Deep venous thrombosis caused by femoral venous catheters in critically ill adult patients. *Chest* 2000; 117:178–83.
49. Mian NZ, Bayly R, Schreck DM, Besserman EB, Richmand D. Incidence of deep venous thrombosis associated with femoral venous catheterization. *Acad Emerg Med* 1997; 4:1118–21.
50. Merrer J, De Jonghe B, Golliot F, et al. Complications of femoral and subclavian venous catheterization in critically ill patients: a randomized controlled trial. *JAMA* 2001; 286:700–7.
51. Goetz AM, Wagener MM, Miller JM, Muder RR. Risk of infection due to central venous catheters: effect of site of placement and catheter type. *Infect Control Hosp Epidemiol* 1998; 19:842–5.
52. Robinson JF, Robinson WA, Cohn A, Garg K, Armstrong JD, 2nd. Perforation of the great vessels during central venous line placement. *Arch Intern Med* 1995; 155:1225–8.
53. Trottier SJ, Veremakis C, O'Brien J, Auer AI. Femoral deep vein thrombosis associated with central venous catheterization: results from a prospective, randomized trial. *Crit Care Med* 1995; 23:52–9.
54. Lorente L, Henry C, Martin MM, Jimenez A, Mora ML. Central venous catheter-related infection in a prospective and observational study of 2,595 catheters. *Crit Care* 2005; 9:R631–5.
55. Schillinger F, Schillinger D, Montagnac R, Milcent T. Post catheterisation vein stenosis in haemodialysis: comparative angiographic study of 50 subclavian and 50 internal jugular accesses. *Nephrol Dial Transplant* 1991; 6:722–4.
56. Cimochowski GE, Worley E, Rutherford WE, Sartain J, Blondin J, Harter H. Superiority of the internal jugular over the subclavian access for temporary dialysis. *Nephron* 1990; 54:154–61.
57. Barrett N, Spencer S, McIvor J, Brown EA. Subclavian stenosis: a major complication of subclavian dialysis catheters. *Nephrol Dial Transplant* 1988; 3:423–5.
58. Trerotola SO, Kuhn-Fulton J, Johnson MS, Shah H, Ambrosius WT, Kneebone PH. Tunneled infusion catheters: increased incidence of symptomatic venous thrombosis after subclavian versus internal jugular venous access. *Radiology* 2000; 217:89–93.
59. National Kidney Foundation. III. NKF-K/DOQI Clinical Practice Guidelines for Vascular Access: update 2000. *Am J Kidney Dis* 2001; 37:S137–81.
60. Hind D, Calvert N, McWilliams R, et al. Ultrasonic locating devices for central venous cannulation: meta-analysis. *BMJ* 2003; 327:361.

61. Randolph AG, Cook DJ, Gonzales CA, Pribble CG. Ultrasound guidance for placement of central venous catheters: a meta-analysis of the literature. *Crit Care Med* 1996; 24:2053–8.
62. Froehlich CD, Rigby MR, Rosenberg ES, et al. Ultrasound-guided central venous catheter placement decreases complications and decreases placement attempts compared with the landmark technique in patients in a pediatric intensive care unit. *Crit Care Med* 2009; 37:1090–6.
63. Lamperti M, Caldiroli D, Cortellazzi P, et al. Safety and efficacy of ultrasound assistance during internal jugular vein cannulation in neurosurgical infants. *Intensive Care Med* 2008; 34:2100–5.
64. Schweickert WD, Herlitz J, Pohlman AS, Gehlbach BK, Hall JB, Kress JP. A randomized, controlled trial evaluating postinsertion neck ultrasound in peripherally inserted central catheter procedures. *Crit Care Med* 2009; 37:1217–21.
65. Clark-Christoff N, Watters VA, Sparks W, Snyder P, Grant JP. Use of triple-lumen subclavian catheters for administration of total parenteral nutrition. *JPEN J Parenter Enteral Nutr* 1992; 16:403–7.
66. Early TF, Gregory RT, Wheeler JR, Snyder SO Jr., Gayle RG. Increased infection rate in double-lumen versus single-lumen Hickman catheters in cancer patients. *South Med J* 1990; 83:34–6.
67. Hilton E, Haslett TM, Borenstein MT, Tucci V, Isenberg HD, Singer C. Central catheter infections: single-versus triple-lumen catheters. Influence of guide wires on infection rates when used for replacement of catheters. *Am J Med* 1988; 84:667–72.
68. Yeung C, May J, Hughes R. Infection rate for single lumen v triple lumen subclavian catheters. *Infect Control Hosp Epidemiol* 1988; 9:154–8.
69. Pronovost P, Needham D, Berenholtz S, et al. An intervention to decrease catheter-related bloodstream infections in the ICU. *N Engl J Med* 2006; 355:2725–32.
70. Berenholtz SM, Pronovost PJ, Lipsett PA, et al. Eliminating catheter-related bloodstream infections in the intensive care unit. *Crit Care Med* 2004; 32:2014–20.
71. Lederle FA, Parenti CM, Berskow LC, Ellingson KJ. The idle intravenous catheter. *Ann Intern Med* 1992; 116:737–8.
72. Parenti CM, Lederle FA, Impola CL, Peterson LR. Reduction of unnecessary intravenous catheter use. Internal medicine house staff participate in a successful quality improvement project. *Arch Intern Med* 1994; 154:1829–32.
73. Abi-Said D, Raad I, Umphrey J, et al. Infusion therapy team and dressing changes of central venous catheters. *Infect Control Hosp Epidemiol* 1999; 20:101–5.
74. Capdevila JA, Segarra A, Pahissa A. Catheter-related bacteremia in patients undergoing hemodialysis. *Ann Intern Med* 1998; 128:600.
75. Mermel LA, Maki DG. Infectious complications of Swan-Ganz pulmonary artery catheters. Pathogenesis, epidemiology, prevention, and management. *Am J Respir Crit Care Med* 1994; 149:1020–36.

76. Raad II, Hohn DC, Gilbreath BJ, et al. Prevention of central venous catheter-related infections by using maximal sterile barrier precautions during insertion. *Infect Control Hosp Epidemiol* 1994; 15:231–8.
77. Boyce JM, Pittet D. Guideline for hand hygiene in health-care settings: recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force. *Infect Control Hosp Epidemiol* 2002; 23:S3–40.
78. Bischoff WE, Reynolds TM, Sessler CN, Edmond MB, Wenzel RP. Handwashing compliance by health care workers: the impact of introducing an accessible, alcohol-based hand antiseptic. *Arch Intern Med* 2000; 160:1017–21.
79. Pittet D, Dharan S, Touveneau S, Sauvan V, Perneger TV. Bacterial contamination of the hands of hospital staff during routine patient care. *Arch Intern Med* 1999; 159:821–6.
80. Carrer S, Bocchi A, Bortolotti M, et al. Effect of different sterile barrier precautions and central venous catheter dressing on the skin colonization around the insertion site. *Minerva Anestesiol* 2005; 71:197–206.
81. Cohen Y, Fosse JP, Karoubi P, et al. The "hands-off" catheter and the prevention of systemic infections associated with pulmonary artery catheter: a prospective study. *Am J Respir Crit Care Med* 1998; 157:284–7.
82. Maki DG, Ringer M, Alvarado CJ. Prospective randomised trial of povidone-iodine, alcohol, and chlorhexidine for prevention of infection associated with central venous and arterial catheters. *Lancet* 1991; 338:339–43.
83. Mimoz O, Pieroni L, Lawrence C, et al. Prospective, randomized trial of two antiseptic solutions for prevention of central venous or arterial catheter colonization and infection in intensive care unit patients. *Crit Care Med* 1996; 24:1818–23.
84. Maki DG, Stolz SS, Wheeler S, Mermel LA. A prospective, randomized trial of gauze and two polyurethane dressings for site care of pulmonary artery catheters: implications for catheter management. *Crit Care Med* 1994; 22:1729–37.
85. Bijma R, Girbes AR, Kleijer DJ, Zwaveling JH. Preventing central venous catheter-related infection in a surgical intensive-care unit. *Infect Control Hosp Epidemiol* 1999; 20:618–20.
86. Madeo M, Martin CR, Turner C, Kirkby V, Thompson DR. A randomized trial comparing Arglaes (a transparent dressing containing silver ions) to Tegaderm (a transparent polyurethane dressing) for dressing peripheral arterial catheters and central vascular catheters. *Intensive Crit Care Nurs* 1998; 14:187–91.
87. Rasero L, Degl'Innocenti M, Mocali M, et al. Comparison of two different time interval protocols for central venous catheter dressing in bone marrow transplant patients: results of a randomized, multicenter study. The Italian Nurse Bone Marrow Transplant Group (GITMO). *Haematologica* 2000; 85:275–9.
88. Zakrzewska-Bode A, Muytjens HL, Liem KD, Hoogkamp-Korstanje JA. Mupirocin resistance in coagulase-negative staphylococci, after topical prophylaxis for the reduction of colonization of central venous catheters. *J Hosp Infect* 1995; 31:189–93.
89. Flowers RH, Schwenzer KJ, Kopel RF, Fisch MJ, Tucker SI, Farr BM. Efficacy of an attachable subcutaneous cuff for the prevention of intravascular catheter-related infection. A randomized, controlled trial. *JAMA* 1989; 261:878–83.

90. Robbins J, Cromwell P, Korones DN. Swimming and central venous catheter-related infections in the child with cancer. *J Pediatr Oncol Nurs* 1999; 16:51–6.
91. Howell PB, Walters PE, Donowitz GR, Farr BM. Risk factors for infection of adult patients with cancer who have tunneled central venous catheters. *Cancer* 1995; 75:1367–75.
92. Ivy DD, Calderbank M, Wagner BD, et al. Closed-hub systems with protected connections and the reduction of risk of catheter-related bloodstream infection in pediatric patients receiving intravenous prostanoid therapy for pulmonary hypertension. *Infect Control Hosp Epidemiol* 2009; 30:823–9.
93. Timsit JF, Schwebel C, Bouadma L, et al. Chlorhexidine-impregnated sponges and less frequent dressing changes for prevention of catheter-related infections in critically ill adults: a randomized controlled trial. *JAMA* 2009; 301:1231–41.
94. Rao SP, Oreopoulos DG. Unusual complications of a polyurethane PD catheter. *Perit Dial Int* 1997; 17:410–2.
95. Riu S, Ruiz CG, Martinez-Vea A, Peralta C, Oliver JA. Spontaneous rupture of polyurethane peritoneal catheter. A possible deleterious effect of mupirocin ointment. *Nephrol Dial Transplant* 1998; 13:1870–1.
96. Garland JS, Alex CP, Mueller CD, et al. A randomized trial comparing povidone-iodine to a chlorhexidine gluconate-impregnated dressing for prevention of central venous catheter infections in neonates. *Pediatrics* 2001; 107:1431–6.
97. Ho KM, Litton E. Use of chlorhexidine-impregnated dressing to prevent vascular and epidural catheter colonization and infection: a meta-analysis. *J Antimicrob Chemother* 2006; 58:281–7.
98. Levy I, Katz J, Solter E, et al. Chlorhexidine-impregnated dressing for prevention of colonization of central venous catheters in infants and children: a randomized controlled study. *Pediatr Infect Dis J* 2005; 24:676–9.
99. Lorenzen AN, Itkin DJ. Surveillance of infection in home care. *Am J Infect Control* 1992; 20:326–9.
100. White MC. Infections and infection risks in home care settings. *Infect Control Hosp Epidemiol* 1992; 13:535–9.
101. White MC, Ragland KE. Surveillance of intravenous catheter-related infections among home care clients. *Am J Infect Control* 1994; 22:231–5.
102. Bleasdale SC, Trick WE, Gonzalez IM, Lyles RD, Hayden MK, Weinstein RA. Effectiveness of chlorhexidine bathing to reduce catheter-associated bloodstream infections in medical intensive care unit patients. *Arch Intern Med* 2007; 167:2073–9.
103. Munoz-Price LS, Hota B, Stemer A, Weinstein RA. Prevention of bloodstream infections by use of daily chlorhexidine baths for patients at a long-term acute care hospital. *Infect Control Hosp Epidemiol* 2009; 30:1031–5.
104. Popovich KJ, Hota B, Hayes R, Weinstein RA, Hayden MK. Effectiveness of routine patient cleansing with chlorhexidine gluconate for infection prevention in the medical intensive care unit. *Infect Control Hosp Epidemiol* 2009; 30:959–63.

105. Yamamoto AJ, Solomon JA, Soulen MC, et al. Sutureless securement device reduces complications of peripherally inserted central venous catheters. *J Vasc Interv Radiol* 2002; 13:77–81.
106. Brun-Buisson C, Doyon F, Sollet JP, Cochard JF, Cohen Y, Nitenberg G. Prevention of intravascular catheter-related infection with newer chlorhexidine-silver sulfadiazine-coated catheters: a randomized controlled trial. *Intensive Care Med* 2004; 30:837–43.
107. Ostendorf T, Meinhold A, Harter C, et al. Chlorhexidine and silver-sulfadiazine coated central venous catheters in haematological patients—a double-blind, randomised, prospective, controlled trial. *Support Care Cancer* 2005; 13:993–1000.
108. Rupp ME, Lisco SJ, Lipsett PA, et al. Effect of a second-generation venous catheter impregnated with chlorhexidine and silver sulfadiazine on central catheter-related infections: a randomized, controlled trial. *Ann Intern Med* 2005; 143:570–80.
109. Darouiche RO, Raad II, Heard SO, et al. A comparison of two antimicrobial-impregnated central venous catheters. Catheter Study Group. *N Engl J Med* 1999; 340:1–8.
110. Raad I, Darouiche R, Dupuis J, et al. Central venous catheters coated with minocycline and rifampin for the prevention of catheter-related colonization and bloodstream infections. A randomized, double-blind trial. The Texas Medical Center Catheter Study Group. *Ann Intern Med* 1997; 127:267–74.
111. Hanna H, Benjamin R, Chatzinikolaou I, et al. Long-term silicone central venous catheters impregnated with minocycline and rifampin decrease rates of catheter-related bloodstream infection in cancer patients: a prospective randomized clinical trial. *J Clin Oncol* 2004; 22:3163–71.
112. Bhutta A, Gilliam C, Honeycutt M, et al. Reduction of bloodstream infections associated with catheters in paediatric intensive care unit: stepwise approach. *BMJ* 2007; 334:362–5.
113. Chelliah A, Heydon KH, Zaoutis TE, et al. Observational trial of antibiotic-coated central venous catheters in critically ill pediatric patients. *Pediatr Infect Dis J* 2007; 26:816–20.
114. van de Wetering MD, van Woensel JBM. Prophylactic antibiotics for preventing early central venous catheter Gram positive infections in oncology patients. *Cochrane Database of Systematic Reviews* 2007; Issue 1. Art. No.: CD003295. DOI: 10.1002/14651858.CD003295.pub2.
115. Maki DG, Band JD. A comparative study of polyantibiotic and iodophor ointments in prevention of vascular catheter-related infection. *Am J Med* 1981; 70:739–44.
116. Fukunaga A, Naritaka H, Fukaya R, Tabuse M, Nakamura T. Povidone-iodine ointment and gauze dressings associated with reduced catheter-related infection in seriously ill neurosurgical patients. *Infect Control Hosp Epidemiol* 2004; 25:696–8.
117. Johnson DW, MacGinley R, Kay TD, et al. A randomized controlled trial of topical exit site mupirocin application in patients with tunnelled, cuffed haemodialysis catheters. *Nephrol Dial Transplant* 2002; 17:1802–7.
118. Fong IW. Prevention of haemodialysis and peritoneal dialysis catheter related infection by topical povidone-iodine. *Postgrad Med J* 1993; 69(Suppl 3):S15–7.
119. Levin A, Mason AJ, Jindal KK, Fong IW, Goldstein MB. Prevention of hemodialysis subclavian vein catheter infections by topical povidone-iodine. *Kidney Int* 1991; 40:934–8.

120. Schwartz C, Henrickson KJ, Roghmann K, Powell K. Prevention of bacteremia attributed to luminal colonization of tunneled central venous catheters with vancomycin-susceptible organisms. *J Clin Oncol* 1990; 8:1591–7.
121. Rackoff WR, Weiman M, Jakobowski D, et al. A randomized, controlled trial of the efficacy of a heparin and vancomycin solution in preventing central venous catheter infections in children. *J Pediatr* 1995; 127:147–51.
122. Carratala J, Niubo J, Fernandez-Sevilla A, et al. Randomized, double-blind trial of an antibiotic-lock technique for prevention of gram-positive central venous catheter-related infection in neutropenic patients with cancer. *Antimicrob Agents Chemother* 1999; 43:2200–4.
123. Jurewitsch B, Lee T, Park J, Jeejeebhoy K. Taurolidine 2% as an antimicrobial lock solution for prevention of recurrent catheter-related bloodstream infections. *J Parenter Enteral Nutr* 1998; 22:242–4.
124. Henrickson KJ, Axtell RA, Hoover SM, et al. Prevention of central venous catheter-related infections and thrombotic events in immunocompromised children by the use of vancomycin/ciprofloxacin/ heparin flush solution: a randomized, multicenter, double-blind trial. *J Clin Oncol* 2000; 18:1269–78.
125. Garland JS, Alex CP, Henrickson KJ, McAuliffe TL, Maki DG. A vancomycin-heparin lock solution for prevention of nosocomial bloodstream infection in critically ill neonates with peripherally inserted central venous catheters: a prospective, randomized trial. *Pediatrics* 2005; 116:e198–205.
126. Daghistani D, Horn M, Rodriguez Z, Schoenike S, Toledano S. Prevention of indwelling central venous catheter sepsis. *Med Pediatr Oncol* 1996; 26:405–8.
127. Barriga FJ, Varas M, Potin M, et al. Efficacy of a vancomycin solution to prevent bacteremia associated with an indwelling central venous catheter in neutropenic and non-neutropenic cancer patients. *Med Pediatr Oncol* 1997; 28:196–200.
128. Dogra GK, Herson H, Hutchison B, et al. Prevention of tunneled hemodialysis catheter-related infections using catheter-restricted filling with gentamicin and citrate: a randomized controlled study. *J Am Soc Nephrol* 2002; 13:2133–9.
129. Allon M. Prophylaxis against dialysis catheter-related bacteremia with a novel antimicrobial lock solution. *Clin Infect Dis* 2003; 36:1539–44.
130. Elhassan NO, Stevens TP, Gigliotti F, Hardy DJ, Cole CA, Sinkin RA. Vancomycin usage in central venous catheters in a neonatal intensive care unit. *Pediatr Infect Dis J* 2004; 23:201–6.
131. McIntyre CW, Hulme LJ, Taal M, Fluck RJ. Locking of tunneled hemodialysis catheters with gentamicin and heparin. *Kidney Int* 2004; 66:801–5.
132. Betjes MG, van Agteren M. Prevention of dialysis catheter-related sepsis with a citrate-taurolidine-containing lock solution. *Nephrol Dial Transplant* 2004; 19:1546–1.
133. Weijmer MC, van den Dorpel MA, Van de Ven PJ, et al. Randomized, clinical trial comparison of trisodium citrate 30% and heparin as catheter-locking solution in hemodialysis patients. *J Am Soc Nephrol* 2005; 16:2769–77.
134. Bleyer AJ, Mason L, Russell G, Raad II, Sherertz RJ. A randomized, controlled trial of a new vascular catheter flush solution (minocycline-EDTA) in temporary hemodialysis access. *Infect Control Hosp Epidemiol* 2005; 26:520–4.

135. Kim SH, Song KI, Chang JW, et al. Prevention of uncuffed hemodialysis catheter-related bacteremia using an antibiotic lock technique: a prospective, randomized clinical trial. *Kidney Int* 2006; 69:161–4.
136. Al-Hwiesh AK, Abdul-Rahman IS. Successful prevention of tunneled, central catheter infection by antibiotic lock therapy using vancomycin and gentamycin. *Saudi J Kidney Dis Transpl* 2007; 18:239–47.
137. Nori US, Manoharan A, Yee J, Besarab A. Comparison of low-dose gentamicin with minocycline as catheter lock solutions in the prevention of catheter-related bacteremia. *Am J Kidney Dis* 2006; 48:596–605.
138. Saxena AK, Panhotra BR, Sundaram DS, et al. Tunneled catheters' outcome optimization among diabetics on dialysis through antibiotic-lock placement. *Kidney Int* 2006; 70:1629–35.
139. Randolph AG, Cook DJ, Gonzales CA, Andrew M. Benefit of heparin in central venous and pulmonary artery catheters: a meta-analysis of randomized controlled trials. *Chest* 1998; 113:165–71.
140. Tager IB, Ginsberg MB, Ellis SE, et al. An epidemiologic study of the risks associated with peripheral intravenous catheters. *Am J Epidemiol* 1983; 118:839–51.
141. Lai KK. Safety of prolonging peripheral cannula and i.v. tubing use from 72 hours to 96 hours. *Am J Infect Control* 1998; 26:66–70.
142. Van Donk P, Rickard CM, McGrail MR, Doolan G. Routine replacement versus clinical monitoring of peripheral intravenous catheters in a regional hospital in the home program: a randomized controlled trial. *Infect Control Hosp Epidemiol* 2009; 30:915–7.
143. Webster J, Clarke S, Paterson D, et al. Routine care of peripheral intravenous catheters versus clinically indicated replacement: randomised controlled trial. *BMJ* 2008; 337:a339.
144. Webster J, Osborne S, Rickard C, Hall J. Clinically-indicated replacement versus routine replacement of peripheral venous catheters. *Cochrane Database Syst Rev* 2010; 3:CD007798.
145. Boo NY, Wong NC, Zulkifli SS, Lye MS. Risk factors associated with umbilical vascular catheter-associated thrombosis in newborn infants. *J Paediatr Child Health* 1999; 35:460–5.
146. Garland JS, Buck RK, Maloney P, et al. Comparison of 10% povidone-iodine and 0.5% chlorhexidine gluconate for the prevention of peripheral intravenous catheter colonization in neonates: a prospective trial. *Pediatr Infect Dis J* 1995; 14:510–6.
147. Krauss AN, Albert RF, Kannan MM. Contamination of umbilical catheters in the newborn infant. *J Pediatr* 1970; 77:965–9.
148. Landers S, Moise AA, Fraley JK, Smith EO, Baker CJ. Factors associated with umbilical catheter-related sepsis in neonates. *Am J Dis Child* 1991; 145:675–80.
149. Cronin WA, Germanson TP, Donowitz LG. Intravascular catheter colonization and related bloodstream infection in critically ill neonates. *Infect Control Hosp Epidemiol* 1990; 11:301–8.
150. Miller KL, Coen PE, White WJ, Hurst WJ, Achey BE, Lang CM. Effectiveness of skin absorption of tincture of I in blocking radioiodine from the human thyroid gland. *Health Phys* 1989; 56:911–4.

Guidelines for the Prevention of Intravascular Catheter-Related Infections

151. Ankola PA, Atakent YS. Effect of adding heparin in very low concentration to the infusate to prolong the patency of umbilical artery catheters. *Am J Perinatol* 1993; 10:229–32.
152. David RJ, Merten DF, Anderson JC, Gross S. Prevention of umbilical artery catheter clots with heparinized infusates. *Dev Pharmacol Ther* 1981; 2:117–26.
153. Horgan MJ, Bartoletti A, Polansky S, Peters JC, Manning TJ, Lamont BM. Effect of heparin infusates in umbilical arterial catheters on frequency of thrombotic complications. *J Pediatr* 1987; 111:774–8.
154. Fletcher MA, Brown DR, Landers S, Seguin J. Umbilical arterial catheter use: report of an audit conducted by the Study Group for Complications of Perinatal Care. *Am J Perinatol* 1994; 11:94–9.
155. Seguin J, Fletcher MA, Landers S, Brown D, Macpherson T. Umbilical venous catheterizations: audit by the Study Group for Complications of Perinatal Care. *Am J Perinatol* 1994; 11:67–70.
156. Loisel DB, Smith MM, MacDonald MG, Martin GR. Intravenous access in newborn infants: impact of extended umbilical venous catheter use on requirement for peripheral venous lines. *J Perinatol* 1996; 16:461–6.
157. Martin C, Saux P, Papazian L, Gouin F. Long-term arterial cannulation in ICU patients using the radial artery or dorsalis pedis artery. *Chest* 2001; 119:901–6.
158. Koh DB, Gowardman JR, Rickard CM, Robertson IK, Brown A. Prospective study of peripheral arterial catheter infection and comparison with concurrently sited central venous catheters. *Crit Care Med* 2008; 36:397–402.
159. Rijnders BJ, Van Wijngaerden E, Wilmer A, Peetermans WE. Use of full sterile barrier precautions during insertion of arterial catheters: a randomized trial. *Clin Infect Dis* 2003; 36:743–8.
160. Donowitz LG, Marsik FJ, Hoyt JW, Wenzel RP. *Serratia marcescens* bacteremia from contaminated pressure transducers. *JAMA* 1979; 242:1749–51.
161. Luskin RL, Weinstein RA, Nathan C, Chamberlin WH, Kabins SA. Extended use of disposable pressure transducers. A bacteriologic evaluation. *JAMA* 1986; 255:916–20.
162. Maki DG, Hassemer CA. Endemic rate of fluid contamination and related septicemia in arterial pressure monitoring. *Am J Med* 1981; 70:733–8.
163. Mermel LA, Maki DG. Epidemic bloodstream infections from hemodynamic pressure monitoring: signs of the times. *Infect Control Hosp Epidemiol* 1989; 10:47–53.
164. Tenold R, Priano L, Kim K, Rourke B, Marrone T. Infection potential of nondisposable pressure transducers prepared prior to use. *Crit Care Med* 1987; 15:582–3.
165. Eyer S, Brummitt C, Crossley K, Siegel R, Cerra F. Catheter-related sepsis: prospective, randomized study of three methods of long-term catheter maintenance. *Crit Care Med* 1990; 18:1073–9.
166. Raad I, Umphrey J, Khan A, Truett LJ, Bodey GP. The duration of placement as a predictor of peripheral and pulmonary arterial catheter infections. *J Hosp Infect* 1993; 23:17–26.
167. Thomas F, Burke JP, Parker J, et al. The risk of infection related to radial vs femoral sites for arterial catheterization. *Crit Care Med* 1983; 11:807–12.

168. Leroy O, Billiau V, Beuscart C, et al. Nosocomial infections associated with long-term radial artery cannulation. *Intensive Care Med* 1989; 15:241–6.
169. Fisher MC, Long SS, Roberts EM, Dunn JM, Balsara RK. *Pseudomonas maltophilia* bacteremia in children undergoing open heart surgery. *JAMA* 1981; 246:1571–4.
170. Stamm WE, Colella JJ, Anderson RL, Dixon RE. Indwelling arterial catheters as a source of nosocomial bacteremia. An outbreak caused by *Flavobacterium* Species. *N Engl J Med* 1975; 292:1099–102.
171. Weinstein RA, Emori TG, Anderson RL, Stamm WE. Pressure transducers as a source of bacteremia after open heart surgery. Report of an outbreak and guidelines for prevention. *Chest* 1976; 69:338–44.
172. Shinozaki T, Deane RS, Mazuzan JE Jr., Hamel AJ, Hazelton D. Bacterial contamination of arterial lines. A prospective study. *JAMA* 1983; 249:223–5.
173. Solomon SL, Alexander H, Eley JW, et al. Nosocomial fungemia in neonates associated with intravascular pressure-monitoring devices. *Pediatr Infect Dis* 1986; 5:680–5.
174. Weems JJ Jr., Chamberland ME, Ward J, Willy M, Padhye AA, Solomon SL. *Candida* parapsilosis fungemia associated with parenteral nutrition and contaminated blood pressure transducers. *J Clin Microbiol* 1987; 25:1029–32.
175. Villarino ME, Jarvis WR, O’Hara C, Bresnahan J, Clark N. Epidemic of *Serratia marcescens* bacteremia in a cardiac intensive care unit. *J Clin Microbiol* 1989; 27:2433–6.
176. Beck-Sague CM, Jarvis WR, Brook JH, et al. Epidemic bacteremia due to *Acinetobacter baumannii* in five intensive care units. *Am J Epidemiol* 1990; 132:723–33.
177. Gillies D, Wallen MM, Morrison AL, Rankin K, Nagy SA, O’Riordan
2. E. Optimal timing for intravenous administration set replacement. *Cochrane Database of Systematic Reviews* 2005; Issue 4. Art. No.: CD003588. DOI: 10.1002/14651858.CD003588.pub2.
178. Sitges-Serra A, Linares J, Perez JL, Jaurrieta E, Lorente L. A randomized trial on the effect of tubing changes on hub contamination
3. and catheter sepsis during parenteral nutrition. *JPEN J Parenter Enteral Nutr* 1985; 9:322–5.
179. Snyderman DR, Donnelly-Reidy M, Perry LK, Martin WJ. Intravenous tubing containing burettes can be safely changed at 72 hour intervals. *Infect Control* 1987; 8:113–6.
180. Maki DG, Botticelli JT, LeRoy ML, Thielke TS. Prospective study of replacing administration sets for intravenous therapy at 48-vs 72hour intervals. 72 hours is safe and cost-effective. *JAMA* 1987; 258:1777–81.
181. Josephson A, Gombert ME, Sierra MF, Karanfil LV, Tansino GF. The relationship between intravenous fluid contamination and the frequency of tubing replacement. *Infect Control* 1985; 6:367–70.
182. Melly MA, Meng HC, Schaffner W. Microbiol growth in lipid emulsions used in parenteral nutrition. *Arch Surg* 1975; 110:1479–81.
183. Mershon J, Nogami W, Williams JM, Yoder C, Eitzen HE, Lemons JA. Bacterial/fungal growth in a combined parenteral nutrition solution. *JPEN J Parenter Enteral Nutr* 1986; 10:498–502.

184. Gilbert M, Gallagher SC, Eads M, Elmore MF. Microbial growth patterns in a total parenteral nutrition formulation containing lipid emulsion. *JPEN J Parenter Enteral Nutr* 1986; 10:494–7.
185. Maki DG, Martin WT. Nationwide epidemic of septicemia caused by contaminated infusion products. IV. Growth of microbial pathogens in fluids for intravenous infusions. *J Infect Dis* 1975; 131:267–72.
186. Bennett SN, McNeil MM, Bland LA, et al. Postoperative infections traced to contamination of an intravenous anesthetic, propofol. *N Engl J Med* 1995; 333:147–54.
187. Arduino MJ, Bland LA, Danzig LE, McAllister SK, Aguero SM. Microbiologic evaluation of needleless and needle-access devices. *Am J Infect Control* 1997; 25:377–80.
188. Brown JD, Moss HA, Elliott TS. The potential for catheter microbial contamination from a needleless connector. *J Hosp Infect* 1997; 36:181–9.
189. Cookson ST, Ihrig M, O’Mara EM, et al. Increased bloodstream infection rates in surgical patients associated with variation from recommended use and care following implementation of a needleless device. *Infect Control Hosp Epidemiol* 1998; 19:23–7.
190. Seymour VM, Dhallu TS, Moss HA, Tebbs SE, Elliot TS. A prospective clinical study to investigate the microbial contamination of a needle-less connector. *J Hosp Infect* 2000; 45:165–8.
191. Luebke MA, Arduino MJ, Duda DL, et al. Comparison of the microbial barrier properties of a needleless and a conventional needle-based intravenous access system. *Am J Infect Control* 1998; 26:437–41.
192. McDonald LC, Banerjee SN, Jarvis WR. Line-associated bloodstream infections in pediatric intensive-care-unit patients associated with a needleless device and intermittent intravenous therapy. *Infect Control Hosp Epidemiol* 1998; 19:772–7.
193. Mendelson MH, Short LJ, Schechter CB, et al. Study of a needleless intermittent intravenous-access system for peripheral infusions: analysis of staff, patient, and institutional outcomes. *Infect Control Hosp Epidemiol* 1998; 19:401–6.
194. Do AN, Ray BJ, Banerjee SN, et al. Bloodstream infection associated with needleless device use and the importance of infection-control practices in the home health care setting. *J Infect Dis* 1999; 179:442–8.
195. Soothill JS, Bravery K, Ho A, Macqueen S, Collins J, Lock P. A fall in bloodstream infections followed a change to 2% chlorhexidine in 70% isopropanol for catheter connection antisepsis: a pediatric single center before/after study on a hemopoietic stem cell transplant ward. *Am J Infect Control* 2009; 37:626–30.
196. Casey AL, Burnell S, Whinn H, Worthington T, Faroqui MH, Elliott TS. A prospective clinical trial to evaluate the microbial barrier of a needleless connector. *J Hosp Infect* 2007; 65:212–8.
197. Rupp ME, Sholtz LA, Jourdan DR, et al. Outbreak of bloodstream infection temporally associated with the use of an intravascular needleless valve. *Clin Infect Dis* 2007; 44:1408–14.
198. Salgado CD, Chinnes L, Paczesny TH, Cantey JR. Increased rate of catheter-related bloodstream infection associated with use of a needleless mechanical valve device at a long-term acute care hospital. *Infect Control Hosp Epidemiol* 2007; 28:684–8.

199. Maragakis LL, Bradley KL, Song X, et al. Increased catheter-related bloodstream infection rates after the introduction of a new mechanical valve intravenous access port. *Infect Control Hosp Epidemiol* 2006; 27:67–70.
200. Field K, McFarlane C, Cheng AC, et al. Incidence of catheter-related bloodstream infection among patients with a needleless, mechanical valve-based intravenous connector in an Australian hematology-oncology unit. *Infect Control Hosp Epidemiol* 2007; 28:610–3.
201. Costello JM, Morrow DF, Graham DA, Potter-Bynoe G, Sandora TJ, Laussen PC. Systematic intervention to reduce central line-associated bloodstream infection rates in a pediatric cardiac intensive care unit. *Pediatrics* 2008; 121:915–23.
202. Frankel HL, Crede WB, Topal JE, Roumanis SA, Devlin MW, Foley AB. Use of corporate Six Sigma performance-improvement strategies to reduce incidence of catheter-related bloodstream infections in a surgical ICU. *J Am Coll Surg* 2005; 201:349–58.
203. Galpern D, Guerrero A, Tu A, Fahoum B, Wise L. Effectiveness of a central line bundle campaign on line-associated infections in the intensive care unit. *Surgery* 2008; 144:492–5; discussion 495.
204. McKee C, Berkowitz I, Cosgrove SE, et al. Reduction of catheter-associated bloodstream infections in pediatric patients: experimentation and reality. *Pediatr Crit Care Med* 2008; 9:40–6.
205. Pronovost PJ, Berenholtz SM, Goeschel CA. Improving the quality of measurement and evaluation in quality improvement efforts. *Am J Med Qual* 2008; 23:143–6.
206. Horan TC, Andrus M, Dudeck MA. CDC/NHSN surveillance definition of health care-associated infection and criteria for specific types of infections in the acute care setting. *Am J Infect Control* 2008; 36:309–32.
207. Edwards JR, Peterson KD, Mu Y, et al. National Healthcare Safety Network (NHSN) report: data summary for 2006 through 2008, issued December 2009. *Am J Infect Control* 2009; 37:783–805.
208. Wisplinghoff H, Bischoff T, Tallent SM, Seifert H, Wenzel RP, Edmond MB. Nosocomial bloodstream infections in US hospitals: analysis of 24,179 cases from a prospective nationwide surveillance study. *Clin Infect Dis* 2004; 39:309–17.
209. Gaynes R, Edwards JR. Overview of nosocomial infections caused by gram-negative bacilli. *Clin Infect Dis* 2005; 41:848–54.
210. Burton DC, Edwards JR, Horan TC, Jernigan JA, Fridkin SK. Methicillin-resistant *Staphylococcus aureus* central line-associated bloodstream infections in US intensive care units, 1997–2007. *JAMA* 2009; 301:727–36.
211. Safdar N, Maki DG. The pathogenesis of catheter-related bloodstream infection with noncuffed short-term central venous catheters. *Intensive Care Med* 2004; 30:62–7.
212. Maki DG, Weise CE, Sarafin HW. A semiquantitative culture method for identifying intravenous-catheter-related infection. *N Engl J Med* 1977; 296:1305–9.
213. Raad I, Costerton W, Sabharwal U, Sacilowski M, Anaissie E, Bodey GP. Ultrastructural analysis of indwelling vascular catheters: a quantitative relationship between luminal colonization and duration of placement. *J Infect Dis* 1993; 168:400–7.

214. Dobbins BM, Kite P, Kindon A, McMahon MJ, Wilcox MH. DNA fingerprinting analysis of coagulase negative staphylococci implicated in catheter related bloodstream infections. *J Clin Pathol* 2002; 55:824–8.
215. Anaissie E, Samonis G, Kontoyiannis D, et al. Role of catheter colonization and infrequent hematogenous seeding in catheter-related infections. *Eur J Clin Microbiol Infect Dis* 1995; 14:134–7.
216. Raad I, Hanna HA, Awad A, et al. Optimal frequency of changing intravenous administration sets: is it safe to prolong use beyond 72 hours? *Infect Control Hosp Epidemiol* 2001; 22:136–9.
217. Mehall JR, Saltzman DA, Jackson RJ, Smith SD. Fibrin sheath enhances central venous catheter infection. *Crit Care Med* 2002; 30:908–12.
218. Donlan RM, Costerton JW. Biofilms: survival mechanisms of clinically relevant microorganisms. *Clin Microbiol Rev* 2002; 15:167–93.
219. Hawser SP, Douglas LJ. Biofilm formation by *Candida* species on the surface of catheter materials in vitro. *Infect Immun* 1994; 62: 915–21.
220. Stillman RM, Soliman F, Garcia L, Sawyer PN. Etiology of catheter-associated sepsis. Correlation with thrombogenicity. *Arch Surg* 1977; 112:1497–9.
221. Raad II, Luna M, Khalil SA, Costerton JW, Lam C, Bodey GP. The relationship between the thrombotic and infectious complications of central venous catheters. *JAMA* 1994; 271:1014–6.
222. Herrmann M, Suchard SJ, Boxer LA, Waldvogel FA, Lew PD. Thrombospondin binds to *Staphylococcus aureus* and promotes staphylococcal adherence to surfaces. *Infect Immun* 1991; 59:279–88.
223. Shanks RM, Sargent JL, Martinez RM, Graber ML, O’Toole GA. Catheter lock solutions influence staphylococcal biofilm formation on abiotic surfaces. *Nephrol Dial Transplant* 2006; 21:2247–55.
224. Chatzinikolaou I, Zipf TF, Hanna H, et al. Minocycline-ethylenediaminetetraacetate lock solution for the prevention of implantable port infections in children with cancer. *Clin Infect Dis* 2003; 36:116–9.
225. McDevitt D, Francois P, Vaudaux P, Foster TJ. Identification of the ligand-binding domain of the surface-located fibrinogen receptor (clumping factor) of *Staphylococcus aureus*. *Mol Microbiol* 1995; 16:895–907.
226. Ni Eidhin D, Perkins S, Francois P, Vaudaux P, Hook M, Foster TJ. Clumping factor B (ClfB), a new surface-located fibrinogen-binding adhesin of *Staphylococcus aureus*. *Mol Microbiol* 1998; 30:245–57.
227. Mack D, Davies AP, Harris LG, Rohde H, Horstkotte MA, Knobloch JK. Microbial interactions in *Staphylococcus epidermidis* biofilms. *Anal Bioanal Chem* 2007; 387:399–408.
228. von Eiff C, Peters G, Heilmann C. Pathogenesis of infections due to coagulase-negative staphylococci. *Lancet Infect Dis* 2002; 2:677–85.
229. Zhu Y, Weiss EC, Otto M, Fey PD, Smeltzer MS, Somerville GA. *Staphylococcus aureus* metabolism in a biofilm: the influence of arginine on polysaccharide intercellular adhesin synthesis, biofilm formation, and pathogenesis. *Infect Immun* 2007; 75:4219–26.
230. Murga R, Miller JM, Donlan RM. Biofilm formation by gram-negative bacteria on central venous catheter connectors: effect of conditioning films in a laboratory model. *J Clin Microbiol* 2001; 39:2294–7.

231. Douglas LJ. *Candida* biofilms and their role in infection. Trends Microbiol 2003; 11:30–6.
232. Donlan RM. Biofilms: microbial life on surfaces. Emerg Infect Dis 2002; 8:881–90.
233. Dunne WM Jr., Burd EM. The effects of magnesium, calcium, EDTA, and pH on the in vitro adhesion of *Staphylococcus epidermidis* to plastic. Microbiol Immunol 1992; 36:1019–27.
234. Ozerdem Akpolat N, Elci S, Atmaca S, Akbayin H, Gul K. The effects of magnesium, calcium and EDTA on slime production by *Staphylococcus epidermidis* strains. Folia Microbiol (Praha) 2003; 48:649–53.
235. Banin E, Brady KM, Greenberg EP. Chelator-induced dispersal and killing of *Pseudomonas aeruginosa* cells in a biofilm. Appl Environ Microbiol 2006; 72:2064–9.
236. Donlan RM. Role of biofilms in antimicrobial resistance. ASAIO J 2000; 46:S47–52.
237. Farber BF, Kaplan MH, Clogston AG. *Staphylococcus epidermidis* extracted slime inhibits the antimicrobial action of glycopeptide antibiotics. J Infect Dis 1990; 161:37–40.
238. Branchini ML, Pfaller MA, Rhine-Chalberg J, Frempong T, Isenberg HD. Genotypic variation and slime production among blood and catheter isolates of *Candida parapsilosis*. J Clin Microbiol 1994; 32:452–6.
239. Sanders RA, Sheldon GF. Septic complications of total parenteral nutrition. A five year experience. Am J Surg 1976; 132:214–20.
240. Ryan JA Jr., Abel RM, Abbott WM, et al. Catheter complications in total parenteral nutrition. A prospective study of 200 consecutive patients. N Engl J Med 1974; 290:757–61.
241. Murphy LM, Lipman TO. Central venous catheter care in parenteral nutrition: a review. JPEN J Parenter Enteral Nutr 1987; 11: 190–201.
242. Armstrong CW, Mayhall CG, Miller KB, et al. Prospective study of catheter replacement and other risk factors for infection of hyperalimentation catheters. J Infect Dis 1986; 154:808–16.
243. Garland JS, Dunne WM Jr., Havens P, et al. Peripheral intravenous catheter complications in critically ill children: a prospective study. Pediatrics 1992; 89:1145–50.
244. Garland JS, Nelson DB, Cheah TE, Hennes HH, Johnson TM. Infectious complications during peripheral intravenous therapy with Teflon catheters: a prospective study. Pediatr Infect Dis J 1987; 6:918–21.
245. Breschan C, Platzer M, Jost R, Schaumberger F, Stettner H, Likar R. Comparison of catheter-related infection and tip colonization between internal jugular and subclavian central venous catheters in surgical neonates. Anesthesiology 2007; 107:946–53.
246. Deshpande KS, Hatem C, Ulrich HL, et al. The incidence of infectious complications of central venous catheters at the subclavian, internal jugular, and femoral sites in an intensive care unit population. Crit Care Med 2005; 33:13–20; discussion 234–5.
247. Durbec O, Viviand X, Potie F, Vialet R, Albanese J, Martin C. A prospective evaluation of the use of femoral venous catheters in critically ill adults. Crit Care Med 1997; 25:1986–9.
248. Venkataraman ST, Thompson AE, Orr RA. Femoral vascular catheterization in critically ill infants and children. Clin Pediatr (Phila) 1997; 36:311–9.

249. Sheridan RL, Weber JM. Mechanical and infectious complications of central venous cannulation in children: lessons learned from a 10-year experience placing more than 1000 catheters. *J Burn Care Res* 2006; 27:713–8.
250. Stenzel JP, Green TP, Fuhrman BP, Carlson PE, Marchessault RP. Percutaneous central venous catheterization in a pediatric intensive care unit: a survival analysis of complications. *Crit Care Med* 1989; 17:984–8.
251. Goldstein AM, Weber JM, Sheridan RL. Femoral venous access is safe in burned children: an analysis of 224 catheters. *J Pediatr* 1997; 130:442–6.
252. Ramos GE, Bolgiani AN, Patino O, et al. Catheter infection risk related to the distance between insertion site and burned area. *J Burn Care Rehabil* 2002; 23:266–71.
253. Sheth NK, Franson TR, Rose HD, Buckmire FL, Cooper JA, Sohnle PG. Colonization of bacteria on polyvinyl chloride and Teflon intravascular catheters in hospitalized patients. *J Clin Microbiol* 1983; 18:1061–3.
254. Maki DG, Ringer M. Evaluation of dressing regimens for prevention of infection with peripheral intravenous catheters. Gauze, a transparent polyurethane dressing, and an iodophor-transparent dressing. *JAMA* 1987; 258:2396–403.
255. Pittet D, Hugonnet S, Harbath S, et al. Effectiveness of a hospital-wide programme to improve compliance with hand hygiene. *Lancet* 2000; 356:1307–9.
256. Humar A, Ostromecki A, Direnfeld J, et al. Prospective randomized trial of 10% povidone-iodine versus 0.5% tincture of chlorhexidine as cutaneous antisepsis for prevention of central venous catheter infection. *Clin Infect Dis* 2000; 31:1001–7.
257. Chaiyakunapruk N, Veenstra DL, Lipsky BA, Saint S. Chlorhexidine compared with povidone-iodine solution for vascular catheter-site care: a meta-analysis. *Ann Intern Med* 2002; 136:792–801.
258. Chaiyakunapruk N, Veenstra DL, Lipsky BA, Sullivan SD, Saint S. Vascular catheter site care: the clinical and economic benefits of chlorhexidine gluconate compared with povidone iodine. *Clin Infect Dis* 2003; 37:764–71.
259. Parienti JJ, du Cheyron D, Ramakers M, et al. Alcoholic povidone-iodine to prevent central venous catheter colonization: a randomized unit-crossover study. *Crit Care Med* 2004; 32:708–13.
260. Hoffmann KK, Weber DJ, Samsa GP, Rutala WA. Transparent polyurethane film as an intravenous catheter dressing. A meta-analysis of the infection risks. *JAMA* 1992; 267:2072–6.
261. Gillies D, O’Riordan E, Carr D, O’Brien I, Frost J, Gunning R. Central venous catheter dressings: a systematic review. *J Adv Nurs* 2003; 44:623–32.
262. Ruschulte H, Franke M, Gastmeier P, et al. Prevention of central venous catheter related infections with chlorhexidine gluconate impregnated wound dressings: a randomized controlled trial. *Ann Hematol* 2009; 88:267–72.
263. Veenstra DL, Saint S, Saha S, Lumley T, Sullivan SD. Efficacy of antiseptic-impregnated central venous catheters in preventing catheter-related bloodstream infection: a meta-analysis. *JAMA* 1999; 281:261–7.

264. Maki DG, Stolz SM, Wheeler S, Mermel LA. Prevention of central venous catheter-related bloodstream infection by use of an antiseptic-impregnated catheter. A randomized, controlled trial. *Ann Intern Med* 1997; 127:257–66.
265. Bassetti S, Hu J, D'Agostino RB Jr., and Sherertz RJ. Prolonged antimicrobial activity of a catheter containing chlorhexidine-silver sulfadiazine extends protection against catheter infections in vivo. *Antimicrob Agents Chemother* 2001; 45:1535–8.
266. Oda T, Hamasaki J, Kanda N, Mikami K. Anaphylactic shock induced by an antiseptic-coated central venous [correction of nervous] catheter. *Anesthesiology* 1997; 87:1242–4.
267. Pittaway A, Ford S. Allergy to chlorhexidine-coated central venous catheters revisited. *Br J Anaesth* 2002; 88:304–5; author reply 305.
268. Stephens R, Mythen M, Kallis P, Davies DW, Egner W, Rickards A. Two episodes of life-threatening anaphylaxis in the same patient to a chlorhexidine-sulphadiazine-coated central venous catheter. *Br J Anaesth* 2001; 87:306–8.
269. Terazawa E, Shimonaka H, Nagase K, Masue T, Dohi S. Severe anaphylactic reaction due to a chlorhexidine-impregnated central venous catheter. *Anesthesiology* 1998; 89:1296–8.
270. Jee R, Nel L, Gnanakumaran G, Williams A, Eren E. Four cases of anaphylaxis to chlorhexidine impregnated central venous catheters: a case cluster or the tip of the iceberg? *Br J Anaesth* 2009; 103:614–5.
271. Veenstra DL, Saint S, Sullivan SD. Cost-effectiveness of antiseptic-impregnated central venous catheters for the prevention of catheter-related bloodstream infection. *JAMA* 1999; 282:554–60.
272. Tambe SM, Sampath L, Modak SM. In vitro evaluation of the risk of developing bacterial resistance to antiseptics and antibiotics used in medical devices. *J Antimicrob Chemother* 2001; 47:589–98.
273. Sampath LA, Tambe SM, Modak SM. In vitro and in vivo efficacy of catheters impregnated with antiseptics or antibiotics: evaluation of the risk of bacterial resistance to the antimicrobials in the catheters. *Infect Control Hosp Epidemiol* 2001; 22:640–6.
274. Marciante KD, Veenstra DL, Lipsky BA, Saint S. Which antimicrobial impregnated central venous catheter should we use? Modeling the costs and outcomes of antimicrobial catheter use. *Am J Infect Control* 2003; 31:1–8.
275. Shorr AF, Humphreys CW, Helman DL. New choices for central venous catheters: potential financial implications. *Chest* 2003; 124:275–84.
276. Hagau N, Studnicska D, Gavrus RL, Csipak G, Hagau R, Slavcovici AV. Central venous catheter colonization and catheter-related bloodstream infections in critically ill patients: a comparison between standard and silver-integrated catheters. *Eur J Anaesthesiol* 2009; 26:752–8.
277. Bong JJ, Kite P, Wilco MH, McMahon MJ. Prevention of catheter related bloodstream infection by silver iontophoretic central venous catheters: a randomised controlled trial. *J Clin Pathol* 2003; 56:731–5.
278. Corral L, Nolla-Salas M, Ibanez-Nolla J, et al. A prospective, randomized study in critically ill patients using the Oligon Vantex catheter. *J Hosp Infect* 2003; 55:212–9.
279. Ranucci M, Isgro G, Giomarelli PP, et al. Impact of oligon central venous catheters on catheter colonization and catheter-related bloodstream infection. *Crit Care Med* 2003; 31:52–9.

280. Raad II, Hachem RY, Abi-Said D, et al. A prospective crossover randomized trial of novobiocin and rifampin prophylaxis for the prevention of intravascular catheter infections in cancer patients treated with interleukin-2. *Cancer* 1998; 82:403–11.
281. McKee R, Dunsmuir R, Whitby M, Garden OJ. Does antibiotic prophylaxis at the time of catheter insertion reduce the incidence of catheter-related sepsis in intravenous nutrition? *J Hosp Infect* 1985; 6:419–25.
282. Sandoe JA, Kumar B, Stoddart B, et al. Effect of extended perioperative antibiotic prophylaxis on intravascular catheter colonization and infection in cardiothoracic surgery patients. *J Antimicrob Chemother* 2003; 52:877–9.
283. Inglis GDT, Jardine LA, Davies MW. Prophylactic antibiotics to reduce morbidity and mortality in neonates with umbilical artery catheters. *Cochrane Database of Systematic Reviews* 2007; Issue 4. Art. No.: CD004697. DOI: 10.1002/14651858.CD004697.pub3.
284. Craft AP, Finer N, Barrington KJ. Vancomycin for prophylaxis against sepsis in preterm neonates. *Cochrane Database of Systematic Reviews* 2000; Issue 1. Art. No.: CD001971. DOI: 10.1002/14651858.CD001971.
285. Norden CW. Application of antibiotic ointment to the site of venous catheterization—a controlled trial. *J Infect Dis* 1969; 120:611–5.
286. Zinner SH, Denny-Brown BC, Braun P, Burke JP, Toala P, Kass EH. Risk of infection with intravenous indwelling catheters: effect of application of antibiotic ointment. *J Infect Dis* 1969; 120:616–9.
287. von Eiff C, Becker K, Machka K, Stammer H, Peters G. Nasal carriage as a source of *Staphylococcus aureus* bacteremia. *N Engl J Med* 2001; 344:11–6.
288. Chow JW, Yu VL. *Staphylococcus aureus* nasal carriage in hemodialysis patients. Its role in infection and approaches to prophylaxis. *Arch Intern Med* 1989; 149:1258–62.
289. Yu VL, Goetz A, Wagener M, et al. *Staphylococcus aureus* nasal carriage and infection in patients on hemodialysis. Efficacy of antibiotic prophylaxis. *N Engl J Med* 1986; 315:91–6.
290. Casewell MW. The nose: an underestimated source of *Staphylococcus aureus* causing wound infection. *J Hosp Infect* 1998; 40:S3–11.
291. Hill RL, Fisher AP, Ware RJ, Wilson S, Casewell MW. Mupirocin for the reduction of colonization of internal jugular cannulae—a randomized controlled trial. *J Hosp Infect* 1990; 15:311–21.
292. Sesso R, Barbosa D, Leme IL, et al. *Staphylococcus aureus* prophylaxis in hemodialysis patients using central venous catheter: effect of mupirocin ointment. *J Am Soc Nephrol* 1998; 9:1085–92.
293. Boelaert JR, Van Landuyt HW, Godard CA, et al. Nasal mupirocin ointment decreases the incidence of *Staphylococcus aureus* bacteraemias in haemodialysis patients. *Nephrol Dial Transplant* 1993; 8:235–9.
294. Netto dos Santos KR, de Souza Fonseca L, Gontijo Filho PP. Emergence of high-level mupirocin resistance in methicillin-resistant *Staphylococcus aureus* isolated from Brazilian university hospitals. *Infect Control Hosp Epidemiol* 1996; 17:813–6.
295. Miller MA, Dascal A, Portnoy J, Mendelson J. Development of mupirocin resistance among methicillin-resistant *Staphylococcus aureus* after widespread use of nasal mupirocin ointment. *Infect Control Hosp Epidemiol* 1996; 17:811–3.

296. Lok CE, Stanley KE, Hux JE, Richardson R, Tobe SW, Conly J. Hemodialysis infection prevention with polysporin ointment. *J Am Soc Nephrol* 2003; 14:169–79.
297. Yahav D, Rozen-Zvi B, Gafter-Gvili A, Leibovici L, Gafter U, Paul M. Antimicrobial lock solutions for the prevention of infections associated with intravascular catheters in patients undergoing hemodialysis: systematic review and meta-analysis of randomized, controlled trials. *Clin Infect Dis* 2008; 47:83–93.
298. Labriola L, Crott R, Jadoul M. Preventing haemodialysis catheter-related bacteraemia with an antimicrobial lock solution: a metaanalysis of prospective randomized trials. *Nephrol Dial Transplant* 2008; 23:1666–72.
299. Jaffer Y, Selby NM, Taal MW, Fluck RJ, McIntyre CW. A meta-analysis of hemodialysis catheter locking solutions in the prevention of catheter-related infection. *Am J Kidney Dis* 2008; 51:233–41.
300. Safdar N, Maki DG. Use of vancomycin-containing lock or flush solutions for prevention of bloodstream infection associated with central venous access devices: a meta-analysis of prospective, randomized trials. *Clin Infect Dis* 2006; 43:474–84.
301. Sanders J, Pithie A, Ganly P, et al. A prospective double-blind randomized trial comparing intraluminal ethanol with heparinized saline for the prevention of catheter-associated bloodstream infection in immunosuppressed haematology patients. *J Antimicrob Chemother* 2008; 62:809–15.
302. Schinabeck MK, Ghannoum MA. Biofilm-related indwelling medical device infections. In: Pace JL, Rupp ME, Finch RG, eds. *Biofilms, infection, and antimicrobial therapy*. Boca Raton: Taylor and Francis, 2006: 39–50.
303. Gristina AG. Biomaterial-centered infection: microbial adhesion versus tissue integration. *Science* 1987; 237:1588–95.
304. Timsit JF, Farkas JC, Boyer JM, et al. Central vein catheter-related thrombosis in intensive care patients: incidence, risks factors, and relationship with catheter-related sepsis. *Chest* 1998; 114:207–13.
305. Eastman ME, Khorsand M, Maki DG, et al. Central venous device-related infection and thrombosis in patients treated with moderate dose continuous-infusion interleukin-2. *Cancer* 2001; 91:806–14.
306. Abdelkefi A, Torjman L, Ladeb S, et al. Randomized trial of prevention of catheter-related bloodstream infection by continuous infusion of low-dose unfractionated heparin in patients with hematologic and oncologic disease. *J Clin Oncol* 2005; 23:7864–70.
307. Mermel LA, Stolz SM, Maki DG. Surface antimicrobial activity of heparin-bonded and antiseptic-impregnated vascular catheters. *J Infect Dis* 1993; 167:920–4.
308. Pierce CM, Wade A, Mok Q. Heparin-bonded central venous lines reduce thrombotic and infective complications in critically ill children. *Intensive Care Med* 2000; 26:967–72.
309. Appelgren P, Ransjo U, Bindslev L, Espersen F, Larm O. Surface heparinization of central venous catheters reduces microbial colonization in vitro and in vivo: results from a prospective, randomized trial. *Crit Care Med* 1996; 24:1482–9.

310. Abdelkefi A, Achour W, Ben Othman T, et al. Use of heparin-coated central venous lines to prevent catheter-related bloodstream infection. *J Support Oncol* 2007; 5:273–8.
311. Carrasco MN, Bueno A, de las Cuevas C, et al. Evaluation of a triple-lumen central venous heparin-coated catheter versus a catheter coated with chlorhexidine and silver sulfadiazine in critically ill patients. *Intensive Care Med* 2004; 30:633–8.
312. Levy JH, Hursting MJ. Heparin-induced thrombocytopenia, a prothrombotic disease. *Hematol Oncol Clin North Am* 2007; 21:65–88.
313. Weijmer MC, Debets-Ossenkopp YJ, Van De Vondervoort FJ, ter Wee PM. Superior antimicrobial activity of trisodium citrate over heparin for catheter locking. *Nephrol Dial Transplant* 2002; 17:2189–95.
314. Boraks P, Seale J, Price J, et al. Prevention of central venous catheter associated thrombosis using minidose warfarin in patients with haematological malignancies. *Br J Haematol* 1998; 101:483–6.
315. Bern MM, Lokich JJ, Wallach SR, et al. Very low doses of warfarin can prevent thrombosis in central venous catheters. A randomized prospective trial. *Ann Intern Med* 1990; 112:423–8.
316. Akl EA, Karmath G, Yosuico VED, Kim SY, Barba M, Sperati F, Cook D, Schunemann H. Anticoagulation for thrombosis prophylaxis in cancer patients with central venous catheters. *Cochrane Database of Systematic Reviews* 2007; Issue 3. Art. No.: CD006468. DOI: 10.1002/14651858.CD006468.pub2.
317. Akl EA, Muti P, Schunemann HJ. Anticoagulation in patients with cancer: an overview of reviews. *Pol Arch Med Wewn* 2008; 118:183–93.
318. Klerk CP, Smorenburg SM, Buller HR. Thrombosis prophylaxis in patient populations with a central venous catheter: a systematic review. *Arch Intern Med* 2003; 163:1913–21.
319. Heaton DC, Han DY, Inder A. Minidose (1 mg) warfarin as prophylaxis for central vein catheter thrombosis. *Intern Med J* 2002; 32:84–8.
320. Masci G, Magagnoli M, Zucali PA, et al. Minidose warfarin prophylaxis for catheter-associated thrombosis in cancer patients: can it be safely associated with fluorouracil-based chemotherapy? *J Clin Oncol* 2003; 21:736–9.
321. Kuter DJ. Thrombotic complications of central venous catheters in cancer patients. *Oncologist* 2004; 9:207–16.
322. Fontaine PJ. Performance of a new softening expanding midline catheter in home intravenous therapy patients. *J Intraven Nurs* 1991; 14:91–9.
323. Harwood IR, Greene LM, Kozakowski-Koch JA, Rasor JS. New peripherally inserted midline catheter: a better alternative for intravenous antibiotic therapy in patients with cystic fibrosis. *Pediatr Pulmonol* 1992; 12:233–9.
324. Mermel LA, Parenteau S, Tow SM. The risk of midline catheterization in hospitalized patients. A prospective study. *Ann Intern Med* 1995; 123:841–4.
325. Uldall PR, Merchant N, Woods F, Yarworski U, Vas S. Changing subclavian haemodialysis cannulas to reduce infection. *Lancet* 1981; 1:1373.

326. Cook D, Randolph A, Kernerman P, et al. Central venous catheter replacement strategies: a systematic review of the literature. *Crit Care Med* 1997; 25:1417–24.
327. Cobb DK, High KP, Sawyer RG, et al. A controlled trial of scheduled replacement of central venous and pulmonary-artery catheters. *N Engl J Med* 1992; 327:1062–8.
328. Beathard GA. Management of bacteremia associated with tunneled-cuffed hemodialysis catheters. *J Am Soc Nephrol* 1999; 10:1045–9.
329. Duszak R Jr., Haskal ZJ, Thomas-Hawkins C, et al. Replacement of failing tunneled hemodialysis catheters through pre-existing subcutaneous tunnels: a comparison of catheter function and infection rates for de novo placements and over-the-wire exchanges. *J Vasc Interv Radiol* 1998; 9:321–7.
330. Robinson D, Suhocki P, Schwab SJ. Treatment of infected tunneled venous access hemodialysis catheters with guidewire exchange. *Kidney Int* 1998; 53:1792–4.
331. Saad TF. Bacteremia associated with tunneled, cuffed hemodialysis catheters. *Am J Kidney Dis* 1999; 34:1114–24.
332. Ainsworth S, Clerihew L, McGuire W. Percutaneous central venous catheters versus peripheral cannulae for delivery of parenteral nutrition in neonates. *Cochrane Database of Systematic Reviews* 2007; Issue 3. Art. No.: CD004219. DOI: 10.1002/14651858.CD004219. pub3.
333. Shah PS, Kalyn A, Satodia P, et al. A randomized, controlled trial of heparin versus placebo infusion to prolong the usability of peripherally placed percutaneous central venous catheters (PCVCs) in neonates: the HIP (Heparin Infusion for PCVC) study. *Pediatrics* 2007; 119:e284–91.
334. Jaar BG, Hermann JA, Furth SL, Briggs W, Powe NR. Septicemia in diabetic hemodialysis patients: comparison of incidence, risk factors, and mortality with nondiabetic hemodialysis patients. *Am J Kidney Dis* 2000; 35:282–92.
335. Powe NR, Jaar B, Furth SL, Hermann J, Briggs W. Septicemia in dialysis patients: incidence, risk factors, and prognosis. *Kidney Int* 1999; 55:1081–90.
336. Hoen B, Paul-Dauphin A, Hestin D, Kessler M. EPIBACDIAL: a multicenter prospective study of risk factors for bacteremia in chronic hemodialysis patients. *J Am Soc Nephrol* 1998; 9:869–76.
337. Blot F, Chachaty E, Raynard B, Antoun S, Bourgain JL, Nitenberg G. Mechanisms and risk factors for infection of pulmonary artery catheters and introducer sheaths in cancer patients admitted to an intensive care unit. *J Hosp Infect* 2001; 48:289–97.
338. Kac G, Durain E, Amrein C, Herisson E, Fiemeyer A, Buu-Hoi A. Colonization and infection of pulmonary artery catheter in cardiac surgery patients: epidemiology and multivariate analysis of risk factors. *Crit Care Med* 2001; 29:971–5.
339. Chen YY, Yen DH, Yang YG, Liu CY, Wang FD, Chou P. Comparison between replacement at 4 days and 7 days of the infection rate for pulmonary artery catheters in an intensive care unit. *Crit Care Med* 2003; 31:1353–8.
340. Balagtas RC, Bell CE, Edwards LD, Levin S. Risk of local and systemic infections associated with umbilical vein catheterization: a prospective study in 86 newborn patients. *Pediatrics* 1971; 48:359–67.

341. Butler-O'Hara M, Buzzard CJ, Reubens L, McDermott MP, DiGrazio W, D'Angio CT. A randomized trial comparing long-term and short-term use of umbilical venous catheters in premature infants with birth weights of less than 1251 grams. *Pediatrics* 2006; 118:e25–35.
342. Scheer B, Perel A, Pfeiffer UJ. Clinical review: complications and risk factors of peripheral arterial catheters used for haemodynamic monitoring in anaesthesia and intensive care medicine. *Crit Care* 2002; 6:199–204.
343. Lorente L, Santacreu R, Martin MM, Jimenez A, Mora ML. Arterial catheter-related infection of 2,949 catheters. *Crit Care* 2006; 10:R83.
344. Furfaro S, Gauthier M, Lacroix J, Nadeau D, Lafleur L, Mathews S. Arterial catheter-related infections in children. A 1-year cohort analysis. *Am J Dis Child* 1991; 145:1037–43.
345. Rickard CM, Lipman J, Courtney M, Siversen R, Daley P. Routine changing of intravenous administration sets does not reduce colonization or infection in central venous catheters. *Infect Control Hosp Epidemiol* 2004; 25:650–5.
346. Hanna HA, Raad I. Blood products: a significant risk factor for long-term catheter-related bloodstream infections in cancer patients. *Infect Control Hosp Epidemiol* 2001; 22:165–6.
347. Saiman L, Ludington E, Dawson JD, et al. Risk factors for *Candida* species colonization of neonatal intensive care unit patients. *Pediatr Infect Dis J* 2001; 20:1119–24.
348. Avila-Figueroa C, Goldmann DA, Richardson DK, Gray JE, Ferrari A, Freeman J. Intravenous lipid emulsions are the major determinant of coagulase-negative staphylococcal bacteremia in very low birth weight newborns. *Pediatr Infect Dis J* 1998; 17:10–7.
349. Crocker KS, Noga R, Filibeck DJ, Krey SH, Markovic M, Steffee WP. Microbial growth comparisons of five commercial parenteral lipid emulsions. *J Parenter Enteral Nutr* 1984; 8:391–5.
350. Jarvis WR, Highsmith AK. Bacterial growth and endotoxin production in lipid emulsion. *J Clin Microbiol* 1984; 19:17–20.
351. Karamanoglu A, Yumuk PF, Gumus M, et al. Port needles: do they need to be removed as frequently in infusional chemotherapy? *J Infus Nurs* 2003; 26:239–42.
352. Niel-Weise BS, Daha TJ, van den Broek PJ. Is there evidence for recommending needleless closed catheter access systems in guidelines? A systematic review of randomized controlled trials. *J Hosp Infect* 2006; 62:406–13.
353. Inoue Y, Nezu R, Matsuda H, et al. Prevention of catheter-related sepsis during parenteral nutrition: effect of a new connection device. *J Parenter Enteral Nutr* 1992; 16:581–5.
354. Yebenes JC, Vidaur L, Serra-Prat M, et al. Prevention of catheter-related bloodstream infection in critically ill patients using a disinfectable, needle-free connector: a randomized controlled trial. *Am J Infect Control* 2004; 32:291–5.
355. Casey AL, Worthington T, Lambert PA, Quinn D, Faroqui MH, Elliott TS. A randomized, prospective clinical trial to assess the potential infection risk associated with the PosiFlow needleless connector. *J Hosp Infect* 2003; 54:288–93.

Guidelines for the Prevention of Intravascular Catheter-Related Infections

356. Esteve F, Pujol M, Limon E, et al. Bloodstream infection related to catheter connections: a prospective trial of two connection systems. *J Hosp Infect* 2007; 67:30–4.
357. Yebenes JC, Delgado M, Sauca G, et al. Efficacy of three different valve systems of needle-free closed connectors in avoiding access of microorganisms to endovascular catheters after incorrect handling. *Crit Care Med* 2008; 36:2558–61.
358. Menyhay SZ, Maki DG. Preventing central venous catheter-associated bloodstream infections: development of an antiseptic barrier cap for needleless connectors. *Am J Infect Control* 2008; 36:(Suppl 174):e1–5.
359. Jarvis WR, Murphy C, Hall KK, et al. Health care-associated bloodstream infections associated with negative-or positive-pressure or displacement mechanical valve needleless connectors. *Clin Infect Dis* 2009; 49:1821–7.
360. Menyhay SZ, Maki DG. Disinfection of needleless catheter connectors and access ports with alcohol may not prevent microbial entry: the promise of a novel antiseptic-barrier cap. *Infect Control Hosp Epidemiol* 2006; 27:23–7.
361. Safdar N, Maki DG. Lost in translation. *Infect Control Hosp Epidemiol* 2006; 27:3–7.
362. Warren DK, Yokoe DS, Climo MW, et al. Preventing catheter-associated bloodstream infections: a survey of policies for insertion and care of central venous catheters from hospitals in the prevention epicenter program. *Infect Control Hosp Epidemiol* 2006; 27:8–13.
363. O’Grady NP, Alexander M, Dellinger EP, et al. Guidelines for the prevention of intravascular catheter-related infections. Centers for Disease Control and Prevention. *MMWR Recomm Rep* 2002;51:1–29.
364. Krein SL, Hofer TP, Kowalski CP, et al. Use of central venous catheter-related bloodstream infection prevention practices by US hospitals. *Mayo Clin Proc* 2007; 82:672–8.
365. Lobo RD, Levin AS, Gomes LM, et al. Impact of an educational program and policy changes on decreasing catheter-associated bloodstream infections in a medical intensive care unit in Brazil. *Am J Infect Control* 2005; 33:83–7.
366. Marschall J, Leone C, Jones M, Nihill D, Fraser VJ, Warren DK. Catheter-associated bloodstream infections in general medical patients outside the intensive care unit: a surveillance study. *Infect Control Hosp Epidemiol* 2007; 28:905–9.
367. Rosenthal VD, McCormick RD, Guzman S, Villamayor C, Orellano PW. Effect of education and performance feedback on handwashing: the benefit of administrative support in Argentinean hospitals. *Am J Infect Control* 2003; 31:85–92.
368. Gastmeier P, Geffers C. Prevention of catheter-related bloodstream infections: analysis of studies published between 2002 and 2005. *J Hosp Infect* 2006; 64:326–35.
369. Pronovost PJ, Goeschel CA, Colantuoni E, et al. Sustaining reductions in catheter related bloodstream infections in Michigan intensive care units: observational study. *BMJ* 2010; 340:c309.
370. Shapey IM, Foster MA, Whitehouse T, Jumaa P, Bion JF. Central venous catheter-related bloodstream infections: improving post-insertion catheter care. *J Hosp Infect* 2009; 71:117–22.

HICPAC

HEALTHCARE INFECTION CONTROL
PRACTICES ADVISORY COMMITTEE

GUIDELINE FOR PREVENTION OF CATHETER- ASSOCIATED URINARY TRACT INFECTIONS 2009

Carolyn V. Gould, MD, MSCR ¹; Craig A. Umscheid, MD, MSCE ²; Rajender K. Agarwal, MD, MPH ²; Gretchen Kuntz, MSW, MSLIS ²; David A. Pegues, MD ³ and the Healthcare Infection Control Practices Advisory Committee (HICPAC) ⁴

¹ Division of Healthcare Quality Promotion
Centers for Disease Control and Prevention
Atlanta, GA

² Center for Evidence-based Practice
University of Pennsylvania Health System
Philadelphia, PA

³ Division of Infectious Diseases
David Geffen School of Medicine at UCLA
Los Angeles, CA



4 Healthcare Infection Control Practices Advisory Committee

(HICPAC)

Chair

BRENNAN, Patrick J., MD
Chief Medical Officer
Division of Infectious Diseases
University of Pennsylvania Health System

Department of Pathology
University of Florida Health Science
Center-Jacksonville

Executive Secretary

BELL, Michael R., MD
Associate Director for Infection Control
Division of Healthcare Quality Promotion
National Center for Infectious Diseases
Centers for Disease Control and
Prevention

MURPHY, Denise M., MPH, RN, CIC
Vice President, Safety and Quality
Barnes-Jewish Hospital at Washington
University Medical Center

OLMSTED, Russell N., MPH
Epidemiologist
Infection Control Services
St. Joseph Mercy Health System

Members

BURNS, Lillian A., MPH
Infection Control Coordinator
Infectious Diseases Department,
Greenwich Hospital

PEGUES, David Alexander, MD
Professor of Medicine, Hospital Epidemiologist
David Geffen School of Medicine at
UCLA

ELWARD, Alexis, MD, MPH
Medical Director of Infection Control
Assistant Professor, Pediatric Infectious
Diseases
Washington University School of Medicine

RAMSEY, Keith M., MD
Professor of Medicine
Medical Director of Infection Control
Pitt County Memorial

ENGEL, Jeffrey, MD
Chief, Epidemiology Section, North
Carolina Division of Public Health
North Carolina State Epidemiologist

SINGH, Nalini, MD, MPH
Professor of Pediatrics
Epidemiology and International Health
George Washington University
Children's National Medical Center

LUNDSTROM, Tammy, MD, JD
Chief Medical Officer
Providence Hospital

SOULE, Barbara M., RN, MPA, CIC
Practice Leader
Infection Prevention Services
Joint Commission Resources/Joint
Commission International

GORDON, Steven M., MD
Chairman, Department of Infectious
Diseases
Hospital Epidemiologist
Cleveland Clinic Foundation

SCHECTER, William, P., MD
Department of Surgery, Ward 3A 17
San Francisco General Hospital

MCCARTER, Yvette S., PhD
Director, Clinical Microbiology
Laboratory

STEVENSON, Kurt Brown, MD, MPH
Division of Infectious Diseases
Department of Internal Medicine
The Ohio State University Medical Center

Ex-officio Members

Agency for Healthcare Research and Quality

Ex-Officio

BAINE, William B., MD
Senior Medical Advisor
Center for Outcomes and Evidence
Agency for Healthcare Research and Quality

National Institute of Health

Ex-Officio

HENDERSON, David, MD
Deputy Director for Clinical Care
National Institute of Health

Health Resources and Services

Administration Ex-Officio

JAY, Lorine J., MPH, RN, CPHQ
Regional Coordinator

Food and Drug Administration

Ex-Officio

MURPHEY, Sheila A., MD
Chief, Infection Control Devices Branch
Division of Anesthesiology, General Hospital
Infection Control and Dental Devices
Center for Devices and Radiology Health
Food and Drug Administration

Center for Medicare & Medicaid Services

(CMS) Ex-Officio

MILLER, Jeannie RN, MPH
Deputy Director, Office of Clinical
Standards and Quality/ Clinical Standards
Group

Department of Veterans Affairs (VA)

ROSELLE, Gary A., MD
National Program Director, Infectious
Diseases
VA Central Office
Cincinnati VA Medical Center

Liaisons

Association of Professionals of Infection Control and Epidemiology, Inc.

BJERKE, Nancy BSN, RN, MPH, CIC

Council of State and Territorial Epidemiologists

KAINER, Marion MD, MPH
Medical Epidemiologist/Infections
Diseases Physician Director, Hospital
Infections and Antimicrobial Resistance
Program, Tennessee Department of Health

Infection Control Consultant
Infection Control Associates

American Health Care Association

FITZLER, Sandra L., RN
Senior Director of Clinical Services
American Health Care Association

American College of Occupational and Environmental Medicine

RUSSI, Mark, MD, MPH
American College of Occupational and
Environmental Medicine

Advisory Council for the Elimination of

Tuberculosis STRICOF, Rachel L., MPH
New York State Department of Health

American Hospital Association

SCHULMAN, Roslyne, MHA, MBA
Senior Associate Director, Policy
Development

Association of periOperative Registered Nurses

BLANCHARD, Joan C., RN, BSN,
MSS, CNOR, CIC
Association of periOperative Registered
Nurses

Society for Healthcare Epidemiology of America

MARAGAKIS, Lisa, MD
Assistant Professor of Medicine
Johns Hopkins Medical Institutions

Joint Commission on Accreditation of Healthcare Organizations

WISE, Robert A., MD
Division of Standards & Survey
Methods
Joint Commission on Accreditation of
Healthcare Organizations

Consumers Union

Senior Policy Analyst on
MCGIFFERT, Lisa
Health Issues, Project Director
Stop Hospital Infections Organization

Acknowledgement

HICPAC thanks the following members who served on the HICPAC CAUTI Guideline subcommittee during the guideline development process: Russell N. Olmsted, MPH, Yvette S. McCarter, PhD, Barbara M. Soule, RN, MPA, CIC, and Nalini Singh, MD, MPH.

HICPAC thanks the following outside experts for reviewing a draft of this guideline: Edward S. Wong, MD, Lindsay E. Nicolle, MD, Anthony J. Schaeffer, MD, and Harriett M. Pitt, RN, MS, CIC. The opinions of the reviewers might not be reflected in all the recommendations contained in this document.

HICPAC would also like to thank the many individuals and organizations who provided valuable feedback on the guideline during the public comment period.

Disclosure of Financial Interests and Relationships

The authors C.V.G., C.A.U., R.K.A., and G.K. report no actual or potential conflicts of interest. D.A.P. is on the Speakers Bureau of Merck, Pfizer, Schering, and Cubist and is a consultant for Dow Pharmaceuticals, DaVita, and Vasonova. C.A.U. and R.K.A. received funding from the CDC to support the guideline development process.

Table of Contents

<i>Abbreviations</i>	6
I. Executive Summary	8
II. Summary of Recommendations.....	10
III. Implementation and Audit	18
IV. Recommendations for Further Research	20
V. Background.....	22
VI. Scope and Purpose	25
VII. Methods	26
VIII. Evidence Review	34
References.....	49

Abbreviations

ADL	Activities of daily living
APACHE II	Acute Physiology and Chronic Health Evaluation II
ASA	American Society of Anesthesiologists
ASB	Asymptomatic bacteriuria
BUN	Blood urea nitrogen
CAUTI	Catheter-associated urinary tract infection
CDC	Centers for Disease Control and Prevention
CFU	Colony-forming units
CI	Confidence interval
CIC	Clean intermittent catheterization
CICU	Coronary intensive care unit
COPD	Chronic obstructive pulmonary disease
ED	Emergency department
F/U	Follow-up
GRADE	Grading of Recommendations Assessment, Development, and Evaluation system
Hb	Hemoglobin concentration
HICPAC	Healthcare Infection Control Practices Advisory Committee
H/O	History of
HPF	High power field
HR	Hazard ratio
ICU	Intensive care unit
IDR	Incidence-density ratio
LOS	Length of stay
MDR	Multi-drug resistant
MICU	Medical intensive care unit
NHSN	National Healthcare Safety Network
NIH	National Institutes of Health
NS	Not significant
OBS	Observational controlled study
OR	Odds ratio
P	P value
PACU	Post-anesthesia care unit
PVC	Polyvinyl chloride

RCT	Randomized controlled trial
RD	Risk difference
RH	Relative hazard
RR	Relative risk
SAPS II	Simplified Acute Physiology Score II
SICU	Surgical intensive care unit
SR	Systematic review
SUTI	Symptomatic urinary tract infection
TMP/SMX	Trimethoprim/sulfamethoxazole
TURP	Transurethral resection of prostate
UTI	Urinary tract infection
VAS	Visual analog scale
WMD	Weighted mean difference

I. Executive Summary

This guideline updates and expands the original Centers for Disease Control and Prevention (CDC) Guideline for Prevention of Catheter-associated Urinary Tract Infections (CAUTI) published in 1981. Several developments necessitated revision of the 1981 guideline, including new research and technological advancements for preventing CAUTI, increasing need to address patients in non-acute care settings and patients requiring long-term urinary catheterization, and greater emphasis on prevention initiatives as well as better defined goals and metrics for outcomes and process measures. In addition to updating the previous guideline, this revised guideline reviews the available evidence on CAUTI prevention for patients requiring chronic indwelling catheters and individuals who can be managed with alternative methods of urinary drainage (e.g., intermittent catheterization). The revised guideline also includes specific recommendations for implementation, performance measurement, and surveillance. Although the general principles of CAUTI prevention have not changed from the previous version, the revised guideline provides clarification and more specific guidance based on a defined, systematic review of the literature through July 2007. For areas where knowledge gaps exist, recommendations for further research are listed. Finally, the revised guideline outlines high-priority recommendations for CAUTI prevention in order to offer guidance for implementation.

This document is intended for use by infection prevention staff, healthcare epidemiologists, healthcare administrators, nurses, other healthcare providers, and persons responsible for developing, implementing, and evaluating infection prevention and control programs for healthcare settings across the continuum of care. The guideline can also be used as a resource for societies or organizations that wish to develop more detailed implementation guidance for prevention of CAUTI.

Our goal was to develop a guideline based on a targeted systematic review of the best available evidence, with explicit links between the evidence and recommendations. To accomplish this, we used an adapted GRADE system approach for evaluating quality of evidence and determining strength of recommendations. The methodology, structure, and components of this guideline are approved by HICPAC and will be used for subsequent guidelines issued by HICPAC. A more detailed description of our approach is available in the [Methods](#) section.

To evaluate the evidence on preventing CAUTI, we examined data addressing three key questions and related subquestions:

1. Who should receive urinary catheters?
 - A. When is urinary catheterization necessary?
 - B. What are the risk factors for CAUTI?
 - C. What populations are at highest risk of mortality related to urinary catheters?
2. For those who may require urinary catheters, what are the best practices?
Specifically, what are the risks and benefits associated with:
 - A. Different approaches to catheterization?
 - B. Different catheters or collecting systems?
 - C. Different catheter management techniques?
 - D. Different systems interventions (i.e., quality improvement programs)?
3. What are the best practices for preventing CAUTI associated with obstructed urinary catheters?

Evidence addressing the key questions was used to formulate recommendations, and explicit links between the evidence and recommendations are available in the Evidence Review in the body of the guideline, and Evidence Tables and GRADE Tables in the Appendices. **It is important to note that Category I recommendations are all considered strong recommendations and should be equally implemented;** it is only the *quality* of the evidence underlying the recommendation that distinguishes between levels A and B. Category IC recommendations are required by state or federal regulation and may have any level of supporting evidence.

The categorization scheme used in this guideline is presented in Table 1 in the Summary of Recommendations and described further in the Methods section.

The Summary of Recommendations is organized as follows: 1) recommendations for who should receive indwelling urinary catheters (or, for certain populations, alternatives to indwelling catheters); 2) recommendations for catheter insertion; 3) recommendations for catheter maintenance; 4) quality improvement programs to achieve appropriate placement, care, and removal of catheters; 5) administrative infrastructure required; and 6) surveillance strategies.

The Implementation and Audit section includes a prioritization of recommendations (i.e., high-priority recommendations that are essential for every healthcare facility), organized by modules, in order to provide facilities more guidance on implementation of these guidelines. A list of recommended performance measures that can potentially be used for internal reporting purposes is also included.

Areas in need of further research identified during the evidence review are outlined in the Recommendations for Further Research. This section includes guidance for specific methodological approaches that should be used in future studies.

Readers who wish to examine the primary evidence underlying the recommendations are referred to the Evidence Review in the body of the guideline, and the Evidence Tables and GRADE Tables in the Appendices. The Evidence Review includes narrative summaries of the data presented in the Evidence Tables and GRADE Tables. The Evidence Tables include all study-level data used in the guideline, and the GRADE Tables assess the overall quality of evidence for each question. The Appendices also contain a clearly delineated search strategy that will be used for periodic updates to ensure that the guideline remains a timely resource as new information becomes available.

II. Summary of Recommendations

Category IA	A strong recommendation supported by high to moderate quality† evidence suggesting net clinical benefits or harms
Category IB	A strong recommendation supported by low quality evidence suggesting net clinical benefits or harms or an accepted practice (e.g., aseptic technique) supported by low to very low quality evidence
Category IC	A strong recommendation required by state or federal regulation.
Category II	A weak recommendation supported by any quality evidence suggesting a trade off between clinical benefits and harms
No recommendation/ unresolved issue	Unresolved issue for which there is low to very low quality evidence with uncertain trade offs between benefits and harms

* Please refer to Methods (p.32) for implications of Category designations

†Please refer to Methods (p. 29-30) for process used to grade quality of evidence

I. Appropriate Urinary Catheter Use

- A. Insert catheters only for appropriate indications (see Table 2 for guidance), and leave in place only as long as needed. **(Category IB)** (Key Questions 1B and 2C)
 1. Minimize urinary catheter use and duration of use in all patients, particularly those at higher risk for CAUTI or mortality from catheterization such as women, the elderly, and patients with impaired immunity. **(Category IB)** (Key Questions 1B and 1C)
 2. Avoid use of urinary catheters in patients and nursing home residents for management of incontinence. **(Category IB)** (Key Question 1A)
 - a. Further research is needed on periodic (e.g., nighttime) use of external catheters (e.g., condom catheters) in incontinent patients or residents and the use of catheters to prevent skin breakdown. **(No recommendation/unresolved issue)** (Key Question 1A)
 3. Use urinary catheters in operative patients only as necessary, rather than routinely. **(Category IB)** (Key Question 1A)
 4. For operative patients who have an indication for an indwelling catheter, remove the catheter as soon as possible postoperatively, preferably within 24 hours, unless there are appropriate indications for continued use. **(Category IB)** (Key Questions 2A and 2C)

Table 2.
A. Examples of Appropriate Indications for Indwelling Urethral Catheter Use ¹⁻⁴
Patient has acute urinary retention or bladder outlet obstruction
Need for accurate measurements of urinary output in critically ill patients
Perioperative use for selected surgical procedures: <ul style="list-style-type: none"> • Patients undergoing urologic surgery or other surgery on contiguous structures of the genitourinary tract • Anticipated prolonged duration of surgery (catheters inserted for this reason should be removed in PACU) • Patients anticipated to receive large-volume infusions or diuretics during surgery • Need for intraoperative monitoring of urinary output
To assist in healing of open sacral or perineal wounds in incontinent patients
Patient requires prolonged immobilization (e.g., potentially unstable thoracic or lumbar spine, multiple traumatic injuries such as pelvic fractures)
To improve comfort for end of life care if needed
B. Examples of Inappropriate Uses of Indwelling Catheters
As a substitute for nursing care of the patient or resident with incontinence
As a means of obtaining urine for culture or other diagnostic tests when the patient can voluntarily void
For prolonged postoperative duration without appropriate indications (e.g., structural repair of urethra or contiguous structures, prolonged effect of epidural anaesthesia, etc.)

Note: These indications are based primarily on expert consensus.

B. Consider using alternatives to indwelling urethral catheterization in selected patients when appropriate.

1. Consider using external catheters as an alternative to indwelling urethral catheters in cooperative male patients without urinary retention or bladder outlet obstruction. **(Category II)** (Key Question 2A)
2. Consider alternatives to chronic indwelling catheters, such as intermittent catheterization, in spinal cord injury patients. **(Category II)** (Key Question 1A)
3. Intermittent catheterization is preferable to indwelling urethral or suprapubic catheters in patients with bladder emptying dysfunction. **(Category II)** (Key Question 2A)
4. Consider intermittent catheterization in children with myelomeningocele and neurogenic bladder to reduce the risk of urinary tract deterioration. **(Category II)** (Key Question 1A)
5. Further research is needed on the benefit of using a urethral stent as an alternative to an indwelling catheter in selected patients with bladder outlet obstruction. **(No recommendation/unresolved issue)** (Key Question 1A)
6. Further research is needed on the risks and benefits of suprapubic catheters as an alternative to indwelling urethral catheters in selected patients requiring short- or long-term catheterization, particularly with respect to complications related to catheter insertion or the catheter site. **(No recommendation/unresolved issue)** (Key Question 2A)

II. Proper Techniques for Urinary Catheter Insertion

- A. Perform hand hygiene immediately before and after insertion or any manipulation of the catheter device or site. **(Category IB)** (Key Question 2D)
- B. Ensure that only properly trained persons (e.g., hospital personnel, family members, or patients themselves) who know the correct technique of aseptic catheter insertion and maintenance are given this responsibility. **(Category IB)** (Key Question 1B)
- C. In the acute care hospital setting, insert urinary catheters using aseptic technique and sterile equipment. **(Category IB)**
 - 1. Use sterile gloves, drape, sponges, an appropriate antiseptic or sterile solution for periurethral cleaning, and a single-use packet of lubricant jelly for insertion. **(Category IB)**
 - 2. Routine use of antiseptic lubricants is not necessary. **(Category II)** (Key Question 2C)
 - 3. Further research is needed on the use of antiseptic solutions vs. sterile water or saline for periurethral cleaning prior to catheter insertion. **(No recommendation/unresolved issue)** (Key Question 2C)
- D. In the non-acute care setting, clean (i.e., non-sterile) technique for intermittent catheterization is an acceptable and more practical alternative to sterile technique for patients requiring chronic intermittent catheterization. **(Category IA)** (Key Question 2A)
 - 1. Further research is needed on optimal cleaning and storage methods for catheters used for clean intermittent catheterization. **(No recommendation/unresolved issue)** (Key Question 2C)
- E. Properly secure indwelling catheters after insertion to prevent movement and urethral traction. **(Category IB)**
- F. Unless otherwise clinically indicated, consider using the smallest bore catheter possible, consistent with good drainage, to minimize bladder neck and urethral trauma. **(Category II)**
- G. If intermittent catheterization is used, perform it at regular intervals to prevent bladder overdistension. **(Category IB)** (Key Question 2A)
- H. Consider using a portable ultrasound device to assess urine volume in patients undergoing intermittent catheterization to assess urine volume and reduce unnecessary catheter insertions. **(Category II)** (Key Question 2C)
 - 1. If ultrasound bladder scanners are used, ensure that indications for use are clearly stated, nursing staff are trained in their use, and equipment is adequately cleaned and disinfected in between patients. **(Category IB)**

III. Proper Techniques for Urinary Catheter Maintenance

- A. Following aseptic insertion of the urinary catheter, maintain a closed drainage system **(Category IB)** (Key Question 1B and 2B)
 - 1. If breaks in aseptic technique, disconnection, or leakage occur, replace the catheter and collecting system using aseptic technique and sterile equipment. **(Category IB)**
 - 2. Consider using urinary catheter systems with preconnected, sealed catheter-tubing junctions. **(Category II)** (Key Question 2B)
- B. Maintain unobstructed urine flow. **(Category IB)** (Key Questions 1B and 2D)
 - 1. Keep the catheter and collecting tube free from kinking. **(Category IB)**
 - 2. Keep the collecting bag below the level of the bladder at all times. Do not rest the bag on the floor. **(Category IB)**
 - 3. Empty the collecting bag regularly using a separate, clean collecting container for each patient; avoid splashing, and prevent contact of the drainage spigot with the nonsterile collecting container. **(Category IB)**
- C. Use Standard Precautions, including the use of gloves and gown as appropriate, during any manipulation of the catheter or collecting system. **(Category IB)**
- D. Complex urinary drainage systems (utilizing mechanisms for reducing bacterial entry such as antiseptic-release cartridges in the drain port) are not necessary for routine use. **(Category II)** (Key Question 2B)
- E. Changing indwelling catheters or drainage bags at routine, fixed intervals is not recommended. Rather, it is suggested to change catheters and drainage bags based on clinical indications such as infection, obstruction, or when the closed system is compromised. **(Category II)** (Key Question 2C)
- F. Unless clinical indications exist (e.g., in patients with bacteriuria upon catheter removal post urologic surgery), do not use systemic antimicrobials routinely to prevent CAUTI in patients requiring either short or long-term catheterization. **(Category IB)** (Key Question 2C)
 - 1. Further research is needed on the use of urinary antiseptics (e.g., methenamine) to prevent UTI in patients requiring short-term catheterization. **(No recommendation/unresolved issue)** (Key Question 2C)
- G. Do not clean the periurethral area with antiseptics to prevent CAUTI while the catheter is in place. Routine hygiene (e.g., cleansing of the meatal surface during daily bathing or showering) is appropriate. **(Category IB)** (Key Question 2C)
- H. Unless obstruction is anticipated (e.g., as might occur with bleeding after prostatic or bladder surgery) bladder irrigation is not recommended. **(Category II)** (Key Question 2C)

1. If obstruction is anticipated, closed continuous irrigation is suggested to prevent obstruction. **(Category II)**
- I. Routine irrigation of the bladder with antimicrobials is not recommended. **(Category II)** (Key Question 2C)
- J. Routine instillation of antiseptic or antimicrobial solutions into urinary drainage bags is not recommended. **(Category II)** (Key Question 2C)
- K. Clamping indwelling catheters prior to removal is not necessary. **(Category II)** (Key Question 2C)
- L. Further research is needed on the use of bacterial interference (i.e., bladder inoculation with a nonpathogenic bacterial strain) to prevent UTI in patients requiring chronic urinary catheterization. **(No recommendation/unresolved issue)** (Key Question 2C)

Catheter Materials

- M. If the CAUTI rate is not decreasing after implementing a comprehensive strategy to reduce rates of CAUTI, consider using antimicrobial/antiseptic-impregnated catheters. The comprehensive strategy should include, at a minimum, the high priority recommendations for urinary catheter use, aseptic insertion, and maintenance (see Section III. Implementation and Audit). **(Category IB)** (Key Question 2B)
 1. Further research is needed on the effect of antimicrobial/antiseptic-impregnated catheters in reducing the risk of symptomatic UTI, their inclusion among the primary interventions, and the patient populations most likely to benefit from these catheters. **(No recommendation/unresolved issue)** (Key Question 2B)
- N. Hydrophilic catheters might be preferable to standard catheters for patients requiring intermittent catheterization. **(Category II)** (Key Question 2B)
- O. Silicone might be preferable to other catheter materials to reduce the risk of encrustation in long-term catheterized patients who have frequent obstruction. **(Category II)** (Key Question 3)
- P. Further research is needed to clarify the benefit of catheter valves in reducing the risk of CAUTI and other urinary complications. **(No recommendation/unresolved issue)** (Key Question 2B)

Management of Obstruction

- Q. If obstruction occurs and it is likely that the catheter material is contributing to obstruction, change the catheter. **(Category IB)**
- R. Further research is needed on the benefit of irrigating the catheter with acidifying solutions or use of oral urease inhibitors in long-term catheterized patients who have frequent catheter obstruction. **(No recommendation/unresolved issue)** (Key Question 3)

- S. Further research is needed on the use of a portable ultrasound device to evaluate for obstruction in patients with indwelling catheters and low urine output. **(No recommendation/unresolved issue)** (Key Question 2C)
- T. Further research is needed on the use of methenamine to prevent encrustation in patients requiring chronic indwelling catheters who are at high risk for obstruction. **(No recommendation/unresolved issue)** (Key Question 2C)

Specimen Collection

- U. Obtain urine samples aseptically. **(Category IB)**
 - 1. If a small volume of fresh urine is needed for examination (i.e., urinalysis or culture), aspirate the urine from the needleless sampling port with a sterile syringe/cannula adapter after cleansing the port with a disinfectant. **(Category IB)**
 - 2. Obtain large volumes of urine for special analyses (not culture) aseptically from the drainage bag. **(Category IB)**

Spatial Separation of Catheterized Patients

- V. Further research is needed on the benefit of spatial separation of patients with urinary catheters to prevent transmission of pathogens colonizing urinary drainage systems. **(No recommendation/unresolved issue)** (Key Question 2D)

IV. Quality Improvement Programs

- A. Implement quality improvement (QI) programs or strategies to enhance appropriate use of indwelling catheters and to reduce the risk of CAUTI based on a facility risk assessment. **(Category IB)** (Key Question 2D)

The purposes of QI programs should be: 1) to assure appropriate utilization of catheters 2) to identify and remove catheters that are no longer needed (e.g., daily review of their continued need) and 3) to ensure adherence to hand hygiene and proper care of catheters. Examples of programs that have been demonstrated to be effective include:

- 1. A system of alerts or reminders to identify all patients with urinary catheters and assess the need for continued catheterization
- 2. Guidelines and protocols for nurse-directed removal of unnecessary urinary catheters
- 3. Education and performance feedback regarding appropriate use, hand hygiene, and catheter care
- 4. Guidelines and algorithms for appropriate peri-operative catheter management, such as:

- a. Procedure-specific guidelines for catheter placement and postoperative catheter removal
- b. Protocols for management of postoperative urinary retention, such as nurse-directed use of intermittent catheterization and use of bladder ultrasound scanners

V. Administrative Infrastructure

A. Provision of guidelines

1. Provide and implement evidence-based guidelines that address catheter use, insertion, and maintenance. **(Category IB)**
 - a. Consider monitoring adherence to facility-based criteria for acceptable indications for indwelling urinary catheter use. **(Category II)**

B. Education and Training

1. Ensure that healthcare personnel and others who take care of catheters are given periodic in-service training regarding techniques and procedures for urinary catheter insertion, maintenance, and removal. Provide education about CAUTI, other complications of urinary catheterization, and alternatives to indwelling catheters. **(Category IB)**
2. When feasible, consider providing performance feedback to these personnel on what proportion of catheters they have placed meet facility-based criteria and other aspects related to catheter care and maintenance. **(Category II)**

C. Supplies

1. Ensure that supplies necessary for aseptic technique for catheter insertion are readily available. **(Category IB)**

D. System of documentation

1. Consider implementing a system for documenting the following in the patient record: indications for catheter insertion, date and time of catheter insertion, individual who inserted catheter, and date and time of catheter removal. **(Category II)**
 - a. Ensuring that documentation is accessible in the patient record and recorded in a standard format for data collection and quality improvement purposes is suggested. Electronic documentation that is searchable is preferable. **(Category II)**

E. Surveillance resources

1. If surveillance for CAUTI is performed, ensure that there are sufficient trained personnel and technology resources to support surveillance for urinary catheter use and outcomes. **(Category IB)**

VI. Surveillance

- A. Consider surveillance for CAUTI when indicated by facility-based risk assessment. **(Category II)**
 - 1. Identify the patient groups or units on which to conduct surveillance based on frequency of catheter use and potential risk of CAUTI.
- B. Use standardized methodology for performing CAUTI surveillance. **(Category IB)**
 - 1. Examples of metrics that should be used for CAUTI surveillance include:
 - a. Number of CAUTI per 1000 catheter-days
 - b. Number of bloodstream infections secondary to CAUTI per 1000 catheter-days
 - c. Catheter utilization ratio: (urinary catheter days/patient days) x 100
 - 2. Use CDC/NHSN criteria for identifying patients who have symptomatic UTI (SUTI) (numerator data) (see NHSN Patient Safety Manual: <http://www.cdc.gov/nhsn/library.html>).
 - 3. For more information on metrics, please see the U.S. Department of Health & Human Services (HHS) Action Plan to Prevent Healthcare-Associated Infections: <http://www.hhs.gov/ophis/initiatives/hai/infection.html>.
- C. Routine screening of catheterized patients for asymptomatic bacteriuria (ASB) is not recommended. **(Category II)** (Key Question 2D)
- D. When performing surveillance for CAUTI, consider providing regular (e.g., quarterly) feedback of unit-specific CAUTI rates to nursing staff and other appropriate clinical care staff. **(Category II)** (Key Question 2D)

III. Implementation and Audit

Prioritization of Recommendations

In this section, the recommendations considered essential for *all* healthcare facilities caring for patients requiring urinary catheterization are organized into modules in order to provide more guidance to facilities on implementation of these guidelines. The high-priority recommendations were chosen by a consensus of experts based on strength of recommendation as well as on the likely impact of the strategy in preventing CAUTI. The administrative functions and infrastructure listed above in the summary of recommendations are necessary to accomplish the high priority recommendations and are therefore critical to the success of a prevention program. In addition, quality improvement programs should be implemented as an active approach to accomplishing these recommendations and when process and outcome measure goals are not being met based on internal reporting.

Priority Recommendations for Appropriate Urinary Catheter Use (Module 1)

- Insert catheters only for appropriate indications (see Table 2), and leave in place only as long as needed. **(Category IB)**
 - Avoid use of urinary catheters in patients and nursing home residents for management of incontinence. **(Category IB)**
 - For operative patients who have an indication for an indwelling catheter, remove the catheter as soon as possible postoperatively, preferably within 24 hours, unless there are appropriate indications for continued use. **(Category IB)**

Priority Recommendations for Aseptic Insertion of Urinary Catheters (Module 2)

- Ensure that only properly trained persons (e.g., hospital personnel, family members, or patients themselves) who know the correct technique of aseptic catheter insertion and maintenance are given this responsibility. **(Category IB)**
- In the acute care hospital setting, insert catheters using aseptic technique and sterile equipment. **(Category IB)**

Priority Recommendations for Proper Urinary Catheter Maintenance (Module 3)

- Following aseptic insertion of the urinary catheter, maintain a closed drainage system **(Category IB)**
- Maintain unobstructed urine flow. **(Category IB)**

Performance Measures

- A. Internal Reporting. Consider reporting both process and outcome measures to senior administrative, medical, and nursing leadership and clinicians who care for patients at risk for CAUTI. **(Category II)**
 1. Examples of process measures:
 - a) Compliance with educational program: Calculate percent of personnel who have proper training:
 - Numerator: number of personnel who insert urinary catheters and who have proper training
 - Denominator: number of personnel who insert urinary catheters
 - Standardization factor: 100 (i.e., multiply by 100 so that measure is expressed as a percentage)

- b) Compliance with documentation of catheter insertion and removal dates: Conduct random audits of selected units and calculate compliance rate:
 - Numerator: number of patients on unit with catheters with proper documentation of insertion and removal dates
 - Denominator: number of patients on the unit with a catheter in place at some point during admission
 - Standardization factor: 100 (i.e., multiply by 100 so that measure is expressed as a percentage)
 - c) Compliance with documentation of indication for catheter placement: Conduct random audits of selected units and calculate compliance rate
 - Numerator: number of patients on unit with catheters with proper documentation of indication
 - Denominator: number of patients on the unit with catheter in place
 - Standardization factor: 100 (i.e., multiply by 100 so that measure is expressed as a percentage)
2. Recommended outcome measures:
- a) Rates of CAUTI: Use NHSN definitions (see <http://www.cdc.gov/nhsn/library.html>). Measurement of rates allows an individual facility to gauge the longitudinal impact of implementation of prevention strategies:
 - Numerator: number of CAUTIs in each location monitored
 - Denominator: total number of urinary catheter-days for all patients that have an indwelling urinary catheter in each location monitored
 - Standardization factor: Multiply by 1000 so that the measure is expressed as cases per 1000 catheter-days
 - b) Rate of bloodstream infections secondary to CAUTI: Use NHSN definitions for laboratory-confirmed bloodstream infection, available at <http://www.cdc.gov/nhsn/library.html>.
 - Numerator: number of episodes of bloodstream infections secondary to CAUTI
 - Denominator: total number of urinary catheter-days for all patients that have an indwelling urinary catheter in each location monitored
 - Standardization factor: Multiply by 1000 so that the measure is expressed as cases per 1000 catheter-days
- B. External Reporting. Current NHSN definitions for CAUTI were developed for monitoring of rates within a facility; however, reporting of CAUTI rates for facility-to-facility comparison might be requested by state requirements and external quality initiatives.

IV. Recommendations for Further Research

Our literature review revealed that many of the studies addressing strategies to prevent CAUTI were not of sufficient quality to allow firm conclusions regarding the benefit of certain interventions. Future studies of CAUTI prevention should:

- 1) Be primary analytic research (i.e. systematic reviews, meta-analyses, interventional studies, and observational studies [cohort, case-control, analytic cross-sectional studies])
- 2) Evaluate clinically relevant outcomes (e.g., SUTI, bloodstream infections secondary to CAUTI)
- 3) Adjust for confounders as needed using multivariable analyses
- 4) Stratify outcomes by patient populations at risk for CAUTI
- 5) Ensure adequate statistical power to detect differences

The following is a compilation of recommendations for further research:

1. Catheter materials
 - a. Antimicrobial and antiseptic-impregnated catheters
 - i. Effect of catheters on reducing the risk of SUTI and other clinically significant outcomes
 - ii. Patient populations most likely to benefit
 - iii. Incidence of antimicrobial resistance in urinary pathogens
 - iv. Role of bacterial biofilms in the pathogenesis of CAUTI
 - b. Standard catheters
 - i. Optimal materials for reducing the risk of CAUTI and other urethral complications
2. Appropriate urinary catheter use
 - a. Incontinent patients
 - i. Risks and benefits of periodic (e.g., nighttime) use of external catheters
 - ii. Risk of local complications (e.g., skin maceration, phimosis) with the use of external catheters
 - iii. Appropriate use of urinary catheters to manage sacral or perineal wounds
 - b. Appropriate indications for continued use in postoperative patients and associated risks
3. Antiseptics
 - a. Use of antiseptic vs. sterile solutions for periurethral cleaning prior to catheter insertion
 - b. Use of antiseptics (e.g., methenamine) to prevent CAUTI
4. Alternatives to indwelling urethral catheters and bag drainage
 - a. Risks and benefits of suprapubic catheters as an alternative to chronic indwelling urethral catheters
 - b. Use of a urethral stent as an alternative to an indwelling catheter in selected patients with bladder outlet obstruction
 - c. Use of catheter valves in reducing the risk of CAUTI and other urinary complications
 - d. Other alternative methods of urinary drainage

5. Optimal methods for preventing encrustation in long-term catheterized patients who have frequent obstruction
 - a. Optimal catheter materials
 - b. Irrigation with acidifying solutions or oral urease inhibitors
 - c. Use of methenamine

6. Other prevention measures
 - a. Use of portable ultrasound in patients with low-urine output to reduce unnecessary catheter insertions or irrigations (in catheterized patients)
 - b. Use of new prevention strategies such as bacterial interference in patients requiring chronic catheterization
 - c. Optimal cleaning and storage procedures (e.g., wet vs. dry storage) for catheters used for clean intermittent catheterization

7. Prevention of transmission
 - a. Spatial separation of patients with urinary catheters (in the absence of epidemic spread or frequent cross-infection) to prevent transmission of pathogens colonizing urinary drainage systems

V. Background

Urinary tract infections are the most common type of healthcare-associated infection, accounting for more than 30% of infections reported by acute care hospitals.¹⁹ Virtually all healthcare-associated UTIs are caused by instrumentation of the urinary tract. Catheter-associated urinary tract infection (CAUTI) has been associated with increased morbidity, mortality, hospital cost, and length of stay.⁶⁻⁹ In addition, bacteriuria commonly leads to unnecessary antimicrobial use, and urinary drainage systems are often reservoirs for multidrug-resistant bacteria and a source of transmission to other patients.^{10,11}

Definitions

An indwelling urinary catheter is a drainage tube that is inserted into the urinary bladder through the urethra, is left in place, and is connected to a closed collection system. Alternative methods of urinary drainage may be employed in some patients. Intermittent (“in-and-out”) catheterization involves brief insertion of a catheter into the bladder through the urethra to drain urine at intervals. An external catheter is a urine containment device that fits over or adheres to the genitalia and is attached to a urinary drainage bag. The most commonly used external catheter is a soft flexible sheath that fits over the penis (“condom” catheter). A suprapubic catheter is surgically inserted into the bladder through an incision above the pubis.

Although UTIs associated with alternative urinary drainage systems are considered device-associated, CAUTI rates reported to the National Healthcare Safety Network (NHSN) only refer to those associated with indwelling urinary catheters. NHSN has recently revised the UTI surveillance definition criteria. Among the changes are removal of the asymptomatic bacteriuria (ASB) criterion and refinement of the criteria for defining symptomatic UTI (SUTI). The time period for follow-up surveillance after catheter removal also has been shortened from 7 days to 48 hours to align with other device-associated infections. The new UTI criteria, which took effect in January 2009, can be found in the NHSN Patient Safety Manual (<http://www.cdc.gov/nhsn/library.html>).

The limitations and heterogeneity of definitions of CAUTI used in various studies present major challenges in appraising the quality of evidence in the CAUTI literature. Study investigators have used numerous different definitions for CAUTI outcomes, ranging from simple bacteriuria at a range of concentrations to, less commonly, symptomatic infection defined by combinations of bacteriuria and various signs and symptoms. Furthermore, most studies that used CDC/NHSN definitions for CAUTI did not distinguish between SUTI and ASB in their analyses.³⁰ The heterogeneity of definitions used for CAUTI may reduce the quality of evidence for a given intervention and often precludes meta-analyses.

The clinical significance of ASB in catheterized patients is undefined. Approximately 75% to 90% of patients with ASB do not develop a systemic inflammatory response or other signs or symptoms to suggest infection.^{6,31} Monitoring and treatment of ASB is also not an effective prevention measure for SUTI, as most cases of SUTI are not preceded by bacteriuria for more than a day.²⁵ Treatment of ASB has not been shown to be clinically beneficial and is associated with the selection of antimicrobial-resistant organisms.

Epidemiology

Between 15% and 25% of hospitalized patients may receive short-term indwelling urinary catheters.^{12,13} In many cases, catheters are placed for inappropriate indications, and healthcare providers are often unaware that their patients have catheters, leading to prolonged, unnecessary use.¹⁴⁻¹⁶ In acute care hospitals reporting to NHSN in 2006, pooled mean urinary catheter utilization ratios in ICU and non-ICU areas ranged from 0.23-0.91 urinary catheter-days/patient-days.¹⁷ While the numbers of units reporting were small, the highest ratios were in trauma ICUs and the lowest in inpatient medical/surgical wards. The overall prevalence of long-term indwelling urethral catheterization use is unknown. The prevalence of urinary catheter use in residents in long-term care facilities in the United States is on the order of 5%, representing approximately 50,000 residents with catheters at any given time.¹⁸ This number appears to be declining over time, likely because of federally mandated nursing home quality measures. However, the high prevalence of urinary catheters in patients transferred to skilled nursing facilities suggests that acute care hospitals should focus more efforts on removing unnecessary catheters prior to transfer.¹⁸

Reported rates of UTI among patients with urinary catheters vary substantially. National data from NHSN acute care hospitals in 2006 showed a range of pooled mean CAUTI rates of 3.1-7.5 infections per 1000 catheter-days.¹⁷ The highest rates were in burn ICUs, followed by inpatient medical wards and neurosurgical ICUs, although these sites also had the fewest numbers of locations reporting. The lowest rates were in medical/surgical ICUs.

Although morbidity and mortality from CAUTI is considered to be relatively low compared to other HAIs, the high prevalence of urinary catheter use leads to a large cumulative burden of infections with resulting infectious complications and deaths. An estimate of annual incidence of HAIs and mortality in 2002, based on a broad survey of US hospitals, found that urinary tract infections made up the highest number of infections (> 560,000) compared to other HAIs, and attributable deaths from UTI were estimated to be over 13,000 (mortality rate 2.3%).¹⁹ And while fewer than 5% of bacteriuric cases develop bacteremia,⁶ CAUTI is the leading cause of secondary nosocomial bloodstream infections; about 17% of hospital-acquired bacteremias are from a urinary source, with an associated mortality of approximately 10%.²⁰ In the nursing home setting, bacteremias are most commonly caused by UTIs, the majority of which are catheter-related.²¹

An estimated 17% to 69% of CAUTI may be preventable with recommended infection control measures, which means that up to 380,000 infections and 9000 deaths related to CAUTI per year could be prevented.²²

Pathogenesis and Microbiology

The source of microorganisms causing CAUTI can be endogenous, typically via meatal, rectal, or vaginal colonization, or exogenous, such as via contaminated hands of healthcare personnel or equipment. Microbial pathogens can enter the urinary tract either by the extraluminal route, via migration along the outside of the catheter in the periurethral mucous sheath, or by the intraluminal route, via movement along the internal lumen of the catheter from a contaminated collection bag or catheter-drainage tube junction. The relative contribution of each route in the pathogenesis of CAUTI is not well known. The marked reduction in risk of bacteriuria with the introduction of the sterile, closed urinary drainage system in the 1960's²³ suggests the importance of the intraluminal route. However, even with the closed drainage system,

bacteriuria inevitably occurs over time either via breaks in the sterile system or via the extraluminal route.²⁴ The daily risk of bacteriuria with catheterization is 3% to 10%,^{25,26} approaching 100% after 30 days, which is considered the delineation between short and long-term catheterization.²⁷

Formation of biofilms by urinary pathogens on the surface of the catheter and drainage system occurs universally with prolonged duration of catheterization.²⁸ Over time, the urinary catheter becomes colonized with microorganisms living in a sessile state within the biofilm, rendering them resistant to antimicrobials and host defenses and virtually impossible to eradicate without removing the catheter. The role of bacteria within biofilms in the pathogenesis of CAUTI is unknown and is an area requiring further research.

The most frequent pathogens associated with CAUTI (combining both ASB and SUTI) in hospitals reporting to NHSN between 2006-2007 were *Escherichia coli* (21.4%) and *Candida* spp (21.0%), followed by *Enterococcus* spp (14.9%), *Pseudomonas aeruginosa* (10.0%), *Klebsiella pneumoniae* (7.7%), and *Enterobacter* spp (4.1%). A smaller proportion was caused by other gram-negative bacteria and *Staphylococcus* spp⁵.

Antimicrobial resistance among urinary pathogens is an ever increasing problem. About a quarter of *E. coli* isolates and one third of *P. aeruginosa* isolates from CAUTI cases were fluoroquinolone-resistant. Resistance of gram-negative pathogens to other agents, including third-generation cephalosporins and carbapenems, was also substantial⁵. The proportion of organisms that were multidrug-resistant, defined by non-susceptibility to all agents in 4 classes, was 4% of *P. aeruginosa*, 9% of *K. pneumoniae*, and 21% of *Acinetobacter baumannii*.²⁹

VI. Scope and Purpose

This guideline updates and expands the original CDC Guideline for Prevention of CAUTI published in 1981. The revised guideline addresses the prevention of CAUTI for patients in need of either short- or long-term (i.e., > 30 days) urinary catheterization in any type of healthcare facility and evaluates evidence for alternative methods of urinary drainage, including intermittent catheterization, external catheters, and suprapubic catheters. The guideline also includes specific recommendations for implementation, performance measurement, and surveillance. Recommendations for further research are also provided to address the knowledge gaps in CAUTI prevention identified during the literature review.

To evaluate the evidence on preventing CAUTI, we examined data addressing three key questions and related subquestions:

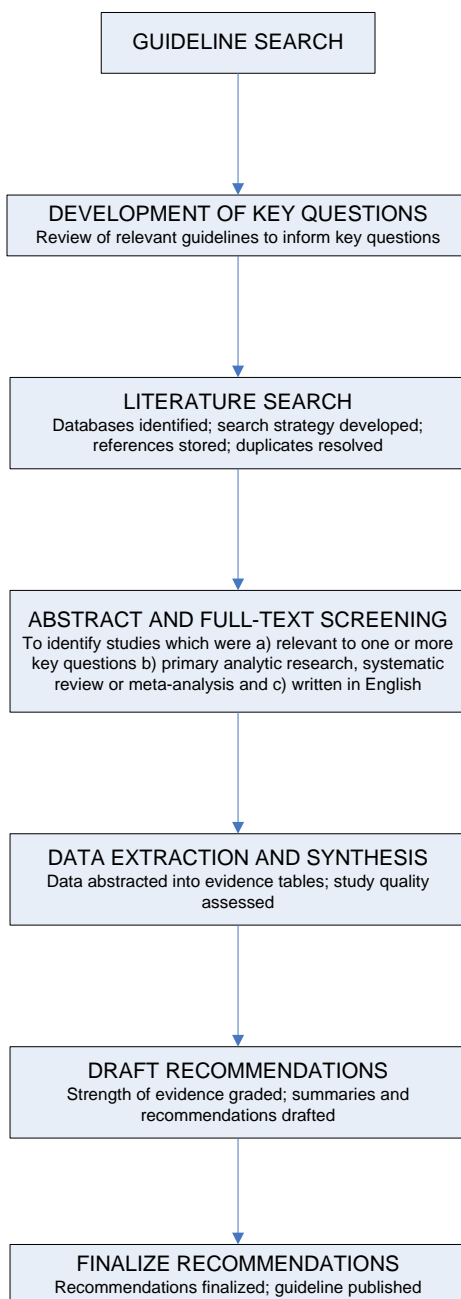
1. Who should receive urinary catheters?
 - A. When is urinary catheterization necessary?
 - B. What are the risk factors for CAUTI?
 - C. What populations are at highest risk of mortality from catheters?
2. For those who may require urinary catheters, what are the best practices?
Specifically, what are the risks and benefits associated with:
 - A. Different approaches to catheterization?
 - B. Different catheters or collecting systems?
 - C. Different catheter management techniques?
 - D. Different systems interventions (i.e., quality improvement programs)?
3. What are the best practices for preventing UTI associated with obstructed urinary catheters?

This document is intended for use by infection prevention staff, healthcare epidemiologists, healthcare administrators, nurses, other healthcare providers, and persons responsible for developing, implementing, and evaluating infection prevention and control programs for healthcare settings across the continuum of care. The guideline can also be used as a resource for societies or organizations that wish to develop more detailed implementation guidance for prevention of CAUTI.

VII. Methods

This guideline was based on a targeted systematic review of the best available evidence on CAUTI prevention. We used the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach³²⁻³⁴ to provide explicit links between the available evidence and the resulting recommendations. Our guideline development process is outlined in *Figure 1*.

Figure 1. The Guideline Development Process



Development of Key Questions

We first conducted an electronic search of the National Guideline Clearinghouse® (Agency for Healthcare Research and Quality), Medline® (National Library of Medicine) using the Ovid® Platform (Ovid Technologies, Wolters Kluwer, New York, NY), the Cochrane® Health Technology Assessment Database (Cochrane Collaboration, Oxford, UK), the NIH Consensus Development Program, and the United States Preventive Services Task Force database for existing national and international guidelines relevant to CAUTI. The strategy used for the guideline search and the search results can be found in [Appendix 1A](#). A preliminary list of key questions was developed from a review of the relevant guidelines identified in the search.^{1,35,36} Key questions were finalized after vetting them with a panel of content experts and HICPAC members.

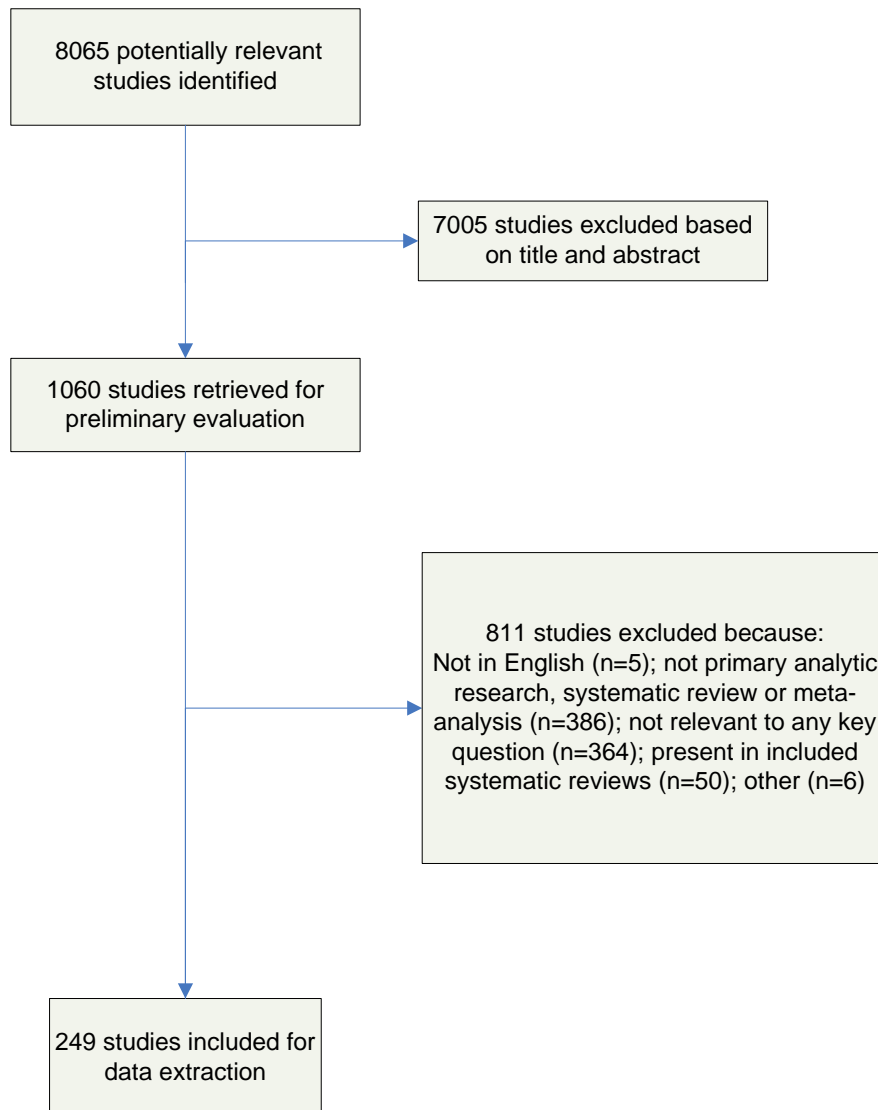
Literature Search

Following the development of the key questions, search terms were developed for identifying literature relevant to the key questions. For the purposes of quality assurance, we compared these terms to those used in relevant seminal studies and guidelines. These search terms were then incorporated into search strategies for the relevant electronic databases. Searches were performed in Medline® (National Library of Medicine) using the Ovid® Platform (Ovid Technologies, Wolters Kluwer, New York, NY), EMBASE® (Elsevier BV, Amsterdam, Netherlands), CINAHL® (Ebsco Publishing, Ipswich, MA) and Cochrane® (Cochrane Collaboration, Oxford, UK) (all databases were searched in July 2007), and the resulting references were imported into a reference manager, where duplicates were resolved. For Cochrane reviews ultimately included in our guideline, we checked for updates in July 2008. The detailed search strategy used for identifying primary literature and the results of the search can be found in [Appendix 1B](#).

Study Selection

Titles and abstracts from references were screened by a single author (C.V.G, R.K.A., or D.A.P.) and the full text articles were retrieved if they were 1) relevant to one or more key questions, 2) primary analytic research, systematic reviews or meta-analyses, and 3) written in English. Likewise, the full-text articles were screened by a single author (C.V.G. or D.A.P.) using the same criteria, and included studies underwent a second review for inclusion by another author (R.K.A.). Disagreements were resolved by the remaining authors. The results of this process are depicted in *Figure 2*.

Figure 2: Results of the Study Selection Process



Data Extraction and Synthesis

Data on the study author, year, design, objective, population, setting, sample size, power, follow-up, and definitions and results of clinically relevant outcomes were extracted into evidence tables ([Appendix 2](#)). Three evidence tables were developed, each of which represented one of our key questions. Studies were extracted into the most relevant evidence table. Then, studies were organized by the common themes that emerged within each evidence table. Data were extracted by one author (R.K.A.) and cross-checked by another (C.V.G.). Disagreements were resolved by the remaining authors. Data and analyses were extracted as originally presented in the included studies. Meta-analyses were performed only where their use was deemed critical to a recommendation, and only in circumstances where multiple studies with sufficiently homogenous populations, interventions, and outcomes could be analyzed. Systematic reviews were included in our review. To avoid duplication of data, we excluded primary studies if they were also included in a systematic review captured by our search. The only exception to this was if the primary study also addressed a relevant question that was outside the scope of the included systematic review. Before exclusion, data from the primary studies that we originally captured were abstracted into the evidence tables and reviewed. We also excluded systematic reviews that analyzed primary studies that were fully captured in a more recent systematic review. The only exception to this was if the older systematic review also addressed a relevant question that was outside the scope of the newer systematic review. To ensure that all relevant studies were captured in the search, the bibliography was vetted by a panel of clinical experts.

Grading of Evidence

First, the quality of each study was assessed using scales adapted from existing methodology checklists, and scores were recorded in the evidence tables. [Appendix 3](#) includes the sets of questions we used to assess the quality of each of the major study designs. Next, the quality of the evidence base was assessed using methods adapted from the GRADE Working Group.³² Briefly, GRADE tables were developed for each of the interventions or questions addressed within the evidence tables. Included in the GRADE tables were the intervention of interest, any outcomes listed in the evidence tables that were judged to be clinically important, the quantity and type of evidence for each outcome, the relevant findings, and the GRADE of evidence for each outcome, as well as an overall GRADE of the evidence base for the given intervention or question. The initial GRADE of evidence for each outcome was deemed high if the evidence base included a randomized controlled trial (RCT) or a systematic review of RCTs, low if the evidence base included only observational studies, or very low if the evidence base consisted only of uncontrolled studies. The initial GRADE could then be modified by eight criteria.³⁴ Criteria which could decrease the GRADE of an evidence base included quality, consistency, directness, precision, and publication bias. Criteria that could increase the GRADE included a large magnitude of effect, a dose-response gradient, or inclusion of unmeasured confounders that would increase the magnitude of effect ([Table 3](#)). GRADE definitions are as follows:

1. High - further research is very unlikely to change confidence in the estimate of effect
2. Moderate - further research is likely to affect confidence in the estimate of effect and may change the estimate
3. Low - further research is very likely to affect confidence in the estimate of effect and is likely to change the estimate
4. Very low - any estimate of effect is very uncertain

After determining the GRADE of the evidence base for each outcome of a given intervention or question, we calculated the overall GRADE of the evidence base for that intervention or question. The overall GRADE was based on the lowest GRADE for the outcomes deemed critical to making a recommendation.

Table 3. Rating the Quality of Evidence Using the GRADE Approach

Type of Evidence	Initial Grade	Criteria to Decrease Grade	Criteria to Increase Grade	Overall Quality Grade
RCT	High	<u>Quality</u> Serious (-1 grade) or very serious (-2 grades) limitation to study quality	<u>Strong association</u> Strong (+1 grade) or very strong evidence of association (+2 grades)	High
				Moderate
Observational study	Low	<u>Consistency</u> Important inconsistency (-1 grade)	<u>Dose-response</u> Evidence of a dose-response gradient (+1 grade)	Low
Any other evidence (e.g., expert opinion)	Very low	<u>Directness</u> Some (-1 grade) or major (-2 grades) uncertainty about directness	<u>Unmeasured Confounders</u> Inclusion of unmeasured confounders increases the magnitude of effect (+1 grade)	Very low
		<u>Precision</u> Imprecise or sparse data (-1 grade)		
		<u>Publication bias</u> High risk of bias (-1 grade)		

Formulating Recommendations

Narrative evidence summaries were then drafted by the working group using the evidence and GRADE tables. One summary was written for each theme that emerged under each key question. The working group then used the narrative evidence summaries to develop guideline recommendations. Factors determining the strength of a recommendation included 1) the values and preferences used to determine which outcomes were "critical," 2) the harms and benefits that result from weighing the "critical" outcomes, and 3) the overall GRADE of the evidence base for the given intervention or question (Table 4).³³ If weighing the "critical outcomes" for a given intervention or question resulted in a "net benefit" or a "net harm," then a "Category I Recommendation" was formulated to strongly recommend for or against the given intervention respectively. If weighing the "critical outcomes" for a given intervention or question resulted in a "trade off" between benefits and harms, then a "Category II Recommendation" was formulated to recommend that providers or institutions consider the intervention when deemed appropriate. If weighing the "critical outcomes" for a given intervention or question resulted in

an "uncertain trade off" between benefits and harms, then a "No Recommendation" was formulated to reflect this uncertainty.

HICPAC Recommendation	Weighing Benefits and Harms for Critical Outcomes	Quality of Evidence
STRONG (I)	Interventions with net benefits or net harms	IA – High to Moderate IB – Low or Very Low (Accepted Practice) IC – High to Very Low (Regulatory)
WEAK (II)	Interventions with trade offs between benefits and harms	High to Very Low
No recommendation/ unresolved issue	Uncertain trade offs between benefits and harms	Low to Very Low

For Category I recommendations, levels A and B represent the quality of the evidence underlying the recommendation, with A representing high to moderate quality evidence and B representing low quality evidence or, in the case of an established standard (e.g., aseptic technique, education and training), very low quality to no evidence based on our literature review. For IB recommendations, although there may be low to very low quality or even no available evidence directly supporting the benefits of the intervention, the theoretical benefits are clear, and the theoretical risks are marginal. Level C represents practices required by state or federal regulation, regardless of the quality of evidence. It is important to note that the strength of a Category IA recommendation is equivalent to that of a Category IB or IC recommendation; it is only the quality of the evidence underlying the IA recommendation that makes it different from a IB.

In some instances, multiple recommendations emerged from a single narrative evidence summary. The new HICPAC categorization scheme for recommendations is provided in [Table 1](#), which is reproduced below.

Category IA	A strong recommendation supported by high to moderate quality evidence suggesting net clinical benefits or harms
Category IB	A strong recommendation supported by low quality evidence suggesting net clinical benefits or harms or an accepted practice (e.g., aseptic technique) supported by low to very low quality evidence
Category IC	A strong recommendation required by state or federal regulation.
Category II	A weak recommendation supported by any quality evidence suggesting a trade off between clinical benefits and harms
No recommendation/ unresolved issue	Unresolved issue for which there is low to very low quality evidence with uncertain trade offs between benefits and harms

Category I recommendations are defined as strong recommendations with the following implications:

1. For patients: Most people in the patient's situation would want the recommended course of action and only a small proportion would not; request discussion if the intervention is not offered.
2. For clinicians: Most patients should receive the recommended course of action.
3. For policymakers: The recommendation may be adopted as a policy.

Category II recommendations are defined as weak recommendations with the following implications:

1. For patients: Most people in the patient's situation would want the recommended course of action, but many would not.
2. For clinicians: Different choices will be appropriate for different patients, and clinicians must help each patient to arrive at a management decision consistent with her or his values and preferences.
3. For policymakers: Policy making will require substantial debate and involvement of many stakeholders.

It should be noted that Category II recommendations are discretionary for the individual institution and are not intended to be enforced.

The wording of each recommendation was carefully selected to reflect the recommendation's strength. In most cases, we used the active voice when writing Category I recommendations - the strong recommendations. Phrases like "do" or "do not" and verbs without auxiliaries or conditionals were used to convey certainty. We used a more passive voice when writing Category II recommendations - the weak recommendations. Words like "consider" and phrases like "is preferable," "is suggested," "is not suggested," or "is not recommended" were chosen to reflect the lesser certainty of the Category II recommendations. Rather than a simple statement of fact, each recommendation is actionable, describing precisely a proposed action to take.

The category "No recommendation/unresolved issue" was most commonly applied to situations where either 1) the overall quality of the evidence base for a given intervention was low to very low and there was no consensus on the benefit of the intervention or 2) there was no published evidence on outcomes deemed critical to weighing the risks and benefits of a given intervention. If the latter was the case, those critical outcomes will be noted at the end of the relevant evidence summary.

Our evidence-based recommendations were cross-checked with those from guidelines identified in our original systematic search. Recommendations from previous guidelines for topics not directly addressed by our systematic review of the evidence were included in our "Summary of Recommendations" if they were deemed critical to the target users of this guideline. Unlike recommendations informed by our literature search, these recommendations are not linked to a key question. These recommendations were agreed upon by expert consensus and are designated either IB if they represent a strong recommendation based on accepted practices (e.g., aseptic technique) or II if they are a suggestion based on a probable net benefit despite limited evidence.

All recommendations were approved by HICPAC. Recommendations focused only on efficacy, effectiveness, and safety. The optimal use of these guidelines should include a consideration of the costs relevant to the local setting of guideline users.

Reviewing and Finalizing the Guideline

After a draft of the tables, narrative summaries, and recommendations was completed, the working group shared the draft with the expert panel for in-depth review. While the expert panel was reviewing this draft, the working group completed the remaining sections of the guideline, including the executive summary, background, scope and purpose, methods, summary of recommendations, and recommendations for guideline implementation, audit, and further research. The working group then made revisions to the draft based on feedback from members of the expert panel and presented the entire draft guideline to HICPAC for review. The guideline was then posted on the Federal Register for public comment. After a period of public comment, the guideline was revised accordingly, and the changes were reviewed and voted on by HICPAC. The final guideline was cleared internally by CDC and published and posted on the HICPAC website.

Updating the Guideline

Future revisions to this guideline will be dictated by new research and technological advancements for preventing CAUTI and will occur at the request of HICPAC.

VIII. Evidence Review

Q1. Who should receive urinary catheters?

To answer this question, we focused on three subquestions: A) When is urinary catheterization necessary? B) What are the risk factors for CAUTI? and C) What populations are at highest risk of mortality from urinary catheters?

Q1A. When is urinary catheterization necessary?

The available data examined five main populations. In all populations, we considered CAUTI outcomes as well as other outcomes we deemed critical to weighing the risks and benefits of catheterization. The evidence for this question consists of 1 systematic review,³⁷ 9 RCTs,³⁸⁻⁴⁶ and 12 observational studies.⁴⁷⁻⁵⁸ The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 1A.

For *operative patients*, low-quality evidence suggested a benefit of avoiding urinary catheterization.^{37-44,47-49} This was based on a decreased risk of bacteriuria/unspecified UTI, no effect on bladder injury, and increased risk of urinary retention in patients without catheters. Urinary retention in patients without catheters was specifically seen following urogenital surgeries. The most common surgeries studied were urogenital, gynecological, laparoscopic, and orthopedic surgeries. Our search did not reveal data on the impact of catheterization on peri-operative hemodynamic management.

For *incontinent patients*, low-quality evidence suggested a benefit of avoiding urinary catheterization.^{45,50-52} This was based on a decreased risk of both SUTI and bacteriuria/unspecified UTI in male nursing home residents without urinary catheters compared to those with continuous condom catheters. We found no difference in the risk of UTI between having a condom catheter only at night and having no catheter. Our search did not reveal data on the impact of catheterization on skin breakdown.

For *patients with bladder outlet obstruction*, very low-quality evidence suggested a benefit of a urethral stent over an indwelling catheter.⁵³ This was based on a reduced risk of bacteriuria in those receiving a urethral stent. Our search did not reveal data on the impact of catheterization versus stent placement on urinary complications.

For *patients with spinal cord injury*, very low-quality evidence suggested a benefit of avoiding indwelling urinary catheters.^{54,56} This was based on a decreased risk of SUTI and bacteriuria in those without indwelling catheters (including patients managed with spontaneous voiding, clean intermittent catheterization [CIC], and external striated sphincterotomy with condom catheter drainage), as well as a lower risk of urinary complications, including hematuria, stones, and urethral injury (fistula, erosion, stricture).

For *children with myelomeningocele and neurogenic bladder*, very low-quality evidence suggested a benefit of CIC compared to urinary diversion or self voiding.^{46,57,58} This was based on a decreased risk of bacteriuria/unspecified UTI in patients receiving CIC compared to urinary diversion, and a lower risk of urinary tract deterioration (defined by febrile urinary tract infection, vesicoureteral reflux, hydronephrosis, or increases in BUN or serum creatinine) compared to self-voiding and in those receiving CIC early (< 1 year of age) versus late (> 3 years of age).

Evidence Review Table 1A. When is urinary catheterization necessary?

1A.1. Use urinary catheters in operative patients only as necessary, rather than routinely. **(Category IB)**

1A.2. Avoid use of urinary catheters in patients and nursing home residents for management of incontinence. **(Category IB)**

1A.2.a. Further research is needed on periodic (e.g., nighttime) use of external catheters in incontinent patients or residents and the use of catheters to prevent skin breakdown. **(No recommendation/unresolved issue)**

1A.3. Further research is needed on the benefit of using a urethral stent as an alternative to an indwelling catheter in selected patients with bladder outlet obstruction. **(No recommendation/unresolved issue)**

1A.4. Consider alternatives to chronic indwelling catheters, such as intermittent catheterization, in spinal cord injury patients. **(Category II)**

1A.5. Consider intermittent catheterization in children with myelomeningocele and neurogenic bladder to reduce the risk of urinary tract deterioration. **(Category II)**

Q1B. What are the risk factors for CAUTI?

To answer this question, we reviewed the quality of evidence for those risk factors examined in more than one study. We considered the critical outcomes for decision-making to be SUTI and bacteriuria. The evidence for this question consists of 11 RCTs⁵⁹⁻⁶⁹ and 37 observational studies.^{9,50,54,70-103} The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 1B.

For *SUTI*,^{50,54,61,62,74,75,79,83,102,103} low-quality evidence suggested that female sex, older age, prolonged catheterization, impaired immunity, and lack of antimicrobial exposure are risk factors. Very low quality evidence suggested that catheter blockage and low albumin level are also risk factors. For *bacteriuria*,^{9,59-61,63-68,72,73,76-78,82,84-86,89-94,96-100} multiple risk factors were identified; there was high quality evidence for prolonged catheterization and moderate quality evidence for female sex, positive meatal cultures, and lack of antimicrobial exposure. Low-quality evidence also implicated the following risk factors for bacteriuria: older age, disconnection of the drainage system, diabetes, renal dysfunction, higher severity of illness, impaired immunity, placement of the catheter outside of the operating room, lower professional training of the person inserting the catheter, incontinence, and being on an orthopaedic or neurology service. Our search did not reveal data on adverse events and antimicrobial resistance associated with antimicrobial use, although one observational study found that the protective effect of antimicrobials lasted only for the first four days of catheterization, and that antimicrobial exposure led to changes in the epidemiology of bacterial flora in the urine.

Evidence Review Table 1B. What are the risk factors for CAUTI?

1B.1. Following aseptic insertion of the urinary catheter, maintain a closed drainage system. **(Category IB)^a**

1B.2. Insert catheters only for appropriate indications, and leave in place only as long as needed. **(Category IB)^b**

1B.3. Minimize urinary catheter use and duration of use in all patients, particularly those at higher risk for CAUTI such as women, the elderly, and patients with impaired immunity. **(Category IB)**

1B.4. Ensure that only properly trained persons (e.g., hospital personnel, family members, or patients themselves) who know the correct technique of aseptic catheter insertion and maintenance are given this responsibility. **(Category IB)**

1B.5. Maintain unobstructed urine flow. **(Category IB)^c**

^a More data are available under Question 2B.

^b More data are available under Question 2C.

^c More data are available under Question 2D.

Q1C. What populations are at highest risk of mortality from urinary catheters?

To answer this question, we reviewed the quality of evidence for those risk factors examined in more than one study. The evidence for this question consists of 2 observational studies.^{7,74} The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 1C.

Low-quality evidence suggested that older age, higher severity of illness, and being on an internal medicine service compared to a surgical service were independent risk factors for mortality in patients with indwelling urinary catheters. Both studies evaluating these risk factors found the highest risk of mortality in patients over 70 years of age. Low-quality evidence also suggested that CAUTI was a risk factor for mortality in patients with catheters.

Evidence Review Table 1C. What populations are at highest risk of mortality from catheters?

1C.1. Minimize urinary catheter use and duration in all patients, particularly those who may be at higher risk for mortality due to catheterization, such as the elderly and patients with severe illness. **(Category IB)**

Q2. For those who may require urinary catheters, what are the best practices?

To answer this question, we focused on four subquestions: A) What are the risks and benefits associated with different approaches to catheterization?, B) What are the risks and benefits associated with different types of catheters or collecting systems?, C) What are the risks and benefits associated with different catheter management techniques, and D) What are the risks and benefits associated with different systems interventions?

Q2A. What are the risks and benefits associated with different approaches to catheterization?

The available data examined the following comparisons of different catheterization approaches:

- 1) External versus indwelling urethral
- 2) Intermittent versus indwelling urethral
- 3) Intermittent versus suprapubic
- 4) Suprapubic versus indwelling urethral
- 5) Clean intermittent versus sterile intermittent

For all comparisons, we considered SUTI, bacteriuria/unspecified UTI, or combinations of these outcomes depending on availability, as well as other outcomes critical to weighing the risks and benefits of different catheterization approaches. The evidence for this question consists of 6 systematic reviews,^{37,104-108} 16 RCTs,^{62,63,109-122} and 18 observational studies.^{54,73,81,84,123-136} The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 2A

Q2A.1. External versus indwelling urethral

Low-quality evidence suggested a benefit of using external catheters over indwelling urethral catheters in male patients who require a urinary collection device but do not have an indication for an indwelling catheter such as urinary retention or bladder outlet obstruction.^{81,109,123} This was based on a decreased risk of a composite outcome of SUTI, bacteriuria, or death as well as increased patient satisfaction with condom catheters. Differences were most pronounced in men without dementia. Statistically significant differences were not found or reported for the individual CAUTI outcomes or death. Our search did not reveal data on differences in local complications such as skin maceration or phimosis.

Q2A.2. Intermittent versus indwelling urethral

Low-quality evidence suggested a benefit of using intermittent catheterization over indwelling urethral catheters in selected populations.^{84,104-106,110-114,124-126,135,136} This was based on a decreased risk of SUTI and bacteriuria/unspecified UTI but an increased risk of urinary retention in postoperative patients with intermittent catheterization. In one study, urinary retention and bladder distension were avoided by performing catheterization at regular intervals (every 6-8 hrs) until return of voiding. Studies of patients with neurogenic bladder most consistently found a decreased risk of CAUTI with intermittent catheterization. Studies in operative patients whose catheters were removed within 24 hrs of surgery found no differences in bacteriuria with intermittent vs. indwelling catheterization, while studies where catheters were left in for longer durations had mixed results. Our search did not reveal data on differences in patient satisfaction.

Q2A.3. Intermittent versus suprapubic

Very low-quality evidence suggested a benefit of intermittent over suprapubic catheterization in selected populations^{115,116,134-136} based on increased patient acceptability and decreased risk of urinary complications (bladder calculi, vesicoureteral reflux, and upper tract abnormalities). Although we found a decreased risk of bacteriuria/unspecified UTI with suprapubic catheterization, there were no differences in SUTI. The populations studied included women undergoing urogynecologic surgery and spinal cord injury patients.

Q2A.4. *Suprapubic versus indwelling urethral*

Low-quality evidence suggested a benefit of suprapubic catheters over indwelling urethral catheters in selected populations.^{37,62,104,107,108,128-133,135,136} This was based on a decreased risk of bacteriuria/unspecified UTI, recatheterization, and urethral stricture, and increased patient comfort and satisfaction. However, there were no differences in SUTI and an increased risk of longer duration of catheterization with suprapubic catheters. Studies involved primarily postoperative and spinal cord injury patients. Our search did not reveal data on differences in complications related to catheter insertion or the catheter site.

Q2A.5. *Clean intermittent versus sterile intermittent*

Moderate-quality evidence suggested no benefit of using sterile over clean technique for intermittent catheterization.^{63,73,105,117-122} No differences were found in the risk of SUTI or bacteriuria/unspecified UTI. Study populations included nursing home residents and adults and children with neurogenic bladder/spinal cord injury.

Evidence Review Table 2A. What are the risks and benefits associated with different approaches to catheterization?

2A.1. Consider using external catheters as an alternative to indwelling urethral catheters in cooperative male patients without urinary retention or bladder outlet obstruction. **(Category II)**

2A.2. Intermittent catheterization is preferable to indwelling urethral or suprapubic catheters in patients with bladder emptying dysfunction. **(Category II)**

2A.3. If intermittent catheterization is used, perform it at regular intervals to prevent bladder overdistension. **(Category IB)**

2A.4. For operative patients who have an indication for an indwelling catheter, remove the catheter as soon as possible postoperatively, preferably within 24 hours, unless there are appropriate indications for continued use. **(Category IB)***

2A.5. Further research is needed on the risks and benefits of suprapubic catheters as an alternative to indwelling urethral catheters in selected patients requiring short- or long-term catheterization, particularly with respect to complications related to catheter insertion or the catheter site. **(No recommendation/unresolved issue)**

2A.6. In the non-acute care setting, clean (i.e., non-sterile) technique for intermittent catheterization is an acceptable and more practical alternative to sterile technique for patients requiring chronic intermittent catheterization. **(Category IA)**

* More data are available under Question 2C

Q2B. What are the risks and benefits associated with different catheters or collecting systems?

The available data examined the following comparisons between different types of catheters and drainage systems:

1. Antimicrobial/antiseptic catheters vs. standard catheters
 - a. Silver-coated catheters vs. standard catheters
 - b. Nitrofurazone-impregnated catheters vs. standard catheters
2. Hydrophilic catheters vs. standard catheters
3. Closed vs. open drainage systems
4. Complex vs. simple drainage systems
5. Preconnected/sealed junction catheters vs. standard catheters
6. Catheter valves vs. catheter bags

For all comparisons, we considered CAUTI outcomes as well as other outcomes critical to weighing the risks and benefits of different types of catheters or collecting systems. The evidence for this question consists of 5 systematic reviews,^{37,137-140} 17 RCTs,^{64,143-158} 23 observational studies,^{82,86,89,97,159-163, 165-178} and 3 economic analyses.^{179,180,181} The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 2B.

Q2B.1.a. Silver-coated catheters vs. standard catheters

Low-quality evidence suggested a benefit of silver-coated catheters over standard latex catheters.^{37,82,86,137-139,143,159-163, 165,166} This was based on a decreased risk of bacteriuria/unspecified UTI with silver-coated catheters and no evidence of increased urethral irritation or antimicrobial resistance in studies that reported data on microbiological outcomes. Differences were significant for silver alloy-coated catheters but not silver oxide-coated catheters. In a meta-analysis of randomized controlled trials (see [Appendix](#)), silver alloy-coated catheters reduced the risk of asymptomatic bacteriuria compared to standard latex catheters (control latex catheters were either uncoated or coated with hydrogel, Teflon®, or silicone), whereas there were no differences when compared to standard, all silicone catheters. The effect of silver alloy catheters compared to latex catheters was more pronounced when used in patients catheterized <1 week. The results were robust to inclusion or exclusion of non peer-reviewed studies. Only one observational study found a decrease in SUTI with silver alloy-coated catheters.¹⁶⁶ The setting was a burn referral center, where the control catheters were latex, and patients in the intervention group had new catheters placed on admission, whereas the control group did not. Recent observational studies in hospitalized patients found mixed results for bacteriuria/unspecified UTI.

Q2B.1.b. Nitrofurazone-impregnated catheters vs. standard catheters

Low-quality evidence suggested a benefit of nitrofurazone-impregnated catheters in patients catheterized for short periods of time.^{137,138} This was based on a decreased risk of bacteriuria and no evidence of increased antimicrobial resistance in studies that reported microbiological outcomes. Differences were significant in a meta-analysis of three studies examining nitrofurazone-impregnated catheters (only one individual study significant) when duration of catheterization was <1 week. No differences were seen when duration of catheterization was >1 week, although the meta-analysis was borderline significant.

Q2B.2. Hydrophilic catheters vs. standard catheters

Very low-quality evidence suggested a benefit of hydrophilic catheters over standard non-hydrophilic catheters in specific populations undergoing clean intermittent catheterization.^{137,144-148,169} This was based on a decreased risk of SUTI, bacteriuria, hematuria, and pain during insertion, and increased patient satisfaction. Differences in CAUTI outcomes were limited to one study of spinal cord injury patients and one study of patients receiving intravesical immunochemoprophylaxis for bladder cancer, while multiple other studies found no significant differences.

Q2B.3. Closed vs. open drainage systems

Very low-quality evidence suggested a benefit of using a closed rather than open urinary drainage system.^{89,171} This was based on a decreased risk of bacteriuria with a closed drainage system. One study also found a suggestion of a decreased risk of SUTI, bacteremia, and UTI-related mortality associated with closed drainage systems, but differences were not statistically significant. Sterile, continuously closed drainage systems became the standard of care based on an uncontrolled study published in 1966 demonstrating a dramatic reduction in the risk of infection in short-term catheterized patients with the use of a closed system.²³ Recent data also include the finding that disconnection of the drainage system is a risk factor for bacteriuria (Q1B).

Q2B.4. Complex vs. simple drainage systems

Low-quality evidence suggested no benefit of complex closed urinary drainage systems over simple closed urinary drainage systems.^{150-152,154,172,176,177} Although there was a decreased risk of bacteriuria with the complex systems, differences were found only in studies published before 1990, and not in more recent studies. The complex drainage systems studied included various mechanisms for reducing bacterial entry, such as antiseptic-releasing cartridges at the drain port of the urine collection bag; see evidence table for systems evaluated.

Q2B.5. Preconnected/sealed junction catheters vs. standard catheters

Low-quality evidence suggested a benefit of using preconnected catheters with junction seals over catheters with unsealed junctions to reduce the risk of disconnections.^{64,153,156,175} This was based on a decreased risk of SUTI and bacteriuria with preconnected sealed catheters. Studies that found differences had higher rates of CAUTI in the control group than studies that did not find an effect.

Q2B.6. Catheter valves vs. drainage bags

Moderate-quality evidence suggested a benefit of catheter valves over drainage bags in selected patients with indwelling urinary catheters.¹⁴⁰ Catheter valves led to greater patient satisfaction but no differences in bacteriuria/unspecified UTI or pain/bladder spasms. Details regarding the setting for recruitment and follow-up of the patients in the studies were unclear, and the majority of subjects were men. Our search did not reveal data on the effect of catheter valves on bladder function, bladder/urethral trauma, or catheter blockage.

Evidence Review Table 2B. What are the risks and benefits associated with different catheters or collecting systems?

2B.1. If the CAUTI rate is not decreasing after implementing a comprehensive strategy to reduce rates of CAUTI, consider using antimicrobial/antiseptic-impregnated catheters. The comprehensive strategy should include, at a minimum, the high priority recommendations for urinary catheter use, aseptic insertion, and maintenance (see Section III. Implementation and Audit). **(Category IB)**

2B.1.a. Further research is needed on the effect of antimicrobial/antiseptic-impregnated catheters in reducing the risk of symptomatic UTI, their inclusion among the primary interventions, and the patient populations most likely to benefit from these catheters. **(No recommendation/unresolved issue)**

2B.2. Hydrophilic catheters might be preferable to standard catheters for patients requiring intermittent catheterization. **(Category II)**

2B.3. Following aseptic insertion of the urinary catheter, maintain a closed drainage system. **(Category IB)**

2B.4. Complex urinary drainage systems (utilizing mechanisms for reducing bacterial entry such as antiseptic-release cartridges in the drain port) are not necessary for routine use. **(Category II)**

2B.5. Urinary catheter systems with preconnected, sealed catheter-tubing junctions are suggested for use. **(Category II)**

2B.6. Further research is needed to clarify the benefit of catheter valves in reducing the risk of CAUTI and other urinary complications. **(No recommendation/unresolved issue)**

Q2C. What are the risks and benefits associated with different catheter management techniques?

The available data examined the following catheter management techniques:

1. Antimicrobial prophylaxis
2. Urinary antiseptics (i.e., methanamine)
3. Bladder irrigation
4. Antiseptic instillation in the drainage bag
5. Periurethral care
6. Routine catheter or bag change
7. Catheter lubricants
8. Securing devices
9. Bacterial interference
10. Catheter cleansing
11. Catheter removal strategies (clamping vs. free drainage prior to removal, postoperative duration of catheterization)
12. Assessment of urine volumes

For all comparisons, we considered CAUTI outcomes as well as other outcomes critical to weighing the risks and benefits of different catheter management techniques. The evidence for this question consists of 6 systematic reviews,^{37,105,106,182-184} 56 RCTs,^{60,61,65-69,143,158,158,185-231} 34 observational studies,^{83,85,88,90,96,102,133,167,178,232-258} and 1 economic analysis.¹⁸⁰ The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 2C.

Q2C.1. Antimicrobial prophylaxis

Low-quality evidence suggested no benefit of antimicrobial prophylaxis in patients undergoing short-term catheterization.^{37,60,61,83,85,133,158,178,182,185,186,189-191,232-234} This was based on heterogeneous results for SUTI and bacteriuria/unspecified UTI and no adverse events related to antimicrobials. Lack of consistency in specific factors, such as patient population, antimicrobial agents, timing of administration, and duration of follow-up, did not allow for a summary of evidence of the effect of antimicrobial prophylaxis on CAUTI in patients undergoing short term catheterization. Only two studies evaluated adverse events related to antimicrobials. Our search did not reveal data on antimicrobial resistance or *Clostridium difficile* infection.

Low-quality evidence suggested no benefit of antimicrobial prophylaxis in patients undergoing long-term catheterization (indwelling and clean intermittent catheterization).^{106,183,192,194,235,238} This was based on a decreased risk of bacteriuria, heterogeneous results for SUTI, and no differences reported for catheter encrustation or adverse events, although data were sparse. One systematic review suggested an increase in antimicrobial resistance with antimicrobial use.

Q2C.2. Urinary antiseptics

Low-quality evidence suggested a benefit of methenamine for short-term catheterized patients.^{196,197} This was based on a reduced risk of SUTI and bacteriuria and no differences in adverse events. Evidence was limited to two studies of patients following gynecological surgery in Norway and Sweden.

Very low-quality evidence suggested a benefit of methanamine for long-term catheterized patients.^{106,236-239} This was based on a reduced risk of encrustation but no differences in risk of SUTI or bacteriuria. Data on encrustation was limited to one study. Studies involved primarily elderly and spinal cord injury patients with chronic indwelling catheters

Q2C.3. Bladder irrigation

Low-quality evidence suggested no benefit of bladder irrigation in patients with indwelling or intermittent catheters.^{66,69,199-206,240-242} This was based on no differences in SUTI and heterogeneous findings for bacteriuria.

Q2C.4. Antiseptic instillation in the drainage bag

Low-quality evidence suggested no benefit of antiseptic instillation in urinary drainage bags.^{90,207-211,243-245} This was based on no differences in SUTI and heterogeneous results for bacteriuria.

Q2C.5. Periurethral care

Low-quality evidence suggested no benefit of antiseptic meatal cleaning regimens before or during catheterization to prevent CAUTI.^{65,67,68,88,158,212-216,246,247} This was based on no difference in the risk of bacteriuria in patients receiving periurethral care regimens compared to those not receiving them. One study found a higher risk of bacteriuria with cleaning of the urethral meatus-catheter junction (either twice daily application of povidine-iodine or once daily cleaning with a non-antiseptic solution of green soap and water) in a subgroup of women with positive meatal cultures and in patients not receiving antimicrobials. Periurethral cleaning with chlorhexidine before catheter insertion did not have an effect in two studies.

Q2C.6. Routine catheter or bag change

Low-quality evidence suggested no benefit of routine catheter or drainage bag changes to prevent CAUTI.^{102,217-219,248,249} This was based on no difference or an increased risk of SUTI and no difference in bacteriuria with routine compared to as-needed changes or with more frequent changing intervals. One study in nursing home residents found no differences in SUTI with routine monthly catheter changes compared to changing only for obstruction or infection, but the study was underpowered to detect a difference. Another study in home care patients found an increased risk of SUTI when catheters were changed more frequently than monthly.

Q2C.7. Catheter lubricants

Very low-quality evidence suggested a benefit of using lubricants for catheter insertion.^{167,220-223,250-254} This was based on a decreased risk of SUTI and bacteriuria with the use of a pre-lubricated catheter compared to a catheter lubricated by the patient and a decreased risk of bacteriuria with use of a lubricant versus no lubricant. Studies were heterogeneous both in the interventions and outcomes studied. Several studies comparing antiseptic lubricants to non-antiseptic lubricants found no significant differences.

Q2C.8. Securing devices

Low-quality evidence suggested no benefit of using catheter securing devices to prevent CAUTI.²²⁴ This was based on no significant difference in the risk of SUTI or meatal erosion. The only study in this category looked at one particular product.

Q2C.9. Bacterial interference

Moderate-quality evidence suggested a benefit of using bacterial interference in catheterized patients.²²⁵ In the one study evaluating this intervention, urinary colonization with a non-pathogenic *Escherichia coli* was associated with a decreased risk of SUTI in adults with spinal cord injury and a history of frequent CAUTI.

Q2C.10. Catheter cleansing

Very low-quality evidence suggested a benefit of wet versus dry storage procedures for catheters used in clean intermittent catheterization.²⁵⁵ This was based on a decreased risk of SUTI with a wet storage procedure in one study of spinal cord injury patients undergoing clean intermittent catheterization compared to a dry storage procedure where the catheter was left to air dry after washing. In the wet procedure, the catheter was stored in a dilute povidone-iodine solution after washing with soap and water.

Q2C.11. Catheter removal strategies

a. Clamping vs. free drainage prior to removal

Low-quality evidence suggested no benefit of clamping versus free drainage before catheter removal.^{37,184} This was based on no difference in risk of bacteriuria, urinary retention, or recatheterization between the two strategies. One study comparing a clamp and release strategy to free drainage over 72 hours found a greater risk of bacteriuria in the clamping group.

b. Postoperative duration of catheterization

Moderate-quality evidence suggested a benefit of shorter versus longer postoperative durations of catheterization.^{37,184,227,228} This was based on a decreased risk of bacteriuria/unspecified UTI, decreased time to ambulation and length of stay, no differences in urinary retention and SUTI, and increased risk of recatheterization. Significant decreases in bacteriuria/unspecified UTI were found specifically for comparisons of 1 day versus 3 or 5 days of postoperative catheterization. Recatheterization risk was greater in only one study comparing immediate removal to removal 6 or 12 hours after hysterectomy.

Q2C.12. Assessment of urine volumes

Low-quality evidence suggested a benefit of using portable ultrasound to assess urine volume in patients undergoing intermittent catheterization.^{229,230} This was based on fewer catheterizations but no reported differences in risk of unspecified UTI. Patients studied were adults with neurogenic bladder in inpatient rehabilitation centers. Our search did not reveal data on the use of ultrasound in catheterized patients in other settings.

Evidence Review Table 2C. What are the risks and benefits associated with different catheter management techniques?

2C.1. Unless clinical indications exist (e.g., in patients with bacteriuria upon catheter removal post urologic surgery), do not use systemic antimicrobials routinely as prophylaxis for UTI in patients requiring either short or long-term catheterization. **(Category IB)**

2C.2.a. Further research is needed on the use of urinary antiseptics (e.g., methanamine) to prevent UTI in patients requiring short-term catheterization. **(No recommendation/unresolved issue)**

2C.2.b. Further research is needed on the use of methanamine to prevent encrustation in patients requiring chronic indwelling catheters who are at high risk for obstruction. **(No recommendation/unresolved issue)**

2C.3.a. Unless obstruction is anticipated (e.g., as might occur with bleeding after prostatic or bladder surgery), bladder irrigation is not recommended. **(Category II)**

2C.3.b. Routine irrigation of the bladder with antimicrobials is not recommended. **(Category II)**

2C.4. Routine instillation of antiseptic or antimicrobial solutions into urinary drainage bags is not recommended. **(Category II)**

2C.5.a. Do not clean the periurethral area with antiseptics to prevent CAUTI while the catheter is in place. Routine hygiene (e.g., cleansing of the meatal surface during daily bathing) is

appropriate. **(Category IB)**

2C.5.b. Further research is needed on the use of antiseptic solutions vs. sterile water or saline for periurethral cleaning prior to catheter insertion. **(No recommendation/unresolved issue)**

2C.6. Changing indwelling catheters or drainage bags at routine, fixed intervals is not recommended. Rather, catheters and drainage bags should be changed based on clinical indications such as infection, obstruction, or when the closed system is compromised. **(Category II)**

2C.7.a. Use a sterile, single-use packet of lubricant jelly for catheter insertion. **(Category IB)**

2C.7.b. Routine use of antiseptic lubricants is not necessary. **(Category II)**

2C.8. Further research is needed on the use of bacterial interference to prevent UTI in patients requiring chronic urinary catheterization. **(No recommendation/unresolved issue)**

2C.9. Further research is needed on optimal cleaning and storage methods for catheters used for clean intermittent catheterization. **(No recommendation/unresolved issue)**

2C.10.a. Clamping indwelling catheters prior to removal is not necessary. **(Category II)**

2C.10.b. Insert catheters only for appropriate indications, and leave in place only as long as needed. **(Category IB)**

2C.10.c. For operative patients who have an indication for an indwelling catheter, remove the catheter as soon as possible postoperatively, preferably within 24 hours, unless there are appropriate indications for continued use. **(Category IB)**

2C.11.a. Consider using a portable ultrasound device to assess urine volume in patients undergoing intermittent catheterization to assess urine volume and reduce unnecessary catheter insertions. **(Category II)**

2C.11.b. Further research is needed on the use of a portable ultrasound device to evaluate for obstruction in patients with indwelling catheters and low urine output. **(No recommendation/unresolved issue)**

Q2D. What are the risks and benefits associated with different systems interventions?

The available data examined the following systems interventions:

1. Infection control/quality improvement programs (multifaceted)
2. Catheter reminders
3. Bacteriologic monitoring
4. Hand hygiene
5. Patient placement
6. Catheter team versus self-catheterization
7. Feedback
8. Nurse-directed catheter removal

We considered CAUTI outcomes, duration of catheterization, recatheterization, and transmission of pathogens when weighing the risks and benefits of different systems interventions. The evidence for this question consists of 1 RCT²⁵⁹ and 19 observational

studies.^{3,25,260-276} The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 2D.

Q2D.1. Multifaceted infection control/quality improvement programs

Low-quality evidence suggested a benefit of multifaceted infection control/quality improvement programs to reduce the risk of CAUTI.^{3,260-267} This was based on a decreased risk of SUTI, bacteriuria/unspecified UTI, and duration of catheter use with implementation of such programs. Studies evaluated various multifaceted interventions. The studies with significant findings included: 1) education and performance feedback regarding compliance with catheter care, emphasizing hand hygiene, and maintaining unobstructed urine flow; 2) computerized alerts to physicians, nurse-driven protocols to remove catheters, and use of handheld bladder scanners to assess for urinary retention; 3) guidelines and education focusing on perioperative catheter management; and 4) a multifaceted infection control program including guidelines for catheter insertion and maintenance. A program using a checklist and algorithm for appropriate catheter use also suggested a decrease in unspecified UTI and catheter duration, but statistical differences were not reported.

Q2D.2. Reminders

Very low-quality evidence suggested a benefit of using urinary catheter reminders to prevent CAUTI.²⁶⁸⁻²⁷⁰ This was based on a decreased risk of bacteriuria and duration of catheterization and no differences in recatheterization or SUTI when reminders were used. Reminders to physicians included both computerized and non-computerized alerts about the presence of urinary catheters and the need to remove unnecessary catheters.

Q2D.3. Bacteriologic monitoring

Very low-quality evidence suggested no benefit of bacteriologic monitoring to prevent CAUTI.^{25,271} Although one study found a decreased risk of bacteriuria during a period of bacteriologic monitoring and feedback, only 2% of SUTI episodes were considered potentially preventable with the use of bacteriologic monitoring.

Q2D.4. Hand hygiene

Very low-quality evidence suggested a benefit of using alcohol hand sanitizer in reducing CAUTI. This was based on one study in a rehabilitation facility that found a decrease in unspecified UTI, although no statistical differences were reported.²⁷² A separate multifaceted study that included education and performance feedback on compliance with catheter care and hand hygiene showed a decrease in risk of SUTI.²⁶⁵

Q2D.5. Patient placement

Very low-quality evidence suggested a benefit of spatially separating patients to prevent transmission of urinary pathogens.²⁷³ This was based on a decreased risk of transmission of urinary bacterial pathogens in nursing home residents in separate rooms compared to residents in the same rooms.

Q2D.6. Catheter team versus self-catheterization

Very low-quality evidence suggested no benefit of a catheter team to prevent CAUTI among patients requiring intermittent catheterization.²⁷⁴ This was based on one study showing no difference in unspecified UTI between use of a catheter care team and self-catheterization for intermittent catheterization in paraplegic patients.

Q2D.7. Feedback

Very low-quality evidence suggested a benefit of using nursing feedback to prevent CAUTI.²⁷⁵ This was based on a decreased risk of unspecified UTI during an intervention where nursing staff were provided with regular reports of unit-specific rates of CAUTI.

Q2D.8. Nurse-directed catheter removal

Very low-quality evidence suggested a benefit of a nurse-directed catheter removal program to prevent CAUTI.²⁷⁶ This was based on a decreased risk of unspecified UTI during an intervention where criteria were developed that allowed a registered nurse to remove a catheter without a physician's order when no longer medically necessary. Of the three intensive care units where the intervention was implemented, differences were significant only in the coronary intensive care unit.

Evidence Review Table 2D. What are the risks and benefits associated with different systems interventions?

2D.1.a. Ensure that healthcare personnel and others who take care of catheters are given periodic in-service training stressing the correct techniques and procedures for urinary catheter insertion, maintenance, and removal. **(Category IB)**

2D.1.b. Implement quality improvement (QI) programs or strategies to enhance appropriate use of indwelling catheters and to reduce the risk of CAUTI based on a facility risk assessment. **(Category IB)**

Examples of programs that have been demonstrated to be effective include:

1. A system of alerts or reminders to identify all patients with urinary catheters and assess the need for continued catheterization
2. Guidelines and protocols for nurse-directed removal of unnecessary urinary catheters
3. Education and performance feedback regarding appropriate use, hand hygiene, and catheter care
4. Guidelines and algorithms for appropriate peri-operative catheter management, such as:
 - a. Procedure-specific guidelines for catheter placement and postoperative catheter removal
 - b. Protocols for management of postoperative urinary retention, such as nurse-directed use of intermittent catheterization and use of ultrasound bladder scanners

2D.2. Routine screening of catheterized patients for asymptomatic bacteriuria is not recommended. **(Category II)**

2D.3. Perform hand hygiene immediately before and after insertion or any manipulation of the catheter site or device. **(Category IB)**

2D.5. Maintain unobstructed urine flow. **(Category IB)**

2D.6. Further research is needed on the benefit of spatial separation of patients with urinary catheters to prevent transmission of pathogens colonizing urinary drainage systems. **(No recommendation/unresolved issue)**

2D.7. When performing surveillance for CAUTI, consider providing regular (e.g., quarterly) feedback of unit-specific CAUTI rates to nursing staff and other appropriate clinical care staff. **(Category II)**

Q3: What are the best practices for preventing UTI associated with obstructed urinary catheters?

The available data examined the following practices:

1. Methods to prevent/reduce encrustations or blockage
2. Catheter materials preventing blockage

For this question, available relevant outcomes included blockage/encrustation. We did not find data on the outcomes of CAUTI. The evidence for this question consists of 1 systematic review,²⁷⁷ 2 RCTs,^{278,279} and 2 observational studies.^{280,281} The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 3.

Q3.1. Methods to prevent/reduce encrustations or blockage

Low-quality evidence suggested a benefit of acidifying solutions or oral acetohydroxamic acid in preventing or reducing catheter encrustations and blockage in long-term catheterized patients.^{277,278,280,281} No differences were seen with daily catheter irrigation with normal saline.

Q3.2. Catheter materials preventing blockage

Low-quality evidence suggested a benefit of silicone over latex or Teflon-coated catheters in prevention or reducing catheter encrustations in long-term catheterized patients who were prone to blockage. No differences were seen with different materials in patients considered “non-blockers.”²⁷⁹

Evidence Review Table 3. What are the best practices for preventing UTI associated with obstructed urinary catheters?

3.1.a. Further research is needed on the benefit of irrigating the catheter with acidifying solutions or use of oral urease inhibitors in long-term catheterized patients who have frequent catheter obstruction. **(No recommendation/unresolved issue)**

3.2.a. Silicone might be preferable to other materials to reduce the risk of encrustation in long-term catheterized patients who have frequent obstruction. **(Category II)**

References

1. Wong ES. Guideline for prevention of catheter-associated urinary tract infections. *Am J Infect Control*. 1983;11(1):28-36.
2. Lo E, Nicolle L, Classen D, et al. Strategies to prevent catheter-associated urinary tract infections in acute care hospitals. *Infect Control Hosp Epidemiol*. 2008;29:S41-S50.
3. Stephan F, Sax H, Wachsmuth M, Hoffmeyer P, Clergue F, Pittet D. Reduction of urinary tract infection and antibiotic use after surgery: A controlled, prospective, before-after intervention study. *Clin Infect Dis*. 2006;42(11):1544-1551.
4. Dolin SJ, Cashman JN. Tolerability of acute postoperative pain management: Nausea, vomiting, sedation, pruritis, and urinary retention. evidence from published data. *Br J Anaesth*. 2005;95(5):584-591.
5. Hidron AI, Edwards JR, Patel J, et al. NHSN annual update: Antimicrobial-resistant pathogens associated with healthcare-associated infections: Annual summary of data reported to the national healthcare safety network at the centers for disease control and prevention, 2006-2007. *Infect Control Hosp Epidemiol*. 2008;29(11):996-1011.
6. Saint S. Clinical and economic consequences of nosocomial catheter-related bacteriuria. *Am J Infect Control*. 2000;28(1):68-75.
7. Platt R, Polk BF, Murdock B, Rosner B. Mortality associated with nosocomial urinary-tract infection. *N Engl J Med*. 1982;307(11):637-642.
8. Givens CD, Wenzel RP. Catheter-associated urinary tract infections in surgical patients: A controlled study on the excess morbidity and costs. *J Urol*. 1980;124(5):646-648.
9. Tambyah PA, Knasinski V, Maki DG. The direct costs of nosocomial catheter-associated urinary tract infection in the era of managed care. *Infect Control Hosp Epidemiol*. 2002;23(1):27-31.
10. Schaberg DR, Weinstein RA, Stamm WE. Epidemics of nosocomial urinary tract infection caused by multiply resistant gram-negative bacilli: Epidemiology and control. *J Infect Dis*. 1976;133(3):363-366.
11. Yoon HJ, Choi JY, Park YS, et al. Outbreaks of *Serratia marcescens* bacteriuria in a neurosurgical intensive care unit of a tertiary care teaching hospital: A clinical, epidemiologic, and laboratory perspective. *Am J Infect Control*. 2005;33(10):595-601.
12. Warren JW. Catheter-associated urinary tract infections. *Int J Antimicrob Agents*. 2001;17(4):299-303.
13. Weinstein JW, Mazon D, Pantelick E, Reagan-Cirincione P, Dembry LM, Hierholzer WJ, Jr. A decade of prevalence surveys in a tertiary-care center: Trends in nosocomial infection rates, device utilization, and patient acuity. *Infect Control Hosp Epidemiol*. 1999;20(8):543-548.
14. Munasinghe RL, Yazdani H, Siddique M, Hafeez W. Appropriateness of use of indwelling urinary catheters in patients admitted to the medical service. *Infect Control Hosp Epidemiol*. 2001;22(10):647-649.

15. Jain P, Parada JP, David A, Smith LG. Overuse of the indwelling urinary tract catheter in hospitalized medical patients. *Arch Intern Med.* 1995;155(13):1425-1429.
16. Saint S, Wiese J, Amory JK, et al. Are physicians aware of which of their patients have indwelling urinary catheters? *Am J Med.* 2000;109(6):476-480.
17. Edwards JR, Peterson KD, Andrus ML, et al. National healthcare safety network (NHSN) report, data summary for 2006, issued june 2007. *Am J Infect Control.* 2007;35(5):290-301.
18. Rogers MA, Mody L, Kaufman SR, Fries BE, McMahon LF, Jr, Saint S. Use of urinary collection devices in skilled nursing facilities in five states. *J Am Geriatr Soc.* 2008;56(5):854-861.
19. Klevens RM, Edwards JR, Richards CL, Jr, et al. Estimating health care-associated infections and deaths in U.S. hospitals, 2002. *Public Health Rep.* 2007;122(2):160-166.
20. Weinstein MP, Towns ML, Quartey SM, et al. The clinical significance of positive blood cultures in the 1990s: A prospective comprehensive evaluation of the microbiology, epidemiology, and outcome of bacteremia and fungemia in adults. *Clin Infect Dis.* 1997;24(4):584-602.
21. Muder RR, Brennen C, Wagener MM, Goetz AM. Bacteremia in a long-term-care facility: A five-year prospective study of 163 consecutive episodes. *Clin Infect Dis.* 1992;14(3):647-654.
22. Umscheid C, Mitchell M, Agarwal R, Williams K, Brennan P. Mortality from reasonably-preventable hospital acquired infections. included in written testimony by the society of healthcare epidemiology of america for the committee on oversight and government reform hearing on healthcare-associated infections: A preventable epidemic, chaired by henry A. waxman, april 16, 2008, washington, DC. [congressional testimony]. .
23. Kunin CM, McCormack RC. Prevention of catheter-induced urinary-tract infections by sterile closed drainage. *N Engl J Med.* 1966;274(21):1155-1161.
24. Tambyah PA, Halvorson KT, Maki DG. A prospective study of pathogenesis of catheter-associated urinary tract infections. *Mayo Clin Proc.* 1999;74(2):131-136.
25. Garibaldi RA, Mooney BR, Epstein BJ, Britt MR. An evaluation of daily bacteriologic monitoring to identify preventable episodes of catheter-associated urinary tract infection. *Infect Control.* 1982;3(6):466-470.
26. Saint S, Lipsky BA, Goold SD. Indwelling urinary catheters: A one-point restraint? *Ann Intern Med.* 2002;137(2):125-127.
27. Warren JW, Tenney JH, Hoopes JM, Muncie HL, Anthony WC. A prospective microbiologic study of bacteriuria in patients with chronic indwelling urethral catheters. *J Infect Dis.* 1982;146(6):719-723.
28. Saint S, Chenoweth CE. Biofilms and catheter-associated urinary tract infections. *Infect Dis Clin North Am.* 2003;17(2):411-432.

29. Kallen AJ, Hidron AI, Edwards JR, Patel J, Srinivasan A. Antibiotic resistance among important gram-negative pathogens causing healthcare-associated infections reported to the national healthcare safety network, 2006-2007 [abstract 376] in: Program and abstracts of the 18th annual scientific meeting of the society for healthcare epidemiology of america, orlando, FL, apr 5-8, 2008. .
30. Horan TC, Gaynes RP. Surveillance of nosocomial infections. In: Mayhall CG, ed. *Hospital Epidemiology and Infection Control*. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2004:1659-1702.
31. Tambyah PA, Maki DG. Catheter-associated urinary tract infection is rarely symptomatic: A prospective study of 1,497 catheterized patients. *Arch Intern Med*. 2000;160(5):678-682.
32. Atkins D, Best D, Briss PA, et al. Grading quality of evidence and strength of recommendations. *BMJ*. 2004;328(7454):1490.
33. Guyatt GH, Oxman AD, Kunz R, et al. Going from evidence to recommendations. *BMJ*. 2008;336(7652):1049-1051.
34. Guyatt GH, Oxman AD, Kunz R, et al. What is "quality of evidence" and why is it important to clinicians? *BMJ*. 2008;336(7651):995-998.
35. Pratt RJ, Pellowe CM, Wilson JA, et al. epic2: National evidence-based guidelines for preventing healthcare-associated infections in NHS hospitals in england. *J Hosp Infect*. 2007;65(Suppl 1):S1-64.
36. Pellowe CM, Pratt RJ, Harper P, et al. Evidence-based guidelines for preventing healthcare-associated infections in primary and community care in england. *J Hosp Infect*. 2003;55(Suppl 2):S2-127.
37. Phipps S, Lim YN, McClinton S, Barry C, Rane A, N'Dow J. Short term urinary catheter policies following urogenital surgery in adults. *Cochrane Database Syst Rev*. 2006(2):CD004374.
38. Tang KK, Wong CK, Lo SF, Ng TK. Is it necessary to catheterise the bladder routinely before gynaecological laparoscopic surgery? *Aust N Z J Obstet Gynaecol*. 2005;45(5):380-383.
39. Iorio R, Healy WL, Patch DA, Appleby D. The role of bladder catheterization in total knee arthroplasty. *Clin Orthop*. 2000(380):80-84.
40. Liu SK, Rassai H, Krasner C, Braun J, Matolo NM. Urinary catheter in laparoscopic cholecystectomy: Is it necessary? *Surg Laparosc Endosc Percutan Tech*. 1999;9(3):184-186.
41. Normelli H, Aaro S, Hedlund R, Svensson O, Stromberg L. Urethral catheterization in spinal surgery: A randomized prospective study. *Eur Spine J*. 1993;2(3):132-135.
42. Carpiello VL, Cendron M, Altman HG, Malloy TR, Booth R. Treatment of urinary complications after total joint replacement in elderly females. *Urology*. 1988;32(3):186-188.
43. Akhtar MS, Beere DM, Wright JT, MacRae KD. Is bladder catheterization really necessary before laparoscopy? *Br J Obstet Gynaecol*. 1985;92(11):1176-1178.

44. Chaudhuri P, Vengadasalam D. Urinary tract infection following abdominal delivery--second thought on indwelling catheter. *Singapore Med J.* 1983;24(4):224-226.
45. McMurdo ME, Davey PG, Elder MA, Miller RM, Old DC, Malek M. A cost-effectiveness study of the management of intractable urinary incontinence by urinary catheterisation or incontinence pads. *J Epidemiol Community Health.* 1992;46(3):222-226.
46. Geraniotis E, Koff SA, Enrile B. The prophylactic use of clean intermittent catheterization in the treatment of infants and young children with myelomeningocele and neurogenic bladder dysfunction. *J Urol.* 1988;139(1):85-86.
47. Kumar P, Mannan K, Chowdhury AM, Kong KC, Pati J. Urinary retention and the role of indwelling catheterization following total knee arthroplasty. *Int Braz J Urol.* 2006;32(1):31-34.
48. Miskry T, Cumbis A, Apostolidis P, Eleftheriou M, Magos A. Do not catheterize the bladder at operative laparoscopy. *Gynaecol Endosc.* 2001;10(1):29-32.
49. Barnes JS. Is it better to avoid urethral catheterization at hysterectomy and caesarean section? *Aust N Z J Obstet Gynaecol.* 1998;38(3):315-316.
50. Ouslander JG, Greengold B, Chen S. External catheter use and urinary tract infections among incontinent male nursing home patients. *J Am Geriatr Soc.* 1987;35(12):1063-1070.
51. Rannikko S, Kyllastinen M, Granqvist B. Comparison of long-term indwelling catheters and bed-pads in the treatment of urinary incontinence in elderly patients. *J Infect.* 1986;12(3):221-227.
52. Nordqvist P, Ekelund P, Edouard L, Svensson ML, Brandberg A, Seeberg S. Catheter-free geriatric care. routines and consequences for clinical infection, care and economy. *J Hosp Infect.* 1984;5(3):298-304.
53. Egilmez T, Aridogan IA, Yachia D, Hassin D. Comparison of nitinol urethral stent infections with indwelling catheter-associated urinary-tract infections. *J Endourol.* 2006;20(4):272-277.
54. Ruz ED, Garcia Leoni E, Herruzo Cabrera R. Epidemiology and risk factors for urinary tract infection in patients with spinal cord injury. *J Urol.* 2000;164(4):1285-1289.
55. Donovan WH, Stolov WC, Clowers DE, et al. Bacteriuria during intermittent catheterization following spinal cord injury. *Arch Phys Med Rehabil.* 1978;59(8):351-357.
56. Larsen LD, Chamberlin DA, Khonsari F, Ahlering TE. Retrospective analysis of urologic complications in male patients with spinal cord injury managed with and without indwelling urinary catheters. *Urology.* 1997;50(3):418-422.
57. Kochakarn W, Ratana-Olarn K, Lertsithichai P, Roongreungsilp U. Follow-up of long-term treatment with clean intermittent catheterization for neurogenic bladder in children. *Asian J.* 2004;27(2):134-136.

58. Ehrlich O, Brem AS. A prospective comparison of urinary tract infections in patients treated with either clean intermittent catheterization or urinary diversion. *Pediatrics*. 1982;70(5):665-669.
59. Darouiche RO, Smith JA, Jr., Hanna H, et al. Efficacy of antimicrobial-impregnated bladder catheters in reducing catheter-associated bacteriuria: A prospective, randomized, multicenter clinical trial. *Urology*. 1999;54(6):976-981.
60. Stricker PD, Grant AB. Relative value of antibiotics and catheter care in the prevention of urinary tract infection after transurethral prostatic resection. *Br J Urol*. 1988;61(6):494-497.
61. Rogers RG, Kammerer-Doak D, Olsen A, et al. A randomized, double-blind, placebo-controlled comparison of the effect of nitrofurantoin monohydrate macrocrystals on the development of urinary tract infections after surgery for pelvic organ prolapse and/or stress urinary incontinence with suprapubic catheterization. *Obstet Gynecol*. 2004;191(1):182-187.
62. Baan AH, Vermeulen H, van der Meulen, J., Bossuyt P, Olszyna D, Gouma DJ. The effect of suprapubic catheterization versus transurethral catheterization after abdominal surgery on urinary tract infection: A randomized controlled trial. *Dig Surg*. 2003;20(4):290-295.
63. Carapeti EA, Andrews SM, Bentley PG. Randomised study of sterile versus non-sterile urethral catheterisation.[erratum appears in ann R coll surg engl 1996 may;78(3 pt 1):167]. *Ann R Coll Surg Engl*. 1996;78(1):59-60.
64. Huth TS, Burke JP, Larsen RA, Classen DC, Stevens LE. Clinical trial of junction seals for the prevention of urinary catheter-associated bacteriuria. *Arch Intern Med*. 1992;152(4):807-812.
65. Huth TS, Burke JP, Larsen RA, Classen DC, Stevens LE. Randomized trial of meatal care with silver sulfadiazine cream for the prevention of catheter-associated bacteriuria. *J Infect Dis*. 1992;165(1):14-18.
66. Schneeberger PM, Vreede RW, Bogdanowicz JF, van Dijk, W. C. A randomized study on the effect of bladder irrigation with povidone-iodine before removal of an indwelling catheter. *J Hosp Infect*. 1992;21(3):223-229.
67. Burke JP, Jacobson JA, Garibaldi RA, Conti MT, Alling DW. Evaluation of daily meatal care with poly-antibiotic ointment in prevention of urinary catheter-associated bacteriuria. *J Urol*. 1983;129(2):331-334.
68. Classen DC, Larsen RA, Burke JP, Alling DW, Stevens LE. Daily meatal care for prevention of catheter-associated bacteriuria: Results using frequent applications of polyantibiotic cream. *Infect Control Hosp Epidemiol*. 1991;12(3):157-162.
69. Warren JW, Platt R, Thomas RJ, Rosner B, Kass EH. Antibiotic irrigation and catheter-associated urinary-tract infections. *N Engl J Med*. 1978;299(11):570-573.
70. Seki N, Masuda K, Kinukawa N, Senoh K, Naito S. Risk factors for febrile urinary tract infection in children with myelodysplasia treated by clean intermittent catheterization. *Int J Urol*. 2004;11(11):973-977.

71. Kelleher RE, Meeropol E, Parks D. Factors influencing the development of symptomatic bacteriuria in children on clean intermittent catheterization. *J Urol Nurs.* 1996;15(2):1256-1265.
72. Waites KB, Canupp KC, DeVivo MJ. Epidemiology and risk factors for urinary tract infection following spinal cord injury. *Arch Phys Med Rehabil.* 1993;74(7):691-695.
73. Anderson RU. Non-sterile intermittent catheterization with antibiotic prophylaxis in the acute spinal cord injured male patient. *J Urol.* 1980;124(3):392-394.
74. Van Der Kooi, T. I. I., De Boer, A. S., Mannien J, et al. Incidence and risk factors of device-associated infections and associated mortality at the intensive care in the dutch surveillance system. *Intensive Care Med.* 2007;33(2):271-278.
75. Bochicchio GV, Joshi M, Shih D, Bochicchio K, Tracy K, Scalea TM. Reclassification of urinary tract infections in critically ill trauma patients: A time-dependent analysis. *Surg Infect.* 2003;4(4):379-385.
76. Leone M, Albanese J, Garnier F, et al. Risk factors of nosocomial catheter-associated urinary tract infection in a polyvalent intensive care unit. *Intensive Care Med.* 2003;29(7):1077-1080.
77. Tissot E, Limat S, Cornette C, Capellier G. Risk factors for catheter-associated bacteriuria in a medical intensive care unit. *Eur J Clin Microbiol Infect Dis.* 2001;20(4):260-262.
78. Colau A, Lucet JC, Rufat P, Botto H, Benoit G, Jardin A. Incidence and risk factors of bacteriuria after transurethral resection of the prostate. *Eur Urol.* 2001;39(3):272-276.
79. Ouslander JG, Greengold B, Chen S. Complications of chronic indwelling urinary catheters among male nursing home patients: A prospective study. *J Urol.* 1987;138(5):1191-1195.
80. Hazelett SE, Tsai M, Gareri M, Allen K. The association between indwelling urinary catheter use in the elderly and urinary tract infection in acute care. *BMC geriatr.* 2006;6:15.
81. Saint S, Kaufman SR, Rogers MAM, Bakers PD, Boyko EJ, Lipsky BA. Risk factors for nosocomial urinary tract-related bacteremia: A case-control study. *Am J Infect Control.* 2006;34(7):401-407.
82. Srinivasan A, Karchmer T, Richards A, Song X, Perl TM. A prospective trial of a novel, silicone-based, silver-coated foley catheter for the prevention of nosocomial urinary tract infections. *Infect Control Hosp Epidemiol.* 2006;27(1):38-43.
83. Cardosi RJ, Cardosi RP, Grendys EC, Jr., Fiorica JV, Hoffman MS. Infectious urinary tract morbidity with prolonged bladder catheterization after radical hysterectomy... includes discussion. *Obstet Gynecol.* 2003;189(2):380-384.
84. Johansson I, Athlin E, Frykholm L, Bolinder H, Larsson G. Intermittent versus indwelling catheters for older patients with hip fractures. *J Clin Nurs.* 2002;11(5):651-656.

85. Hustinx WN, Mintjes-de Groot, Verkooyen RP, Verbrugh HA. Impact of concurrent antimicrobial therapy on catheter-associated urinary tract infection.[see comment]. *J Hosp Infect.* 1991;18(1):45-56.
86. Johnson JR, Roberts PL, Olsen RJ, Moyer KA, Stamm WE. Prevention of catheter-associated urinary tract infection with a silver oxide-coated urinary catheter: Clinical and microbiologic correlates. *J Infect Dis.* 1990;162(5):1145-1150.
87. Lima NL, Guerrant RL, Kaiser DL, Germanson T, Farr BM. A retrospective cohort study of nosocomial diarrhea as a risk factor for nosocomial infection. *J Infect Dis.* 1990;161(5):948-952.
88. Jacono JJ, Talamelli CL. Nosocomial urinary tract infection in catheterized patients. *Infect Control Can.* 1988;3(1):16-20.
89. Lanara V, Plati C, Paniara O, et al. The prevalence of urinary tract infection in patients related to type of drainage bag. *Scand J Caring Sci.* 1988;2(4):163-170.
90. Holliman R, Seal DV, Archer H, Doman S. Controlled trial of chemical disinfection of urinary drainage bags. reduction in hospital-acquired catheter-associated infection. *Br J Urol.* 1987;60(5):419-422.
91. Mulhall AB, Chapman RG, Crow RA. Bacteriuria during indwelling urethral catheterization. *J Hosp Infect.* 1988;11(3):253-262.
92. Pien FD, Landers JQ, Jr. Indwelling urinary catheter infections in small community hospital. role of urinary drainage bag. *Urology.* 1983;22(3):255-258.
93. Shapiro M, Simchen E, Izraeli S, Sacks TG. A multivariate analysis of risk factors for acquiring bacteriuria in patients with indwelling urinary catheters for longer than 24 hours. *Infect Control.* 1984;5(11):525-532.
94. Platt R, Polk BF, Murdock B, Rosner B. Risk factors for nosocomial urinary tract infection. *Am J Epidemiol.* 1986;124(6):977-985.
95. Burke JP, Larsen RA, Stevens LE. Nosocomial bacteriuria: Estimating the potential for prevention by closed sterile urinary drainage. *Infect Control.* 1986;7(2):96-99.
96. Saramma PP. Catheter-associated urinary tract infection: A follow-up study in prevention. *Nurs J India.* 1987;78(10):257-259.
97. Islam AK, Chapman J. Closed catheter drainage and urinary infection--a comparison of two methods of catheter drainage. *Br J Urol.* 1977;49(3):215-220.
98. Hartstein AI, Garber SB, Ward TT, Jones SR, Morthland VH. Nosocomial urinary tract infection: A prospective evaluation of 108 catheterized patients. *Infect Control.* 1981;2(5):380-386.
99. Garibaldi RA, Burke JP, Britt MR, Miller MA, Smith CB. Meatal colonization and catheter-associated bacteriuria. *N Engl J Med.* 1980;303(6):316-318.
100. Garibaldi RA, Burke JP, Dickman ML, Smith CB. Factors predisposing to bacteriuria during indwelling urethral catheterization. *N Engl J Med.* 1974;291(5):215-219.

101. Hirsh DD, Fainstein V, Musher DM. Do condom catheter collecting systems cause urinary tract infection? *JAMA*. 1979;242(4):340-341.
102. White MC, Ragland KE. Urinary catheter-related infections among home care patients. *J Wound Ostomy Continence Nurs*. 1995;22(6):286-290.
103. Wilde MH, Carrigan MJ. A chart audit of factors related to urine flow and urinary tract infection. *J Adv Nurs*. 2003;43(3):254-262.
104. Niel-Weise BS, van den Broek, P. J. Urinary catheter policies for short-term bladder drainage in adults. *Cochrane Libr*. 2006;4.
105. Shekelle PG, Morton SC, Clark KA, Pathak M, Vickrey BG. Systematic review of risk factors for urinary tract infection in adults with spinal cord dysfunction. *J Spinal Cord Med*. 1999;22(4):258-272.
106. Vickrey BG, Shekelle P, Morton S, Clark K, Pathak M, Kamberg C. Prevention and management of urinary tract infections in paralyzed persons. *Evid Rep Technol Assess*. 1999(6):1-3.
107. Branagan GW, Moran BJ. Published evidence favors the use of suprapubic catheters in pelvic colorectal surgery. *Dis Colon Rectum*. 2002;45(8):1104-1108.
108. McPhail MJ, Abu-Hilal M, Johnson CD. A meta-analysis comparing suprapubic and transurethral catheterization for bladder drainage after abdominal surgery. *Br J Surg*. 2006;93(9):1038-1044.
109. Saint S, Kaufman SR, Rogers MA, Baker PD, Ossenkop K, Lipsky BA. Condom versus indwelling urinary catheters: A randomized trial. *J Am Geriatr Soc*. 2006;54(7):1055-1061.
110. Michelson JD, Lotke PA, Steinberg ME. Urinary-bladder management after total joint-replacement surgery. *N Engl J Med*. 1988;319(6):321-326.
111. Tang MW, Kwok TC, Hui E, Woo J. Intermittent versus indwelling urinary catheterization in older female patients. *Maturitas*. 2006;53(3):274-281.
112. Turi MH, Hanif S, Fasih Q, Shaikh MA. Proportion of complications in patients practicing clean intermittent self-catheterization (CISC) vs indwelling catheter. *J Pak Med Assoc*. 2006;56(9):401-404.
113. Tangtrakul S, Taechaiya S, Suthutvoravut S, Linasmita V. Post-cesarean section urinary tract infection: A comparison between intermittent and indwelling catheterization. *J Med Assoc Thai*. 1994;77(5):244-248.
114. Skelly JM, Guyatt GH, Kalbfleisch R, Singer J, Winter L. Management of urinary retention after surgical repair of hip fracture. *Can Med Assoc J*. 1992;146(7):1185-1189.
115. Jannelli ML, Wu JM, Plunkett LW, Williams KS, Visco AG. A randomized controlled trial of clean intermittent self-catheterization versus suprapubic catheterization after urogynecologic surgery. *Am J Obstet Gynecol*. 2007;197(1).
116. Roberts K, Naik R. Catheterization options following radical surgery for cervical cancer. *Br J Nurs*. 2006;15(19):1038-1044.

117. Duffy LM, Cleary J, Ahern S, et al. Clean intermittent catheterization: Safe, cost-effective bladder management for male residents of VA nursing homes. *J Am Geriatr Soc.* 1995;43(8):865-870.
118. Moore KN, Burt J, Voaklander DC. Intermittent catheterization in the rehabilitation setting: A comparison of clean and sterile technique. *Clin Rehabil.* 2006;20(6):461-468.
119. Moore KN, Kelm M, Sinclair O, Cadrain G. Bacteriuria in intermittent catheterization users: The effect of sterile versus clean reused catheters. *Rehabil Nurs.* 1993;18(5):306-309.
120. Prieto-Fingerhut T, Banovac K, Lynne CM. A study comparing sterile and nonsterile urethral catheterization in patients with spinal cord injury. *Rehabil Nurs.* 1997;22(6):299-302.
121. Schlager TA, Clark M, Anderson S. Effect of a single-use sterile catheter for each void on the frequency of bacteriuria in children with neurogenic bladder on intermittent catheterization for bladder emptying. *Pediatrics.* 2001;108(4):E71.
122. Joseph C, Jacobson C, Strausbaugh L, Maxwell M, French M, Colling J. Sterile vs clean urinary catheterization. *J Am Geriatr Soc.* 1991;39(10):1042-1043.
123. Saint S, Lipsky BA, Baker PD, McDonald LL, Ossenkop K. Urinary catheters: What type do men and their nurses prefer? *J Am Geriatr Soc.* 1999;47(12):1453-1457.
124. Ritter MA, Faris PM, Keating EM. Urinary tract catheterization protocols following total joint arthroplasty. *Orthopedics.* 1989;12(8):1085-1087.
125. Oishi CS, Williams VJ, Hanson PB, Schneider JE, Colwell CW, Jr., Walker RH. Perioperative bladder management after primary total hip arthroplasty. *J Arthroplasty.* 1995;10(6):732-736.
126. Furuhashi A, Ogawa K, Saito K, Yamaguchi T. Preoperative intermittent catheterization in patients with prostatic hypertrophy. *Clin Ther.* 1988;10 Spec No:47-51.
127. Alli MO, Singh B, Moodley J, Shaik AS. Prospective evaluation of combined suprapubic and urethral catheterization to urethral drainage alone for intraperitoneal bladder injuries. *Journal of Trauma-Injury Infection & Critical Care.* 2003;55(6):1152-1154.
128. Dunn TS, Figge J, Wolf D. A comparison of outcomes of transurethral versus suprapubic catheterization after burch cystourethropexy. *Int Urogynecol J Pelvic Floor Dysfunct.* 2005;16(1):60-2; discussion 62.
129. Dinneen MD, Wetter LA, May AR. Urethral strictures and aortic surgery. suprapubic rather than urethral catheters. *Eur J Vasc Surg.* 1990;4(5):535-538.
130. Horgan AF, Prasad B, Waldron DJ, O'Sullivan DC. Acute urinary retention. comparison of suprapubic and urethral catheterisation. *Br J Urol.* 1992;70(2):149-151.
131. Hofmeister FJ, Martens WE, Strelbel RL. Foley catheter or suprapubic tube? *Obstet Gynecol.* 1970;107(5):767-779.

132. Van Nagell, JR, Penny RM, Roddick JW. Suprapubic bladder drainage following radical hysterectomy. *Obstet Gynecol.* 1972;113(6):849-850.
133. Verbrugh HA, Mintjes-de Groot, Andriess R, Hamersma K, van Dijk A. Postoperative prophylaxis with norfloxacin in patients requiring bladder catheters. *Eur J Clin Microbiol Infect Dis.* 1988;7(4):490-494.
134. Noll F, Russe O, Kling E, Botel U, Schreiter F. Intermittent catheterisation versus percutaneous suprapubic cystostomy in the early management of traumatic spinal cord lesions. *Paraplegia.* 1988;26(1):4-9.
135. Weld KJ, Dmochowski RR, Foster HE, Jr. Effect of bladder management on urological complications in spinal cord injured patients. *J Urol.* 2000;163(3):768-772.
136. Lloyd LK, Kuhlemeier KV, Fine PR, Stover SL. Initial bladder management in spinal cord injury: Does it make a difference? *J Urol.* 1986;135(3):523-527.
137. Schumm K, Lam TB. Types of urethral catheters for management of short-term voiding problems in hospitalised adults.[update of cochrane database syst rev. 2004;(1):CD004013; PMID: 14974052]. *Cochrane Database Syst Rev.* 2008(2):004013.
138. Johnson JR, Kuskowski MA, Wilt TJ. Systematic review: Antimicrobial urinary catheters to prevent catheter-associated urinary tract infection in hospitalized patients. *Ann Intern Med.* 2006;144(2):116-126.
139. Niel-Weise BS, Arend SM, van den Broek, PJ. Is there evidence for recommending silver-coated urinary catheters in guidelines? *J Hosp Infect.* 2002;52(2):81-87.
140. van den Eijkel, E., Griffiths P. Catheter valves for indwelling urinary catheters: A systematic review. *Br J Community Nurs.* 2006;11(3):111-2, 114.
141. Crnich CJ, Drinka PJ. Does the composition of urinary catheters influence clinical outcomes and the results of research studies? *Infect Control Hosp Epidemiol.* 2007;28(1):102-103.
142. Reiche T, Lisby G, Jorgensen S, Christensen AB, Nordling J. A prospective, controlled, randomized study of the effect of a slow-release silver device on the frequency of urinary tract infection in newly catheterized patients. *BJU Int.* 2000;85(1):54-59.
143. Schaeffer AJ, Story KO, Johnson SM. Effect of silver oxide/trichloroisocyanuric acid antimicrobial urinary drainage system on catheter-associated bacteriuria. *J Urol.* 1988;139(1):69-73.
144. De Ridder, D. J., Everaert K, Fernandez LG, et al. Intermittent catheterisation with hydrophilic-coated catheters (SpeediCath) reduces the risk of clinical urinary tract infection in spinal cord injured patients: A prospective randomised parallel comparative trial. *Eur Urol.* 2005;48(6):991-995.
145. Cindolo L, Palmieri EA, Autorino R, Salzano L, Altieri V. Standard versus hydrophilic catheterization in the adjuvant treatment of patients with superficial bladder cancer. *Urol Int.* 2004;73(1):19-22.
146. Stensballe J, Looms D, Nielsen PN, Tvede M. Hydrophilic-coated catheters for intermittent catheterisation reduce urethral micro trauma: A prospective, randomised, participant-blinded, crossover study of three different types of catheters. *Eur Urol.* 2005;48(6):978-983.

147. Vapnek JM, Maynard FM, Kim J, Vapnek JM, Maynard FM, Kim J. A prospective randomized trial of the LoFric hydrophilic coated catheter versus conventional plastic catheter for clean intermittent catheterization. *J Urol.* 2003;169(3):994-998.
148. Monson T, Kunin CM. Evaluation of a polymer-coated indwelling catheter in prevention of infection. *J Urol.* 1974;111(2):220-222.
149. Shafik A. The electrified catheter. role in sterilizing urine and decreasing bacteriuria. *World J Urol.* 1993;11(3):183-185.
150. Leone M, Garnier F, Antonini F, Bimar MC, Albanese J, Martin C. Comparison of effectiveness of two urinary drainage systems in intensive care unit: A prospective, randomized clinical trial. *Intensive Care Med.* 2003;29(4):551-554.
151. Classen DC, Larsen RA, Burke JP, Stevens LE. Prevention of catheter-associated bacteriuria: Clinical trial of methods to block three known pathways of infection. *Am J Infect Control.* 1991;19(3):136-142.
152. al-Juburi AZ, Cicmanec J. New apparatus to reduce urinary drainage associated with urinary tract infections. *Urology.* 1989;33(2):97-101.
153. DeGroot-Kosolcharoen J, Guse R, Jones JM. Evaluation of a urinary catheter with a preconnected closed drainage bag. *Infect Control Hosp Epidemiol.* 1988;9(2):72-76.
154. Klarskov P, Bischoff N, Bremmelgaard A, Hald T. Catheter-associated bacteriuria. A controlled trial with bardex(TM) urinary drainage system. *Acta Obstet Gynecol Scand.* 1986;65(4):295-299.
155. Keys TF, Maker MD, Segura JW. Bacteriuria during closed urinary drainage: An evaluation of top-vented versus bag-vented systems. *J Urol.* 1979;122(1):49-51.
156. Platt R, Polk BF, Murdock B, Rosner B. Reduction of mortality associated with nosocomial urinary tract infection. *Lancet.* 1983;1(8330):893-897.
157. Monson TP, Macalalad FV, Hamman JW, Kunin CM. Evaluation of a vented drainage system in prevention of bacteriuria. *J Urol.* 1977;117(2):216-219.
158. Cleland V, Cox F, Berggren H, MacInnis MR. Prevention of bacteriuria in female patients with welling catheters. *Nurs Res.* 1971;20(4):309-318.
159. Seymour C. Audit of catheter-associated UTI using silver alloy-coated foley catheters. *Br J Nurs.* 2006;15(11):598-603.
160. Gentry H, Cope S. Using silver to reduce catheter-associated urinary tract infections. *Nurs Stand.* 2005;19(50):51-54.
161. Madeo M, Davies D, Johnson G, Owen E, Wadsworth P, Martin CR. The impact of using silver alloy urinary catheters in reducing the incidence of urinary tract infections in the critical care setting. *Br J Infect Control.* 2004;5(1):21-24.
162. Rupp ME, Fitzgerald T, Marion N, et al. Effect of silver-coated urinary catheters: Efficacy, cost-effectiveness, and antimicrobial resistance. *Am J Infect Control.* 2004;32(8):445-450.

163. Lai KK, Fontecchio SA, Lai KK, Fontecchio SA. Use of silver-hydrogel urinary catheters on the incidence of catheter-associated urinary tract infections in hospitalized patients. *Am J Infect Control*. 2002;30(4):221-225.
164. Akiyama H, Okamoto S. Prophylaxis of indwelling urethral catheter infection: Clinical experience with a modified foley catheter and drainage system. *J Urol*. 1979;121(1):40-42.
165. Bologna RA, Tu LM, Polansky M, Fraimow HD, Gordon DA, Whitmore KE. Hydrogel/silver ion-coated urinary catheter reduces nosocomial urinary tract infection rates in intensive care unit patients: A multicenter study. *Urology*. 1999;54(6):982-987.
166. Newton T, Still JM, Law E. A comparison of the effect of early insertion of standard latex and silver-impregnated latex foley catheters on urinary tract infections in burn patients. *Infect Control Hosp Epidemiol*. 2002;23(4):217-218.
167. Butler HK, Kunin CM. Evaluation of polymyxin catheter lubricant and impregnated catheters. *J Urol*. 1968;100(4):560-566.
168. Mooro H, Kamel M. Prevention of catheter fever by the use of furacin urethral inserts. *J Egypt Med Assoc*. 1966;49(8):550-553.
169. Pachler J, Frimodt-Moller C. A comparison of prelubricated hydrophilic and non-hydrophilic polyvinyl chloride catheters for urethral catheterization. *BJU Int*. 1999;83(7):767-769.
170. Chen TY, Ponsot Y, Carmel M, Bouffard N, Kennelly MJ, Tu LM. Multi-centre study of intraurethral valve-pump catheter in women with a hypocontractile or acontractile bladder. *Eur Urol*. 2005;48(4):628-633.
171. Danchaivijitr S, Chokeloidkaew S, Prutsachativuthi S, Trakulsomboon S. Should the open urinary drainage system be continued? *J Med Assoc Thai*. 1988;71 Suppl 3:14-18.
172. Wille JC, Blusse van Oud Alblas, A., Thewessen EA. Nosocomial catheter-associated bacteriuria: A clinical trial comparing two closed urinary drainage systems. *J Hosp Infect*. 1993;25(3):191-198.
173. Hardyck C, Petrinovich L. Reducing urinary tract infections in catheterized patients. *Ostomy Wound*. 1998;44(12):36-8, 40, 42-3.
174. Montagnino BA, Gonzales ET, Jr., Roth DR. Open catheter drainage after urethral surgery. *J Urol*. 1988;140(5):1250-1252.
175. Madeo M, Barr B, Owen E. A study to determine whether the use of a pre-connect urinary catheter system reduces the incidence of nosocomial urinary tract infections. *Br J Infect Control*. 2005;6(4):22-25.
176. Leone M, Garnier F, Dubuc M, Bimar MC, Martin C. Prevention of nosocomial urinary tract infection in ICU patients: Comparison of effectiveness of two urinary drainage systems. *Chest*. 2001;120(1):220-224.
177. Blenkarn JJ. Prevention of bacteriuria during urinary catheterization of patients in an intensive care unit: Evaluation of the 'ureofix 500' closed drainage system. *J Hosp Infect*. 1985;6(2):187-193.

178. Drach GW, Lacy SS, Cox CE, 2nd. Prevention of catheter-induced post-prostatectomy infection. effects of systemic cephaloridine and local irrigation with neomycin-polymyxin through closed-drainage catheter system. *J Urol*. 1971;105(6):840-842.
179. Plowman R, Graves N, Esquivel J, Roberts JA. An economic model to assess the cost and benefits of the routine use of silver alloy coated urinary catheters to reduce the risk of urinary tract infections in catheterized patients. *J Hosp Infect*. 2001;48(1):33-42.
180. Platt R, Polk BF, Murdock B, Rosner B. Prevention of catheter-associated urinary tract infection: A cost-benefit analysis. *Infect Control Hosp Epidemiol*. 1989;10(2):60-64.
181. Saint S, Veenstra DL, Sullivan SD, Chenoweth C, Fendrick AM. The potential clinical and economic benefits of silver alloy urinary catheters in preventing urinary tract infection. *Arch Intern Med*. 2000;160(17):2670-2675.
182. Niel-Weise BS, van den Broek, P. J. Antibiotic policies for short-term catheter bladder drainage in adults. *Cochrane Libr*. 2006;4.
183. Niel-Weise BS, van den Broek, P. J. Urinary catheter policies for long-term bladder drainage. *Cochrane Libr*. 2006;4.
184. Griffiths R, Fernandez R. Strategies for the removal of short-term indwelling urethral catheters in adults. *Cochrane Libr*. 2007(2).
185. Esposito S, Noviello S, Leone S, Marvaso A, Drago L, Marchetti F, The LSG. A pilot study on prevention of catheter-related urinary tract infections with fluoroquinolones. *J Chemother*. 2006;18(5):494-501.
186. Wazait HD, Patel HR, Van Der Meulen, J. H. P., et al. A pilot randomized double-blind placebo-controlled trial on the use of antibiotics on urinary catheter removal to reduce the rate of urinary tract infection: The pitfalls of ciprofloxacin. *BJU Int*. 2004;94(7):1048-1050.
187. Lukkarinen O, Hellstrom P, Leppilahti M, Kontituri M, Tammela T. Antibiotic prophylaxis in patients with urinary retention undergoing transurethral prostatectomy. *Ann Chir Gynaecol*. 1997;86(3):239-242.
188. Lukkarinen O, Hellstrom P, Leppilahti M, Kontturi M, Tammela T. Prospective randomized open study between ciprofloxacin and a combination of sulfadiazine and trimethoprim in antibiotic prophylaxis in connection with transurethral prostatectomy. *Scand J Urol Nephrol*. 1996;30(1):33-36.
189. Vollaard EJ, Clasener HA, Zambon JV, Joosten HJ, van Griethuysen, AJ. Prevention of catheter-associated gram-negative bacilluria with norfloxacin by selective decontamination of the bowel and high urinary concentration. *J Antimicrob Chemother*. 1989;23(6):915-922.
190. Grabe M, Forsgren A, Hellsten S. A short antibiotic course given in conjunction with and after catheter removal consecutive to transurethral prostatic resection. *Scand J Urol Nephrol*. 1984;18(3):193-199.
191. Little PJ, Pearson S, Peddie BA, Greenslade NF, Utley WL. Amoxicillin in the prevention of catheter-induced urinary infection. *J Infect Dis*. 1974;129(0):suppl:S241-2.
192. Clarke SA, Samuel M, Boddy SA. Are prophylactic antibiotics necessary with clean intermittent catheterization? A randomized controlled trial. *J Pediatr Surg*. 2005;40(3):568-571.

193. Waites KB, Canupp KC, Armstrong S, DeVivo MJ. Effect of cranberry extract on bacteriuria and pyuria in persons with neurogenic bladder secondary to spinal cord injury. *J Spinal Cord Med.* 2004;27(1):35-40.
194. Firestein M, Mendelson G, Gronich D, Granot E, Ben-Israel J, Raz R. Can antibiotic use during routine replacement of long-term urinary catheter prevent bacteriuria? *Infectious Diseases in Clinical Practice.* 2001;10(3):133-135.
195. Schlager TA, Anderson S, Trudell J, Hendley JO. Effect of cranberry juice on bacteriuria in children with neurogenic bladder receiving intermittent catheterization. *J Pediatr.* 1999;135(6):698-702.
196. Schiotz HA, Guttu K. Value of urinary prophylaxis with methenamine in gynecologic surgery. *Acta Obstet Gynecol Scand.* 2002;81(8):743-746.
197. Tyreman NO, Andersson PO, Kroon L, Orstam S. Urinary tract infection after vaginal surgery. effect of prophylactic treatment with methenamine hippurate. *Acta Obstet Gynecol Scand.* 1986;65(7):731-733.
198. Pearman JW, Bailey M, Harper WE, Pearman JW, Bailey M, Harper WE. Comparison of the efficacy of "trisdine" and kanamycin-colistin bladder instillations in reducing bacteriuria during intermittent catheterisation of patients with acute spinal cord trauma. *Br J Urol.* 1988;62(2):140-144.
199. Pearman JW, Bailey M, Riley LP. Bladder instillations of trisdine compared with catheter introducer for reduction of bacteriuria during intermittent catheterisation of patients with acute spinal cord trauma. *Br J Urol.* 1991;67(5):483-490.
200. Adesanya AA, Osegbe DN, Amaku EO. The use of intermittent chlorhexidine bladder irrigation in the prevention of post-prostatectomy infective complications. *Int Urol Nephrol.* 1993;25(4):359-367.
201. Ball AJ, Carr TW, Gillespie WA, Kelly M, Simpson RA, Smith PJ. Bladder irrigation with chlorhexidine for the prevention of urinary infection after transurethral operations: A prospective controlled study.[see comment]. *J Urol.* 1987;138(3):491-494.
202. van den Broek, P. J., Daha TJ, Mouton RP. Bladder irrigation with povidone-iodine in prevention of urinary-tract infections associated with intermittent urethral catheterisation. *Lancet.* 1985;1(8428):563-565.
203. Savage JE, Phillips B, Lifshitz S, et al. Bacteriuria in closed bladder drainage versus continuous irrigation in patients undergoing intracavitary radium for treatment of gynecologic cancer. *Gynecol Oncol.* 1982;13(1):26-30.
204. Kirk D, Dunn M, Bullock DW, Mitchell JP, Hobbs SJ. Hibitane bladder irrigation in the prevention of catheter-associated urinary infection. *Br J Urol.* 1979;51(6):528-531.
205. Chamberlain G, Holt E. Automatic bladder irrigation after vaginal surgery. *Br J Urol.* 1975;47(6):641-643.
206. Clark LW. Neomycin in the prevention of postcatheterization bacteriuria. *Med J Aust.* 1973;1(21):1034-1036.
207. Washington EA. Instillation of 3% hydrogen peroxide or distilled vinegar in urethral catheter drainage bag to decrease catheter-associated bacteriuria. *Biol Res Nurs.* 2001;3(2):78-87.

208. Sweet DE, Goodpasture HC, Holl K, Smart S, Alexander H, Hedari A. Evaluation of H₂O₂ prophylaxis of bacteriuria in patients with long-term indwelling foley catheters: A randomized controlled study. *Infect Control*. 1985;6(7):263-266.
209. Thompson RL, Haley CE, Searcy MA, et al. Catheter-associated bacteriuria. failure to reduce attack rates using periodic instillations of a disinfectant into urinary drainage systems. *JAMA*. 1984;251(6):747-751.
210. Gillespie WA, Simpson RA, Jones JE, Nashef L, Teasdale C, Speller DC. Does the addition of disinfectant to urine drainage bags prevent infection in catheterised patients? *Lancet*. 1983;1(8332):1037-1039.
211. Maizels M, Schaeffer AJ. Decreased incidence of bacteriuria associated with periodic instillations of hydrogen peroxide into the urethral catheter drainage bag. *J Urol*. 1980;123(6):841-845.
212. Webster J, Hood RH, Burrige CA, Doidge ML, Phillips CM, George N. Water or antiseptic for periurethral cleaning before urinary catheterization: A randomized controlled trial. *Am J Infect Control*. 2001;29(6):389-394.
213. Bardwell TA. Comparing meatal cleansing. *Aust Nurs J*. 1999;7(2):30-31.
214. Sanderson PJ, Weissler S. A comparison of the effect of chlorhexidine antiseptics, soap and antibiotics on bacteriuria, perineal colonization and environmental contamination in spinally injured patients. *J Hosp Infect*. 1990;15(3):235-243.
215. Burke JP, Garibaldi RA, Britt MR, Jacobson JA, Conti M, Alling DW. Prevention of catheter-associated urinary tract infections. efficacy of daily meatal care regimens. *Am J Med*. 1981;70(3):655-658.
216. Ross PW, Smylie HG. A comparison of two regimes of catheterisation in the prophylaxis of urinary tract infection in gynaecological patients. *Br J Urol*. 1966;38(2):194-198.
217. Keerasuntonpong A, Thearawiboon W, Panthawanan A, et al. Incidence of urinary tract infections in patients with short-term indwelling urethral catheters: A comparison between a 3-day urinary drainage bag change and no change regimens. *Am J Infect Control*. 2003;31(1):9-12.
218. Dille CA, Kirchhoff KT, Sullivan JJ, Larson E. Increasing the wearing time of vinyl urinary drainage bags by decontamination with bleach. *Arch Phys Med Rehabil*. 1993;74(4):431-437.
219. Priefer BA, Duthie EH, Jr., Gambert SR. Frequency of urinary catheter change and clinical urinary tract infection. study in hospital-based, skilled nursing home. *Urology*. 1982;20(2):141-142.
220. Fera P, Glashan RQ, Bruschini H, Lelis MAS, Baretta MCC, Srougi M. Lubricated urethral catheters with lidocaine versus gentamycin for clean intermittent catheterization. *International Braz J Urol*. 2002;28(1):50-56.
221. Giannantoni A, Di Stasi SM., Scivoletto G, Virgili G, Dolci S, Porena M. Intermittent catheterization with a prelubricated catheter in spinal cord injured patients: A prospective randomized crossover study. *J Urol*. 2001;166(1):130-133.

222. Cohen A. A microbiological comparison of a povidone-iodine lubricating gel and a control as catheter lubricants. *J Hosp Infect.* 1985;6 Suppl A:155-161.
223. Kunin CM, Finkelberg Z. Evaluation of an intraurethral lubricating catheter in prevention of catheter-induced urinary tract infections. *J Urol.* 1971;106(6):928-930.
224. Darouiche RO, Goetz L, Kaldis T, Cerra-Stewart C, AlSharif A, Priebe M. Impact of StatLock securing device on symptomatic catheter-related urinary tract infection: A prospective, randomized, multicenter clinical trial. *Am J Infect Control.* 2006;34(9):555-560.
225. Darouiche RO, Thornby JI, Cerra-Stewart C, Donovan WH, Hull RA. Bacterial interference for prevention of urinary tract infection: A prospective, randomized, placebo-controlled, double-blind pilot trial. *Clinical infectious diseases : an official publication of the Infectious Diseases Society of America.* 2005;41(10):1531-1534.
226. Moore KN. Two methods for cleaning catheters used for intermittent catheterization: Sunlight(TM) liquid detergent and cetrimide 1:30 (savlon(TM)). *Canadian Journal of Rehabilitation.* 1990;4(2):87-92.
227. Alessandri F, Mistrangelo E, Lijoi D, Ferrero S, Ragni N. A prospective, randomized trial comparing immediate versus delayed catheter removal following hysterectomy. *Acta Obstet Gynecol Scand.* 2006;85(6):716-720.
228. Schiotz HA. Comparison of 1 and 3 days' transurethral foley catheterization after vaginal plastic surgery. *International Urogynecology Journal.* 1995;6(3):158-161.
229. Polliack T, Bluvshstein V, Philo O, et al. Clinical and economic consequences of volume- or time-dependent intermittent catheterization in patients with spinal cord lesions and neuropathic bladder (structured abstract). *Spinal Cord.* 2005;43(10):615-619.
230. Anton HA, Chambers K, Clifton J, Tasaka J. Clinical utility of a portable ultrasound device in intermittent catheterization. *Archives of Physical Medicine & Rehabilitation.* 1998;79(2):172-175.
231. Moyad RH, Persky L. Vesical irrigation and urosepsis. *Invest Urol.* 1968;6(1):21-25.
232. Shohet I, Alagam M, Shafir R, Tsur H, Cohen B. Postoperative catheterization and prophylactic antimicrobials in children with hypospadias. *Urology.* 1983;22(4):391-393.
233. Anonymous. Evaluation of aseptic techniques and chlorhexidine on the rate of catheter-associated urinary-tract infection. southampton infection control team. *Lancet.* 1982;1(8263):89-91.
234. Cafferkey MT, Conneely B, Falkiner FR, Gillespie WA, Murphy D. Post-operative urinary infection and septicaemia in urology. *J Hosp Infect.* 1980;1(4):315-320.
235. Salomon J, Denys P, Merle C, et al. Prevention of urinary tract infection in spinal cord-injured patients: Safety and efficacy of a weekly oral cyclic antibiotic (WOCA) programme with a 2 year follow-up - an observational prospective study. *J Antimicrob Chemother.* 2006;57(4):784-788.

236. Wibell L, Scheynius A, Norrman K. Methenamine-hippurate and bacteriuria in the geriatric patient with a catheter. *Acta Med Scand.* 1980;207(6):469-473.
237. Kostiala AA, Nyren P, Jokinen EJ, Runeberg L. Prospective study on the appearance of antibody-coated bacteria in patients with an indwelling urinary catheter. *Nephron.* 1982;30(3):279-285.
238. Nyren P, Runeberg L, Kostiala AI, Renkonen OV, Roine R. Prophylactic methenamine hippurate or nitrofurantoin in patients with an indwelling urinary catheter. *Ann Clin Res.* 1981;13(1):16-21.
239. Norrman K, Wibell L. Treatment with methenamine hippurate in the patient with a catheter. *J Int Med Res.* 1976;4(2):115-117.
240. Pearman JW. The value of kanamycin-colistin bladder instillations in reducing bacteriuria during intermittent catheterisation of patients with acute spinal cord injury. *Br J Urol.* 1979;51(5):367-374.
241. Cox F, Smith RF, Elliott JP, Quinn EL. Neomycin-polymyxin prophylaxis of urinary-tract infection associated with indwelling catheters. *Antimicrob Agents Chemother.* 1966;6:165-168.
242. Thornton GF, Lytton B, Andriole VT. Bacteriuria during indwelling catheter drainage. effect of constant bladder rinse. *JAMA.* 1966;195(3):179-183.
243. Wongsatanapong W. Prevention of urinary tract infection by instillation of chlorhexidine into urinary drainage bags. *J Med Assoc Thai.* 1988;71 Suppl 3:19-21.
244. Sujka SK, Petrelli NJ, Herrera L. Incidence of urinary tract infections in patients requiring long-term catheterization after abdominoperineal resection for rectal carcinoma: Does betadine(TM) in the foley drainage bag make a difference? *European Journal of Surgical Oncology.* 1987;13(4):341-343.
245. Samuels P, Fisher P. The use of hydrogen peroxide in catheter drainage units. *AUAA J.* 1983;3(3):5-9.
246. Koskeroglu N, Durmaz G, Bahar M, Kural M, Yelken B. The role of meatal disinfection in preventing catheter-related bacteriuria in an intensive care unit: A pilot study in turkey. *J Hosp Infect.* 2004;56(3):236-238.
247. Matsumoto T, Sakumoto M, Takahashi K, Kumazawa J. Prevention of catheter-associated urinary tract infection by meatal disinfection. *Dermatology.* 1997;195 Suppl 2:73-77.
248. Stelling JD, Hale AM. Protocol for changing condom catheters in males with spinal cord injury. *SCI Nurs.* 1996;13(2):28-34.
249. Reid RI, Webster O, Pead PJ, Maskell R. Comparison of urine bag-changing regimens in elderly catheterised patients. *Lancet.* 1982;2(8301):754-756.
250. Kambal C, Chance J, Cope S, Beck J. Catheter-associated UTIs in patients after major gynaecological surgery. *Prof Nurse.* 2004;19(9):515-518.
251. Schiotz HA. Antiseptic catheter gel and urinary tract infection after short-term postoperative catheterization in women. *Arch Gynecol Obstet.* 1996;258(2):97-100.
252. Harrison LH. Comparison of a microbicidal povidone-iodine gel and a placebo gel as catheter lubricants. *J Urol.* 1980;124(3):347-349.

253. Chavigny KH. The use of polymixin B as a urethral lubricant to reduce the post-instrumental incidence of bacteriuria in females. an exploratory study. *Int J Nurs Stud.* 1975;12(1):33-42.
254. Nooyen RL. Prophylaxis of catheter-induced urinary tract infection in obstetric patients. *J Am Osteopath Assoc.* 1966;66(3):324-326.
255. Sims L, Ballard N. A comparison of two methods of catheter cleansing and storage used with clean intermittent catheterization. *Rehabil Nurs Res.* 1993;2(2):87-92.
256. Wald H, Epstein A, Kramer A. Extended use of indwelling urinary catheters in postoperative hip fracture patients. *Med Care.* 2005;43(10):1009-1017.
257. Wyatt TD, Timoney R. The effect of introducing a policy for catheter care on the catheter infection rate in a small hospital. *J Hosp Infect.* 1987;9(3):230-234.
258. Hoy WE, Kissel SM, Freeman RB, Sterling WA, Jr. Altered patterns of posttransplant urinary tract infections associated with perioperative antibiotics and curtailed catheterization. *Am J Kidney Dis.* 1985;6(4):212-216.
259. Pickard WG, Grundy DJ. A comparison of two methods of sterile urethral catheterisation in spinal cord injured adults. *Paraplegia.* 1996;34(1):30-33.
260. Jain M, Miller L, Belt D, King D, Berwick DM. Decline in ICU adverse events, nosocomial infections and cost through a quality improvement initiative focusing on teamwork and culture change. *Qual Saf Health Care.* 2006;15(4):235-239.
261. Reilly L, Sullivan P, Ninni S, Fochesto D, Williams K, Fetherman B. Reducing foley catheter device days in an intensive care unit: Using the evidence to change practice. *AACN Adv Crit Care.* 2006;17(3):272-283.
262. Verdier R, Parer S, Jean-Pierre H, Dujols P, Picot MC. Impact of an infection control program in an intensive care unit in france. *Infect Control Hosp Epidemiol.* 2006;27(1):60-66.
263. Topal J, Conklin S, Camp K, Morris V, Balcezak T, Herbert P. Prevention of nosocomial catheter-associated urinary tract infections through computerized feedback to physicians and a nurse-directed protocol. *Am J Med Qual.* 2005;20(3):121-126.
264. Misset B, Timsit JF, Dumay MF, et al. A continuous quality-improvement program reduces nosocomial infection rates in the ICU. *Intensive Care Med.* 2004;30(3):395-400.
265. Rosenthal VD, Guzman S, Safdar N. Effect of education and performance feedback on rates of catheter-associated urinary tract infection in intensive care units in argentina. *Infect Control Hosp Epidemiol.* 2004;25(1):47-50.
266. Berg DE, Hershow RC, Ramirez CA, Weinstein RA. Control of nosocomial infections in an intensive care unit in guatemala city. *Clin Infect Dis.* 1995;21(3):588-593.
267. Cools HJ, van der Meer JW. Infection control in a skilled nursing facility: A 6-year survey. *J Hosp Infect.* 1988;12(2):117-124.

268. Saint S, Kaufman SR, Thompson M, Rogers MA, Chenoweth CE. A reminder reduces urinary catheterization in hospitalized patients. *Jt Comm J Qual Patient Saf.* 2005;31(8):455-462.
269. Huang WC, Wann SR, Lin SL, et al. Catheter-associated urinary tract infections in intensive care units can be reduced by prompting physicians to remove unnecessary catheters. *Infect Control Hosp Epidemiol.* 2004;25(11):974-978.
270. Cornia PB, Amory JK, Fraser S, Saint S, Lipsky BA. Computer-based order entry decreases duration of indwelling urinary catheterization in hospitalized patients. *Am J Med.* 2003;114(5):404-407.
271. Jacobson JA, Burke JP, Kasworm E. Effect of bacteriologic monitoring of urinary catheters on recognition and treatment of hospital-acquired urinary tract infections. *Infect Control.* 1981;2(3):227-232.
272. Fendler EJ, Ali Y, Hammond BS, Lyons MK, Kelley MB, Vowell NA. The impact of alcohol and sanitizer use on infection rates in an extended care facility. *Am J Infect Control.* 2002;30(4):226-233.
273. Fryklund B, Haeggman S, Burman LG. Transmission of urinary bacterial strains between patients with indwelling catheters - nursing in the same room and in separate rooms compared. *J Hosp Infect.* 1997;36(2):147-153.
274. Wyndaele JJ, De Taeye N. Early intermittent self-catheterisation after spinal cord injury. *Paraplegia.* 1990;28(2):76-80.
275. Goetz AM, Kedzuf S, Wagener M, Muder RR. Feedback to nursing staff as an intervention to reduce catheter-associated urinary tract infections. *Am J Infect Control.* 1999;27(5):402-404.
276. Dumigan DG, Kohan CA, Reed CR, Jekel JF, Fikrig MK. Utilizing national nosocomial infection surveillance system data to improve urinary tract infection rates in three intensive-care units. *Clin Perform Qual Health Care.* 1998;6(4):172-178.
277. Mayes J, Bliss J, Griffiths P. Preventing blockage of long-term indwelling catheters in adults: Are citric acid solutions effective? *Br J Community Nurs.* 2003;8(4):172-175.
278. Muncie HL, Jr., Hoopes JM, Damron DJ, Tenney JH, Warren JW. Once-daily irrigation of long-term urethral catheters with normal saline. lack of benefit. *Arch Intern Med.* 1989;149(2):441-443.
279. Kunin CM, Chin QF, Chambers S. Formation of encrustations on indwelling urinary catheters in the elderly: A comparison of different types of catheter materials in "blockers" and "nonblockers". *J Urol.* 1987;138(4):899-902.
280. Burns JR, Gauthier JF. Prevention of urinary catheter encrustations by acetohydroxamic acid. *J Urol.* 1984;132(3):455-456.
281. Ruwaldt MM. Irrigation of indwelling urinary catheters. *Urology.* 1983;21(2):127-129.



Infection Prevention and Control Certificate of Mastery

Course Reference Guide



Guidelines for the Prevention of Intravascular Catheter-Related Infections, 2011

Naomi P. O'Grady, M.D.¹, Mary Alexander, R.N.², Lillian A. Burns, M.T., M.P.H., C.I.C.³, E. Patchen Dellinger, M.D.⁴, Jeffery Garland, M.D., S.M.⁵, Stephen O. Heard, M.D.⁶, Pamela A. Lipsett, M.D.⁷, Henry Masur, M.D.¹, Leonard A. Mermel, D.O., Sc.M.⁸, Michele L. Pearson, M.D.⁹, Issam I. Raad, M.D.¹⁰, Adrienne Randolph, M.D., M.Sc.¹¹, Mark E. Rupp, M.D.¹², Sanjay Saint, M.D., M.P.H.¹³ and the Healthcare Infection Control Practices Advisory Committee (HICPAC)¹⁴.

1National Institutes of Health, Bethesda, Maryland

2Infusion Nurses Society, Norwood, Massachusetts

3Greenwich Hospital, Greenwich, Connecticut

4University of Washington, Seattle, Washington

5Wheaton Franciscan Healthcare-St. Joseph, Milwaukee, Wisconsin

6 University of Massachusetts Medical School, Worcester, Massachusetts

7Johns Hopkins University School of Medicine, Baltimore, Maryland

8Warren Alpert Medical School of Brown University and Rhode Island Hospital, Providence, Rhode Island

9Office of Infectious Diseases, CDC, Atlanta, Georgia

10MD Anderson Cancer Center, Houston, Texas

11The Children's Hospital, Boston, Massachusetts

12University of Nebraska Medical Center, Omaha, Nebraska

13Ann Arbor VA Medical Center and University of Michigan, Ann Arbor, Michigan

¹⁴ Healthcare Infection Control Practices Advisory Committee

CHAIRMAN

BRENNAN, Patrick J., MD
Chief Medical Officer
Division of Infectious Diseases
University of Pennsylvania Health System

MEMBERSHIP

BRATZLER, Dale, DO, MPH
President and CEO
Oklahoma Foundation for Medical Quality

BURNS, Lillian A., MT, MPH
Infection Control Coordinator
Greenwich Hospital, Infectious Diseases
Department

ELWARD, Alexis, MD
Assistant Professor, Pediatrics Infectious
Diseases
Washington University School of Medicine
Department of Pediatrics
Division of Infectious Diseases

HUANG, Susan, MD, MPH
Assistant Professor
Medical Director, Epidemiology and Infection
Prevention
Division of Infectious Diseases
UC Irvine School of Medicine

LUNDSTROM, Tammy, MD, JD
Chief Medical Officer
Providence Hospital

MCCARTER, Yvette S., PhD
Director, Clinical Microbiology
Laboratory
Department of Pathology
University of Florida Health Science
Center

MURPHY, Denise M. RN, MPH, CIC
Vice President, Quality and Patient Safety
Main Line Health System

EXECUTIVE SECRETARY

BELL, Michael R., MD
Deputy Director
Division of Healthcare Quality Promotion
Centers for Disease Control and Prevention

OSTROFF, Stephen, MD
Director, Bureau of Epidemiology
Pennsylvania Department of Health

OLMSTED, Russell N., MPH, CIC
Epidemiologist
Infection Control Services
St. Joseph Mercy Health System

PEGUES, David Alexander, MD
Professor of Medicine, Hospital Epidemiologist
David Geffen School of Medicine at
UCLA

PRONOVOST, Peter J., MD, PhD, FCCM
Director, Johns Hopkins Quality and Safety Research
Group
Johns Hopkins Quality and Safety Research Group

SOULE, Barbara M., RN, MPA, CIC
Practice Leader
Infection Prevention and Control Services
Joint Commission Resources/Joint
Commission International

SCHECTER, William, P., MD
Professor of Surgery
Department of Surgery
San Francisco General Hospital

Guidelines for the Prevention of Intravascular Catheter-Related Infections

EX-OFFICIO MEMBERS

Agency for Healthcare Research and Quality (AHRQ)

BAINE, William B., MD
Senior Medical Advisor
Center for Outcomes and Evidence

Center for Medicare & Medicaid Services (CMS)

MILLER, Jeannie, RN, MPH
Deputy Director, Clinical Standards Group

Food and Drug Administration (FDA)

MURPHEY, Sheila A., MD
Division of Anesthesiology, General Hospital Infection
Control Dental Devices
Center for Devices and Radiology Health

LIAISONS

Advisory Council for the Elimination of Tuberculosis (ACET)

STRICOF, Rachel L., MPH

American College of Occupational and Environmental Medicine

RUSSI, Mark, MD, MPH
Professor of Medicine
Yale University School of Medicine
Director, Occupational Health
Yale-New Haven Hospital

American Health Care Assn (AHCA)

FITZLER, Sandra L., RN
Senior Director of Clinical Services

American Hospital Association (AHA)

SCHULMAN, Roslyne, MHA, MBA
Director, Policy Development

Association of Professionals of Infection Control and Epidemiology, Inc. (APIC)

DeBAUN, Barbara, MSN, RN, CIC

Association of periOperative Registered Nurses (AORN)

BLANCHARD, Joan C., RN, BSN

Council of State and Territorial Epidemiologists (CSTE)

KAINER, Marion MD, MPH
Director, Hospital Infections and Antimicrobial Resistance
Program
Tennessee Department Health

National Institute of Health (NIH)

HENDERSON, David, MD
Deputy Director for Clinical Care
Associate Director for Hospital Epidemiology and Quality
Improvement
NIH Clinical Center

Department of Veterans Affairs (VA)

ROSELLE, Gary A., MD
National Program Director, Infectious Diseases
VA Central Office
Cincinnati VA Medical Center

Consumers Union

MCGIFFERT, Lisa
Senior Policy Analyst on Health Issues
Project Director Stop Hospital Infections Organization

Infectious Disease Society of America (IDSA)

HUSKINS, W. Charles MD, MSc
Division of Pediatric Infectious Diseases
Assistant Professor of Pediatrics
Mayo Clinic

Public Health Agency of Canada

PATON, Shirley, RN, MN
Senior Advisor Healthcare Acquired Infections
Center for Communicable Diseases and Infection Control

Society for Healthcare Epidemiology of America (SHEA)

MARAGAKIS, Lisa, MD
Assistant Professor of Medicine
John Hopkins Medical Institutions

Society of Hospital Medicine

SAINT, Sanjay, MD, MPH
Director, Ann Arbor VA Medical Center/University of
Michigan Patient Safety Enhancement Program

The Joint Commission

WISE, Robert A., MD
Vice President
Division of Standards & Survey Methods

Use of trade names and commercial sources is for identification only and does not imply endorsement by the U.S. Department of Health and Human Services.

Acknowledgments

We wish to acknowledge Ingi Lee, MD, MSCE and Craig A. Umscheid, MD, MSCE from Center for Evidence-based Practice, University of Pennsylvania Health System Philadelphia, PA who performed a systematic review for issues raised the during the guideline process.

Disclosure of potential conflicts of interests. N.P.O.'G. served as a board member for the ABIM Subspecialty Board for Critical Care Medicine. M.A. is an employee of the Infusion Nurses Society, Honoraria from 3M, Becton Dickinson, Smiths Medical. L.A.B. is a consultant for Institute of Healthcare Improvement, Board membership for Theradoc, Medline. Honoraria from APIC, Clorox. E.P.D. consulting from Merck, Baxter, Ortho-McNeil, Targanta, Schering-Plough, Optimer, Cadence, Cardinal, BDGeneOhm, WebEx, Cerebrio, and Tyco. Grant support through the NIH. Payment for lecture from Merck. Payment for development of educational presentation from Medscape. Travel and meeting expenses paid for by ASHP, IDSA, ASM, American College of Surgeons, NQF, SHEA/CDC, HHS, Trauma Shock Inflammation and Sepsis Meeting (Munich), University of Minnesota. J.G. Honoria from Ethicon. S.O.H. provides research support from Angiotech; Honoraria from Angiotech, Merck. L.A.M provides research support from Astellas, Theravance, Pfizer; Consulting for Ash Access, Cadence, CorMedix, Catheter Connections, Carefusion, Sage, Bard, Teleflex; Payment for manuscript preparation from Catheter Connections. I.I.R. provides research support from Cubist, Enzon, and Basilea; Consulting for Clorox; Stock Equity or Options in Great Lakes Pharmaceuticals and Inventive Protocol; Speakers Bureau for Cook, Inc.; Royalty income (patents owned by MD Anderson on which Dr. Raad is an inventor: American Medical Systems, Cook, Inc., Cook urological, Teleflex, TyRx, Medtronic, Biomet, Great Lakes Pharmaceuticals. A.R. consulting income from Eisai Pharmaceuticals, Discovery Laboratories. M.E.R. provides research support from Molnlycke, Cardinal Healthcare Foundation, Sanofi-Pasteur, 3M, and Cubist; Consulting from Semprus; Honorarium for lectures from 3M, Carefusion, Baxter and Becton Dickinson. Previously served on Board of Directors for Society for Healthcare Epidemiology of America. All other authors: no conflicts.

Table of Contents

NOTICE TO READERS: 7

Introduction 8

Summary Of Recommendations 9

 Education, Training and Staffing 9

 Selection of Catheters and Sites 10

 Peripheral Catheters and Midline Catheters 10

 Central Venous Catheters 11

 Hand Hygiene and Aseptic Technique 12

 Maximal Sterile Barrier Precautions 12

 Skin Preparation 13

 Catheter Site Dressing Regimens 13

 Patient Cleansing 15

 Catheter Securement Devices 15

 Antimicrobial/Antiseptic Impregnated Catheters and Cuffs 15

 Systemic Antibiotic Prophylaxis 15

 Antibiotic/Antiseptic Ointments 15

 Antibiotic Lock Prophylaxis, Antimicrobial Catheter Flush and Catheter Lock Prophylaxis 16

 Anticoagulants 16

 Replacement of Peripheral and Midline Catheters 16

 Replacement of CVCs, Including PICCs and Hemodialysis Catheters 16

 Umbilical Catheters 17

 Peripheral Arterial Catheters and Pressure Monitoring Devices for Adult and Pediatric Patients 18

 Replacement of Administration Sets 19

 Needleless Intravascular Catheter Systems 19

 Performance Improvement 20

Background Information 20

 Terminology and Estimates of Risk 20

 Epidemiology and Microbiology in Adult and Pediatric Patients 23

 Pathogenesis 23

 Strategies for Prevention of Catheter-Related Infections in Adult and Pediatric Patients 25

 Education, Training and Staffing 25

 Selection of Catheters and Sites 26

 Peripheral and Midline Catheter Recommendations 26

 Central Venous Catheters Recommendations 27

 Hand Hygiene and Aseptic Technique 29

 Maximal Sterile Barrier Precautions 30

 Skin Preparation 31

 Catheter Site Dressing Regimens 32

 Patient Cleansing 35

 Catheter Securement Devices 36

Antimicrobial/Antiseptic Impregnated Catheters and Cuffs	36
Systemic Antibiotic Prophylaxis	39
Antibiotic/Antiseptic Ointments	40
Antibiotic Lock Prophylaxis, Antimicrobial Catheter Flush and Catheter Lock Prophylaxis	41
Anticoagulants	43
Replacement of Peripheral and Midline Catheters	45
Replacement of CVCs, Including PICCs and Hemodialysis Catheters	46
Umbilical Catheters	49
Peripheral Arterial Catheters and Pressure Monitoring Devices for Adult and Pediatric Patients	51
Replacement of Administration Sets	53
Needleless Intravascular Catheter Systems	54
Performance Improvement	57
References	59

NOTICE TO READERS:

In 2009, the Centers for Disease Control and Prevention (CDC) and Healthcare Infection Control Practices Advisory Committee (HICPAC) integrated current advances in guideline production and implementation into its development process (<http://www.cdc.gov/hicpac/guidelineMethod/guidelineMethod.html>). The new methodology enables CDC and HICPAC to improve the validity and usability of its guidelines while also addressing emerging challenges in guideline development in the area of infection prevention and control. However, the *Guidelines for the Prevention of Intravascular Catheter-Related Infections* were initiated before the methodology was revised. Therefore, this guideline reflects the development methods that were used for guidelines produced prior to 2009. Future revisions will be performed using the updated methodology.

These guidelines have been developed for healthcare personnel who insert intravascular catheters and for persons responsible for surveillance and control of infections in hospital, outpatient, and home healthcare settings. This report was prepared by a working group comprising members from professional organizations representing the disciplines of critical care medicine, infectious diseases, healthcare infection control, surgery, anesthesiology, interventional radiology, pulmonary medicine, pediatric medicine, and nursing. The working group was led by the Society of Critical Care Medicine (SCCM), in collaboration with the Infectious Diseases Society of America (IDSA), Society for Healthcare Epidemiology of America (SHEA), Surgical Infection Society (SIS), American College of Chest Physicians (ACCP), American Thoracic Society (ATS), American Society of Critical Care Anesthesiologists (ASCCA), Association for Professionals in Infection Control and Epidemiology (APIC), Infusion Nurses Society (INS), Oncology Nursing Society (ONS), American Society for Parenteral and Enteral Nutrition (ASPEN), Society of Interventional Radiology (SIR), American Academy of Pediatrics (AAP), Pediatric Infectious Diseases Society (PIDS), and the Healthcare Infection Control Practices Advisory Committee (HICPAC) of the Centers for Disease Control and Prevention (CDC) and is intended to replace the Guideline for Prevention of Intravascular Catheter-Related Infections published in

2002. These guidelines are intended to provide evidence-based recommendations for preventing intravascular catheter-related infections. Major areas of emphasis include 1) educating and training healthcare personnel who insert and maintain catheters; 2) using maximal sterile barrier precautions during central venous catheter insertion; 3) using a > 0.5% chlorhexidine skin preparation with alcohol for antisepsis; 4) avoiding routine replacement of central venous catheters as a strategy to prevent infection; and 5) using antiseptic/antibiotic impregnated short-term central venous catheters and chlorhexidine impregnated sponge dressings if the rate of infection is not decreasing despite adherence to other strategies (i.e., education and training, maximal sterile barrier precautions, and >0.5% chlorhexidine preparations with alcohol for skin antisepsis). These guidelines also emphasize performance improvement by implementing bundled strategies, and documenting and reporting rates of compliance with all components of the bundle as benchmarks for quality assurance and performance improvement.

As in previous guidelines issued by CDC and HICPAC, each recommendation is categorized on the basis of existing scientific data, theoretical rationale, applicability, and economic impact. The system for categorizing recommendations in this guideline is as follows:

Category IA. Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiologic studies.

Category IB. Strongly recommended for implementation and supported by some experimental, clinical, or epidemiologic studies and a strong theoretical rationale; or an accepted practice (e.g., aseptic technique) supported by limited evidence.

Category IC. Required by state or federal regulations, rules, or standards.

Category II. Suggested for implementation and supported by suggestive clinical or epidemiologic studies or a theoretical rationale.

Unresolved issue. Represents an unresolved issue for which evidence is insufficient or no consensus regarding efficacy exists.

Introduction

In the United States, 15 million central vascular catheter (CVC) days (i.e., the total number of days of exposure to CVCs among all patients in the selected population during the

selected time period) occur in intensive care units (ICUs) each year [1]. Studies have variously addressed catheter-related bloodstream infections (CRBSI). These infections independently increase hospital costs and length of stay [2-5], but have not generally been shown to independently increase mortality. While 80,000 CRBSIs occur in ICUs each year [1], a total of 250,000 cases of BSIs have been estimated to occur annually, if entire hospitals are assessed [6]. By several analyses, the cost of these infections is substantial, both in terms of morbidity and financial resources expended. To improve patient outcome and to reduce healthcare costs, there is considerable interest by healthcare providers, insurers, regulators, and patient advocates in reducing the incidence of these infections. This effort should be multidisciplinary, involving healthcare professionals who order the insertion and removal of CVCs, those personnel who insert and maintain intravascular catheters, infection control personnel, healthcare managers including the chief executive officer (CEO) and those who allocate resources, and patients who are capable of assisting in the care of their catheters.

The goal of an effective prevention program should be the elimination of CRBSI from all patient-care areas. Although this is challenging, programs have demonstrated success, but sustained elimination requires continued effort. The goal of the measures discussed in this document is to reduce the rate to as low as feasible given the specific patient population being served, the universal presence of microorganisms in the human environment, and the limitations of current strategies and technologies.

Summary of Recommendations

Education, Training and Staffing

1. Educate healthcare personnel regarding the indications for intravascular catheter use, proper procedures for the insertion and maintenance of intravascular catheters, and appropriate infection control measures to prevent intravascular catheter-related infections [7–15]. Category IA
2. Periodically assess knowledge of and adherence to guidelines for all personnel involved in the insertion and maintenance of intravascular catheters [7–15]. Category IA

3. Designate only trained personnel who demonstrate competence for the insertion and maintenance of peripheral and central intravascular catheters. [14–28]. Category IA
4. Ensure appropriate nursing staff levels in ICUs. Observational studies suggest that a higher proportion of "pool nurses" or an elevated patient–to-nurse ratio is associated with CRBSI in ICUs where nurses are managing patients with CVCs [29–31]. Category IB

Selection of Catheters and Sites

Peripheral Catheters and Midline Catheters

1. In adults, use an upper-extremity site for catheter insertion. Replace a catheter inserted in a lower extremity site to an upper extremity site as soon as possible. Category II
2. In pediatric patients, the upper or lower extremities or the scalp (in neonates or young infants) can be used as the catheter insertion site [32, 33]. Category II
3. Select catheters on the basis of the intended purpose and duration of use, known infectious and non-infectious complications (e.g., phlebitis and infiltration), and experience of individual catheter operators [33–35]. Category IB
4. Avoid the use of steel needles for the administration of fluids and medication that might cause tissue necrosis if extravasation occurs [33, 34]. Category IA
5. Use a midline catheter or peripherally inserted central catheter (PICC), instead of a short peripheral catheter, when the duration of IV therapy will likely exceed six days. Category II
6. Evaluate the catheter insertion site daily by palpation through the dressing to discern tenderness and by inspection if a transparent dressing is in use. Gauze and opaque dressings should not be removed if the patient has no clinical signs of infection. If the patient has local tenderness or other signs of possible CRBSI, an opaque dressing should be removed and the site inspected visually. Category II
7. Remove peripheral venous catheters if the patients develops signs of phlebitis (warmth, tenderness, erythema or palpable venous cord), infection, or a malfunctioning catheter [36]. Category IB

Central Venous Catheters

1. Weigh the risks and benefits of placing a central venous device at a recommended site to reduce infectious complications against the risk for mechanical complications (e.g., pneumothorax, subclavian artery puncture, subclavian vein laceration, subclavian vein stenosis, hemothorax, thrombosis, air embolism, and catheter misplacement) [37–53].
Category IA
2. Avoid using the femoral vein for central venous access in adult patients [38, 50, 51, 54].
Category 1A
3. Use a subclavian site, rather than a jugular or a femoral site, in adult patients to minimize infection risk for nontunneled CVC placement [50–52]. Category IB
4. No recommendation can be made for a preferred site of insertion to minimize infection risk for a tunneled CVC. Unresolved issue
5. Avoid the subclavian site in hemodialysis patients and patients with advanced kidney disease, to avoid subclavian vein stenosis [53,55–58]. Category IA
6. Use a fistula or graft in patients with chronic renal failure instead of a CVC for permanent access for dialysis [59]. Category 1A
7. Use ultrasound guidance to place central venous catheters (if this technology is available) to reduce the number of cannulation attempts and mechanical complications. Ultrasound guidance should only be used by those fully trained in its technique. [60–64].
Category 1B
8. Use a CVC with the minimum number of ports or lumens essential for the management of the patient [65–68]. Category IB
9. No recommendation can be made regarding the use of a designated lumen for parenteral nutrition. Unresolved issue
10. Promptly remove any intravascular catheter that is no longer essential [69–72].
Category IA
11. When adherence to aseptic technique cannot be ensured (i.e catheters inserted during a medical emergency), replace the catheter as soon as possible, i.e, within 48 hours [37,73–76]. Category IB

Hand Hygiene and Aseptic Technique

1. Perform hand hygiene procedures, either by washing hands with conventional soap and water or with alcohol-based hand rubs (ABHR). Hand hygiene should be performed before and after palpating catheter insertion sites as well as before and after inserting, replacing, accessing, repairing, or dressing an intravascular catheter. Palpation of the insertion site should not be performed after the application of antiseptic, unless aseptic technique is maintained [12, 77–79]. Category IB
2. Maintain aseptic technique for the insertion and care of intravascular catheters [37, 73, 74, 76]. Category IB
3. Wear clean gloves, rather than sterile gloves, for the insertion of peripheral intravascular catheters, if the access site is not touched after the application of skin antiseptics. Category IC
4. Sterile gloves should be worn for the insertion of arterial, central, and midline catheters [37, 73, 74, 76]. Category IA
5. Use new sterile gloves before handling the new catheter when guidewire exchanges are performed. Category II
6. Wear either clean or sterile gloves when changing the dressing on intravascular catheters. Category IC

Maximal Sterile Barrier Precautions

1. Use maximal sterile barrier precautions, including the use of a cap, mask, sterile gown, sterile gloves, and a sterile full body drape, for the insertion of CVCs, PICCs, or guidewire exchange [14, 75, 76, 80]. Category IB
2. Use a sterile sleeve to protect pulmonary artery catheters during insertion [81]. Category IB

Skin Preparation

1. Prepare clean skin with an antiseptic (70% alcohol, tincture of iodine, or alcoholic chlorhexidine gluconate solution) before peripheral venous catheter insertion [82].
Category IB
2. Prepare clean skin with a >0.5% chlorhexidine preparation with alcohol before central venous catheter and peripheral arterial catheter insertion and during dressing changes. If there is a contraindication to chlorhexidine, tincture of iodine, an iodophor, or 70% alcohol can be used as alternatives [82, 83]. Category IA
3. No comparison has been made between using chlorhexidine preparations with alcohol and povidone-iodine in alcohol to prepare clean skin. Unresolved issue.
4. No recommendation can be made for the safety or efficacy of chlorhexidine in infants aged <2 months. Unresolved issue
5. Antiseptics should be allowed to dry according to the manufacturer's recommendation prior to placing the catheter [82, 83]. Category IB

Catheter Site Dressing Regimens

1. Use either sterile gauze or sterile, transparent, semipermeable dressing to cover the catheter site [84–87]. Category IA
2. If the patient is diaphoretic or if the site is bleeding or oozing, use a gauze dressing until this is resolved [84–87]. Category II
3. Replace catheter site dressing if the dressing becomes damp, loosened, or visibly soiled [84, 85]. Category IB
4. Do not use topical antibiotic ointment or creams on insertion sites, except for dialysis catheters, because of their potential to promote fungal infections and antimicrobial resistance [88, 89]. Category IB
5. Do not submerge the catheter or catheter site in water. Showering should be permitted if precautions can be taken to reduce the likelihood of introducing organisms into the

- catheter (e.g., if the catheter and connecting device are protected with an impermeable cover during the shower) [90–92]. Category IB
6. Replace dressings used on short-term CVC sites every 2 days for gauze dressings.
Category II
 7. Replace dressings used on short-term CVC sites at least every 7 days for transparent dressings, except in those pediatric patients in which the risk for dislodging the catheter may outweigh the benefit of changing the dressing [87, 93]. Category IB
 8. Replace transparent dressings used on tunneled or implanted CVC sites no more than once per week (unless the dressing is soiled or loose), until the insertion site has healed.
Category II
 9. No recommendation can be made regarding the necessity for any dressing on well-healed exit sites of long-term cuffed and tunneled CVCs. Unresolved issue
 10. Ensure that catheter site care is compatible with the catheter material [94, 95].
Category IB
 11. Use a sterile sleeve for all pulmonary artery catheters [81]. Category IB
 12. Use a chlorhexidine-impregnated sponge dressing for temporary short-term catheters in patients older than 2 months of age if the CLABSI rate is not decreasing despite adherence to basic prevention measures, including education and training, appropriate use of chlorhexidine for skin antisepsis, and MSB [93, 96–98]. Category 1B
 13. No recommendation is made for other types of chlorhexidine dressings. Unresolved issue
 14. Monitor the catheter sites visually when changing the dressing or by palpation through an intact dressing on a regular basis, depending on the clinical situation of the individual patient. If patients have tenderness at the insertion site, fever without obvious source, or other manifestations suggesting local or bloodstream infection, the dressing should be removed to allow thorough examination of the site [99–101]. Category IB
 15. Encourage patients to report any changes in their catheter site or any new discomfort to their provider. Category II

Patient Cleansing

Use a 2% chlorhexidine wash for daily skin cleansing to reduce CRBSI [102–104].

Category II

Catheter Securement Devices

Use a sutureless securement device to reduce the risk of infection for intravascular catheters [105]. Category II

Antimicrobial/Antiseptic Impregnated Catheters and Cuffs

Use a chlorhexidine/silver sulfadiazine or minocycline/rifampin -impregnated CVC in patients whose catheter is expected to remain in place >5 days if, after successful implementation of a comprehensive strategy to reduce rates of CLABSI, the CLABSI rate is not decreasing. The comprehensive strategy should include at least the following three components: educating persons who insert and maintain catheters, use of maximal sterile barrier precautions, and a >0.5% chlorhexidine preparation with alcohol for skin antisepsis during CVC insertion [106–113]. Category IA

Systemic Antibiotic Prophylaxis

Do not administer systemic antimicrobial prophylaxis routinely before insertion or during use of an intravascular catheter to prevent catheter colonization or CRBSI [114].

Category IB

Antibiotic/Antiseptic Ointments

Use povidone iodine antiseptic ointment or bacitracin/gramicidin/ polymyxin B ointment at the hemodialysis catheter exit site after catheter insertion and at the end of each dialysis session only if this ointment does not interact with the material of the hemodialysis catheter per manufacturer's recommendation [59, 115–119]. Category IB

Antibiotic Lock Prophylaxis, Antimicrobial Catheter Flush and Catheter Lock Prophylaxis

Use prophylactic antimicrobial lock solution in patients with long term catheters who have a history of multiple CRBSI despite optimal maximal adherence to aseptic technique [120– 138]. Category II

Anticoagulants

Do not routinely use anticoagulant therapy to reduce the risk of catheter-related infection in general patient populations [139]. Category II

Replacement of Peripheral and Midline Catheters

1. There is no need to replace peripheral catheters more frequently than every 72-96 hours to reduce risk of infection and phlebitis in adults [36, 140, 141]. Category 1B
2. No recommendation is made regarding replacement of peripheral catheters in adults only when clinically indicated [142–144]. Unresolved issue
3. Replace peripheral catheters in children only when clinically indicated [32, 33]. Category 1B
4. Replace midline catheters only when there is a specific indication. Category II

Replacement of CVCs, Including PICCs and Hemodialysis Catheters

1. Do not routinely replace CVCs, PICCs, hemodialysis catheters, or pulmonary artery catheters to prevent catheter-related infections. Category IB
2. Do not remove CVCs or PICCs on the basis of fever alone. Use clinical judgment regarding the appropriateness of removing the catheter if infection is evidenced elsewhere or if a noninfectious cause of fever is suspected. Category II
3. Do not use guidewire exchanges routinely for non-tunneled catheters to prevent infection. Category IB
4. Do not use guidewire exchanges to replace a non-tunneled catheter suspected of infection. Category IB

5. Use a guidewire exchange to replace a malfunctioning non-tunneled catheter if no evidence of infection is present. Category IB
6. Use new sterile gloves before handling the new catheter when guidewire exchanges are performed. Category II

Umbilical Catheters

1. Remove and do not replace umbilical artery catheters if any signs of CRBSI, vascular insufficiency in the lower extremities, or thrombosis are present [145]. Category II
2. Remove and do not replace umbilical venous catheters if any signs of CRBSI or thrombosis are present [145]. Category II
3. No recommendation can be made regarding attempts to salvage an umbilical catheter by administering antibiotic treatment through the catheter. Unresolved issue
4. Cleanse the umbilical insertion site with an antiseptic before catheter insertion. Avoid tincture of iodine because of the potential effect on the neonatal thyroid. Other iodine-containing products (e.g., povidone iodine) can be used [146– 150]. Category IB
5. Do not use topical antibiotic ointment or creams on umbilical catheter insertion sites because of the potential to promote fungal infections and antimicrobial resistance [88, 89]. Category IA
6. Add low-doses of heparin (0.25—1.0 U/ml) to the fluid infused through umbilical arterial catheters [151–153]. Category IB
7. Remove umbilical catheters as soon as possible when no longer needed or when any sign of vascular insufficiency to the lower extremities is observed. Optimally, umbilical artery catheters should not be left in place >5 days [145, 154]. Category II
8. Umbilical venous catheters should be removed as soon as possible when no longer needed, but can be used up to 14 days if managed aseptically [155, 156]. Category II
9. An umbilical catheter may be replaced if it is malfunctioning, and there is no other indication for catheter removal, and the total duration of catheterization has not exceeded 5 days for an umbilical artery catheter or 14 days for an umbilical vein catheter. Category II

Peripheral Arterial Catheters and Pressure Monitoring Devices for Adult and Pediatric

Patients

1. In adults, use of the radial, brachial or dorsalis pedis sites is preferred over the femoral or axillary sites of insertion to reduce the risk of infection [46, 47, 157, 158]. Category IB
2. In children, the brachial site should not be used. The radial, dorsalis pedis, and posterior tibial sites are preferred over the femoral or axillary sites of insertion [46]. Category II
3. A minimum of a cap, mask, sterile gloves and a small sterile fenestrated drape should be used during peripheral arterial catheter insertion [47, 158, 159]. Category IB
4. During axillary or femoral artery catheter insertion, maximal sterile barriers precautions should be used. Category II
5. Replace arterial catheters only when there is a clinical indication. Category II
6. Remove the arterial catheter as soon as it is no longer needed. Category II
7. Use disposable, rather than reusable, transducer assemblies when possible [160–164]. Category IB
8. Do not routinely replace arterial catheters to prevent catheter-related infections [165, 166, 167, 168]. Category II
9. Replace disposable or reusable transducers at 96-hour intervals. Replace other components of the system (including the tubing, continuous-flush device, and flush solution) at the time the transducer is replaced [37, 161]. Category IB
10. Keep all components of the pressure monitoring system (including calibration devices and flush solution) sterile [160, 169–171]. Category IA
11. Minimize the number of manipulations of and entries into the pressure monitoring system. Use a closed flush system (i.e, continuous flush), rather than an open system (i.e, one that requires a syringe and stopcock), to maintain the patency of the pressure monitoring catheters [163, 172]. Category II
12. When the pressure monitoring system is accessed through a diaphragm, rather than a stopcock, scrub the diaphragm with an appropriate antiseptic before accessing the system [163]. Category IA

13. Do not administer dextrose-containing solutions or parenteral nutrition fluids through the pressure monitoring circuit [163, 173, 174]. Category IA
14. Sterilize reusable transducers according to the manufacturers' instructions if the use of disposable transducers is not feasible [163, 173–176]. Category IA

Replacement of Administration Sets

1. In patients not receiving blood, blood products or fat emulsions, replace administration sets that are continuously used, including secondary sets and add-on devices, no more frequently than at 96-hour intervals, [177] but at least every 7 days [178–181]. Category IA
2. No recommendation can be made regarding the frequency for replacing intermittently used administration sets. Unresolved issue
3. No recommendation can be made regarding the frequency for replacing needles to access implantable ports. Unresolved issue
4. Replace tubing used to administer blood, blood products, or fat emulsions (those combined with amino acids and glucose in a 3-in-1 admixture or infused separately) within 24 hours of initiating the infusion [182–185]. Category IB
5. Replace tubing used to administer propofol infusions every 6 or 12 hours, when the vial is changed, per the manufacturer's recommendation (FDA website Medwatch) [186]. Category IA
6. No recommendation can be made regarding the length of time a needle used to access implanted ports can remain in place. Unresolved issue

Needleless Intravascular Catheter Systems

1. Change the needleless components at least as frequently as the administration set. There is no benefit to changing these more frequently than every 72 hours. [39, 187–193]. Category II

2. Change needleless connectors no more frequently than every 72 hours or according to manufacturers' recommendations for the purpose of reducing infection rates [187, 189, 192, 193]. Category II
3. Ensure that all components of the system are compatible to minimize leaks and breaks in the system [194]. Category II
4. Minimize contamination risk by scrubbing the access port with an appropriate antiseptic (chlorhexidine, povidone iodine, an iodophor, or 70% alcohol) and accessing the port only with sterile devices [189, 192, 194–196]. Category IA
5. Use a needleless system to access IV tubing. Category IC
6. When needleless systems are used, a split septum valve may be preferred over some mechanical valves due to increased risk of infection with the mechanical valves [197–200]. Category II

Performance Improvement

Use hospital-specific or collaborative-based performance improvement initiatives in which multifaceted strategies are "bundled" together to improve compliance with evidence-based recommended practices [15, 69, 70, 201–205]. Category IB

Background Information

Terminology and Estimates of Risk

The terminology used to identify different types of catheters is confusing, because many clinicians and researchers use different aspects of the catheter for informal reference. A catheter can be designated by the type of vessel it occupies (e.g., peripheral venous, central venous, or arterial); its intended life span (e.g., temporary or short-term versus permanent or long-term); its site of insertion (e.g., subclavian, femoral, internal jugular, peripheral, and peripherally inserted central catheter [PICC]); its pathway from skin to vessel (e.g., tunneled versus nontunneled); its physical length (e.g., long versus short); or some special characteristic of the catheter (e.g., presence or absence of a cuff, impregnation with heparin, antibiotics or antiseptics, and the number of lumens). To accurately define a specific type of catheter, all of these aspects should be described (Table 1).

Likewise the terms used to describe intravascular catheter-related infections can also be confusing because catheter-related bloodstream infection (CRBSI) and central line–associated bloodstream infection (CLABSI) are often used interchangeably even though the meanings differ.

CRBSI is a clinical definition, used when diagnosing and treating patients, that requires specific laboratory testing that more thoroughly identifies the catheter as the source of the BSI. It is not typically used for surveillance purposes. It is often problematic to precisely establish if a BSI is a CRBSI due to the clinical needs of the patient (the catheter is not always pulled), limited availability of microbiologic methods (many labs do not use quantitative blood cultures or differential time to positivity), and procedural compliance by direct care personnel (labeling must be accurate). Simpler definitions are often used for surveillance purposes. For example, CLABSI is a term used by CDC’s National Healthcare Safety Network (NHSN) (visit NHSN CLABSI information) [206]. A CLABSI is a primary BSI in a patient that had a central line within the 48-hour period before the development of the BSI and is not bloodstream related to an infection at another site. However, since some BSIs are secondary to other sources other than the central line (e.g., pancreatitis, mucositis) that may not be easily recognized, the CLABSI surveillance definition may overestimate the true incidence of CRBSI.

Table 1. Catheters used for venous and arterial access.

Catheter type	Entry Site	Length	Comments
Peripheral venous catheters	Usually inserted in veins of forearm or hand	<3 inches	Phlebitis with prolonged use; rarely associated with bloodstream infection
Peripheral arterial catheters	Usually inserted in radial artery; can be placed in femoral, axillary, brachial, posterior tibial arteries	<3 inches	Low infection risk; rarely associated with bloodstream infection
Midline catheters	Inserted via the antecubital fossa into the proximal basilic or cephalic veins; does not enter central veins, peripheral catheters	3 to 8 inches	Anaphylactoid reactions have been reported with catheters made of elastomeric hydrogel; lower rates of phlebitis than short peripheral catheters
Nontunneled central venous catheters	Percutaneously inserted into central veins (subclavian, internal jugular, or femoral)	≥8 cm depending on patient size	Account for majority of CRBSI
Pulmonary artery catheters	Inserted through a Teflon® introducer in a central vein (subclavian, internal jugular, or femoral)	≥30 cm depending on patient size	Usually heparin bonded; similar rates of bloodstream infection as CVCs; subclavian site preferred to reduce infection risk
Peripherally inserted central venous catheters (PICC)	Inserted into basilic, cephalic, or brachial veins and enter the superior vena cava	≥20 cm depending on patient size	Lower rate of infection than nontunneled CVCs
Tunneled central venous catheters	Implanted into subclavian, internal jugular, or femoral veins	≥8 cm depending on patient size	Cuff inhibits migration of organisms into catheter tract; lower rate of infection than nontunneled CVC
Totally implantable	Tunneled beneath skin and have subcutaneous port accessed with a needle; implanted in subclavian or internal jugular vein	≥8 cm depending on patient size	Lowest risk for CRBSI; improved patient self-image; no need for local catheter-site care; surgery required for catheter removal
Umbilical catheters	Inserted into either umbilical vein or umbilical artery	≤6 cm depending on patient size	Risk for CRBSI similar with catheters placed in umbilical vein versus artery

Epidemiology and Microbiology in Adult and Pediatric Patients

National estimates of CLABSI rates are available through CDC's NHSN, a surveillance system for healthcare-associated infections, and are available on CDC's website. A recent report highlights data from 1,545 hospitals in 48 States and the District of Columbia that monitor infections in one or more ICUs and/or non-ICUs (e.g., patient care areas, wards) [207]. Because BSI rates are influenced by patient-related factors, such as severity of illness and type of illness (e.g., third-degree burns versus post-cardiac surgery), by catheter-related factors, (such as the condition under which the catheter was placed and catheter type), and by institutional factors (e.g., bed-size, academic affiliation), these aggregate, risk-adjusted rates can be used as benchmarks against which hospitals can make intra-and inter-facility comparisons.

The most commonly reported causative pathogens remain coagulase-negative staphylococci, *Staphylococcus aureus*, enterococci, and *Candida* spp [208]. Gram negative bacilli accounted for 19% and 21% of CLABSIs reported to CDC [209] and the Surveillance and Control of Pathogens of Epidemiological Importance (SCOPE) database, respectively [208].

For all common pathogens causing CLABSIs, antimicrobial resistance is a problem, particularly in ICUs. Although methicillin-resistant *Staphylococcus aureus* (MRSA) now account for more than 50% of all *Staphylococcus aureus* isolates obtained in ICUs, the incidence of MRSA CLABSIs has decreased in recent years, perhaps as a result of prevention efforts [210]. For gram negative rods, antimicrobial resistance to third generation cephalosporins among *Klebsiella pneumoniae* and *E. coli* has increased significantly as has imipenem and ceftazidime resistance among *Pseudomonas aeruginosa* [209]. *Candida* spp. are increasingly noted to be fluconazole resistant.

Pathogenesis

There are four recognized routes for contamination of catheters: 1) migration of skin organisms at the insertion site into the cutaneous catheter tract and along the surface of the catheter with colonization of the catheter tip; this is the most common route of infection for short-term catheters [37, 211, 212]; 2) direct contamination of the catheter or catheter hub by

contact with hands or contaminated fluids or devices [213, 214]; 3) less commonly, catheters might become hematogenously seeded from another focus of infection [215]; and 4) rarely, infusate contamination might lead to CRBSI [216].

Important pathogenic determinants of CRBSI are 1) the material of which the device is made; 2) the host factors consisting of protein adhesions, such as fibrin and fibronectin, that form a sheath around the catheter [217]; and 3) the intrinsic virulence factors of the infecting organism, including the extracellular polymeric substance (EPS) produced by the adherent organisms [218]. Some catheter materials also have surface irregularities that enhance the microbial adherence of certain species (e.g., *S. epidermidis* and *C. albicans*) [219, 220]. Catheters made of these materials are especially vulnerable to microbial colonization and subsequent infection. Due to the formation of the fibrin sheath, silastic catheters are associated with higher risk of catheter infections than polyurethane catheters [217]. On the other hand, biofilm formation by *C. albicans* occurs more readily on silicone elastomer catheter surfaces than polyurethane catheters [219]. Modification of the biomaterial surface properties has been shown to influence the ability of *C. albicans* to form biofilm [220]. Additionally, certain catheter materials are more thrombogenic than others, a characteristic that also might predispose to catheter colonization and infection [221, 222]. This association has led to emphasis on preventing catheter-related thrombus as an additional mechanism for reducing CRBSI [223, 224].

The adherence properties of a given microorganism in relationship to host factors are also important in the pathogenesis of CRBSI. For example, *S. aureus* can adhere to host proteins (e.g., fibrinogen, fibronectin) commonly present on catheters by expressing clumping factors (ClfA and ClfB) that bind to the protein adhesins [217, 222, 225, 226]. Furthermore, adherence is enhanced through the production by microbial organisms, such as coagulase negative staphylococci [227, 228], *S. aureus* [229], *Pseudomonas aeruginosa* [230], and *Candida* species [231] of an extracellular polymeric substance (EPS) consisting mostly of an exopolysaccharide that forms a microbial biofilm layer [218, 232]. This biofilm matrix is enriched by divalent metallic cations, such as calcium, magnesium and iron, which make it a solid enclave in which microbial organisms can embed themselves [233–235]. Such a biofilm

potentiates the pathogenicity of various microbes by allowing them to withstand host defense mechanisms (e.g., acting as a barrier to engulfment and killing by polymorphonuclear leukocytes) or by making them less susceptible to antimicrobial agents (e.g., forming a matrix that binds antimicrobials before their contact with the organism cell wall or providing for a population of metabolically quiescent, antimicrobial tolerant "persister" cells) [228, 236, 237]. Some *Candida* spp., in the presence of dextrose-containing fluids, produce slime similar to that of their bacterial counterparts, potentially explaining the increased proportion of BSIs caused by fungal pathogens among patients receiving parenteral nutrition fluids [238].

Strategies for Prevention of Catheter-Related Infections in Adult and Pediatric Patients

Education, Training and Staffing

Recommendations

1. Educate healthcare personnel regarding the indications for intravascular catheter use, proper procedures for the insertion and maintenance of intravascular catheters, and appropriate infection control measures to prevent intravascular catheter-related infections [7–15]. Category IA
2. Periodically assess knowledge of and adherence to guidelines for all personnel involved in the insertion and maintenance of intravascular catheters [7–15].
Category IA
3. Designate only trained personnel who demonstrate competence for the insertion and maintenance of peripheral and central intravascular catheters. [14–28]. Category IA
4. Ensure appropriate nursing staff levels in ICUs. Observational studies suggest that a higher proportion of "pool nurses" or an elevated patient-to-nurse ratio is associated with CRBSI in ICUs where nurses are managing patients with CVCs [29–31]. Category IB

Background

Well-organized programs that enable healthcare providers to become educated and to provide, monitor, and evaluate care are critical to the success of this effort. Reports spanning

the past four decades have consistently demonstrated that risk for infection declines following standardization of aseptic care [7, 12, 14, 15, 239–241] and that insertion and maintenance of intravascular catheters by inexperienced staff might increase the risk for catheter colonization and CRBSI [15, 242]. Specialized "IV teams" have shown unequivocal effectiveness in reducing the incidence of CRBSI, associated complications, and costs [16–26]. Additionally, infection risk increases with nursing staff reductions below a critical level [30].

Selection of Catheters and Sites

Peripheral and Midline Catheter Recommendations

1. In adults, use an upper-extremity site for catheter insertion. Replace a catheter inserted in a lower extremity site to an upper extremity site as soon as possible. Category II
2. In pediatric patients, the upper or lower extremities or the scalp (in neonates or young infants) can be used as the catheter insertion site [32, 33]. Category II
3. Select catheters on the basis of the intended purpose and duration of use, known infectious and non-infectious complications (e.g., phlebitis and infiltration), and experience of individual catheter operators [33–35]. Category IB
4. Avoid the use of steel needles for the administration of fluids and medication that might cause tissue necrosis if extravasation occurs [33, 34]. Category IA
5. Use a midline catheter or peripherally inserted central catheter (PICC), instead of a short peripheral catheter, when the duration of IV therapy will likely exceed six days. Category II
6. Evaluate the catheter insertion site daily by palpation through the dressing to discern tenderness and by inspection if a transparent dressing is in use. Gauze and opaque dressings should not be removed if the patient has no clinical signs of infection. If the patient has local tenderness or other signs of possible CRBSI, an opaque dressing should be removed and the site inspected visually. Category II
7. Remove peripheral venous catheters if the patients develops signs of phlebitis (warmth, tenderness, erythema or palpable venous cord), infection, or a malfunctioning catheter [36]. Category IB

Central Venous Catheters Recommendations

1. Weigh the risks and benefits of placing a central venous device at a recommended site to reduce infectious complications against the risk for mechanical complications (e.g., pneumothorax, subclavian artery puncture, subclavian vein laceration, subclavian vein stenosis, hemothorax, thrombosis, air embolism, and catheter misplacement) [37–53].
Category IA
2. Avoid using the femoral vein for central venous access in adult patients [38, 50, 51, 54].
Category 1A
3. Use a subclavian site, rather than a jugular or a femoral site, in adult patients to minimize infection risk for nontunneled CVC placement [50–52]. Category IB
4. No recommendation can be made for a preferred site of insertion to minimize infection risk for a tunneled CVC. Unresolved issue
5. Avoid the subclavian site in hemodialysis patients and patients with advanced kidney disease, to avoid subclavian vein stenosis [53, 55–58]. Category IA
6. Use a fistula or graft in patients with chronic renal failure instead of a CVC for permanent access for dialysis [59]. Category 1A
7. Use ultrasound guidance to place central venous catheters (if this technology is available) to reduce the number of cannulation attempts and mechanical complications. Ultrasound guidance should only be used by those fully trained in its technique. [60–64].
Category 1B
8. Use a CVC with the minimum number of ports or lumens essential for the management of the patient [65–68]. Category IB
9. No recommendation can be made regarding the use of a designated lumen for parenteral nutrition. Unresolved issue
10. Promptly remove any intravascular catheter that is no longer essential [69–72].
Category IA

11. When adherence to aseptic technique cannot be ensured (i.e catheters inserted during a medical emergency), replace the catheter as soon as possible, i.e, within 48 hours [37, 73–76]. Category IB

Background

The site at which a catheter is placed influences the subsequent risk for catheter-related infection and phlebitis. The influence of site on the risk for catheter infections is related in part to the risk for thrombophlebitis and density of local skin flora.

As in adults, the use of peripheral venous catheters in pediatric patients might be complicated by phlebitis, infusion extravasation, and catheter infection [243]. Catheter location, infusion of parenteral nutritional fluids with continuous IV fat emulsions, and length of ICU stay before catheter insertion, have all increased pediatric patients' risk for phlebitis. However, contrary to the risk in adults, the risk for phlebitis in children has not increased with the duration of catheterization [243, 244].

The density of skin flora at the catheter insertion site is a major risk factor for CRBSI. No single trial has satisfactorily compared infection rates for catheters placed in jugular, subclavian, and femoral veins. In retrospective observational studies, catheters inserted into an internal jugular vein have usually been associated with higher risk for colonization and/or CRBSI than those inserted into a subclavian [37–47]. Similar findings were noted in neonates in a single retrospective study [245]. Femoral catheters have been demonstrated to have high colonization rates compared with subclavian and internal jugular sites when used in adults and, in some studies, higher rates of CLABSIs [40, 45–47, 50, 51, 246]. Femoral catheters should also be avoided, when possible, because they are associated with a higher risk for deep venous thrombosis than are internal jugular or subclavian catheters [48–50, 53, 247]. One study [38] found that the risk of infection associated with catheters placed in the femoral vein is accentuated in obese patients. In contrast to adults, studies in pediatric patients have demonstrated that femoral catheters have a low incidence of mechanical complications and might have an equivalent infection rate to that of non-femoral catheters [248–251]. Thus, in adult patients, a subclavian site is preferred for infection control purposes, although other

factors (e.g., the potential for mechanical complications, risk for subclavian vein stenosis, and catheter-operator skill) should be considered when deciding where to place the catheter.

In two meta-analyses, the use of real-time two-dimensional ultrasound for the placement of CVCs substantially decreased mechanical complications and reduced the number of attempts at required cannulation and failed attempts at cannulation compared with the standard landmark placement [60, 61]. Evidence favors the use of two-dimensional ultrasound guidance over Doppler ultrasound guidance [60]. Site selection should be guided by patient comfort, ability to secure the catheter, and maintenance of asepsis as well as patient-specific factors (e.g., preexisting catheters, anatomic deformity, and bleeding diathesis), relative risk of mechanical complications (e.g., bleeding and pneumothorax), the availability of bedside ultrasound, the experience of the person inserting the catheter, and the risk for infection.

Catheters should be inserted as great a distance as possible from open wounds. In one study, catheters inserted close to open burn wounds (i.e, 25 cm² overlapped a wound) were 1.79 times more likely to be colonized and 5.12 times more likely to be associated with bacteremia than catheters inserted farther from the wounds [252].

Type of Catheter Material. Polytetrafluoroethylene (Teflon[®]) or polyurethane catheters have been associated with fewer infectious complications than catheters made of polyvinyl chloride or polyethylene [36, 253, 254]. Steel needles used as an alternative to catheters for peripheral venous access have the same rate of infectious complications as do Teflon[®] catheters [33, 34]. However, the use of steel needles frequently is complicated by infiltration of intravenous (IV) fluids into the subcutaneous tissues, a potentially serious complication if the infused fluid is a vesicant [34].

Hand Hygiene and Aseptic Technique

Recommendations

1. Perform hand hygiene procedures, either by washing hands with conventional soap and water or with alcohol-based hand rubs (ABHR). Hand hygiene should be performed before and after palpating catheter insertion sites as well as before and after inserting, replacing, accessing, repairing, or dressing an intravascular catheter. Palpation of the

insertion site should not be performed after the application of antiseptic, unless aseptic technique is maintained [12, 77–79]. Category IB

2. Maintain aseptic technique for the insertion and care of intravascular catheters [37, 73, 74, 76]. Category IB
3. Wear clean gloves, rather than sterile gloves, for the insertion of peripheral intravascular catheters, if the access site is not touched after the application of skin antiseptics. Category IC
4. Sterile gloves should be worn for the insertion of arterial, central, and midline catheters [37, 73, 74, 76]. Category IA
5. Use new sterile gloves before handling the new catheter when guidewire exchanges are performed. Category II
6. Wear either clean or sterile gloves when changing the dressing on intravascular catheters. Category IC

Background

Hand hygiene before catheter insertion or maintenance, combined with proper aseptic technique during catheter manipulation, provides protection against infection [12]. Proper hand hygiene can be achieved through the use of either an alcohol-based product [255] or with soap and water with adequate rinsing [77]. Appropriate aseptic technique does not necessarily require sterile gloves for insertion of peripheral catheters; a new pair of disposable nonsterile gloves can be used in conjunction with a "no-touch" technique for the insertion of peripheral venous catheters. Sterile gloves must be worn for placement of central catheters since a "no-touch" technique is not possible.

Maximal Sterile Barrier Precautions

Recommendations

1. Use maximal sterile barrier precautions, including the use of a cap, mask, sterile gown, sterile gloves, and a sterile full body drape, for the insertion of CVCs, PICCs, or guidewire exchange [14, 75, 76, 80]. Category IB
2. Use a sterile sleeve to protect pulmonary artery catheters during insertion [81]. Category IB

Background

Maximum sterile barrier (MSB) precautions are defined as wearing a sterile gown, sterile gloves, and cap and using a full body drape (similar to the drapes used in the operating room) during the placement of CVC. Maximal sterile barrier precautions during insertion of CVC were compared with sterile gloves and a small drape in a randomized controlled trial. The MSB group had fewer episodes of both catheter colonization (RR = .32, 95% CI, .10–.96, P = .04) and CR-BSI (RR = .16, 95% CI, .02–1.30, P = .06). In addition, the group using MSB precautions had infections that occurred much later and contained gram negative, rather than gram positive, organisms [76]. A study of pulmonary artery catheters also secondarily demonstrated that use of MSB precautions lowered risk of infection [37]. Another study evaluated an educational program directed at improving infection control practices, especially MSB precautions. In this study, MSB precautions use increased and CRBSI decreased [14]. A small trial demonstrated a reduced risk of skin colonization at the insertion site when MSB precautions were used [OR 3.40, 95%CI 1.32 to 3.67] [80].

Skin Preparation

Recommendations

1. Prepare clean skin with an antiseptic (70% alcohol, tincture of iodine, an iodophor or chlorhexidine gluconate) before peripheral venous catheter insertion [82]. Category IB
2. Prepare clean skin with a >0.5% chlorhexidine preparation with alcohol before central venous catheter and peripheral arterial catheter insertion and during dressing changes. If there is a contraindication to chlorhexidine, tincture of iodine, an iodophor, or 70% alcohol can be used as alternatives [82, 83]. Category IA
3. No comparison has been made between using chlorhexidine preparations with alcohol and povidone-iodine in alcohol to prepare clean skin. Unresolved issue.
4. No recommendation can be made for the safety or efficacy of chlorhexidine in infants aged <2 months. Unresolved issue
5. Antiseptics should be allowed to dry according to the manufacturer's recommendation prior to placing the catheter [82, 83]. Category IB

Background

Two well-designed studies evaluating the chlorhexidine-containing cutaneous antiseptic regimen in comparison with either povidone iodine or alcohol for the care of an intravascular catheter insertion site have shown lower rates of catheter colonization or CRBSI associated with the chlorhexidine preparation [82, 83]. (The comparison of chlorhexidine gluconate alcohol to povidone iodine alcohol has not been done.) When 0.5% tincture of chlorhexidine was compared with 10% povidone iodine, no differences were seen in central venous catheter (CVC) colonization or in CRBSI [256]. In a three-armed study (2% aqueous chlorhexidine gluconate vs 10% povidone-iodine vs 70% alcohol), 2% aqueous chlorhexidine gluconate tended to decrease CRBSI compared with 10% povidone iodine or 70% alcohol [82]. A meta-analysis of 4,143 catheters suggested that chlorhexidine preparation reduced the risk of catheter related infection by 49% (95% CI .28 to .88) relative to povidone iodine [257]. An economic decision analysis based on available evidence suggested that the use of chlorhexidine, rather than povidone iodine, for CVC care would result in a 1.6% decrease in the incidence of CRBSI, a 0.23% decrease in the incidence of death, and a savings of \$113 per catheter used [258]. While chlorhexidine has become a standard antiseptic for skin preparation for the insertion of both central and peripheral venous catheters, 5% povidone iodine solution in 70% ethanol was associated with a substantial reduction of CVC-related colonization and infection compared with 10% aqueous povidone iodine [259].

Catheter Site Dressing Regimens

Recommendations

1. Use either sterile gauze or sterile, transparent, semipermeable dressing to cover the catheter site [84–87]. Category IA
2. If the patient is diaphoretic or if the site is bleeding or oozing, use gauze dressing until this is resolved [84–87]. Category II
3. Replace catheter site dressing if the dressing becomes damp, loosened, or visibly soiled [84, 85]. Category IB

4. Do not use topical antibiotic ointment or creams on insertion sites, except for dialysis catheters, because of their potential to promote fungal infections and antimicrobial resistance [88, 89]. Category IB
5. Do not submerge the catheter or catheter site in water. Showering should be permitted if precautions can be taken to reduce the likelihood of introducing organisms into the catheter (e.g., if the catheter and connecting device are protected with an impermeable cover during the shower) [90–92]. Category IB
6. Replace dressings used on short-term CVC sites every 2 days for gauze dressings. Category II
7. Replace dressings used on short-term CVC sites at least every 7 days for transparent dressings, except in those pediatric patients in which the risk for dislodging the catheter may outweigh the benefit of changing the dressing [87, 93]. Category IB
8. Replace transparent dressings used on tunneled or implanted CVC sites no more than once per week (unless the dressing is soiled or loose), until the insertion site has healed. Category II
9. No recommendation can be made regarding the necessity for any dressing on well-healed exit sites of long-term cuffed and tunneled CVCs. Unresolved issue
10. Ensure that catheter site care is compatible with the catheter material [94, 95]. Category IB
11. Use a sterile sleeve for all pulmonary artery catheters [80]. Category IB
12. Use a chlorhexidine-impregnated sponge dressing for temporary short-term catheters in patients older than 2 months of age if the CLABSI rate is not decreasing despite adherence to basic prevention measures, including education and training, appropriate use of chlorhexidine for skin antisepsis, and MSB [93, 96–98]. Category 1B
13. No recommendation is made for other types of chlorhexidine dressings. Unresolved issue
14. Monitor the catheter sites visually when changing the dressing or by palpation through an intact dressing on a regular basis, depending on the clinical situation of the individual patient. If patients have tenderness at the insertion site, fever without obvious source,

or other manifestations suggesting local or bloodstream infection, the dressing should be removed to allow thorough examination of the site [99–101]. Category IB

15. Encourage patients to report any changes in their catheter site or any new discomfort to their provider. Category II

Background

Transparent, semi-permeable polyurethane dressings permit continuous visual inspection of the catheter site and require less frequent changes than do standard gauze and tape dressings. In the largest controlled trial of dressing regimens on peripheral catheters, the infectious morbidity associated with the use of transparent dressings on approximately 2,000 peripheral catheters was examined [254]. Data from this study suggest that the rate of colonization among catheters dressed with transparent dressings (5.7%) is comparable to that of those dressed with gauze (4.6%) and that no clinically substantial differences exist in the incidence of either catheter site colonization or phlebitis. Furthermore, these data suggest that transparent dressings can be safely left on peripheral venous catheters for the duration of catheter insertion without increasing the risk for thrombophlebitis [254].

A meta-analysis has assessed studies that compared the risk for CRBSIs using transparent dressings versus using gauze dressing [260]. The risk for CRBSIs did not differ between the groups. The choice of dressing can be a matter of preference. If blood is oozing from the catheter insertion site, gauze dressing is preferred. Another systemic review of randomized controlled trials comparing gauze and tape to transparent dressings found no significant differences between dressing types in CRBSIs, catheter tip colonization, or skin colonization [261].

Chlorhexidine impregnated dressings have been used to reduce the risk of CRBSI. In the largest multicenter randomized controlled trial published to date comparing chlorhexidine impregnated sponge dressings vs standard dressings in ICU patients, rates of CRBSIs were reduced even when background rates of infection were low. In this study, 1636 patients (3778 catheters, 28 931 catheter-days) were evaluated. The chlorhexidine- impregnated sponge dressings decreased the rates of major CRBSIs (10/1953 [0.5%], 0.6 per 1000 catheter-days vs 19/1825 [1.1%], 1.4 per 1000 catheter-days; hazard ratio [HR], 0.39 [95% confidence interval

{CI}, .17–.93]; $P = .03$) and CRBSIs (6/1953 catheters, 0.40 per 1000 catheter-days vs 17/1825 catheters, 1.3 per 1000 catheter-days; HR, 0.24 [95% CI, .09–.65]) [93]. A randomized controlled study of polyurethane or a chlorhexidine impregnated sponge dressing in 140 children showed no statistical difference in BSIs; however, the chlorhexidine group had lower rates of CVC colonization [98]. In 601 cancer patients receiving chemotherapy, the incidence of CRBSI was reduced in patients receiving the chlorhexidine impregnated sponge dressing compared with standard dressings ($P = .016$, relative risk 0.54; confidence interval 0.31–.94) [262]. A meta-analysis that included eight randomized controlled trials demonstrated that chlorhexidine impregnated sponge dressings are associated with a reduction of vascular and epidural catheter exit site colonization but no significant reduction in CRBSI (2.2% versus 3.8%, OR 0.58, 95% CI: .29–1.14, $p = .11$) [97].

Although data regarding the use of a chlorhexidine impregnated sponge dressing in children are limited, one randomized, controlled study involving 705 neonates reported a substantial decrease in colonized catheters in infants in the chlorhexidine impregnated sponge dressing group compared with the group that had standard dressings (15% versus 24%; RR = .6; 95% CI 0.5–.9), but no difference in the rates of CRBSI or BSI without a source. Chlorhexidine impregnated sponge dressings were associated with localized contact dermatitis in infants of very low birth weight. In 98 neonates with very low birth weight, 15 (15%) developed localized contact dermatitis; four (1.5%) of 237 neonates weighing >1,000 g developed this reaction ($P < .0001$). Infants with gestational age <26 weeks who had CVCs placed at age <8 days were at increased risk for having localized contact dermatitis, whereas no infants in the control group developed this local reaction [96].

Patient Cleansing

Recommendation

Use a 2% chlorhexidine wash for daily skin cleansing to reduce CRBSI [102–104].

Category II

Background

Daily cleansing of ICU patients with a 2% chlorhexidine impregnated washcloth may be a simple, effective strategy to decrease the rate of primary BSIs. In a single center study of 836 ICU patients, patients receiving the chlorhexidine intervention were significantly less likely to acquire a primary BSI (4.1 vs 10.4 infections per 1000 patient days; incidence difference, 6.3 [95% confidence interval, 1.2–11.0]) than those bathed with soap and water [102].

Catheter Securement Devices

Recommendation

Use a sutureless securement device to reduce the risk of infection for intravascular catheters [105]. Category II

Background

Catheter stabilization is recognized as an intervention to decrease the risk for phlebitis, catheter migration and dislodgement, and may be advantageous in preventing CRBSIs. Pathogenesis of CRBSI occurs via migration of skin flora through the percutaneous entry site. Sutureless securement devices avoid disruption around the catheter entry site and may decrease the degree of bacterial colonization. [105]. Using a sutureless securement device also mitigates the risk of sharps injury to the healthcare provider from inadvertent needlestick injury.

Antimicrobial/Antiseptic Impregnated Catheters and Cuffs

Recommendation

Use a chlorhexidine/silver sulfadiazine or minocycline/ rifampin -impregnated CVC in patients whose catheter is expected to remain in place >5 days if, after successful implementation of a comprehensive strategy to reduce rates of CLABSI, the CLABSI rate is not decreasing. The comprehensive strategy should include at least the following three components: educating persons who insert and maintain catheters, use of maximal sterile barrier precautions, and a >0.5% chlorhexidine preparation with alcohol for skin antisepsis during CVC insertion [106–113]. Category IA

Background

Certain catheters and cuffs that are coated or impregnated with antimicrobial or antiseptic agents can decrease the risk for CRBSI and potentially decrease hospital costs associated with treating CRBSIs, despite the additional acquisition cost of an antimicrobial/antiseptic impregnated catheter [110]. Nearly all of the studies involving antimicrobial/antiseptic-impregnated catheters have been conducted using triple-lumen, uncuffed catheters in adult patients whose catheters remained in place <30 days. While most of the studies have been conducted in adults, these catheters have been approved by FDA for use in patients weighing >3 kg. Two non-randomized studies [112, 113] in pediatric ICU patients suggest that these catheters might reduce risk of catheter-associated infection. No antiseptic or antimicrobial impregnated catheters currently are available for use in infants weighing <3kg.

Chlorhexidine/Silver Sulfadiazine Catheters coated with chlorhexidine/silver sulfadiazine only on the external luminal surface have been studied as a means to reduce CRBSI. Two meta-analyses of first-generation catheters [1, 263] demonstrated that such catheters reduced the risk for CRBSI compared with standard non-coated catheters. The duration of catheter placement in one study ranged from 5.1 to 11.2 days [264]. A second-generation catheter is now available with chlorhexidine coating the internal surface extending into the extension set and hubs while the external luminal surface is coated with chlorhexidine and silver sulfadiazine. The external surface has three times the amount of chlorhexidine and extended release of the surface bound antiseptics than that in the first generation catheters. All three prospective, randomized studies of second-generation catheters demonstrated a significant reduction in catheter colonization, but they were underpowered to show a difference in CRBSI [106–108]. Prolonged anti-infective activity provides improved efficacy in preventing infections [265]. Although rare, anaphylaxis with the use of these chlorhexidine/silver sulfadiazine catheters has been observed [266–270].

Chlorhexidine/silver sulfadiazine catheters are more expensive than standard catheters. However, one analysis has suggested that the use of chlorhexidine/silver sulfadiazine catheters should lead to a cost savings of \$68 to \$391 per catheter [271] in settings in which the risk for CRBSI is high, despite adherence to other preventive strategies (e.g., maximal barrier precautions and aseptic techniques). Use of these catheters might be cost effective in ICU

patients, burn patients, neutropenic patients, and other patient populations in which the rate of infection exceeds 3.3 per 1,000 catheter days [264].

Minocycline/Rifampin In a multicenter randomized trial, CVCs impregnated on both the external and internal surfaces with minocycline/rifampin were associated with lower rates of CRBSI when compared with the first generation chlorhexidine/ silver sulfadiazine impregnated catheters [109]. The beneficial effect began after day 6 of catheterization. Silicone minocycline/ rifampin impregnated CVCs with an average dwell time of over 60 days have been shown to be effective in reducing CRBSI [111]. No minocycline/rifampin-resistant organisms were reported in these studies. Two trials demonstrated that use of these catheters significantly reduced CRBSI compared with uncoated catheters [110, 111]. No comparative studies have been published using the second-generation chlorhexidine/silver sulfadiazine catheter. Although there have been concerns related to the potential for development of resistance, several prospective clinical studies have shown that the risk is low [272, 273]. Further, no resistance to minocycline or rifampin related to the use of the catheter has been documented in the clinical setting. Two studies using decision model analysis revealed these catheters were associated with superior cost savings compared with first generation chlorhexidine/ silver sulfadiazine catheters [274, 275]. Such analysis needs to be done compared with the second-generation catheters. However, as baseline rates of infection decrease and the cost of catheters decrease, the cost-benefit ratio will likely change.

The decision to use chlorhexidine/silver sulfadiazine or minocycline/rifampin impregnated catheters should be based on the need to enhance prevention of CRBSI after bundled standard procedures have been implemented (e.g., educating personnel, using maximal sterile barrier precautions, and using >0.5% chlorhexidine preparation with alcohol for skin antiseptics) and then balanced against the concern for emergence of resistant pathogens and the cost of implementing this strategy.

Platinum/Silver A combination platinum/silver impregnated catheter (i.e., a silver iontophoretic catheter) is available for use in the United States. Several prospective, randomized studies have been published comparing these catheters to uncoated catheters [276–279]. One study showed a reduction in the incidence density of catheter colonization and

CRBSI [278], but the other studies found no difference in catheter colonization or CRBSI between the impregnated catheter and a non-impregnated catheter [39, 276, 277]. In light of this, a firm recommendation for or against the use of these catheters cannot be made.

Systemic Antibiotic Prophylaxis

Recommendation

Do not administer systemic antimicrobial prophylaxis routinely before insertion or during use of an intravascular catheter to prevent catheter colonization or CRBSI [114].

Category IB

Background

Several studies have examined the role of systemic antibiotic prophylaxis in prevention of catheter-related infection. A recent meta-analysis reviewed these studies in oncology patients [114]. Four studies used a prophylactic glycopeptide prior to catheter insertion. However, heterogeneity in these studies precludes making any conclusion regarding efficacy.

In a study examining the effect of ongoing oral prophylaxis with rifampin and novobiocin on catheter-related infection in cancer patients treated with interleukin-2 [280], a reduction in CRBSI was observed, even though 9 of 26 subjects (35%) discontinued the prophylactic antibiotics due to side effects or toxicity. In non-oncology patients, no benefit was associated with vancomycin administration prior to catheter insertion in 55 patients undergoing catheterization for parenteral nutrition [281]. Similarly, extending perioperative prophylactic antibiotics in cardiovascular surgery patients did not reduce central venous catheter colonization [282]. A recent Cochrane review of prophylactic antibiotics in neonates with umbilical venous catheters concluded that there is insufficient evidence from randomized trials to support or refute the use of prophylactic antibiotics [283].

Late onset neonatal sepsis is often due to coagulase negative staphylococci and is thought to frequently stem from infected central venous catheters. Five trials involved a total of 371 neonates comparing vancomycin by continuous infusion via parenteral nutrition or intermittent dosing, and placebo. The infants treated with vancomycin experienced less sepsis (RR .11; 95% CI .05-.24) and less sepsis due to coagulase negative staphylococci (RR .33; 95% CI

.19–.59) [284]. However, mortality and length of stay were not significantly different between the two groups. There were insufficient data to evaluate the risk of selection for vancomycin resistant organisms.

Antibiotic/Antiseptic Ointments

Recommendation

Use povidone iodine antiseptic ointment or bacitracin/ gramicidin/polymyxin B ointment at the hemodialysis catheter exit site after catheter insertion and at the end of each dialysis session only if this ointment does not interact with the material of the hemodialysis catheter per manufacturer’s recommendation [59, 115–119]. Category IB

Background

A variety of topical antibiotic or antiseptic ointments have been utilized in attempts to lower the antimicrobial burden at the catheter insertion site and thus prevent infection. A number of older studies, examining primarily peripheral venous catheters, yielded varying conclusions [82, 285, 286]. In addition, the use of antibiotic ointments that have limited antifungal activity may serve to increase colonization and/or infection due to *Candida* species [89].

More recent studies have examined this approach in high-risk patients, particularly those undergoing hemodialysis [116–119]. Three randomized, controlled trials have evaluated the use of 10% povidone iodine [117–119]. A significant decrease in colonization, exit-site infection, or bloodstream infection was observed. The beneficial effect was most prominent in subjects with nasal colonization by *Staphylococcus aureus* [117–119].

Nasal carriers of *S. aureus* are more likely to experience a CRBSI than non-colonized persons [287–289]. This has prompted investigators to assess the utility of topical mupirocin, a potent anti-staphylococcal agent. Several studies have demonstrated a reduced risk of CRBSI when mupirocin ointment was applied at the catheter insertion site [117, 290–292]. Others have shown similar benefits when mupirocin was applied nasally [288, 289, 293]. However, enthusiasm for this measure has been dampened by the rapid emergence of mupirocin

resistance observed at some centers [88, 294, 295], and the potential degrading effect that mupirocin has on polyurethane catheters [94, 95].

In the only study demonstrating a significant effect on mortality, the application of bacitracin/gramicidin/polymyxin B ointment at the catheter insertion site was compared with placebo in 169 hemodialysis patients [296]. Infections were observed in more patients in the placebo group than in the bacitracin/gramicidin/polymyxin B group (34 versus 12%; relative risk, 0.35; 95% CI, .18 to .68). The number of infections per 1,000 catheter days (4.10 versus 1.02; $P < .0001$) and the number of bacteremias per 1,000 catheter days (2.48 versus .63; $P = .0004$) were also greater in the placebo group. Within the 6-month study period, there were 13 deaths in the placebo group as compared with three deaths in the bacitracin/gramicidin/polymyxin B group ($P = .004$). Thus, there is evidence from one study in hemodialysis patients that bacitracin/gramicidin/polymyxin B ointment can improve outcome, but no similar data exist for use in other patient populations [296]. It should be noted that the gramicidin-containing ointment is not currently available in the United States.

Antibiotic Lock Prophylaxis, Antimicrobial Catheter Flush and Catheter Lock Prophylaxis

Recommendation

Use prophylactic antimicrobial lock solution in patients with long term catheters who have a history of multiple CRBSI despite optimal maximal adherence to aseptic technique [120– 138]. Category II

Background

To prevent CRBSI, a wide variety of antibiotic and antiseptic solutions have been used to flush or lock catheter lumens [120– 138]. Catheter lock is a technique by which an antimicrobial solution is used to fill a catheter lumen and then allowed to dwell for a period of time while the catheter is idle. Antibiotics of various concentrations that have been used either alone (when directed at a specific organism) or in combination (to achieve broad empiric coverage) to prophylactically flush or lock central venous catheters include vancomycin, gentamicin, ciprofloxacin, minocycline, amikacin, cefazolin, cefotaxime, and ceftazidime; while antiseptics have included alcohol, taurolidine, trisodium citrate. (Taurolidine and trisodium citrate are not

approved for this use in the United States). These agents are usually combined with a compound acting as an anticoagulant, such as heparin or EDTA. Most of these studies have been conducted in relatively small numbers of high-risk patients, such as hemodialysis patients, neonates, or neutropenic oncology patients. Although most studies indicate a beneficial effect of the antimicrobial flush or lock solution in terms of prevention of catheter-related infection, this must be balanced by the potential for side effects, toxicity, allergic reactions, or emergence of resistance associated with the antimicrobial agent. The wide variety of compounds used, the heterogeneity of the patient populations studied, and limitations in the size or design of studies preclude a general recommendation for use. In addition, there are no FDA approved formulations approved for marketing, and most formulations have been prepared in hospital pharmacies. A brief overview of some of the studies follows.

At least 10 studies regarding catheter flush or lock solutions have been performed in hemodialysis patients [128, 129, 131– 138]. Three meta-analyses have all demonstrated that catheter lock solutions reduce risk of CRBSI in hemodialysis patients [297–299]. In the largest of these studies, 291 subjects were enrolled in a prospective randomized comparison of 30% trisodium citrate versus heparin [133]. The rate of CRBSI was significantly lower in the group whose catheters were locked with trisodium citrate (4.1 BSI/1,000 CVC days vs. 1.1 BSI/1,000 CVC days, $P < .001$), and no significant difference in thrombosis or occlusion of the catheter was noted. However, if infused rapidly, concentrated citrate can result in serious hypocalcaemia, cardiac dysrhythmia, and death. The second largest study in hemodialysis subjects examined the effect of a catheter lock solution containing cefazolin, gentamicin, and heparin compared with control patients receiving only heparin [135]. In 120 subjects, the rate of CRBSI was significantly lower in those receiving the antibiotic lock solution (0.44 BSI/1,000 CVC days vs. 3.12 BSI/1,000 CVC days, $P = .03$) [135]. Other trials in hemodialysis patients have studied minocycline, gentamicin, EDTA, heparin, taurolidine, vancomycin, and cefotaxime.

At least five studies have been conducted in pediatric oncology patients [120, 121, 124, 126, 127]. In the largest trial, 126 subjects were enrolled in a prospective, randomized, double blind study comparing vancomycin/ciprofloxacin/heparin (VCH) to vancomycin/heparin (VH) to heparin (H) alone [124]. The time to CVC-related infection was significantly longer in the VCH or

VH arms of the study compared with heparin, and the rate of infection was significantly lower with either of the antibiotic containing solutions compared with heparin alone (1.72/1,000 CVC days [H] vs. 0.55/1,000 CVC days [VCH] vs. 0.37/1,000 CVC days [VH]).

In a meta-analysis of seven randomized, controlled trials examining the utility of vancomycin-containing lock or flush solutions compared with heparin alone, the risk ratio for vancomycin/heparin solutions was 0.49 (95% CI .26–.95, P = .03) [300]. Use of the catheter lock technique appeared to have greater benefit than simply flushing vancomycin through the catheter.

Recently, a prospective, double blind, randomized trial compared the utility of 70% ethanol lock versus heparinized saline for the prevention of primary CRBSI in oncology patients. Patients receiving the ethanol lock preventive therapy were significantly less likely to experience a primary CRBSI (0.60/ 1,000 CVC days vs. 3.11/1,000 CVC days; OR 0.18, 95% CI .05-.65, P5 .008) [301].

Anticoagulants

Recommendation

Do not routinely use anticoagulant therapy to reduce the risk of catheter-related infection in general patient populations [139]. Category II

Background

Shortly after insertion, intravascular catheters are coated with a conditioning film, consisting of fibrin, plasma proteins, and cellular elements, such as platelets and red blood cells [213, 302]. Microbes interact with the conditioning film, resulting in colonization of the catheter [303]. There is a close association between thrombosis of central venous catheters and infection [221, 304, 305]. Therefore, anticoagulants have been used to prevent catheter thrombosis and presumably reduce the risk of infection.

In a meta-analysis evaluating the benefit of heparin prophylaxis (3 units/mL in parenteral nutrition, 5,000 units every 6 or 12 hours flush or 2,500 units low molecular weight heparin subcutaneously) in patients with short-term CVCs, the risk for catheter-related central venous thrombosis was reduced with the use of prophylactic heparin [139]. However, no

substantial difference in the rate of CRBSI was observed. In a more recent prospective, randomized trial, 204 patients with non-tunneled catheters were assigned to receive a continuous infusion of heparin (100 units/kg/ d) or saline (50 mL/d) [306]. The rate of CRBSI was significantly decreased in the group receiving heparin (2.5 BSI/1,000 CVC days vs. 6.4 BSI/1,000 CVC days). Because the majority of heparin solutions contain preservatives with antimicrobial activity, whether any decrease in the rate of CRBSI is a result of the reduced thrombus formation, the preservative, or both is unclear. The majority of pulmonary artery, umbilical, and central venous catheters are available as heparin-bonded devices. The majority of catheters are heparin bonded with benzalkonium, which provides the catheters with antimicrobial activity [307] and provides an anti-thrombotic effect [308]. However, some catheters have heparin bound directly to the catheter without benzalkonium [309]. Studies have shown that heparin-bonded catheters reduce risk of thrombosis and risk of CRBSI [306, 308– 310], but are less effective at reducing catheter colonization than catheters impregnated with chlorhexidine/silver sulfadiazine [311]. Unfortunately, heparin-induced thrombocytopenia can occur and has prompted many clinicians to avoid heparin [312]. Trisodium citrate has been recommended as a catheter lock solution because it possesses both anticoagulant and antimicrobial properties [133]. In a prospective, randomized, double blind study in hemodialysis patients, use of interdialytic heparin (5,000 U/mL) was associated with a significantly greater rate of CRBSIs compared with use of 30% trisodium citrate (4.1 BSI/ 1,000 CVC days vs. 1.1BSI/1,000 CVC days [313].

Warfarin has been evaluated as a means to reduce CVC thrombus formation and, hence, infection [314–318]. In patients with long-term CVCs, low dose warfarin (i.e., 1 mg/day) reduced the incidence of catheter thrombus [142, 143]. However, other studies have not confirmed reduced thrombosis and still others have found untoward interactions in patients receiving 5-FU [319, 320]. Data are limited; although low dose warfarin decreases the risk of thrombus formation in cancer patients, it has not been shown to reduce infectious complications. Over 20% of patients in some studies develop prolonged prothrombin times and required dosage adjustment [321]. Other anticoagulants, such as factor Xa inhibitors or direct

thrombin inhibitors, have not been adequately assessed in terms of reducing the risk of catheter-associated infection.

Replacement of Peripheral and Midline Catheters

Recommendations

1. There is no need to replace peripheral catheters more frequently than every 72–96 hours to reduce risk of infection and phlebitis in adults [36, 140, 141]. Category 1B
2. No recommendation is made regarding replacement of peripheral catheters in adults only when clinically indicated [142–144]. Unresolved issue
3. Replace peripheral catheters in children only when clinically indicated [32, 33]. Category 1B
4. Replace midline catheters only when there is a specific indication. Category II

Background

Scheduled replacement of intravascular catheters has been proposed as a method to prevent phlebitis and catheter-related infections. Studies of short peripheral venous catheters indicate that the incidence of thrombophlebitis and bacterial colonization of catheters increases when catheters are left in place >72 hours [258]. However, rates of phlebitis are not substantially different in peripheral catheters left in place 72 hours compared with 96 hours [141]. Because phlebitis and catheter colonization have been associated with an increased risk for catheter-related infection, short peripheral catheter sites commonly are replaced at 72–96 hour intervals to reduce both the risk for infection and patient discomfort associated with phlebitis.

Some studies have suggested that planned removal at 72 hours vs. removing as needed resulted in similar rates of phlebitis and catheter failure [142–144]. However, these studies did not address the issue of CRBSI, and the risk of CRBSIs with this strategy is not well studied.

Midline catheters are associated with lower rates of phlebitis than short peripheral catheters and with lower rates of infection than CVCs [322–324]. In one prospective study of 140 midline catheters, their use was associated with a BSI rate of 0.8 per 1,000 catheter days [324]. No specific risk factors, including duration of catheterization, were associated with

infection. Midline catheters were in place a median of 7 days, but for as long as 49 days. Although the findings of this study suggested that midline catheters could be changed only when there is a specific indication, no prospective, randomized studies have assessed the benefit of routine replacement as a strategy to prevent CRBSI associated with midline catheters.

Replacement of CVCs, Including PICCs and Hemodialysis Catheters

Recommendations

1. Do not routinely replace CVCs, PICCs, hemodialysis catheters, or pulmonary artery catheters to prevent catheter-related infections. Category IB
2. Do not remove CVCs or PICCs on the basis of fever alone. Use clinical judgment regarding the appropriateness of removing the catheter if infection is evidenced elsewhere or if a noninfectious cause of fever is suspected. Category II
3. Do not use guidewire exchanges routinely for non-tunneled catheters to prevent infection. Category IB
4. Do not use guidewire exchanges to replace a non-tunneled catheter suspected of infection. Category IB
5. Use a guidewire exchange to replace a malfunctioning non-tunneled catheter if no evidence of infection is present. Category IB
6. Use new sterile gloves before handling the new catheter when guidewire exchanges are performed. Category II

Background

Catheter replacement at scheduled time intervals as a method to reduce CRBSI has not lowered rates. Two trials have assessed a strategy of changing the catheter every 7 days compared with a strategy of changing catheters as needed [165, 325]. One of these studies involved 112 surgical ICU patients needing CVCs, pulmonary artery catheters, or peripheral arterial catheters [165], whereas the other study involved only subclavian hemodialysis catheters [325]. In both studies, no difference in CRBSI was observed in patients undergoing

scheduled catheter replacement every 7 days compared with patients whose catheters were replaced as needed.

Scheduled guidewire exchange of CVCs is another proposed strategy for preventing CRBSI. The results of a meta-analysis of 12 randomized, controlled trials assessing CVC management failed to demonstrate any reduction of CRBSI rates through routine replacement of CVCs by guidewire exchange compared with catheter replacement on an as needed basis [326]. Thus, routine replacement of CVCs is not necessary for catheters that are functioning and have no evidence of causing local or systemic complications.

Catheter replacement over a guidewire has become an accepted technique for replacing a malfunctioning catheter or exchanging a pulmonary artery catheter for a CVC when invasive monitoring no longer is needed. Catheter insertion over a guidewire is associated with less discomfort and a significantly lower rate of mechanical complications than are those percutaneously inserted at a new site [327]. In addition, this technique provides a means of preserving limited venous access in some patients. Replacement of temporary catheters over a guidewire in the presence of bacteremia is not an acceptable replacement strategy because the source of infection is usually colonization of the skin tract from the insertion site to the vein [37, 327]. However, in selected patients with tunneled hemodialysis catheters and bacteremia, catheter exchange over a guidewire, in combination with antibiotic therapy, is an alternative as a salvage strategy in patients with limited venous access [328–331].

Because of the increased difficulty obtaining vascular access in children, attention should be given to the frequency with which catheters are replaced in these patients. In a study in which survival analysis techniques were used to examine the relation between the duration of central venous catheterization and complications in pediatric ICU patients, all of the patients studied ($n = 397$) remained uninfected for a median of 23.7 days [250]. In addition, no relation was found between duration of catheterization and the daily probability of infection ($r = 0.21$; $P > .1$), suggesting that routine replacement of CVCs likely does not reduce the incidence of catheter-related infection [250].

Vascular access sites can be even more limited among neonates. Four randomized trials ($n = 368$) summarized in a recent Cochrane Database Systemic Review compared the effects of

giving parenteral nutrition through percutaneous central venous catheters vs. peripheral intravenous catheters. Fewer painful procedures (venipunctures) were required in neonates randomized to percutaneously placed CVCs, and there was no evidence for increased risk of BSIs [332].

CVC occlusion due to thrombus formation is one of the most common reasons for CVC removal in neonates. Various methods have been tried to prevent catheter occlusion. Recently, a randomized trial (n = 201) evaluated whether a continuous heparin infusion (0.5 units/kg/hour) could effectively prolong the duration of catheterization when compared with a placebo infusion. The rate of catheter occlusion requiring catheter removal was lower in the heparin group (6% vs. 31%, P = .001; NNT = 4). Rates of CRBSI were similar, although the study was not powered to evaluate CRBSI rate differences. Heparin associated antibody levels were not routinely measured [333].

Hemodialysis Catheters. The use of catheters for hemodialysis is the most common factor contributing to bacteremia in dialysis patients [334, 335]. The relative risk for bacteremia in patients with dialysis catheters is sevenfold the risk for patients with arteriovenous (AV) fistulas [336]. AV fistulas and grafts are preferred over hemodialysis catheters in patients with chronic renal failure, due to their lower associated risk of infection. If temporary access is needed for dialysis, a tunneled cuffed catheter is preferable to a non-cuffed catheter, even in the ICU setting, if the catheter is expected to stay in place for >3weeks [59].

Pulmonary Artery Catheters. Pulmonary artery catheters are inserted through a Teflon® introducer and typically remain in place an average of 3 days. The majority of pulmonary artery catheters are heparin bonded, which reduces not only catheter thrombosis but also microbial adherence to the catheter [307]. Meta-analysis indicates that the CRBSI rate associated with pulmonary artery catheterization is 3.7 per 1,000 catheter days and somewhat higher than the rate observed for unmedicated and non-tunnelled CVCs (2.7 per 1,000 catheter days)[6, 45].

Data from prospective studies indicate that the risk of significant catheter colonization and CRBSI increases the longer the catheter remains in place. In general, the risk of significant catheter colonization increases after 4 days of catheterization [75, 337, 338], whereas the risk of CRBSI increases beyond 5-7 days of catheterization [75, 84, 166]. Efforts must be made to

differentiate between infection related to the introducer and that related to the pulmonary artery catheter. Significant colonization of the introducer occurs earlier than that of the pulmonary artery catheter [337, 339]. However, no studies indicate that catheter replacement at scheduled time intervals is an effective method to reduce risk of CRBSI [165, 327, 339]. In patients who continue to require hemodynamic monitoring, pulmonary artery catheters do not need to be changed more frequently than every 7 days [339]. No specific recommendation can be made regarding routine replacement of catheters that need to be in place for >7 days.

Pulmonary artery catheters are usually packaged with a thin plastic sleeve that prevents touch contamination when placed over the catheter. In a study of 166 catheters, patients who were randomly assigned to have their catheters self-contained within this sleeve had a reduced risk for CRBSI compared with those who had a pulmonary artery catheter placed without the sleeve ($P = .002$) [81].

Umbilical Catheters

Recommendations

1. Remove and do not replace umbilical artery catheters if any signs of CRBSI, vascular insufficiency in the lower extremities, or thrombosis are present [145]. Category II
2. Remove and do not replace umbilical venous catheters if any signs of CRBSI or thrombosis are present [145]. Category II
3. No recommendation can be made regarding attempts to salvage an umbilical catheter by administering antibiotic treatment through the catheter. Unresolved issue
4. Cleanse the umbilical insertion site with an antiseptic before catheter insertion. Avoid tincture of iodine because of the potential effect on the neonatal thyroid. Other iodine-containing products (e.g., povidone iodine) can be used [146– 150]. Category IB
5. Do not use topical antibiotic ointment or creams on umbilical catheter insertion sites because of the potential to promote fungal infections and antimicrobial resistance [88, 89]. Category IA
6. Add low-doses of heparin (0.25–1.0 U/ml) to the fluid infused through umbilical arterial catheters [151–153]. Category IB

7. Remove umbilical catheters as soon as possible when no longer needed or when any sign of vascular insufficiency to the lower extremities is observed. Optimally, umbilical artery catheters should not be left in place >5 days [145, 154]. Category II
8. Umbilical venous catheters should be removed as soon as possible when no longer needed, but can be used up to 14 days if managed aseptically [155, 156]. Category II
9. An umbilical catheter may be replaced if it is malfunctioning, and there is no other indication for catheter removal, and the total duration of catheterization has not exceeded 5 days for an umbilical artery catheter or 14 days for an umbilical vein catheter. Category II

Background

Although the umbilical stump becomes heavily colonized soon after birth, umbilical vessel catheterization often is used for vascular access in newborn infants. Umbilical vessels can be cannulated easily and permit both collection of blood samples and measurement of hemodynamic status. The incidences of catheter colonization and BSI are similar for umbilical vein catheters and umbilical artery catheters. In several studies, an estimated 40%–55% of umbilical artery catheters were colonized and 5% resulted in CRBSI; umbilical vein catheters were associated with colonization in 22%–59% of cases [147, 148, 340] and with CRBSI in 3%–8% of cases [148]. Although CRBSI rates are similar for umbilical catheters in the high position (i.e, above the diaphragm) compared with the low position (i.e, below the diaphragm and above the aortic bifurcation), catheters placed in the high position result in a lower incidence of vascular complications without an increase in adverse sequelae [148].

Risk factors for infection differ for umbilical artery and umbilical vein catheters. In one study, neonates with very low birth weight who also received antibiotics for >10 days were at increased risk for umbilical artery CRBSIs [148]. In comparison, those with higher birth weight and receipt of parenteral nutrition fluids were at increased risk for umbilical vein CRBSI. Duration of catheterization was not an independent risk factor for infection of either type of umbilical catheter.

A recent randomized trial (n = 210) evaluated whether long-term umbilical venous catheterization (up to 28 days) would result in the same or fewer CRBSIs when compared with

neonates who were randomized to short-term umbilical venous catheterization for 7–10 days followed by percutaneous central venous catheterization. CRBSI rate was higher (20%) among long term catheterized neonates when compared with short term catheterized neonates (13%). The difference was not statistically significant ($P = .17$), although the study was underpowered. The study was not powered to evaluate differences in venous thrombosis rates [341].

Peripheral Arterial Catheters and Pressure Monitoring Devices for Adult and Pediatric

Patients

Recommendations

1. In adults, use of the radial, brachial or dorsalis pedis sites is preferred over the femoral or axillary sites of insertion to reduce the risk of infection [46, 47, 157, 158]. Category IB
2. In children, the brachial site should not be used. The radial, dorsalis pedis, and posterior tibial sites are preferred over the femoral or axillary sites of insertion [46]. Category II
3. A minimum of a cap, mask, sterile gloves and a small sterile fenestrated drape should be used during peripheral arterial catheter insertion [47, 158, 159]. Category IB
4. During axillary or femoral artery catheter insertion, maximal sterile barriers precautions should be used. Category II
5. Replace arterial catheters only when there is a clinical indication. Category II
6. Remove the arterial catheter as soon as it is no longer needed. Category II
7. Use disposable, rather than reusable, transducer assemblies when possible [160–164]. Category IB
8. Do not routinely replace arterial catheters to prevent catheter-related infections [165, 166, 167, 168]. Category II
9. Replace disposable or reusable transducers at 96-hour intervals. Replace other components of the system (including the tubing, continuous-flush device, and flush solution) at the time the transducer is replaced [37, 161]. Category IB
10. Keep all components of the pressure monitoring system (including calibration devices and flush solution) sterile [160, 169–171]. Category IA

11. Minimize the number of manipulations of and entries into the pressure monitoring system. Use a closed flush system (i.e, continuous flush), rather than an open system (i.e, one that requires a syringe and stopcock), to maintain the patency of the pressure monitoring catheters [163, 172]. Category II
12. When the pressure monitoring system is accessed through a diaphragm, rather than a stopcock, scrub the diaphragm with an appropriate antiseptic before accessing the system [163]. Category IA
13. Do not administer dextrose-containing solutions or parenteral nutrition fluids through the pressure monitoring circuit [163, 173, 174]. Category IA
14. Sterilize reusable transducers according to the manufacturers' instructions if the use of disposable transducers is not feasible [163, 173–176]. Category IA

Background

Arterial catheters are usually inserted into the radial or femoral artery and permit continuous blood pressure monitoring and blood gas measurements. The risk of CRBSI for arterial catheters is lower than that associated with non-coated, uncuffed, non-tunneled short term CVCs (1.7 versus 2.7 per 1,000 catheter days) [6]. However, risk of CRBSI rates are comparable between arterial catheters and coated, uncuffed, non-tunneled short term CVCs [6]. Unlike CVCs, use of full barrier precautions during arterial cannulation does not appear to reduce the risk of arterial CRBSI [158, 159]. Nonetheless, when arterial catheters are inserted using a protocol which includes maximum barrier precautions, a very low risk of CRBSI (0.41/1,000 catheter days) can be achieved [47]. Although a meta-analysis failed to discern a difference in rates of CRBSI among three sites of insertion (radial, femoral, and axillary) [342], colonization of catheters inserted in the femoral site occurs more often [158]. In addition, a prospective observational study of over 2,900 arterial catheters that were inserted using maximum barrier precautions demonstrated an almost 8-fold increase in the incidence of CRBSI when the femoral site was used compared with the radial site [343]. Furthermore, there is a greater risk of CRBSI caused by gram-negative bacteria when the femoral site is used [343]. The rates of catheter colonization and CRBSI appear similar between the radial and dorsalis pedis sites [157]. The risk of developing a CRBSI increases with the duration of catheterization [166,

344]; however, the routine changing of arterial catheters at scheduled times does not result in a diminution of the risk of CRBSI [165]. Catheters that need to be in place for >5 days should not be routinely changed if no evidence of infection is observed.

Replacement of Administration Sets

Recommendations

1. In patients not receiving blood, blood products or fat emulsions, replace administration sets that are continuously used, including secondary sets and add-on devices, no more frequently than at 96-hour intervals, [177] but at least every 7 days [178–181]. Category IA
2. No recommendation can be made regarding the frequency for replacing intermittently used administration sets. Unresolved issue
3. No recommendation can be made regarding the frequency for replacing needles to access implantable ports. Unresolved issue
4. Replace tubing used to administer blood, blood products, or fat emulsions (those combined with amino acids and glucose in a 3-in-1 admixture or infused separately) within 24 hours of initiating the infusion [182–185]. Category IB
5. Replace tubing used to administer propofol infusions every 6 or 12 hours, when the vial is changed, per the manufacturer's recommendation (FDA website Medwatch) [186]. Category IA
6. No recommendation can be made regarding the length of time a needle used to access implanted ports can remain in place. Unresolved issue

Background

The optimal interval for routine replacement of IV administration sets has been examined in a number of well-controlled studies and meta-analyses. Data from these studies reveal that replacing administration sets no more frequently than 72–96 hours after initiation of use is safe and cost-effective [141, 177, 179–181]. More recent studies suggest that administration sets may be used safely for up to 7 days if used in conjunction with antiseptic catheters or if fluids that enhance microbial growth (e.g., parenteral nutrition or blood) have

not been used [216, 345]. When a fluid that enhances microbial growth is infused (e.g., fat emulsions and blood products), more frequent changes of administration sets are indicated as these products have been identified as independent risk factors for CRBSI [182, 216, 346–350]. Little data exist regarding the length of time a needle used to access implanted ports can remain in place and the risk of CRBSI. While some centers have left them in place for several weeks without CRBSI, [351], this practice has not been adequately studied.

Needleless Intravascular Catheter Systems

Recommendations

1. Change the needleless components at least as frequently as the administration set. There is no benefit to changing these more frequently than every 72 hours. [39, 187–193]. Category II
2. Change needleless connectors no more frequently than every 72 hours or according to manufacturers' recommendations for the purpose of reducing infection rates [187, 189, 192, 193]. Category II
3. Ensure that all components of the system are compatible to minimize leaks and breaks in the system [194]. Category II
4. Minimize contamination risk by scrubbing the access port with an appropriate antiseptic (chlorhexidine, povidone iodine, an iodophor, or 70% alcohol) and accessing the port only with sterile devices [189, 192, 194–196]. Category IA
5. Use a needleless system to access IV tubing. Category IC
6. When needleless systems are used, a split septum valve may be preferred over some mechanical valves due to increased risk of infection with the mechanical valves [197–200]. Category II

Background

Stopcocks used for injection of medications, administration of IV infusions, and collection of blood samples represent a potential portal of entry for microorganisms into vascular access catheters and IV fluids. Whether such contamination is a substantial entry point of microorganisms that cause CRBSI has not been demonstrated. Nonetheless, stopcocks

should be capped when not being used. In general, closed catheter access systems are associated with fewer CRBSIs than open systems and should be used preferentially [352].

"Piggyback" systems (secondary intermittent infusions delivered through a port on a primary infusion set) are used as an alternative to stopcocks. However, they also pose a risk for contamination of the intravascular fluid if the device entering the rubber membrane of an injection port is exposed to air or if it comes into direct contact with nonsterile tape used to fix the needle to the port. Modified piggyback systems have the potential to prevent contamination at these sites [353].

Attempts to reduce the incidence of sharps injuries and the resultant risk for transmission of bloodborne infections to healthcare personnel have led to the introduction and mandating of needleless infusion systems. There are several types of needleless connectors on the market.

The first type of needleless system connectors consisted of a split septum connector, which is accessed with a blunt cannula instead of a needle (external cannulae activated split septums). Because of the large amount of space in the connector to accommodate the cannula, when the cannula is removed it may result in the creation of negative pressure which may cause blood to be aspirated into the distal lumen, possibly increasing the risk of catheter occlusion or thrombosis. A luer-activated device, which incorporates a valve preventing the outflow of fluid through the connector, was designed to eliminate this problem. Some luer devices require a cap to be attached to the valve when not in use, which can be difficult to maintain aseptically, and therefore they may be prone to contamination.

Another type of second-generation needleless system addressed the occlusion issue by incorporating positive or neutral fluid displacement to either flush out aspirated blood or prevent its aspiration into infusion catheters.

Use of needleless connectors or mechanical valves appear to be effective in reducing connector colonization in some [196, 354, 355], but not all studies [356] when compared with stopcocks and caps. In one study [354], the incidence of CRBSI was reduced when the needleless connector was compared with standard stopcocks. Appropriate disinfectants must be used to prevent transmission of microbes through connectors [357]. Some studies have

shown that disinfection of the devices with chlorhexidine/alcohol solutions appears to be most effective in reducing colonization [195, 196]. In addition, the time spent applying the disinfectant may be important. One study found that swiping the luer-activated device with 70% alcohol for only 3 to 5 seconds did not adequately disinfect the septal surface [358]. However, a number of outbreak investigations have reported increases in CRBSIs associated with a switch from external cannulae activated split septum needleless devices to mechanical valve devices [197, 198, 200, 359]. The reasons for these associations are not known and it is also not known if this is a device-specific or class association, particularly as physical and mechanical properties of needleless connectors vary from device to device. In addition, one investigation found CRBSIs increased with the switch from a luer-activated negative displacement mechanical valve to a luer-activated positive fluid displacement mechanical valve [199]. However in an observational study, a switch from a luer-activated negative displacement mechanical valve to a different luer-activated positive displacement mechanical valve as part of a bundled intervention resulted in a significant decrease in CRBSIs [201]. Potential explanations for outbreaks associated with these devices include difficulty encountered in adequate disinfection of the surface of the connector due to physical characteristics of the plastic housing diaphragm interface, fluid flow properties (laminar vs. turbulent), internal surface area, potential fluid dead space, inadequate flushing of the device due to poor visualization of the fluid flow pathway in opaque devices, and the presence of internal corrugations that could harbor organisms, particularly if the catheters are used to withdraw blood [199]. Some studies have shown that the increase in CRBSIs with the change to lueractivated devices may be related to improper cleaning and infection control practices such as infrequently changing the devices [192, 194]. Additionally, silver-coated connector valves have been FDA approved; however, there are no published randomized trials with this device and no recommendation can be made regarding its use. Likewise, an antiseptic-barrier cap for needleless connectors has been studied in a laboratory setting and appears to be effective in preventing the entry of microorganisms [360], but has not yet been studied in a clinical trial.

Performance Improvement

Recommendation

Use hospital-specific or collaborative-based performance improvement initiatives in which multifaceted strategies are "bundled" together to improve compliance with evidence-based recommended practices [15, 69, 70, 201–205]. Category IB

Background

Clinical decision makers, healthcare payers, and patient safety advocates emphasize the importance of translating research findings into everyday practice. Rigorous evaluations of CRBSI preventive practices using study designs with high internal validity and including study populations that optimize external validity remain necessary. Once practices have been determined to be effective and economically efficient, the next step is to implement these evidence-based practices so they become part of routine clinical care. Unfortunately, implementation of evidence-based CRBSI preventive practices in U.S. hospitals has been suboptimal [361, 362]. In a national survey conducted in March 2005 of over 700 U.S. hospitals, approximately one quarter of U.S. hospitals indicated that either maximal sterile barrier precautions during central line insertion or chlorhexidine gluconate as site disinfectant, two practices widely recommended in the guidelines published in 2002 [363], were not being used routinely [364]. Approximately 15% of U.S. hospitals reported routinely changing CVCs to prevent infection despite evidence that this practice should no longer be used [362, 364].

Accordingly, investigators have attempted various approaches to better translate research findings and evidence-based recommendations into clinical practice. Numerous quality improvement studies have been published during the past several years that have used various methods, such as education of healthcare personnel, audit and feedback, organizational change, and clinical reminders [8–11, 69, 70, 202, 365–367]. The educational interventions primarily targeted hand hygiene, use of maximal sterile barriers during insertion, appropriate insertion site selection, proper site care using chlorhexidine gluconate, and prompt removal of unnecessary catheters. While a large number of before-and-after studies with a few using concurrent control groups [15, 70] have been published, no randomized, controlled trial

evaluating a quality improvement strategy to prevent CRBSI has been reported [368]. The vast majority of before-and-after studies reported statistically significant decreases in CRBSI rates after a quality improvement strategy was implemented [368]. Additionally, both controlled trials also found statistically significant reductions of CRBSI in the intervention units compared with control units [15, 70].

Investigators have also employed multifaceted approaches in which several strategies are bundled together to improve compliance with evidence-based guidelines [15, 69, 70]. One such collaborative cohort study [69] of 108 ICUs in Michigan targeted clinicians' use of five evidence-based practices: hand hygiene, maximum barrier precautions, chlorhexidine site disinfection, avoiding the femoral site, and promptly removing unnecessary central venous catheters. In addition to educating clinicians about CRBSI prevention, interventions used included: 1) a central venous catheter cart that contained all the necessary supplies; 2) a checklist to ensure adherence to proper practices; 3) stoppage of procedures in non-emergent situations, if evidence-based practices were not being followed; 4) prompt removal of unnecessary central catheters identified during daily patient rounds; 5) feedback to the clinical teams regarding the number of CRBSI episodes and overall rates; and 6) buy-in from the chief executive officers of the participating hospitals that chlorhexidine gluconate products/solutions would be stocked prior to study initiation. Using an interrupted time series analysis and multivariable regression, the investigators reported a statistically significant 66% decrease in CRBSI rates approximately 18 months after the intervention began [69] and sustained reductions over time [369]. Specific process and outcome measures for tracking and feedback (i.e rate of central line infections, proportion of central lines placed with all or individual bundle elements performed AND documented) should be identified in individual institutions based on areas that have been identified for performance improvement.

Finally, emphasis on the care and maintenance of catheters once they are in place should be a focus of performance improvement and quality assurance in all programs. A study to assess practice and staff knowledge of CVC post-insertion care and identify aspects of CVC care with potential for improvement revealed several areas of opportunity to improve post-insertion care [370]. Data were recorded on 151 CVCs in 106 patients giving a total of 721

catheter days. In all, 323 breaches in care were identified giving a failure rate of 44.8%, with significant differences between intensive care unit (ICU) and non-ICU wards. Dressings (not intact) and caps (incorrectly placed) were identified as the major lapses in CVC care with 158 and 156 breaches per 1000 catheter days, respectively. Interventions to improve reliability of care should focus on making the implementation of best practice easier to achieve.

References

1. Mermel LA. Prevention of intravascular catheter-related infections. (Erratum: Ann Intern Med 133:395, 2000). *Ann Intern Med* 2000; 132:391–402.
2. Dimick JB, Pelz RK, Consunji R, Swoboda SM, Hendrix CW, Lipsett PA. Increased resource use associated with catheter-related bloodstream infection in the surgical intensive care unit. *Arch Surg* 2001; 136:229–34.
3. Warren DK, Quadir WW, Hollenbeak CS, Elward AM, Cox MJ, Fraser VJ. Attributable cost of catheter-associated bloodstream infections among intensive care patients in a nonteaching hospital. *Crit Care Med* 2006; 34:2084–9.
4. Blot SI, Depuydt P, Annemans L, et al. Clinical and economic outcomes in critically ill patients with nosocomial catheter-related bloodstream infections. *Clin Infect Dis* 2005; 41:1591–8.
5. Renaud B, Brun-Buisson C. Outcomes of primary and catheter-related bacteremia. A cohort and case-control study in critically ill patients. *Am J Respir Crit Care Med* 2001; 163:1584–90.
6. Maki DG, Kluger DM, Crnich CJ. The risk of bloodstream infection in adults with different intravascular devices: a systematic review of 200 published prospective studies. *Mayo Clin Proc* 2006; 81:1159–71.
7. Yoo S, Ha M, Choi D, Pai H. Effectiveness of surveillance of central catheter-related bloodstream infection in an ICU in Korea. *Infect Control Hosp Epidemiol* 2001; 22:433–6.
8. Warren DK, Zack JE, Cox MJ, Cohen MM, Fraser VJ. An educational intervention to prevent catheter-associated bloodstream infections in a non-teaching community medical center. *Crit Care Med* 2003; 31:1959–63.
9. Warren DK, Zack JE, Mayfield JL, et al. The effect of an education program on the incidence of central venous catheter-associated bloodstream infection in a medical ICU. *Chest* 2004; 126:1612–8.
10. Warren DK, Cosgrove SE, Diekema DJ, et al. A multicenter intervention to prevent catheter-associated bloodstream infections. *Infect Control Hosp Epidemiol* 2006; 27:662–9.
11. Higuera F, Rosenthal VD, Duarte P, Ruiz J, Franco G, Safdar N. The effect of process control on the incidence of central venous catheter-associated bloodstream infections and mortality in intensive care units in Mexico. *Crit Care Med* 2005; 33:2022–7.
12. Coopersmith CM, Rebmann TL, Zack JE, et al. Effect of an education program on decreasing catheter-related bloodstream infections in the surgical intensive care unit. *Crit Care Med* 2002; 30:59–64.

13. Coopersmith CM, Zack JE, Ward MR, et al. The impact of bedside behavior on catheter-related bacteremia in the intensive care unit. *Arch Surg* 2004; 139:131–6.
14. Sherertz RJ, Ely EW, Westbrook DM, et al. Education of physicians-in-training can decrease the risk for vascular catheter infection. *Ann Intern Med* 2000; 132:641–8.
15. Eggimann P, Harbarth S, Constantin MN, Touveneau S, Chevrollet JC, Pittet D. Impact of a prevention strategy targeted at vascular-access care on incidence of infections acquired in intensive care. *Lancet* 2000; 355:1864–8.
16. Nehme AE. Nutritional support of the hospitalized patient. The team concept. *JAMA* 1980; 243:1906–8.
17. Soifer NE, Borzak S, Edlin BR, Weinstein RA. Prevention of peripheral venous catheter complications with an intravenous therapy team: a randomized controlled trial. *Arch Intern Med* 1998; 158:473–7.
18. Tomford JW, Hershey CO, McLaren CE, Porter DK, Cohen DI. Intravenous therapy team and peripheral venous catheter-associated complications. A prospective controlled study. *Arch Intern Med* 1984; 144:1191–4.
19. Scalley RD, Van CS, Cochran RS. The impact of an i.v. team on the occurrence of intravenous-related phlebitis. A 30-month study. *J Intraven Nurs* 1992; 15:100–9.
20. Palefski SS, Stoddard GJ. The infusion nurse and patient complication rates of peripheral-short catheters. A prospective evaluation. *J Intraven Nurs* 2001; 24:113–23.
21. Miller JM, Goetz AM, Squier C, Muder RR. Reduction in nosocomial intravenous device-related bacteremias after institution of an intravenous therapy team. *J Intraven Nurs* 1996; 19:103–6.
22. Hunter MR. Development of a Vascular Access Team in an acute care setting. *J Infus Nurs* 2003; 26:86–91.
23. Hawes ML. A proactive approach to combating venous depletion in the hospital setting. *J Infus Nurs* 2007; 30:33–44.
24. Brunelle D. Impact of a dedicated infusion therapy team on the reduction of catheter-related nosocomial infections. *J Infus Nurs* 2003; 26:362–6.
25. Bosma TL, Jewesson PJ. An infusion program resource nurse consult service: our experience in a major Canadian teaching hospital. *J Infus Nurs* 2002; 25:310–5.
26. Pierce CA, Baker JJ. A nursing process model: quantifying infusion therapy resource consumption. *J Infus Nurs* 2004; 27:232–44.
27. Tomford JW, Hershey CO. The i.v. therapy team: impact on patient care and costs of hospitalization. *NITA* 1985; 8:387–9.
28. Davis D, O'Brien MA, Freemantle N, Wolf FM, Mazmanian P, Taylor-Vaisey A. Impact of formal continuing medical education: do conferences, workshops, rounds, and other traditional continuing education activities change physician behavior or health care outcomes? *JAMA* 1999; 282:867–74.

29. Alonso-Echanove J, Edwards JR, Richards MJ, et al. Effect of nurse staffing and antimicrobial-impregnated central venous catheters on the risk for bloodstream infections in intensive care units. *Infect Control Hosp Epidemiol* 2003; 24:916–25.
30. Fridkin SK, Pear SM, Williamson TH, Galgiani JN, Jarvis WR. The role of understaffing in central venous catheter-associated bloodstream infections. *Infect Control Hosp Epidemiol* 1996; 17: 150–8.
31. Robert J, Fridkin SK, Blumberg HM, et al. The influence of the composition of the nursing staff on primary bloodstream infection rates in a surgical intensive care unit. *Infect Control Hosp Epidemiol* 2000; 21:12–7.
32. Maki DG, Goldman DA, Rhame FS. Infection control in intravenous therapy. *Ann Intern Med* 1973; 79:867–87.
33. Band JD, Maki DG. Steel needles used for intravenous therapy. Morbidity in patients with hematologic malignancy. *Arch Intern Med* 1980; 140:31–4.
34. Tully JL, Friedland GH, Baldini LM, Goldmann DA. Complications of intravenous therapy with steel needles and Teflon catheters. A comparative study. *Am J Med* 1981; 70:702–6.
35. Ryder MA. Peripheral access options. *Surg Oncol Clin N Am* 1995; 4:395–427.
36. Maki DG, Ringer M. Risk factors for infusion-related phlebitis with small peripheral venous catheters. A randomized controlled trial. *Ann Intern Med* 1991; 114:845–54.
37. Mermel LA, McCormick RD, Springman SR, Maki DG. The pathogenesis and epidemiology of catheter-related infection with pulmonary artery Swan-Ganz catheters: a prospective study utilizing molecular subtyping. *Am J Med* 1991; 91:197S–205.
38. Parienti JJ, Thirion M, Megarbane B, et al. Femoral vs jugular venous catheterization and risk of nosocomial events in adults requiring acute renal replacement therapy: a randomized controlled trial. *JAMA* 2008; 299:2413–22.
39. Moretti EW, Ofstead CL, Kristy RM, Wetzler HP. Impact of central venous catheter type and methods on catheter-related colonization and bacteraemia. *J Hosp Infect* 2005; 61:139–45.
40. Nagashima G, Kikuchi T, Tsuyuzaki H, et al. To reduce catheter-related bloodstream infections: is the subclavian route better than the jugular route for central venous catheterization? *J Infect Chemother* 2006; 12:363–5.
41. Ruesch S, Walder B, Tramer MR. Complications of central venous catheters: internal jugular versus subclavian access—a systematic review. *Crit Care Med* 2002; 30:454–60.
42. Sadoyama G, Gontijo Filho PP. Comparison between the jugular and subclavian vein as insertion site for central venous catheters: microbiological aspects and risk factors for colonization and infection. *Braz J Infect Dis* 2003; 7:142–8.
43. Heard SO, Wagle M, Vijayakumar E, et al. Influence of triple-lumen central venous catheters coated with chlorhexidine and silver sulfadiazine on the incidence of catheter-related bacteremia. *Arch Intern Med* 1998; 158:81–7.
44. Richet H, Hubert B, Nitemberg G, et al. Prospective multicenter study of vascular-catheter-related complications and risk factors for positive central-catheter cultures in intensive care unit patients. *J Clin Microbiol* 1990; 28:2520–5.

45. Safdar N, Kluger DM, Maki DG. A review of risk factors for catheter-related bloodstream infection caused by percutaneously inserted, noncuffed central venous catheters: implications for preventive strategies. *Medicine (Baltimore)* 2002; 81:466–79.
46. Lorente L, Jimenez A, Iribarren JL, Jimenez JJ, Martin MM, Mora ML. The micro-organism responsible for central venous catheter related bloodstream infection depends on catheter site. *Intensive Care Med* 2006; 32:1449–50.
47. Traore O, Liotier J, Souweine B. Prospective study of arterial and central venous catheter colonization and of arterial-and central venous catheter-related bacteremia in intensive care units. *Crit Care Med* 2005; 33:1276–80.
48. Joynt GM, Kew J, Gomersall CD, Leung VY, Liu EK. Deep venous thrombosis caused by femoral venous catheters in critically ill adult patients. *Chest* 2000; 117:178–83.
49. Mian NZ, Bayly R, Schreck DM, Besserman EB, Richmand D. Incidence of deep venous thrombosis associated with femoral venous catheterization. *Acad Emerg Med* 1997; 4:1118–21.
50. Merrer J, De Jonghe B, Golliot F, et al. Complications of femoral and subclavian venous catheterization in critically ill patients: a randomized controlled trial. *JAMA* 2001; 286:700–7.
51. Goetz AM, Wagener MM, Miller JM, Muder RR. Risk of infection due to central venous catheters: effect of site of placement and catheter type. *Infect Control Hosp Epidemiol* 1998; 19:842–5.
52. Robinson JF, Robinson WA, Cohn A, Garg K, Armstrong JD, 2nd. Perforation of the great vessels during central venous line placement. *Arch Intern Med* 1995; 155:1225–8.
53. Trottier SJ, Veremakis C, O'Brien J, Auer AI. Femoral deep vein thrombosis associated with central venous catheterization: results from a prospective, randomized trial. *Crit Care Med* 1995; 23:52–9.
54. Lorente L, Henry C, Martin MM, Jimenez A, Mora ML. Central venous catheter-related infection in a prospective and observational study of 2,595 catheters. *Crit Care* 2005; 9:R631–5.
55. Schillinger F, Schillinger D, Montagnac R, Milcent T. Post catheterisation vein stenosis in haemodialysis: comparative angiographic study of 50 subclavian and 50 internal jugular accesses. *Nephrol Dial Transplant* 1991; 6:722–4.
56. Cimochowski GE, Worley E, Rutherford WE, Sartain J, Blondin J, Harter H. Superiority of the internal jugular over the subclavian access for temporary dialysis. *Nephron* 1990; 54:154–61.
57. Barrett N, Spencer S, McIvor J, Brown EA. Subclavian stenosis: a major complication of subclavian dialysis catheters. *Nephrol Dial Transplant* 1988; 3:423–5.
58. Trerotola SO, Kuhn-Fulton J, Johnson MS, Shah H, Ambrosius WT, Kneebone PH. Tunneled infusion catheters: increased incidence of symptomatic venous thrombosis after subclavian versus internal jugular venous access. *Radiology* 2000; 217:89–93.
59. National Kidney Foundation. III. NKF-K/DOQI Clinical Practice Guidelines for Vascular Access: update 2000. *Am J Kidney Dis* 2001; 37:S137–81.
60. Hind D, Calvert N, McWilliams R, et al. Ultrasonic locating devices for central venous cannulation: meta-analysis. *BMJ* 2003; 327:361.

61. Randolph AG, Cook DJ, Gonzales CA, Pribble CG. Ultrasound guidance for placement of central venous catheters: a meta-analysis of the literature. *Crit Care Med* 1996; 24:2053–8.
62. Froehlich CD, Rigby MR, Rosenberg ES, et al. Ultrasound-guided central venous catheter placement decreases complications and decreases placement attempts compared with the landmark technique in patients in a pediatric intensive care unit. *Crit Care Med* 2009; 37:1090–6.
63. Lamperti M, Caldiroli D, Cortellazzi P, et al. Safety and efficacy of ultrasound assistance during internal jugular vein cannulation in neurosurgical infants. *Intensive Care Med* 2008; 34:2100–5.
64. Schweickert WD, Herlitz J, Pohlman AS, Gehlbach BK, Hall JB, Kress JP. A randomized, controlled trial evaluating postinsertion neck ultrasound in peripherally inserted central catheter procedures. *Crit Care Med* 2009; 37:1217–21.
65. Clark-Christoff N, Watters VA, Sparks W, Snyder P, Grant JP. Use of triple-lumen subclavian catheters for administration of total parenteral nutrition. *JPEN J Parenter Enteral Nutr* 1992; 16:403–7.
66. Early TF, Gregory RT, Wheeler JR, Snyder SO Jr., Gayle RG. Increased infection rate in double-lumen versus single-lumen Hickman catheters in cancer patients. *South Med J* 1990; 83:34–6.
67. Hilton E, Haslett TM, Borenstein MT, Tucci V, Isenberg HD, Singer C. Central catheter infections: single-versus triple-lumen catheters. Influence of guide wires on infection rates when used for replacement of catheters. *Am J Med* 1988; 84:667–72.
68. Yeung C, May J, Hughes R. Infection rate for single lumen v triple lumen subclavian catheters. *Infect Control Hosp Epidemiol* 1988; 9:154–8.
69. Pronovost P, Needham D, Berenholtz S, et al. An intervention to decrease catheter-related bloodstream infections in the ICU. *N Engl J Med* 2006; 355:2725–32.
70. Berenholtz SM, Pronovost PJ, Lipsett PA, et al. Eliminating catheter-related bloodstream infections in the intensive care unit. *Crit Care Med* 2004; 32:2014–20.
71. Lederle FA, Parenti CM, Berskow LC, Ellingson KJ. The idle intravenous catheter. *Ann Intern Med* 1992; 116:737–8.
72. Parenti CM, Lederle FA, Impola CL, Peterson LR. Reduction of unnecessary intravenous catheter use. Internal medicine house staff participate in a successful quality improvement project. *Arch Intern Med* 1994; 154:1829–32.
73. Abi-Said D, Raad I, Umphrey J, et al. Infusion therapy team and dressing changes of central venous catheters. *Infect Control Hosp Epidemiol* 1999; 20:101–5.
74. Capdevila JA, Segarra A, Pahissa A. Catheter-related bacteremia in patients undergoing hemodialysis. *Ann Intern Med* 1998; 128:600.
75. Mermel LA, Maki DG. Infectious complications of Swan-Ganz pulmonary artery catheters. Pathogenesis, epidemiology, prevention, and management. *Am J Respir Crit Care Med* 1994; 149:1020–36.

76. Raad II, Hohn DC, Gilbreath BJ, et al. Prevention of central venous catheter-related infections by using maximal sterile barrier precautions during insertion. *Infect Control Hosp Epidemiol* 1994; 15:231–8.
77. Boyce JM, Pittet D. Guideline for hand hygiene in health-care settings: recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force. *Infect Control Hosp Epidemiol* 2002; 23:S3–40.
78. Bischoff WE, Reynolds TM, Sessler CN, Edmond MB, Wenzel RP. Handwashing compliance by health care workers: the impact of introducing an accessible, alcohol-based hand antiseptic. *Arch Intern Med* 2000; 160:1017–21.
79. Pittet D, Dharan S, Touveneau S, Sauvan V, Perneger TV. Bacterial contamination of the hands of hospital staff during routine patient care. *Arch Intern Med* 1999; 159:821–6.
80. Carrer S, Bocchi A, Bortolotti M, et al. Effect of different sterile barrier precautions and central venous catheter dressing on the skin colonization around the insertion site. *Minerva Anestesiol* 2005; 71:197–206.
81. Cohen Y, Fosse JP, Karoubi P, et al. The "hands-off" catheter and the prevention of systemic infections associated with pulmonary artery catheter: a prospective study. *Am J Respir Crit Care Med* 1998; 157:284–7.
82. Maki DG, Ringer M, Alvarado CJ. Prospective randomised trial of povidone-iodine, alcohol, and chlorhexidine for prevention of infection associated with central venous and arterial catheters. *Lancet* 1991; 338:339–43.
83. Mimoz O, Pieroni L, Lawrence C, et al. Prospective, randomized trial of two antiseptic solutions for prevention of central venous or arterial catheter colonization and infection in intensive care unit patients. *Crit Care Med* 1996; 24:1818–23.
84. Maki DG, Stolz SS, Wheeler S, Mermel LA. A prospective, randomized trial of gauze and two polyurethane dressings for site care of pulmonary artery catheters: implications for catheter management. *Crit Care Med* 1994; 22:1729–37.
85. Bijma R, Girbes AR, Kleijer DJ, Zwaveling JH. Preventing central venous catheter-related infection in a surgical intensive-care unit. *Infect Control Hosp Epidemiol* 1999; 20:618–20.
86. Madeo M, Martin CR, Turner C, Kirkby V, Thompson DR. A randomized trial comparing Arglaes (a transparent dressing containing silver ions) to Tegaderm (a transparent polyurethane dressing) for dressing peripheral arterial catheters and central vascular catheters. *Intensive Crit Care Nurs* 1998; 14:187–91.
87. Rasero L, Degl'Innocenti M, Mocali M, et al. Comparison of two different time interval protocols for central venous catheter dressing in bone marrow transplant patients: results of a randomized, multicenter study. *The Italian Nurse Bone Marrow Transplant Group (GITMO). Haematologica* 2000; 85:275–9.
88. Zakrzewska-Bode A, Muytjens HL, Liem KD, Hoogkamp-Korstanje JA. Mupirocin resistance in coagulase-negative staphylococci, after topical prophylaxis for the reduction of colonization of central venous catheters. *J Hosp Infect* 1995; 31:189–93.
89. Flowers RH, Schwenzer KJ, Kopel RF, Fisch MJ, Tucker SI, Farr BM. Efficacy of an attachable subcutaneous cuff for the prevention of intravascular catheter-related infection. A randomized, controlled trial. *JAMA* 1989; 261:878–83.

90. Robbins J, Cromwell P, Korones DN. Swimming and central venous catheter-related infections in the child with cancer. *J Pediatr Oncol Nurs* 1999; 16:51–6.
91. Howell PB, Walters PE, Donowitz GR, Farr BM. Risk factors for infection of adult patients with cancer who have tunneled central venous catheters. *Cancer* 1995; 75:1367–75.
92. Ivy DD, Calderbank M, Wagner BD, et al. Closed-hub systems with protected connections and the reduction of risk of catheter-related bloodstream infection in pediatric patients receiving intravenous prostanoid therapy for pulmonary hypertension. *Infect Control Hosp Epidemiol* 2009; 30:823–9.
93. Timsit JF, Schwebel C, Bouadma L, et al. Chlorhexidine-impregnated sponges and less frequent dressing changes for prevention of catheter-related infections in critically ill adults: a randomized controlled trial. *JAMA* 2009; 301:1231–41.
94. Rao SP, Oreopoulos DG. Unusual complications of a polyurethane PD catheter. *Perit Dial Int* 1997; 17:410–2.
95. Riu S, Ruiz CG, Martinez-Vea A, Peralta C, Oliver JA. Spontaneous rupture of polyurethane peritoneal catheter. A possible deleterious effect of mupirocin ointment. *Nephrol Dial Transplant* 1998; 13:1870–1.
96. Garland JS, Alex CP, Mueller CD, et al. A randomized trial comparing povidone-iodine to a chlorhexidine gluconate-impregnated dressing for prevention of central venous catheter infections in neonates. *Pediatrics* 2001; 107:1431–6.
97. Ho KM, Litton E. Use of chlorhexidine-impregnated dressing to prevent vascular and epidural catheter colonization and infection: a meta-analysis. *J Antimicrob Chemother* 2006; 58:281–7.
98. Levy I, Katz J, Solter E, et al. Chlorhexidine-impregnated dressing for prevention of colonization of central venous catheters in infants and children: a randomized controlled study. *Pediatr Infect Dis J* 2005; 24:676–9.
99. Lorenzen AN, Itkin DJ. Surveillance of infection in home care. *Am J Infect Control* 1992; 20:326–9.
100. White MC. Infections and infection risks in home care settings. *Infect Control Hosp Epidemiol* 1992; 13:535–9.
101. White MC, Ragland KE. Surveillance of intravenous catheter-related infections among home care clients. *Am J Infect Control* 1994; 22:231–5.
102. Bleasdale SC, Trick WE, Gonzalez IM, Lyles RD, Hayden MK, Weinstein RA. Effectiveness of chlorhexidine bathing to reduce catheter-associated bloodstream infections in medical intensive care unit patients. *Arch Intern Med* 2007; 167:2073–9.
103. Munoz-Price LS, Hota B, Stemer A, Weinstein RA. Prevention of bloodstream infections by use of daily chlorhexidine baths for patients at a long-term acute care hospital. *Infect Control Hosp Epidemiol* 2009; 30:1031–5.
104. Popovich KJ, Hota B, Hayes R, Weinstein RA, Hayden MK. Effectiveness of routine patient cleansing with chlorhexidine gluconate for infection prevention in the medical intensive care unit. *Infect Control Hosp Epidemiol* 2009; 30:959–63.

105. Yamamoto AJ, Solomon JA, Soulen MC, et al. Sutureless securement device reduces complications of peripherally inserted central venous catheters. *J Vasc Interv Radiol* 2002; 13:77–81.
106. Brun-Buisson C, Doyon F, Sollet JP, Cochard JF, Cohen Y, Nitenberg G. Prevention of intravascular catheter-related infection with newer chlorhexidine-silver sulfadiazine-coated catheters: a randomized controlled trial. *Intensive Care Med* 2004; 30:837–43.
107. Ostendorf T, Meinhold A, Harter C, et al. Chlorhexidine and silver-sulfadiazine coated central venous catheters in haematological patients—a double-blind, randomised, prospective, controlled trial. *Support Care Cancer* 2005; 13:993–1000.
108. Rupp ME, Lisco SJ, Lipsett PA, et al. Effect of a second-generation venous catheter impregnated with chlorhexidine and silver sulfadiazine on central catheter-related infections: a randomized, controlled trial. *Ann Intern Med* 2005; 143:570–80.
109. Darouiche RO, Raad II, Heard SO, et al. A comparison of two antimicrobial-impregnated central venous catheters. Catheter Study Group. *N Engl J Med* 1999; 340:1–8.
110. Raad I, Darouiche R, Dupuis J, et al. Central venous catheters coated with minocycline and rifampin for the prevention of catheter-related colonization and bloodstream infections. A randomized, double-blind trial. The Texas Medical Center Catheter Study Group. *Ann Intern Med* 1997; 127:267–74.
111. Hanna H, Benjamin R, Chatzinikolaou I, et al. Long-term silicone central venous catheters impregnated with minocycline and rifampin decrease rates of catheter-related bloodstream infection in cancer patients: a prospective randomized clinical trial. *J Clin Oncol* 2004; 22:3163–71.
112. Bhutta A, Gilliam C, Honeycutt M, et al. Reduction of bloodstream infections associated with catheters in paediatric intensive care unit: stepwise approach. *BMJ* 2007; 334:362–5.
113. Chelliah A, Heydon KH, Zaoutis TE, et al. Observational trial of antibiotic-coated central venous catheters in critically ill pediatric patients. *Pediatr Infect Dis J* 2007; 26:816–20.
114. van de Wetering MD, van Woensel JBM. Prophylactic antibiotics for preventing early central venous catheter Gram positive infections in oncology patients. *Cochrane Database of Systematic Reviews* 2007; Issue 1. Art. No.: CD003295. DOI: 10.1002/14651858.CD003295.pub2.
115. Maki DG, Band JD. A comparative study of polyantibiotic and iodophor ointments in prevention of vascular catheter-related infection. *Am J Med* 1981; 70:739–44.
116. Fukunaga A, Naritaka H, Fukaya R, Tabuse M, Nakamura T. Povidone-iodine ointment and gauze dressings associated with reduced catheter-related infection in seriously ill neurosurgical patients. *Infect Control Hosp Epidemiol* 2004; 25:696–8.
117. Johnson DW, MacGinley R, Kay TD, et al. A randomized controlled trial of topical exit site mupirocin application in patients with tunnelled, cuffed haemodialysis catheters. *Nephrol Dial Transplant* 2002; 17:1802–7.
118. Fong IW. Prevention of haemodialysis and peritoneal dialysis catheter related infection by topical povidone-iodine. *Postgrad Med J* 1993; 69(Suppl 3):S15–7.
119. Levin A, Mason AJ, Jindal KK, Fong IW, Goldstein MB. Prevention of hemodialysis subclavian vein catheter infections by topical povidone-iodine. *Kidney Int* 1991; 40:934–8.

120. Schwartz C, Henrickson KJ, Roghmann K, Powell K. Prevention of bacteremia attributed to luminal colonization of tunneled central venous catheters with vancomycin-susceptible organisms. *J Clin Oncol* 1990; 8:1591–7.
121. Rackoff WR, Weiman M, Jakobowski D, et al. A randomized, controlled trial of the efficacy of a heparin and vancomycin solution in preventing central venous catheter infections in children. *J Pediatr* 1995; 127:147–51.
122. Carratala J, Niubo J, Fernandez-Sevilla A, et al. Randomized, double-blind trial of an antibiotic-lock technique for prevention of gram-positive central venous catheter-related infection in neutropenic patients with cancer. *Antimicrob Agents Chemother* 1999; 43:2200–4.
123. Jurewitsch B, Lee T, Park J, Jeejeebhoy K. Taurolidine 2% as an antimicrobial lock solution for prevention of recurrent catheter-related bloodstream infections. *J Parenter Enteral Nutr* 1998; 22:242–4.
124. Henrickson KJ, Axtell RA, Hoover SM, et al. Prevention of central venous catheter-related infections and thrombotic events in immunocompromised children by the use of vancomycin/ciprofloxacin/ heparin flush solution: a randomized, multicenter, double-blind trial. *J Clin Oncol* 2000; 18:1269–78.
125. Garland JS, Alex CP, Henrickson KJ, McAuliffe TL, Maki DG. A vancomycin-heparin lock solution for prevention of nosocomial bloodstream infection in critically ill neonates with peripherally inserted central venous catheters: a prospective, randomized trial. *Pediatrics* 2005; 116:e198–205.
126. Daghistani D, Horn M, Rodriguez Z, Schoenike S, Toledano S. Prevention of indwelling central venous catheter sepsis. *Med Pediatr Oncol* 1996; 26:405–8.
127. Barriga FJ, Varas M, Potin M, et al. Efficacy of a vancomycin solution to prevent bacteremia associated with an indwelling central venous catheter in neutropenic and non-neutropenic cancer patients. *Med Pediatr Oncol* 1997; 28:196–200.
128. Dogra GK, Herson H, Hutchison B, et al. Prevention of tunneled hemodialysis catheter-related infections using catheter-restricted filling with gentamicin and citrate: a randomized controlled study. *J Am Soc Nephrol* 2002; 13:2133–9.
129. Allon M. Prophylaxis against dialysis catheter-related bacteremia with a novel antimicrobial lock solution. *Clin Infect Dis* 2003; 36:1539–44.
130. Elhassan NO, Stevens TP, Gigliotti F, Hardy DJ, Cole CA, Sinkin RA. Vancomycin usage in central venous catheters in a neonatal intensive care unit. *Pediatr Infect Dis J* 2004; 23:201–6.
131. McIntyre CW, Hulme LJ, Taal M, Fluck RJ. Locking of tunneled hemodialysis catheters with gentamicin and heparin. *Kidney Int* 2004; 66:801–5.
132. Betjes MG, van Agteren M. Prevention of dialysis catheter-related sepsis with a citrate-taurolidine-containing lock solution. *Nephrol Dial Transplant* 2004; 19:1546–1.
133. Weijmer MC, van den Dorpel MA, Van de Ven PJ, et al. Randomized, clinical trial comparison of trisodium citrate 30% and heparin as catheter-locking solution in hemodialysis patients. *J Am Soc Nephrol* 2005; 16:2769–77.
134. Bleyer AJ, Mason L, Russell G, Raad II, Sherertz RJ. A randomized, controlled trial of a new vascular catheter flush solution (minocycline-EDTA) in temporary hemodialysis access. *Infect Control Hosp Epidemiol* 2005; 26:520–4.

135. Kim SH, Song KI, Chang JW, et al. Prevention of uncuffed hemodialysis catheter-related bacteremia using an antibiotic lock technique: a prospective, randomized clinical trial. *Kidney Int* 2006; 69:161–4.
136. Al-Hwiesh AK, Abdul-Rahman IS. Successful prevention of tunneled, central catheter infection by antibiotic lock therapy using vancomycin and gentamycin. *Saudi J Kidney Dis Transpl* 2007; 18:239–47.
137. Nori US, Manoharan A, Yee J, Besarab A. Comparison of low-dose gentamicin with minocycline as catheter lock solutions in the prevention of catheter-related bacteremia. *Am J Kidney Dis* 2006; 48:596–605.
138. Saxena AK, Panhotra BR, Sundaram DS, et al. Tunneled catheters' outcome optimization among diabetics on dialysis through antibiotic-lock placement. *Kidney Int* 2006; 70:1629–35.
139. Randolph AG, Cook DJ, Gonzales CA, Andrew M. Benefit of heparin in central venous and pulmonary artery catheters: a meta-analysis of randomized controlled trials. *Chest* 1998; 113:165–71.
140. Tager IB, Ginsberg MB, Ellis SE, et al. An epidemiologic study of the risks associated with peripheral intravenous catheters. *Am J Epidemiol* 1983; 118:839–51.
141. Lai KK. Safety of prolonging peripheral cannula and i.v. tubing use from 72 hours to 96 hours. *Am J Infect Control* 1998; 26:66–70.
142. Van Donk P, Rickard CM, McGrail MR, Doolan G. Routine replacement versus clinical monitoring of peripheral intravenous catheters in a regional hospital in the home program: a randomized controlled trial. *Infect Control Hosp Epidemiol* 2009; 30:915–7.
143. Webster J, Clarke S, Paterson D, et al. Routine care of peripheral intravenous catheters versus clinically indicated replacement: randomised controlled trial. *BMJ* 2008; 337:a339.
144. Webster J, Osborne S, Rickard C, Hall J. Clinically-indicated replacement versus routine replacement of peripheral venous catheters. *Cochrane Database Syst Rev* 2010; 3:CD007798.
145. Boo NY, Wong NC, Zulkifli SS, Lye MS. Risk factors associated with umbilical vascular catheter-associated thrombosis in newborn infants. *J Paediatr Child Health* 1999; 35:460–5.
146. Garland JS, Buck RK, Maloney P, et al. Comparison of 10% povidone-iodine and 0.5% chlorhexidine gluconate for the prevention of peripheral intravenous catheter colonization in neonates: a prospective trial. *Pediatr Infect Dis J* 1995; 14:510–6.
147. Krauss AN, Albert RF, Kannan MM. Contamination of umbilical catheters in the newborn infant. *J Pediatr* 1970; 77:965–9.
148. Landers S, Moise AA, Fraley JK, Smith EO, Baker CJ. Factors associated with umbilical catheter-related sepsis in neonates. *Am J Dis Child* 1991; 145:675–80.
149. Cronin WA, Germanson TP, Donowitz LG. Intravascular catheter colonization and related bloodstream infection in critically ill neonates. *Infect Control Hosp Epidemiol* 1990; 11:301–8.
150. Miller KL, Coen PE, White WJ, Hurst WJ, Achey BE, Lang CM. Effectiveness of skin absorption of tincture of I in blocking radioiodine from the human thyroid gland. *Health Phys* 1989; 56:911–4.

Guidelines for the Prevention of Intravascular Catheter-Related Infections

151. Ankola PA, Atakent YS. Effect of adding heparin in very low concentration to the infusate to prolong the patency of umbilical artery catheters. *Am J Perinatol* 1993; 10:229–32.
152. David RJ, Merten DF, Anderson JC, Gross S. Prevention of umbilical artery catheter clots with heparinized infusates. *Dev Pharmacol Ther* 1981; 2:117–26.
153. Horgan MJ, Bartoletti A, Polansky S, Peters JC, Manning TJ, Lamont BM. Effect of heparin infusates in umbilical arterial catheters on frequency of thrombotic complications. *J Pediatr* 1987; 111:774–8.
154. Fletcher MA, Brown DR, Landers S, Seguin J. Umbilical arterial catheter use: report of an audit conducted by the Study Group for Complications of Perinatal Care. *Am J Perinatol* 1994; 11:94–9.
155. Seguin J, Fletcher MA, Landers S, Brown D, Macpherson T. Umbilical venous catheterizations: audit by the Study Group for Complications of Perinatal Care. *Am J Perinatol* 1994; 11:67–70.
156. Loisel DB, Smith MM, MacDonald MG, Martin GR. Intravenous access in newborn infants: impact of extended umbilical venous catheter use on requirement for peripheral venous lines. *J Perinatol* 1996; 16:461–6.
157. Martin C, Saux P, Papazian L, Gouin F. Long-term arterial cannulation in ICU patients using the radial artery or dorsalis pedis artery. *Chest* 2001; 119:901–6.
158. Koh DB, Gowardman JR, Rickard CM, Robertson IK, Brown A. Prospective study of peripheral arterial catheter infection and comparison with concurrently sited central venous catheters. *Crit Care Med* 2008; 36:397–402.
159. Rijnders BJ, Van Wijngaerden E, Wilmer A, Peetermans WE. Use of full sterile barrier precautions during insertion of arterial catheters: a randomized trial. *Clin Infect Dis* 2003; 36:743–8.
160. Donowitz LG, Marsik FJ, Hoyt JW, Wenzel RP. *Serratia marcescens* bacteremia from contaminated pressure transducers. *JAMA* 1979; 242:1749–51.
161. Luskin RL, Weinstein RA, Nathan C, Chamberlin WH, Kabins SA. Extended use of disposable pressure transducers. A bacteriologic evaluation. *JAMA* 1986; 255:916–20.
162. Maki DG, Hassemer CA. Endemic rate of fluid contamination and related septicemia in arterial pressure monitoring. *Am J Med* 1981; 70:733–8.
163. Mermel LA, Maki DG. Epidemic bloodstream infections from hemodynamic pressure monitoring: signs of the times. *Infect Control Hosp Epidemiol* 1989; 10:47–53.
164. Tenold R, Priano L, Kim K, Rourke B, Marrone T. Infection potential of nondisposable pressure transducers prepared prior to use. *Crit Care Med* 1987; 15:582–3.
165. Eyer S, Brummitt C, Crossley K, Siegel R, Cerra F. Catheter-related sepsis: prospective, randomized study of three methods of long-term catheter maintenance. *Crit Care Med* 1990; 18:1073–9.
166. Raad I, Umphrey J, Khan A, Truett LJ, Bodey GP. The duration of placement as a predictor of peripheral and pulmonary arterial catheter infections. *J Hosp Infect* 1993; 23:17–26.
167. Thomas F, Burke JP, Parker J, et al. The risk of infection related to radial vs femoral sites for arterial catheterization. *Crit Care Med* 1983; 11:807–12.

168. Leroy O, Billiau V, Beuscart C, et al. Nosocomial infections associated with long-term radial artery cannulation. *Intensive Care Med* 1989; 15:241–6.
169. Fisher MC, Long SS, Roberts EM, Dunn JM, Balsara RK. *Pseudomonas maltophilia* bacteremia in children undergoing open heart surgery. *JAMA* 1981; 246:1571–4.
170. Stamm WE, Colella JJ, Anderson RL, Dixon RE. Indwelling arterial catheters as a source of nosocomial bacteremia. An outbreak caused by *Flavobacterium* Species. *N Engl J Med* 1975; 292:1099–102.
171. Weinstein RA, Emori TG, Anderson RL, Stamm WE. Pressure transducers as a source of bacteremia after open heart surgery. Report of an outbreak and guidelines for prevention. *Chest* 1976; 69:338–44.
172. Shinozaki T, Deane RS, Mazuzan JE Jr., Hamel AJ, Hazelton D. Bacterial contamination of arterial lines. A prospective study. *JAMA* 1983; 249:223–5.
173. Solomon SL, Alexander H, Eley JW, et al. Nosocomial fungemia in neonates associated with intravascular pressure-monitoring devices. *Pediatr Infect Dis* 1986; 5:680–5.
174. Weems JJ Jr., Chamberland ME, Ward J, Willy M, Padhye AA, Solomon SL. *Candida* parapsilosis fungemia associated with parenteral nutrition and contaminated blood pressure transducers. *J Clin Microbiol* 1987; 25:1029–32.
175. Villarino ME, Jarvis WR, O’Hara C, Bresnahan J, Clark N. Epidemic of *Serratia marcescens* bacteremia in a cardiac intensive care unit. *J Clin Microbiol* 1989; 27:2433–6.
176. Beck-Sague CM, Jarvis WR, Brook JH, et al. Epidemic bacteremia due to *Acinetobacter baumannii* in five intensive care units. *Am J Epidemiol* 1990; 132:723–33.
177. Gillies D, Wallen MM, Morrison AL, Rankin K, Nagy SA, O’Riordan
2. E. Optimal timing for intravenous administration set replacement. *Cochrane Database of Systematic Reviews* 2005; Issue 4. Art. No.: CD003588. DOI: 10.1002/14651858.CD003588.pub2.
178. Sitges-Serra A, Linares J, Perez JL, Jaurrieta E, Lorente L. A randomized trial on the effect of tubing changes on hub contamination
3. and catheter sepsis during parenteral nutrition. *JPEN J Parenter Enteral Nutr* 1985; 9:322–5.
179. Snyderman DR, Donnelly-Reidy M, Perry LK, Martin WJ. Intravenous tubing containing burettes can be safely changed at 72 hour intervals. *Infect Control* 1987; 8:113–6.
180. Maki DG, Botticelli JT, LeRoy ML, Thielke TS. Prospective study of replacing administration sets for intravenous therapy at 48-vs 72hour intervals. 72 hours is safe and cost-effective. *JAMA* 1987; 258:1777–81.
181. Josephson A, Gombert ME, Sierra MF, Karanfil LV, Tansino GF. The relationship between intravenous fluid contamination and the frequency of tubing replacement. *Infect Control* 1985; 6:367–70.
182. Melly MA, Meng HC, Schaffner W. Microbiol growth in lipid emulsions used in parenteral nutrition. *Arch Surg* 1975; 110:1479–81.
183. Mershon J, Nogami W, Williams JM, Yoder C, Eitzen HE, Lemons JA. Bacterial/fungal growth in a combined parenteral nutrition solution. *JPEN J Parenter Enteral Nutr* 1986; 10:498–502.

184. Gilbert M, Gallagher SC, Eads M, Elmore MF. Microbial growth patterns in a total parenteral nutrition formulation containing lipid emulsion. *JPEN J Parenter Enteral Nutr* 1986; 10:494–7.
185. Maki DG, Martin WT. Nationwide epidemic of septicemia caused by contaminated infusion products. IV. Growth of microbial pathogens in fluids for intravenous infusions. *J Infect Dis* 1975; 131:267–72.
186. Bennett SN, McNeil MM, Bland LA, et al. Postoperative infections traced to contamination of an intravenous anesthetic, propofol. *N Engl J Med* 1995; 333:147–54.
187. Arduino MJ, Bland LA, Danzig LE, McAllister SK, Aguero SM. Microbiologic evaluation of needleless and needle-access devices. *Am J Infect Control* 1997; 25:377–80.
188. Brown JD, Moss HA, Elliott TS. The potential for catheter microbial contamination from a needleless connector. *J Hosp Infect* 1997; 36:181–9.
189. Cookson ST, Ihrig M, O’Mara EM, et al. Increased bloodstream infection rates in surgical patients associated with variation from recommended use and care following implementation of a needleless device. *Infect Control Hosp Epidemiol* 1998; 19:23–7.
190. Seymour VM, Dhallu TS, Moss HA, Tebbs SE, Elliot TS. A prospective clinical study to investigate the microbial contamination of a needle-less connector. *J Hosp Infect* 2000; 45:165–8.
191. Luebke MA, Arduino MJ, Duda DL, et al. Comparison of the microbial barrier properties of a needleless and a conventional needle-based intravenous access system. *Am J Infect Control* 1998; 26:437–41.
192. McDonald LC, Banerjee SN, Jarvis WR. Line-associated bloodstream infections in pediatric intensive-care-unit patients associated with a needleless device and intermittent intravenous therapy. *Infect Control Hosp Epidemiol* 1998; 19:772–7.
193. Mendelson MH, Short LJ, Schechter CB, et al. Study of a needleless intermittent intravenous-access system for peripheral infusions: analysis of staff, patient, and institutional outcomes. *Infect Control Hosp Epidemiol* 1998; 19:401–6.
194. Do AN, Ray BJ, Banerjee SN, et al. Bloodstream infection associated with needleless device use and the importance of infection-control practices in the home health care setting. *J Infect Dis* 1999; 179:442–8.
195. Soothill JS, Bravery K, Ho A, Macqueen S, Collins J, Lock P. A fall in bloodstream infections followed a change to 2% chlorhexidine in 70% isopropanol for catheter connection antisepsis: a pediatric single center before/after study on a hemopoietic stem cell transplant ward. *Am J Infect Control* 2009; 37:626–30.
196. Casey AL, Burnell S, Whinn H, Worthington T, Faroqui MH, Elliott TS. A prospective clinical trial to evaluate the microbial barrier of a needleless connector. *J Hosp Infect* 2007; 65:212–8.
197. Rupp ME, Sholtz LA, Jourdan DR, et al. Outbreak of bloodstream infection temporally associated with the use of an intravascular needleless valve. *Clin Infect Dis* 2007; 44:1408–14.
198. Salgado CD, Chinnes L, Paczesny TH, Cantey JR. Increased rate of catheter-related bloodstream infection associated with use of a needleless mechanical valve device at a long-term acute care hospital. *Infect Control Hosp Epidemiol* 2007; 28:684–8.

199. Maragakis LL, Bradley KL, Song X, et al. Increased catheter-related bloodstream infection rates after the introduction of a new mechanical valve intravenous access port. *Infect Control Hosp Epidemiol* 2006; 27:67–70.
200. Field K, McFarlane C, Cheng AC, et al. Incidence of catheter-related bloodstream infection among patients with a needleless, mechanical valve-based intravenous connector in an Australian hematology-oncology unit. *Infect Control Hosp Epidemiol* 2007; 28:610–3.
201. Costello JM, Morrow DF, Graham DA, Potter-Bynoe G, Sandora TJ, Laussen PC. Systematic intervention to reduce central line-associated bloodstream infection rates in a pediatric cardiac intensive care unit. *Pediatrics* 2008; 121:915–23.
202. Frankel HL, Crede WB, Topal JE, Roumanis SA, Devlin MW, Foley AB. Use of corporate Six Sigma performance-improvement strategies to reduce incidence of catheter-related bloodstream infections in a surgical ICU. *J Am Coll Surg* 2005; 201:349–58.
203. Galpern D, Guerrero A, Tu A, Fahoum B, Wise L. Effectiveness of a central line bundle campaign on line-associated infections in the intensive care unit. *Surgery* 2008; 144:492–5; discussion 495.
204. McKee C, Berkowitz I, Cosgrove SE, et al. Reduction of catheter-associated bloodstream infections in pediatric patients: experimentation and reality. *Pediatr Crit Care Med* 2008; 9:40–6.
205. Pronovost PJ, Berenholtz SM, Goeschel CA. Improving the quality of measurement and evaluation in quality improvement efforts. *Am J Med Qual* 2008; 23:143–6.
206. Horan TC, Andrus M, Dudeck MA. CDC/NHSN surveillance definition of health care-associated infection and criteria for specific types of infections in the acute care setting. *Am J Infect Control* 2008; 36:309–32.
207. Edwards JR, Peterson KD, Mu Y, et al. National Healthcare Safety Network (NHSN) report: data summary for 2006 through 2008, issued December 2009. *Am J Infect Control* 2009; 37:783–805.
208. Wisplinghoff H, Bischoff T, Tallent SM, Seifert H, Wenzel RP, Edmond MB. Nosocomial bloodstream infections in US hospitals: analysis of 24,179 cases from a prospective nationwide surveillance study. *Clin Infect Dis* 2004; 39:309–17.
209. Gaynes R, Edwards JR. Overview of nosocomial infections caused by gram-negative bacilli. *Clin Infect Dis* 2005; 41:848–54.
210. Burton DC, Edwards JR, Horan TC, Jernigan JA, Fridkin SK. Methicillin-resistant *Staphylococcus aureus* central line-associated bloodstream infections in US intensive care units, 1997–2007. *JAMA* 2009; 301:727–36.
211. Safdar N, Maki DG. The pathogenesis of catheter-related bloodstream infection with noncuffed short-term central venous catheters. *Intensive Care Med* 2004; 30:62–7.
212. Maki DG, Weise CE, Sarafin HW. A semiquantitative culture method for identifying intravenous-catheter-related infection. *N Engl J Med* 1977; 296:1305–9.
213. Raad I, Costerton W, Sabharwal U, Sacilowski M, Anaissie E, Bodey GP. Ultrastructural analysis of indwelling vascular catheters: a quantitative relationship between luminal colonization and duration of placement. *J Infect Dis* 1993; 168:400–7.

214. Dobbins BM, Kite P, Kindon A, McMahon MJ, Wilcox MH. DNA fingerprinting analysis of coagulase negative staphylococci implicated in catheter related bloodstream infections. *J Clin Pathol* 2002; 55:824–8.
215. Anaissie E, Samonis G, Kontoyiannis D, et al. Role of catheter colonization and infrequent hematogenous seeding in catheter-related infections. *Eur J Clin Microbiol Infect Dis* 1995; 14:134–7.
216. Raad I, Hanna HA, Awad A, et al. Optimal frequency of changing intravenous administration sets: is it safe to prolong use beyond 72 hours? *Infect Control Hosp Epidemiol* 2001; 22:136–9.
217. Mehall JR, Saltzman DA, Jackson RJ, Smith SD. Fibrin sheath enhances central venous catheter infection. *Crit Care Med* 2002; 30:908–12.
218. Donlan RM, Costerton JW. Biofilms: survival mechanisms of clinically relevant microorganisms. *Clin Microbiol Rev* 2002; 15:167–93.
219. Hawser SP, Douglas LJ. Biofilm formation by *Candida* species on the surface of catheter materials in vitro. *Infect Immun* 1994; 62: 915–21.
220. Stillman RM, Soliman F, Garcia L, Sawyer PN. Etiology of catheter-associated sepsis. Correlation with thrombogenicity. *Arch Surg* 1977; 112:1497–9.
221. Raad II, Luna M, Khalil SA, Costerton JW, Lam C, Bodey GP. The relationship between the thrombotic and infectious complications of central venous catheters. *JAMA* 1994; 271:1014–6.
222. Herrmann M, Suchard SJ, Boxer LA, Waldvogel FA, Lew PD. Thrombospondin binds to *Staphylococcus aureus* and promotes staphylococcal adherence to surfaces. *Infect Immun* 1991; 59:279–88.
223. Shanks RM, Sargent JL, Martinez RM, Graber ML, O’Toole GA. Catheter lock solutions influence staphylococcal biofilm formation on abiotic surfaces. *Nephrol Dial Transplant* 2006; 21:2247–55.
224. Chatzinikolaou I, Zipf TF, Hanna H, et al. Minocycline-ethylenediaminetetraacetate lock solution for the prevention of implantable port infections in children with cancer. *Clin Infect Dis* 2003; 36:116–9.
225. McDevitt D, Francois P, Vaudaux P, Foster TJ. Identification of the ligand-binding domain of the surface-located fibrinogen receptor (clumping factor) of *Staphylococcus aureus*. *Mol Microbiol* 1995; 16:895–907.
226. Ni Eidhin D, Perkins S, Francois P, Vaudaux P, Hook M, Foster TJ. Clumping factor B (ClfB), a new surface-located fibrinogen-binding adhesin of *Staphylococcus aureus*. *Mol Microbiol* 1998; 30:245–57.
227. Mack D, Davies AP, Harris LG, Rohde H, Horstkotte MA, Knobloch JK. Microbial interactions in *Staphylococcus epidermidis* biofilms. *Anal Bioanal Chem* 2007; 387:399–408.
228. von Eiff C, Peters G, Heilmann C. Pathogenesis of infections due to coagulase-negative staphylococci. *Lancet Infect Dis* 2002; 2:677–85.
229. Zhu Y, Weiss EC, Otto M, Fey PD, Smeltzer MS, Somerville GA. *Staphylococcus aureus* metabolism in a biofilm: the influence of arginine on polysaccharide intercellular adhesin synthesis, biofilm formation, and pathogenesis. *Infect Immun* 2007; 75:4219–26.
230. Murga R, Miller JM, Donlan RM. Biofilm formation by gram-negative bacteria on central venous catheter connectors: effect of conditioning films in a laboratory model. *J Clin Microbiol* 2001; 39:2294–7.

231. Douglas LJ. *Candida* biofilms and their role in infection. *Trends Microbiol* 2003; 11:30–6.
232. Donlan RM. Biofilms: microbial life on surfaces. *Emerg Infect Dis* 2002; 8:881–90.
233. Dunne WM Jr., Burd EM. The effects of magnesium, calcium, EDTA, and pH on the in vitro adhesion of *Staphylococcus epidermidis* to plastic. *Microbiol Immunol* 1992; 36:1019–27.
234. Ozerdem Akpolat N, Elci S, Atmaca S, Akbayin H, Gul K. The effects of magnesium, calcium and EDTA on slime production by *Staphylococcus epidermidis* strains. *Folia Microbiol (Praha)* 2003; 48:649–53.
235. Banin E, Brady KM, Greenberg EP. Chelator-induced dispersal and killing of *Pseudomonas aeruginosa* cells in a biofilm. *Appl Environ Microbiol* 2006; 72:2064–9.
236. Donlan RM. Role of biofilms in antimicrobial resistance. *ASAIO J* 2000; 46:S47–52.
237. Farber BF, Kaplan MH, Clogston AG. *Staphylococcus epidermidis* extracted slime inhibits the antimicrobial action of glycopeptide antibiotics. *J Infect Dis* 1990; 161:37–40.
238. Branchini ML, Pfaller MA, Rhine-Chalberg J, Frempong T, Isenberg HD. Genotypic variation and slime production among blood and catheter isolates of *Candida parapsilosis*. *J Clin Microbiol* 1994; 32:452–6.
239. Sanders RA, Sheldon GF. Septic complications of total parenteral nutrition. A five year experience. *Am J Surg* 1976; 132:214–20.
240. Ryan JA Jr., Abel RM, Abbott WM, et al. Catheter complications in total parenteral nutrition. A prospective study of 200 consecutive patients. *N Engl J Med* 1974; 290:757–61.
241. Murphy LM, Lipman TO. Central venous catheter care in parenteral nutrition: a review. *JPEN J Parenter Enteral Nutr* 1987; 11: 190–201.
242. Armstrong CW, Mayhall CG, Miller KB, et al. Prospective study of catheter replacement and other risk factors for infection of hyperalimentation catheters. *J Infect Dis* 1986; 154:808–16.
243. Garland JS, Dunne WM Jr., Havens P, et al. Peripheral intravenous catheter complications in critically ill children: a prospective study. *Pediatrics* 1992; 89:1145–50.
244. Garland JS, Nelson DB, Cheah TE, Hennes HH, Johnson TM. Infectious complications during peripheral intravenous therapy with Teflon catheters: a prospective study. *Pediatr Infect Dis J* 1987; 6:918–21.
245. Breschan C, Platzer M, Jost R, Schaumberger F, Stettner H, Likar R. Comparison of catheter-related infection and tip colonization between internal jugular and subclavian central venous catheters in surgical neonates. *Anesthesiology* 2007; 107:946–53.
246. Deshpande KS, Hatem C, Ulrich HL, et al. The incidence of infectious complications of central venous catheters at the subclavian, internal jugular, and femoral sites in an intensive care unit population. *Crit Care Med* 2005; 33:13–20; discussion 234–5.
247. Durbec O, Viviand X, Potie F, Vialet R, Albanese J, Martin C. A prospective evaluation of the use of femoral venous catheters in critically ill adults. *Crit Care Med* 1997; 25:1986–9.
248. Venkataraman ST, Thompson AE, Orr RA. Femoral vascular catheterization in critically ill infants and children. *Clin Pediatr (Phila)* 1997; 36:311–9.

249. Sheridan RL, Weber JM. Mechanical and infectious complications of central venous cannulation in children: lessons learned from a 10-year experience placing more than 1000 catheters. *J Burn Care Res* 2006; 27:713–8.
250. Stenzel JP, Green TP, Fuhrman BP, Carlson PE, Marchessault RP. Percutaneous central venous catheterization in a pediatric intensive care unit: a survival analysis of complications. *Crit Care Med* 1989; 17:984–8.
251. Goldstein AM, Weber JM, Sheridan RL. Femoral venous access is safe in burned children: an analysis of 224 catheters. *J Pediatr* 1997; 130:442–6.
252. Ramos GE, Bolgiani AN, Patino O, et al. Catheter infection risk related to the distance between insertion site and burned area. *J Burn Care Rehabil* 2002; 23:266–71.
253. Sheth NK, Franson TR, Rose HD, Buckmire FL, Cooper JA, Sohnle PG. Colonization of bacteria on polyvinyl chloride and Teflon intravascular catheters in hospitalized patients. *J Clin Microbiol* 1983; 18:1061–3.
254. Maki DG, Ringer M. Evaluation of dressing regimens for prevention of infection with peripheral intravenous catheters. Gauze, a transparent polyurethane dressing, and an iodophor-transparent dressing. *JAMA* 1987; 258:2396–403.
255. Pittet D, Hugonnet S, Harbath S, et al. Effectiveness of a hospital-wide programme to improve compliance with hand hygiene. *Lancet* 2000; 356:1307–9.
256. Humar A, Ostromecki A, Direnfeld J, et al. Prospective randomized trial of 10% povidone-iodine versus 0.5% tincture of chlorhexidine as cutaneous antisepsis for prevention of central venous catheter infection. *Clin Infect Dis* 2000; 31:1001–7.
257. Chaiyakunapruk N, Veenstra DL, Lipsky BA, Saint S. Chlorhexidine compared with povidone-iodine solution for vascular catheter-site care: a meta-analysis. *Ann Intern Med* 2002; 136:792–801.
258. Chaiyakunapruk N, Veenstra DL, Lipsky BA, Sullivan SD, Saint S. Vascular catheter site care: the clinical and economic benefits of chlorhexidine gluconate compared with povidone iodine. *Clin Infect Dis* 2003; 37:764–71.
259. Parienti JJ, du Cheyron D, Ramakers M, et al. Alcoholic povidone-iodine to prevent central venous catheter colonization: a randomized unit-crossover study. *Crit Care Med* 2004; 32:708–13.
260. Hoffmann KK, Weber DJ, Samsa GP, Rutala WA. Transparent polyurethane film as an intravenous catheter dressing. A meta-analysis of the infection risks. *JAMA* 1992; 267:2072–6.
261. Gillies D, O’Riordan E, Carr D, O’Brien I, Frost J, Gunning R. Central venous catheter dressings: a systematic review. *J Adv Nurs* 2003; 44:623–32.
262. Ruschulte H, Franke M, Gastmeier P, et al. Prevention of central venous catheter related infections with chlorhexidine gluconate impregnated wound dressings: a randomized controlled trial. *Ann Hematol* 2009; 88:267–72.
263. Veenstra DL, Saint S, Saha S, Lumley T, Sullivan SD. Efficacy of antiseptic-impregnated central venous catheters in preventing catheter-related bloodstream infection: a meta-analysis. *JAMA* 1999; 281:261–7.

264. Maki DG, Stolz SM, Wheeler S, Mermel LA. Prevention of central venous catheter-related bloodstream infection by use of an antiseptic-impregnated catheter. A randomized, controlled trial. *Ann Intern Med* 1997; 127:257–66.
265. Bassetti S, Hu J, D'Agostino RB Jr., and Sherertz RJ. Prolonged antimicrobial activity of a catheter containing chlorhexidine-silver sulfadiazine extends protection against catheter infections in vivo. *Antimicrob Agents Chemother* 2001; 45:1535–8.
266. Oda T, Hamasaki J, Kanda N, Mikami K. Anaphylactic shock induced by an antiseptic-coated central venous [correction of nervous] catheter. *Anesthesiology* 1997; 87:1242–4.
267. Pittaway A, Ford S. Allergy to chlorhexidine-coated central venous catheters revisited. *Br J Anaesth* 2002; 88:304–5; author reply 305.
268. Stephens R, Mythen M, Kallis P, Davies DW, Egner W, Rickards A. Two episodes of life-threatening anaphylaxis in the same patient to a chlorhexidine-sulphadiazine-coated central venous catheter. *Br J Anaesth* 2001; 87:306–8.
269. Terazawa E, Shimonaka H, Nagase K, Masue T, Dohi S. Severe anaphylactic reaction due to a chlorhexidine-impregnated central venous catheter. *Anesthesiology* 1998; 89:1296–8.
270. Jee R, Nel L, Gnanakumaran G, Williams A, Eren E. Four cases of anaphylaxis to chlorhexidine impregnated central venous catheters: a case cluster or the tip of the iceberg? *Br J Anaesth* 2009; 103:614–5.
271. Veenstra DL, Saint S, Sullivan SD. Cost-effectiveness of antiseptic-impregnated central venous catheters for the prevention of catheter-related bloodstream infection. *JAMA* 1999; 282:554–60.
272. Tambe SM, Sampath L, Modak SM. In vitro evaluation of the risk of developing bacterial resistance to antiseptics and antibiotics used in medical devices. *J Antimicrob Chemother* 2001; 47:589–98.
273. Sampath LA, Tambe SM, Modak SM. In vitro and in vivo efficacy of catheters impregnated with antiseptics or antibiotics: evaluation of the risk of bacterial resistance to the antimicrobials in the catheters. *Infect Control Hosp Epidemiol* 2001; 22:640–6.
274. Marciante KD, Veenstra DL, Lipsky BA, Saint S. Which antimicrobial impregnated central venous catheter should we use? Modeling the costs and outcomes of antimicrobial catheter use. *Am J Infect Control* 2003; 31:1–8.
275. Shorr AF, Humphreys CW, Helman DL. New choices for central venous catheters: potential financial implications. *Chest* 2003; 124:275–84.
276. Hagau N, Studnicska D, Gavrus RL, Csipak G, Hagau R, Slavcovici AV. Central venous catheter colonization and catheter-related bloodstream infections in critically ill patients: a comparison between standard and silver-integrated catheters. *Eur J Anaesthesiol* 2009; 26:752–8.
277. Bong JJ, Kite P, Wilco MH, McMahon MJ. Prevention of catheter related bloodstream infection by silver iontophoretic central venous catheters: a randomised controlled trial. *J Clin Pathol* 2003; 56:731–5.
278. Corral L, Nolla-Salas M, Ibanez-Nolla J, et al. A prospective, randomized study in critically ill patients using the Oligon Vantex catheter. *J Hosp Infect* 2003; 55:212–9.
279. Ranucci M, Isgro G, Giomarelli PP, et al. Impact of oligon central venous catheters on catheter colonization and catheter-related bloodstream infection. *Crit Care Med* 2003; 31:52–9.

280. Raad II, Hachem RY, Abi-Said D, et al. A prospective crossover randomized trial of novobiocin and rifampin prophylaxis for the prevention of intravascular catheter infections in cancer patients treated with interleukin-2. *Cancer* 1998; 82:403–11.
281. McKee R, Dunsmuir R, Whitby M, Garden OJ. Does antibiotic prophylaxis at the time of catheter insertion reduce the incidence of catheter-related sepsis in intravenous nutrition? *J Hosp Infect* 1985; 6:419–25.
282. Sandoe JA, Kumar B, Stoddart B, et al. Effect of extended perioperative antibiotic prophylaxis on intravascular catheter colonization and infection in cardiothoracic surgery patients. *J Antimicrob Chemother* 2003; 52:877–9.
283. Inglis GDT, Jardine LA, Davies MW. Prophylactic antibiotics to reduce morbidity and mortality in neonates with umbilical artery catheters. *Cochrane Database of Systematic Reviews* 2007; Issue 4. Art. No.: CD004697. DOI: 10.1002/14651858.CD004697.pub3.
284. Craft AP, Finer N, Barrington KJ. Vancomycin for prophylaxis against sepsis in preterm neonates. *Cochrane Database of Systematic Reviews* 2000; Issue 1. Art. No.: CD001971. DOI: 10.1002/14651858. CD001971.
285. Norden CW. Application of antibiotic ointment to the site of venous catheterization—a controlled trial. *J Infect Dis* 1969; 120:611–5.
286. Zinner SH, Denny-Brown BC, Braun P, Burke JP, Toala P, Kass EH. Risk of infection with intravenous indwelling catheters: effect of application of antibiotic ointment. *J Infect Dis* 1969; 120:616–9.
287. von Eiff C, Becker K, Machka K, Stammer H, Peters G. Nasal carriage as a source of *Staphylococcus aureus* bacteremia. *N Engl J Med* 2001; 344:11–6.
288. Chow JW, Yu VL. *Staphylococcus aureus* nasal carriage in hemodialysis patients. Its role in infection and approaches to prophylaxis. *Arch Intern Med* 1989; 149:1258–62.
289. Yu VL, Goetz A, Wagener M, et al. *Staphylococcus aureus* nasal carriage and infection in patients on hemodialysis. Efficacy of antibiotic prophylaxis. *N Engl J Med* 1986; 315:91–6.
290. Casewell MW. The nose: an underestimated source of *Staphylococcus aureus* causing wound infection. *J Hosp Infect* 1998; 40:S3–11.
291. Hill RL, Fisher AP, Ware RJ, Wilson S, Casewell MW. Mupirocin for the reduction of colonization of internal jugular cannulae—a randomized controlled trial. *J Hosp Infect* 1990; 15:311–21.
292. Sesso R, Barbosa D, Leme IL, et al. *Staphylococcus aureus* prophylaxis in hemodialysis patients using central venous catheter: effect of mupirocin ointment. *J Am Soc Nephrol* 1998; 9:1085–92.
293. Boelaert JR, Van Landuyt HW, Godard CA, et al. Nasal mupirocin ointment decreases the incidence of *Staphylococcus aureus* bacteraemias in haemodialysis patients. *Nephrol Dial Transplant* 1993; 8:235–9.
294. Netto dos Santos KR, de Souza Fonseca L, Gontijo Filho PP. Emergence of high-level mupirocin resistance in methicillin-resistant *Staphylococcus aureus* isolated from Brazilian university hospitals. *Infect Control Hosp Epidemiol* 1996; 17:813–6.
295. Miller MA, Dascal A, Portnoy J, Mendelson J. Development of mupirocin resistance among methicillin-resistant *Staphylococcus aureus* after widespread use of nasal mupirocin ointment. *Infect Control Hosp Epidemiol* 1996; 17:811–3.

296. Lok CE, Stanley KE, Hux JE, Richardson R, Tobe SW, Conly J. Hemodialysis infection prevention with polysporin ointment. *J Am Soc Nephrol* 2003; 14:169–79.
297. Yahav D, Rozen-Zvi B, Gafter-Gvili A, Leibovici L, Gafter U, Paul M. Antimicrobial lock solutions for the prevention of infections associated with intravascular catheters in patients undergoing hemodialysis: systematic review and meta-analysis of randomized, controlled trials. *Clin Infect Dis* 2008; 47:83–93.
298. Labriola L, Crott R, Jadoul M. Preventing haemodialysis catheter-related bacteraemia with an antimicrobial lock solution: a metaanalysis of prospective randomized trials. *Nephrol Dial Transplant* 2008; 23:1666–72.
299. Jaffer Y, Selby NM, Taal MW, Fluck RJ, McIntyre CW. A meta-analysis of hemodialysis catheter locking solutions in the prevention of catheter-related infection. *Am J Kidney Dis* 2008; 51:233–41.
300. Safdar N, Maki DG. Use of vancomycin-containing lock or flush solutions for prevention of bloodstream infection associated with central venous access devices: a meta-analysis of prospective, randomized trials. *Clin Infect Dis* 2006; 43:474–84.
301. Sanders J, Pithie A, Ganly P, et al. A prospective double-blind randomized trial comparing intraluminal ethanol with heparinized saline for the prevention of catheter-associated bloodstream infection in immunosuppressed haematology patients. *J Antimicrob Chemother* 2008; 62:809–15.
302. Schinabeck MK, Ghannoum MA. Biofilm-related indwelling medical device infections. In: Pace JL, Rupp ME, Finch RG, eds. *Biofilms, infection, and antimicrobial therapy*. Boca Raton: Taylor and Francis, 2006: 39–50.
303. Gristina AG. Biomaterial-centered infection: microbial adhesion versus tissue integration. *Science* 1987; 237:1588–95.
304. Timsit JF, Farkas JC, Boyer JM, et al. Central vein catheter-related thrombosis in intensive care patients: incidence, risks factors, and relationship with catheter-related sepsis. *Chest* 1998; 114:207–13.
305. Eastman ME, Khorsand M, Maki DG, et al. Central venous device-related infection and thrombosis in patients treated with moderate dose continuous-infusion interleukin-2. *Cancer* 2001; 91:806–14.
306. Abdelkefi A, Torjman L, Ladeb S, et al. Randomized trial of prevention of catheter-related bloodstream infection by continuous infusion of low-dose unfractionated heparin in patients with hematologic and oncologic disease. *J Clin Oncol* 2005; 23:7864–70.
307. Mermel LA, Stolz SM, Maki DG. Surface antimicrobial activity of heparin-bonded and antiseptic-impregnated vascular catheters. *J Infect Dis* 1993; 167:920–4.
308. Pierce CM, Wade A, Mok Q. Heparin-bonded central venous lines reduce thrombotic and infective complications in critically ill children. *Intensive Care Med* 2000; 26:967–72.
309. Appelgren P, Ransjo U, Bindslev L, Espersen F, Larm O. Surface heparinization of central venous catheters reduces microbial colonization in vitro and in vivo: results from a prospective, randomized trial. *Crit Care Med* 1996; 24:1482–9.

310. Abdelkefi A, Achour W, Ben Othman T, et al. Use of heparin-coated central venous lines to prevent catheter-related bloodstream infection. *J Support Oncol* 2007; 5:273–8.
311. Carrasco MN, Bueno A, de las Cuevas C, et al. Evaluation of a triple-lumen central venous heparin-coated catheter versus a catheter coated with chlorhexidine and silver sulfadiazine in critically ill patients. *Intensive Care Med* 2004; 30:633–8.
312. Levy JH, Hursting MJ. Heparin-induced thrombocytopenia, a prothrombotic disease. *Hematol Oncol Clin North Am* 2007; 21:65–88.
313. Weijmer MC, Debets-Ossenkopp YJ, Van De Vondervoort FJ, ter Wee PM. Superior antimicrobial activity of trisodium citrate over heparin for catheter locking. *Nephrol Dial Transplant* 2002; 17:2189–95.
314. Boraks P, Seale J, Price J, et al. Prevention of central venous catheter associated thrombosis using minidose warfarin in patients with haematological malignancies. *Br J Haematol* 1998; 101:483–6.
315. Bern MM, Lokich JJ, Wallach SR, et al. Very low doses of warfarin can prevent thrombosis in central venous catheters. A randomized prospective trial. *Ann Intern Med* 1990; 112:423–8.
316. Akl EA, Karmath G, Yosuíco VED, Kim SY, Barba M, Sperati F, Cook D, Schunemann H. Anticoagulation for thrombosis prophylaxis in cancer patients with central venous catheters. *Cochrane Database of Systematic Reviews* 2007; Issue 3. Art. No.: CD006468. DOI: 10.1002/14651858.CD006468.pub2.
317. Akl EA, Muti P, Schunemann HJ. Anticoagulation in patients with cancer: an overview of reviews. *Pol Arch Med Wewn* 2008; 118:183–93.
318. Klerk CP, Smorenburg SM, Buller HR. Thrombosis prophylaxis in patient populations with a central venous catheter: a systematic review. *Arch Intern Med* 2003; 163:1913–21.
319. Heaton DC, Han DY, Inder A. Minidose (1 mg) warfarin as prophylaxis for central vein catheter thrombosis. *Intern Med J* 2002; 32:84–8.
320. Masci G, Magagnoli M, Zucali PA, et al. Minidose warfarin prophylaxis for catheter-associated thrombosis in cancer patients: can it be safely associated with fluorouracil-based chemotherapy? *J Clin Oncol* 2003; 21:736–9.
321. Kuter DJ. Thrombotic complications of central venous catheters in cancer patients. *Oncologist* 2004; 9:207–16.
322. Fontaine PJ. Performance of a new softening expanding midline catheter in home intravenous therapy patients. *J Intraven Nurs* 1991; 14:91–9.
323. Harwood IR, Greene LM, Kozakowski-Koch JA, Rasor JS. New peripherally inserted midline catheter: a better alternative for intravenous antibiotic therapy in patients with cystic fibrosis. *Pediatr Pulmonol* 1992; 12:233–9.
324. Mermel LA, Parenteau S, Tow SM. The risk of midline catheterization in hospitalized patients. A prospective study. *Ann Intern Med* 1995; 123:841–4.
325. Uldall PR, Merchant N, Woods F, Yarworski U, Vas S. Changing subclavian haemodialysis cannulas to reduce infection. *Lancet* 1981; 1:1373.

326. Cook D, Randolph A, Kernerman P, et al. Central venous catheter replacement strategies: a systematic review of the literature. *Crit Care Med* 1997; 25:1417–24.
327. Cobb DK, High KP, Sawyer RG, et al. A controlled trial of scheduled replacement of central venous and pulmonary-artery catheters. *N Engl J Med* 1992; 327:1062–8.
328. Beathard GA. Management of bacteremia associated with tunneled-cuffed hemodialysis catheters. *J Am Soc Nephrol* 1999; 10:1045–9.
329. Duszak R Jr., Haskal ZJ, Thomas-Hawkins C, et al. Replacement of failing tunneled hemodialysis catheters through pre-existing subcutaneous tunnels: a comparison of catheter function and infection rates for de novo placements and over-the-wire exchanges. *J Vasc Interv Radiol* 1998; 9:321–7.
330. Robinson D, Suhocki P, Schwab SJ. Treatment of infected tunneled venous access hemodialysis catheters with guidewire exchange. *Kidney Int* 1998; 53:1792–4.
331. Saad TF. Bacteremia associated with tunneled, cuffed hemodialysis catheters. *Am J Kidney Dis* 1999; 34:1114–24.
332. Ainsworth S, Clerihew L, McGuire W. Percutaneous central venous catheters versus peripheral cannulae for delivery of parenteral nutrition in neonates. *Cochrane Database of Systematic Reviews* 2007; Issue 3. Art. No.: CD004219. DOI: 10.1002/14651858.CD004219. pub3.
333. Shah PS, Kalyn A, Satodia P, et al. A randomized, controlled trial of heparin versus placebo infusion to prolong the usability of peripherally placed percutaneous central venous catheters (PCVCs) in neonates: the HIP (Heparin Infusion for PCVC) study. *Pediatrics* 2007; 119:e284–91.
334. Jaar BG, Hermann JA, Furth SL, Briggs W, Powe NR. Septicemia in diabetic hemodialysis patients: comparison of incidence, risk factors, and mortality with nondiabetic hemodialysis patients. *Am J Kidney Dis* 2000; 35:282–92.
335. Powe NR, Jaar B, Furth SL, Hermann J, Briggs W. Septicemia in dialysis patients: incidence, risk factors, and prognosis. *Kidney Int* 1999; 55:1081–90.
336. Hoen B, Paul-Dauphin A, Hestin D, Kessler M. EPIBACDIAL: a multicenter prospective study of risk factors for bacteremia in chronic hemodialysis patients. *J Am Soc Nephrol* 1998; 9:869–76.
337. Blot F, Chachaty E, Raynard B, Antoun S, Bourgain JL, Nitenberg G. Mechanisms and risk factors for infection of pulmonary artery catheters and introducer sheaths in cancer patients admitted to an intensive care unit. *J Hosp Infect* 2001; 48:289–97.
338. Kac G, Durain E, Amrein C, Herisson E, Fiemeyer A, Buu-Hoi A. Colonization and infection of pulmonary artery catheter in cardiac surgery patients: epidemiology and multivariate analysis of risk factors. *Crit Care Med* 2001; 29:971–5.
339. Chen YY, Yen DH, Yang YG, Liu CY, Wang FD, Chou P. Comparison between replacement at 4 days and 7 days of the infection rate for pulmonary artery catheters in an intensive care unit. *Crit Care Med* 2003; 31:1353–8.
340. Balagtas RC, Bell CE, Edwards LD, Levin S. Risk of local and systemic infections associated with umbilical vein catheterization: a prospective study in 86 newborn patients. *Pediatrics* 1971; 48:359–67.

341. Butler-O'Hara M, Buzzard CJ, Reubens L, McDermott MP, DiGrazio W, D'Angio CT. A randomized trial comparing long-term and short-term use of umbilical venous catheters in premature infants with birth weights of less than 1251 grams. *Pediatrics* 2006; 118:e25–35.
342. Scheer B, Perel A, Pfeiffer UJ. Clinical review: complications and risk factors of peripheral arterial catheters used for haemodynamic monitoring in anaesthesia and intensive care medicine. *Crit Care* 2002; 6:199–204.
343. Lorente L, Santacreu R, Martin MM, Jimenez A, Mora ML. Arterial catheter-related infection of 2,949 catheters. *Crit Care* 2006; 10:R83.
344. Furfaro S, Gauthier M, Lacroix J, Nadeau D, Lafleur L, Mathews S. Arterial catheter-related infections in children. A 1-year cohort analysis. *Am J Dis Child* 1991; 145:1037–43.
345. Rickard CM, Lipman J, Courtney M, Siversen R, Daley P. Routine changing of intravenous administration sets does not reduce colonization or infection in central venous catheters. *Infect Control Hosp Epidemiol* 2004; 25:650–5.
346. Hanna HA, Raad I. Blood products: a significant risk factor for long-term catheter-related bloodstream infections in cancer patients. *Infect Control Hosp Epidemiol* 2001; 22:165–6.
347. Saiman L, Ludington E, Dawson JD, et al. Risk factors for *Candida* species colonization of neonatal intensive care unit patients. *Pediatr Infect Dis J* 2001; 20:1119–24.
348. Avila-Figueroa C, Goldmann DA, Richardson DK, Gray JE, Ferrari A, Freeman J. Intravenous lipid emulsions are the major determinant of coagulase-negative staphylococcal bacteremia in very low birth weight newborns. *Pediatr Infect Dis J* 1998; 17:10–7.
349. Crocker KS, Noga R, Filibeck DJ, Krey SH, Markovic M, Steffee WP. Microbial growth comparisons of five commercial parenteral lipid emulsions. *J Parenter Enteral Nutr* 1984; 8:391–5.
350. Jarvis WR, Highsmith AK. Bacterial growth and endotoxin production in lipid emulsion. *J Clin Microbiol* 1984; 19:17–20.
351. Karamanoglu A, Yumuk PF, Gumus M, et al. Port needles: do they need to be removed as frequently in infusional chemotherapy? *J Infus Nurs* 2003; 26:239–42.
352. Niel-Weise BS, Daha TJ, van den Broek PJ. Is there evidence for recommending needleless closed catheter access systems in guidelines? A systematic review of randomized controlled trials. *J Hosp Infect* 2006; 62:406–13.
353. Inoue Y, Nezu R, Matsuda H, et al. Prevention of catheter-related sepsis during parenteral nutrition: effect of a new connection device. *J Parenter Enteral Nutr* 1992; 16:581–5.
354. Yebenes JC, Vidaur L, Serra-Prat M, et al. Prevention of catheter-related bloodstream infection in critically ill patients using a disinfectable, needle-free connector: a randomized controlled trial. *Am J Infect Control* 2004; 32:291–5.
355. Casey AL, Worthington T, Lambert PA, Quinn D, Faroqui MH, Elliott TS. A randomized, prospective clinical trial to assess the potential infection risk associated with the PosiFlow needleless connector. *J Hosp Infect* 2003; 54:288–93.

Guidelines for the Prevention of Intravascular Catheter-Related Infections

356. Esteve F, Pujol M, Limon E, et al. Bloodstream infection related to catheter connections: a prospective trial of two connection systems. *J Hosp Infect* 2007; 67:30–4.
357. Yebenes JC, Delgado M, Sauca G, et al. Efficacy of three different valve systems of needle-free closed connectors in avoiding access of microorganisms to endovascular catheters after incorrect handling. *Crit Care Med* 2008; 36:2558–61.
358. Menyhay SZ, Maki DG. Preventing central venous catheter-associated bloodstream infections: development of an antiseptic barrier cap for needleless connectors. *Am J Infect Control* 2008; 36:(Suppl 174):e1–5.
359. Jarvis WR, Murphy C, Hall KK, et al. Health care-associated bloodstream infections associated with negative-or positive-pressure or displacement mechanical valve needleless connectors. *Clin Infect Dis* 2009; 49:1821–7.
360. Menyhay SZ, Maki DG. Disinfection of needleless catheter connectors and access ports with alcohol may not prevent microbial entry: the promise of a novel antiseptic-barrier cap. *Infect Control Hosp Epidemiol* 2006; 27:23–7.
361. Safdar N, Maki DG. Lost in translation. *Infect Control Hosp Epidemiol* 2006; 27:3–7.
362. Warren DK, Yokoe DS, Climo MW, et al. Preventing catheter-associated bloodstream infections: a survey of policies for insertion and care of central venous catheters from hospitals in the prevention epicenter program. *Infect Control Hosp Epidemiol* 2006; 27:8–13.
363. O’Grady NP, Alexander M, Dellinger EP, et al. Guidelines for the prevention of intravascular catheter-related infections. Centers for Disease Control and Prevention. *MMWR Recomm Rep* 2002;51:1–29.
364. Krein SL, Hofer TP, Kowalski CP, et al. Use of central venous catheter-related bloodstream infection prevention practices by US hospitals. *Mayo Clin Proc* 2007; 82:672–8.
365. Lobo RD, Levin AS, Gomes LM, et al. Impact of an educational program and policy changes on decreasing catheter-associated bloodstream infections in a medical intensive care unit in Brazil. *Am J Infect Control* 2005; 33:83–7.
366. Marschall J, Leone C, Jones M, Nihill D, Fraser VJ, Warren DK. Catheter-associated bloodstream infections in general medical patients outside the intensive care unit: a surveillance study. *Infect Control Hosp Epidemiol* 2007; 28:905–9.
367. Rosenthal VD, McCormick RD, Guzman S, Villamayor C, Orellano PW. Effect of education and performance feedback on handwashing: the benefit of administrative support in Argentinean hospitals. *Am J Infect Control* 2003; 31:85–92.
368. Gastmeier P, Geffers C. Prevention of catheter-related bloodstream infections: analysis of studies published between 2002 and 2005. *J Hosp Infect* 2006; 64:326–35.
369. Pronovost PJ, Goeschel CA, Colantuoni E, et al. Sustaining reductions in catheter related bloodstream infections in Michigan intensive care units: observational study. *BMJ* 2010; 340:c309.
370. Shapey IM, Foster MA, Whitehouse T, Jumaa P, Bion JF. Central venous catheter-related bloodstream infections: improving post-insertion catheter care. *J Hosp Infect* 2009; 71:117–22.



MMWRTM

Morbidity and Mortality Weekly Report

Recommendations and Reports

October 25, 2002 / Vol. 51 / No. RR-16

Guideline for Hand Hygiene in Health-Care Settings

**Recommendations of the Healthcare Infection Control Practices
Advisory Committee and the HICPAC/SHEA/APIC/IDSA
Hand Hygiene Task Force**

INSIDE: Continuing Education Examination

CENTERS FOR DISEASE CONTROL AND PREVENTION

SAFER • HEALTHIER • PEOPLETM

The *MMWR* series of publications is published by the Epidemiology Program Office, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30333.

SUGGESTED CITATION

Centers for Disease Control and Prevention. Guideline for Hand Hygiene in Health-Care Settings: Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force. *MMWR* 2002;51(No. RR-16):[inclusive page numbers].

Centers for Disease Control and Prevention

Julie L. Gerberding, M.D., M.P.H.

Director

David W. Fleming, M.D.

Deputy Director for Science and Public Health

Dixie E. Snider, Jr., M.D., M.P.H.

Associate Director for Science

Epidemiology Program Office

Stephen B. Thacker, M.D., M.Sc.

Director

Office of Scientific and Health Communications

John W. Ward, M.D.

Director

Editor, MMWR Series

Suzanne M. Hewitt, M.P.A.

Managing Editor

Rachel J. Wilson

Douglas W. Weatherwax

Project Editors

Malbea A. Heilman

Beverly J. Holland

Visual Information Specialists

Quang M. Doan

Erica R. Shaver

Information Technology Specialists

CONTENTS

Part I. Review of the Scientific Data Regarding

Hand Hygiene	1
Historical Perspective	1
Normal Bacterial Skin Flora	2
Physiology of Normal Skin	2
Definition of Terms	3
Evidence of Transmission of Pathogens on Hands	4
Models of Hand Transmission	5
Relation of Hand Hygiene and Acquisition of Health-Care–Associated Pathogens	5
Methods Used To Evaluate the Efficacy of Hand-Hygiene Products	6
Review of Preparations Used for Hand Hygiene	8
Activity of Antiseptic Agents Against Spore-Forming Bacteria	16
Reduced Susceptibility of Bacteria to Antiseptics	17
Surgical Hand Antisepsis	17
Relative Efficacy of Plain Soap, Antiseptic Soap/Detergent, and Alcohols	18
Irritant Contact Dermatitis Resulting from Hand-Hygiene Measures	18
Proposed Methods for Reducing Adverse Effects of Agents	19
Factors To Consider When Selecting Hand-Hygiene Products	20
Hand-Hygiene Practices Among HCWs	21
Lessons Learned from Behavioral Theories	25
Methods Used To Promote Improved Hand Hygiene	26
Efficacy of Promotion and Impact of Improved Hand Hygiene	27
Other Policies Related to Hand Hygiene	29
Hand-Hygiene Research Agenda	30
Web-Based Hand-Hygiene Resources	30
Part II. Recommendations	31
Categories	31
Recommendations	32
Part III. Performance Indicators	34
References	34
Appendix	45
Continuing Education Activity	CE-1

Guideline for Hand Hygiene in Health-Care Settings

Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force

Prepared by
John M. Boyce, M.D.¹
Didier Pittet, M.D.²
¹Hospital of Saint Raphael
New Haven, Connecticut
²University of Geneva
Geneva, Switzerland

Summary

The Guideline for Hand Hygiene in Health-Care Settings provides health-care workers (HCWs) with a review of data regarding handwashing and hand antisepsis in health-care settings. In addition, it provides specific recommendations to promote improved hand-hygiene practices and reduce transmission of pathogenic microorganisms to patients and personnel in health-care settings. This report reviews studies published since the 1985 CDC guideline (Garner JS, Favero MS. CDC guideline for handwashing and hospital environmental control, 1985. Infect Control 1986;7:231–43) and the 1995 APIC guideline (Larson EL, APIC Guidelines Committee. APIC guideline for handwashing and hand antisepsis in health care settings. Am J Infect Control 1995;23:251–69) were issued and provides an in-depth review of hand-hygiene practices of HCWs, levels of adherence of personnel to recommended handwashing practices, and factors adversely affecting adherence. New studies of the in vivo efficacy of alcohol-based hand rubs and the low incidence of dermatitis associated with their use are reviewed. Recent studies demonstrating the value of multidisciplinary hand-hygiene promotion programs and the potential role of alcohol-based hand rubs in improving hand-hygiene practices are summarized. Recommendations concerning related issues (e.g., the use of surgical hand antiseptics, hand lotions or creams, and wearing of artificial fingernails) are also included.

Part I. Review of the Scientific Data Regarding Hand Hygiene

Historical Perspective

For generations, handwashing with soap and water has been considered a measure of personal hygiene (1). The concept of cleansing hands with an antiseptic agent probably emerged in the early 19th century. As early as 1822, a French pharmacist demonstrated that solutions containing chlorides of lime or soda could eradicate the foul odors associated with human corpses and that such solutions could be used as disinfectants and antiseptics (2). In a paper published in 1825, this pharmacist stated that physicians and other persons attending patients with contagious diseases would benefit from moistening their hands with a liquid chloride solution (2).

In 1846, Ignaz Semmelweis observed that women whose babies were delivered by students and physicians in the First Clinic at the General Hospital of Vienna consistently had a

higher mortality rate than those whose babies were delivered by midwives in the Second Clinic (3). He noted that physicians who went directly from the autopsy suite to the obstetrics ward had a disagreeable odor on their hands despite washing their hands with soap and water upon entering the obstetrics clinic. He postulated that the puerperal fever that affected so many parturient women was caused by “cadaverous particles” transmitted from the autopsy suite to the obstetrics ward via the hands of students and physicians. Perhaps because of the known deodorizing effect of chlorine compounds, as of May 1847, he insisted that students and physicians clean their hands with a chlorine solution between each patient in the clinic. The maternal mortality rate in the First Clinic subsequently dropped dramatically and remained low for years. This intervention by Semmelweis represents the first evidence indicating that cleansing heavily contaminated hands with an antiseptic agent between patient contacts may reduce health-care–associated transmission of contagious diseases more effectively than handwashing with plain soap and water.

In 1843, Oliver Wendell Holmes concluded independently that puerperal fever was spread by the hands of health personnel (1). Although he described measures that could be taken to limit its spread, his recommendations had little impact on

The material in this report originated in the National Center for Infectious Diseases, James M. Hughes, M.D., Director; and the Division of Healthcare Quality Promotion, Steve Solomon, M.D., Acting Director.

obstetric practices at the time. However, as a result of the seminal studies by Semmelweis and Holmes, handwashing gradually became accepted as one of the most important measures for preventing transmission of pathogens in health-care facilities.

In 1961, the U. S. Public Health Service produced a training film that demonstrated handwashing techniques recommended for use by health-care workers (HCWs) (4). At the time, recommendations directed that personnel wash their hands with soap and water for 1–2 minutes before and after patient contact. Rinsing hands with an antiseptic agent was believed to be less effective than handwashing and was recommended only in emergencies or in areas where sinks were unavailable.

In 1975 and 1985, formal written guidelines on handwashing practices in hospitals were published by CDC (5,6). These guidelines recommended handwashing with non-antimicrobial soap between the majority of patient contacts and washing with antimicrobial soap before and after performing invasive procedures or caring for patients at high risk. Use of waterless antiseptic agents (e.g., alcohol-based solutions) was recommended only in situations where sinks were not available.

In 1988 and 1995, guidelines for handwashing and hand antisepsis were published by the Association for Professionals in Infection Control (APIC) (7,8). Recommended indications for handwashing were similar to those listed in the CDC guidelines. The 1995 APIC guideline included more detailed discussion of alcohol-based hand rubs and supported their use in more clinical settings than had been recommended in earlier guidelines. In 1995 and 1996, the Healthcare Infection Control Practices Advisory Committee (HICPAC) recommended that either antimicrobial soap or a waterless antiseptic agent be used for cleaning hands upon leaving the rooms of patients with multidrug-resistant pathogens (e.g., vancomycin-resistant enterococci [VRE] and methicillin-resistant *Staphylococcus aureus* [MRSA]) (9,10). These guidelines also provided recommendations for handwashing and hand antisepsis in other clinical settings, including routine patient care. Although the APIC and HICPAC guidelines have been adopted by the majority of hospitals, adherence of HCWs to recommended handwashing practices has remained low (11,12).

Recent developments in the field have stimulated a review of the scientific data regarding hand hygiene and the development of new guidelines designed to improve hand-hygiene practices in health-care facilities. This literature review and accompanying recommendations have been prepared by a Hand Hygiene Task Force, comprising representatives from HICPAC, the Society for Healthcare Epidemiology of America (SHEA), APIC, and the Infectious Diseases Society of America (IDSA).

Normal Bacterial Skin Flora

To understand the objectives of different approaches to hand cleansing, a knowledge of normal bacterial skin flora is essential. Normal human skin is colonized with bacteria; different areas of the body have varied total aerobic bacterial counts (e.g., 1×10^6 colony forming units (CFUs)/cm² on the scalp, 5×10^5 CFUs/cm² in the axilla, 4×10^4 CFUs/cm² on the abdomen, and 1×10^4 CFUs/cm² on the forearm) (13). Total bacterial counts on the hands of medical personnel have ranged from 3.9×10^4 to 4.6×10^6 (14–17). In 1938, bacteria recovered from the hands were divided into two categories: transient and resident (14). Transient flora, which colonize the superficial layers of the skin, are more amenable to removal by routine handwashing. They are often acquired by HCWs during direct contact with patients or contact with contaminated environmental surfaces within close proximity of the patient. Transient flora are the organisms most frequently associated with health-care-associated infections. Resident flora, which are attached to deeper layers of the skin, are more resistant to removal. In addition, resident flora (e.g., coagulase-negative staphylococci and diphtheroids) are less likely to be associated with such infections. The hands of HCWs may become persistently colonized with pathogenic flora (e.g., *S. aureus*), gram-negative bacilli, or yeast. Investigators have documented that, although the number of transient and resident flora varies considerably from person to person, it is often relatively constant for any specific person (14,18).

Physiology of Normal Skin

The primary function of the skin is to reduce water loss, provide protection against abrasive action and microorganisms, and act as a permeability barrier to the environment. The basic structure of skin includes, from outer- to innermost layer, the superficial region (i.e., the stratum corneum or horny layer, which is 10- to 20- μ m thick), the viable epidermis (50- to 100- μ m thick), the dermis (1- to 2-mm thick), and the hypodermis (1- to 2-mm thick). The barrier to percutaneous absorption lies within the stratum corneum, the thinnest and smallest compartment of the skin. The stratum corneum contains the corneocytes (or horny cells), which are flat, polyhedral-shaped nonnucleated cells, remnants of the terminally differentiated keratinocytes located in the viable epidermis. Corneocytes are composed primarily of insoluble bundled keratins surrounded by a cell envelope stabilized by cross-linked proteins and covalently bound lipid. Interconnecting the corneocytes of the stratum corneum are polar structures (e.g., corneodesmosomes), which contribute to stratum corneum cohesion.

The intercellular region of the stratum corneum is composed of lipid primarily generated from the exocytosis of lamellar bodies during the terminal differentiation of the keratinocytes. The intercellular lipid is required for a competent skin barrier and forms the only continuous domain. Directly under the stratum corneum is a stratified epidermis, which is composed primarily of 10–20 layers of keratinizing epithelial cells that are responsible for the synthesis of the stratum corneum. This layer also contains melanocytes involved in skin pigmentation; Langerhans cells, which are important for antigen presentation and immune responses; and Merkel cells, whose precise role in sensory reception has yet to be fully delineated. As keratinocytes undergo terminal differentiation, they begin to flatten out and assume the dimensions characteristic of the corneocytes (i.e., their diameter changes from 10–12 μm to 20–30 μm , and their volume increases by 10- to 20-fold). The viable epidermis does not contain a vascular network, and the keratinocytes obtain their nutrients from below by passive diffusion through the interstitial fluid.

The skin is a dynamic structure. Barrier function does not simply arise from the dying, degeneration, and compaction of the underlying epidermis. Rather, the processes of cornification and desquamation are intimately linked; synthesis of the stratum corneum occurs at the same rate as loss. Substantial evidence now confirms that the formation of the skin barrier is under homeostatic control, which is illustrated by the epidermal response to barrier perturbation by skin stripping or solvent extraction. Circumstantial evidence indicates that the rate of keratinocyte proliferation directly influences the integrity of the skin barrier. A general increase in the rate of proliferation results in a decrease in the time available for 1) uptake of nutrients (e.g., essential fatty acids), 2) protein and lipid synthesis, and 3) processing of the precursor molecules required for skin-barrier function. Whether chronic but quantitatively smaller increases in rate of epidermal proliferation also lead to changes in skin-barrier function remains unclear. Thus, the extent to which the decreased barrier function caused by irritants is caused by an increased epidermal proliferation also is unknown.

The current understanding of the formation of the stratum corneum has come from studies of the epidermal responses to perturbation of the skin barrier. Experimental manipulations that disrupt the skin barrier include 1) extraction of skin lipids with apolar solvents, 2) physical stripping of the stratum corneum using adhesive tape, and 3) chemically induced irritation. All of these experimental manipulations lead to a decreased skin barrier as determined by transepidermal water loss (TEWL). The most studied experimental system is the treatment of mouse skin with acetone. This experiment

results in a marked and immediate increase in TEWL, and therefore a decrease in skin-barrier function. Acetone treatment selectively removes glycerolipids and sterols from the skin, which indicates that these lipids are necessary, though perhaps not sufficient in themselves, for barrier function. Detergents act like acetone on the intercellular lipid domain. The return to normal barrier function is biphasic: 50%–60% of barrier recovery typically occurs within 6 hours, but complete normalization of barrier function requires 5–6 days.

Definition of Terms

Alcohol-based hand rub. An alcohol-containing preparation designed for application to the hands for reducing the number of viable microorganisms on the hands. In the United States, such preparations usually contain 60%–95% ethanol or isopropanol.

Antimicrobial soap. Soap (i.e., detergent) containing an antiseptic agent.

Antiseptic agent. Antimicrobial substances that are applied to the skin to reduce the number of microbial flora. Examples include alcohols, chlorhexidine, chlorine, hexachlorophene, iodine, chloroxylenol (PCMX), quaternary ammonium compounds, and triclosan.

Antiseptic handwash. Washing hands with water and soap or other detergents containing an antiseptic agent.

Antiseptic hand rub. Applying an antiseptic hand-rub product to all surfaces of the hands to reduce the number of microorganisms present.

Cumulative effect. A progressive decrease in the numbers of microorganisms recovered after repeated applications of a test material.

Decontaminate hands. To Reduce bacterial counts on hands by performing antiseptic hand rub or antiseptic handwash.

Detergent. Detergents (i.e., surfactants) are compounds that possess a cleaning action. They are composed of both hydrophilic and lipophilic parts and can be divided into four groups: anionic, cationic, amphoteric, and nonionic detergents. Although products used for handwashing or antiseptic handwash in health-care settings represent various types of detergents, the term “soap” is used to refer to such detergents in this guideline.

Hand antiseptics. Refers to either antiseptic handwash or antiseptic hand rub.

Hand hygiene. A general term that applies to either handwashing, antiseptic handwash, antiseptic hand rub, or surgical hand antiseptics.

Handwashing. Washing hands with plain (i.e., non-antimicrobial) soap and water.

Persistent activity. Persistent activity is defined as the prolonged or extended antimicrobial activity that prevents or inhibits the proliferation or survival of microorganisms after application of the product. This activity may be demonstrated by sampling a site several minutes or hours after application and demonstrating bacterial antimicrobial effectiveness when compared with a baseline level. This property also has been referred to as “residual activity.” Both substantive and nonsubstantive active ingredients can show a persistent effect if they substantially lower the number of bacteria during the wash period.

Plain soap. Plain soap refers to detergents that do not contain antimicrobial agents or contain low concentrations of antimicrobial agents that are effective solely as preservatives.

Substantivity. Substantivity is an attribute of certain active ingredients that adhere to the stratum corneum (i.e., remain on the skin after rinsing or drying) to provide an inhibitory effect on the growth of bacteria remaining on the skin.

Surgical hand antisepsis. Antiseptic handwash or antiseptic hand rub performed preoperatively by surgical personnel to eliminate transient and reduce resident hand flora. Antiseptic detergent preparations often have persistent antimicrobial activity.

Visibly soiled hands. Hands showing visible dirt or visibly contaminated with proteinaceous material, blood, or other body fluids (e.g., fecal material or urine).

Waterless antiseptic agent. An antiseptic agent that does not require use of exogenous water. After applying such an agent, the hands are rubbed together until the agent has dried.

Food and Drug Administration (FDA) product categories. The 1994 FDA Tentative Final Monograph for Health-Care Antiseptic Drug Products divided products into three categories and defined them as follows (19):

- **Patient preoperative skin preparation.** A fast-acting, broad-spectrum, and persistent antiseptic-containing preparation that substantially reduces the number of microorganisms on intact skin.
- **Antiseptic handwash or HCW handwash.** An antiseptic-containing preparation designed for frequent use; it reduces the number of microorganisms on intact skin to an initial baseline level after adequate washing, rinsing, and drying; it is broad-spectrum, fast-acting, and if possible, persistent.
- **Surgical hand scrub.** An antiseptic-containing preparation that substantially reduces the number of microorganisms on intact skin; it is broad-spectrum, fast-acting, and persistent.

Evidence of Transmission of Pathogens on Hands

Transmission of health-care-associated pathogens from one patient to another via the hands of HCWs requires the following sequence of events:

- Organisms present on the patient’s skin, or that have been shed onto inanimate objects in close proximity to the patient, must be transferred to the hands of HCWs.
- These organisms must then be capable of surviving for at least several minutes on the hands of personnel.
- Next, handwashing or hand antisepsis by the worker must be inadequate or omitted entirely, or the agent used for hand hygiene must be inappropriate.
- Finally, the contaminated hands of the caregiver must come in direct contact with another patient, or with an inanimate object that will come into direct contact with the patient.

Health-care-associated pathogens can be recovered not only from infected or draining wounds, but also from frequently colonized areas of normal, intact patient skin (20–31). The perineal or inguinal areas are usually most heavily colonized, but the axillae, trunk, and upper extremities (including the hands) also are frequently colonized (23,25,26,28,30–32). The number of organisms (e.g., *S. aureus*, *Proteus mirabilis*, *Klebsiella* spp., and *Acinetobacter* spp.) present on intact areas of the skin of certain patients can vary from 100 to $10^6/\text{cm}^2$ (25,29,31,33). Persons with diabetes, patients undergoing dialysis for chronic renal failure, and those with chronic dermatitis are likely to have areas of intact skin that are colonized with *S. aureus* (34–41). Because approximately 10^6 skin squames containing viable microorganisms are shed daily from normal skin (42), patient gowns, bed linen, bedside furniture, and other objects in the patient’s immediate environment can easily become contaminated with patient flora (30,43–46). Such contamination is particularly likely to be caused by staphylococci or enterococci, which are resistant to desiccation.

Data are limited regarding the types of patient-care activities that result in transmission of patient flora to the hands of personnel (26,45–51). In the past, attempts have been made to stratify patient-care activities into those most likely to cause hand contamination (52), but such stratification schemes were never validated by quantifying the level of bacterial contamination that occurred. Nurses can contaminate their hands with 100–1,000 CFUs of *Klebsiella* spp. during “clean” activities (e.g., lifting a patient; taking a patient’s pulse, blood pressure, or oral temperature; or touching a patient’s hand, shoulder, or groin) (48). Similarly, in another study, hands were cultured of nurses who touched the groins of patients heavily colonized with *P. mirabilis* (25); 10–600 CFUs/mL of this

organism were recovered from glove juice samples from the nurses' hands. Recently, other researchers studied contamination of HCWs' hands during activities that involved direct patient-contact wound care, intravascular catheter care, respiratory-tract care, and the handling of patient secretions (51). Agar fingertip impression plates were used to culture bacteria; the number of bacteria recovered from fingertips ranged from 0 to 300 CFUs. Data from this study indicated that direct patient contact and respiratory-tract care were most likely to contaminate the fingers of caregivers. Gram-negative bacilli accounted for 15% of isolates and *S. aureus* for 11%. Duration of patient-care activity was strongly associated with the intensity of bacterial contamination of HCWs' hands.

HCWs can contaminate their hands with gram-negative bacilli, *S. aureus*, enterococci, or *Clostridium difficile* by performing "clean procedures" or touching intact areas of the skin of hospitalized patients (26,45,46,53). Furthermore, personnel caring for infants with respiratory syncytial virus (RSV) infections have acquired RSV by performing certain activities (e.g., feeding infants, changing diapers, and playing with infants) (49). Personnel who had contact only with surfaces contaminated with the infants' secretions also acquired RSV by contaminating their hands with RSV and inoculating their oral or conjunctival mucosa. Other studies also have documented that HCWs may contaminate their hands (or gloves) merely by touching inanimate objects in patient rooms (46,53–56). None of the studies concerning hand contamination of hospital personnel were designed to determine if the contamination resulted in transmission of pathogens to susceptible patients.

Other studies have documented contamination of HCWs' hands with potential health-care-associated pathogens, but did not relate their findings to the specific type of preceding patient contact (15,17,57–62). For example, before glove use was common among HCWs, 15% of nurses working in an isolation unit carried a median of 1×10^4 CFUs of *S. aureus* on their hands (61). Of nurses working in a general hospital, 29% had *S. aureus* on their hands (median count: 3,800 CFUs), whereas 78% of those working in a hospital for dermatology patients had the organism on their hands (median count: 14.3×10^6 CFUs). Similarly, 17%–30% of nurses carried gram-negative bacilli on their hands (median counts: 3,400–38,000 CFUs). One study found that *S. aureus* could be recovered from the hands of 21% of intensive-care-unit personnel and that 21% of physician and 5% of nurse carriers had $>1,000$ CFUs of the organism on their hands (59). Another study found lower levels of colonization on the hands of personnel working in a neurosurgery unit, with an average of 3 CFUs of *S. aureus* and 11 CFUs of gram-negative bacilli (16). Serial

cultures revealed that 100% of HCWs carried gram-negative bacilli at least once, and 64% carried *S. aureus* at least once.

Models of Hand Transmission

Several investigators have studied transmission of infectious agents by using different experimental models. In one study, nurses were asked to touch the groins of patients heavily colonized with gram-negative bacilli for 15 seconds — as though they were taking a femoral pulse (25). Nurses then cleaned their hands by washing with plain soap and water or by using an alcohol hand rinse. After cleaning their hands, they touched a piece of urinary catheter material with their fingers, and the catheter segment was cultured. The study revealed that touching intact areas of moist skin of the patient transferred enough organisms to the nurses' hands to result in subsequent transmission to catheter material, despite handwashing with plain soap and water.

The transmission of organisms from artificially contaminated "donor" fabrics to clean "recipient" fabrics via hand contact also has been studied. Results indicated that the number of organisms transmitted was greater if the donor fabric or the hands were wet upon contact (63). Overall, only 0.06% of the organisms obtained from the contaminated donor fabric were transferred to recipient fabric via hand contact. *Staphylococcus saprophyticus*, *Pseudomonas aeruginosa*, and *Serratia* spp. were also transferred in greater numbers than was *Escherichia coli* from contaminated fabric to clean fabric after hand contact (64). Organisms are transferred to various types of surfaces in much larger numbers (i.e., $>10^4$) from wet hands than from hands that are thoroughly dried (65).

Relation of Hand Hygiene and Acquisition of Health-Care-Associated Pathogens

Hand antisepsis reduces the incidence of health-care-associated infections (66,67). An intervention trial using historical controls demonstrated in 1847 that the mortality rate among mothers who delivered in the First Obstetrics Clinic at the General Hospital of Vienna was substantially lower when hospital staff cleaned their hands with an antiseptic agent than when they washed their hands with plain soap and water (3).

In the 1960s, a prospective, controlled trial sponsored by the National Institutes of Health and the Office of the Surgeon General demonstrated that infants cared for by nurses who did not wash their hands after handling an index infant colonized with *S. aureus* acquired the organism more often and more rapidly than did infants cared for by nurses who used hexachlorophene to clean their hands between infant

contacts (68). This trial provided evidence that, when compared with no handwashing, washing hands with an antiseptic agent between patient contacts reduces transmission of health-care-associated pathogens.

Trials have studied the effects of handwashing with plain soap and water versus some form of hand antiseptics on health-care-associated infection rates (69,70). Health-care-associated infection rates were lower when antiseptic handwashing was performed by personnel (69). In another study, antiseptic handwashing was associated with lower health-care-associated infection rates in certain intensive-care units, but not in others (70).

Health-care-associated infection rates were lower after antiseptic handwashing using a chlorhexidine-containing detergent compared with handwashing with plain soap or use of an alcohol-based hand rinse (71). However, because only a minimal amount of the alcohol rinse was used during periods when the combination regimen also was in use and because adherence to policies was higher when chlorhexidine was available, determining which factor (i.e., the hand-hygiene regimen or differences in adherence) accounted for the lower infection rates was difficult. Investigators have determined also that health-care-associated acquisition of MRSA was reduced when the antimicrobial soap used for hygienic handwashing was changed (72,73).

Increased handwashing frequency among hospital staff has been associated with decreased transmission of *Klebsiella* spp. among patients (48); these studies, however, did not quantify the level of handwashing among personnel. In a recent study, the acquisition of various health-care-associated pathogens was reduced when hand antiseptics was performed more frequently by hospital personnel (74); both this study and another (75) documented that the prevalence of health-care-associated infections decreased as adherence to recommended hand-hygiene measures improved.

Outbreak investigations have indicated an association between infections and understaffing or overcrowding; the association was consistently linked with poor adherence to hand hygiene. During an outbreak investigation of risk factors for central venous catheter-associated bloodstream infections (76), after adjustment for confounding factors, the patient-to-nurse ratio remained an independent risk factor for bloodstream infection, indicating that nursing staff reduction below a critical threshold may have contributed to this outbreak by jeopardizing adequate catheter care. The understaffing of nurses can facilitate the spread of MRSA in intensive-care settings (77) through relaxed attention to basic control measures (e.g., hand hygiene). In an outbreak of *Enterobacter cloacae* in a neonatal intensive-care unit (78), the daily number of

hospitalized children was above the maximum capacity of the unit, resulting in an available space per child below current recommendations. In parallel, the number of staff members on duty was substantially less than the number necessitated by the workload, which also resulted in relaxed attention to basic infection-control measures. Adherence to hand-hygiene practices before device contact was only 25% during the workload peak, but increased to 70% after the end of the understaffing and overcrowding period. Surveillance documented that being hospitalized during this period was associated with a fourfold increased risk of acquiring a health-care-associated infection. This study not only demonstrates the association between workload and infections, but it also highlights the intermediate cause of antimicrobial spread: poor adherence to hand-hygiene policies.

Methods Used To Evaluate the Efficacy of Hand-Hygiene Products

Current Methods

Investigators use different methods to study the in vivo efficacy of handwashing, antiseptic handwash, and surgical hand antiseptics protocols. Differences among the various studies include 1) whether hands are purposely contaminated with bacteria before use of test agents, 2) the method used to contaminate fingers or hands, 3) the volume of hand-hygiene product applied to the hands, 4) the time the product is in contact with the skin, 5) the method used to recover bacteria from the skin after the test solution has been used, and 6) the method of expressing the efficacy of the product (i.e., either percent reduction in bacteria recovered from the skin or log reduction of bacteria released from the skin). Despite these differences, the majority of studies can be placed into one of two major categories: studies focusing on products to remove transient flora and studies involving products that are used to remove resident flora from the hands. The majority of studies of products for removing transient flora from the hands of HCWs involve artificial contamination of the volunteer's skin with a defined inoculum of a test organism before the volunteer uses a plain soap, an antimicrobial soap, or a waterless antiseptic agent. In contrast, products tested for the preoperative cleansing of surgeons' hands (which must comply with surgical hand-antiseptics protocols) are tested for their ability to remove resident flora from without artificially contaminating the volunteers' hands.

In the United States, antiseptic handwash products intended for use by HCWs are regulated by FDA's Division of Over-the-Counter Drug Products (OTC). Requirements for in vitro and in vivo testing of HCW handwash products and surgical

hand scrubs are outlined in the FDA Tentative Final Monograph for Healthcare Antiseptic Drug Products (TFM) (19). Products intended for use as HCW handwashes are evaluated by using a standardized method (19). Tests are performed in accordance with use directions for the test material. Before baseline bacterial sampling and before each wash with the test material, 5 mL of a standardized suspension of *Serratia marcescens* are applied to the hands and then rubbed over the surfaces of the hands. A specified volume of the test material is dispensed into the hands and is spread over the hands and lower one third of the forearms. A small amount of tap water is added to the hands, and hands are completely lathered for a specified time, covering all surfaces of the hands and the lower third of the forearms. Volunteers then rinse hands and forearms under 40°C tap water for 30 seconds. Ten washes with the test formulation are required. After the first, third, seventh, and tenth washes, rubber gloves or polyethylene bags used for sampling are placed on the right and left hands, and 75 mL of sampling solution is added to each glove; gloves are secured above the wrist. All surfaces of the hand are massaged for 1 minute, and samples are obtained aseptically for quantitative culture. No neutralizer of the antimicrobial is routinely added to the sampling solution, but if dilution of the antimicrobial in the sampling fluid does not result in demonstrable neutralization, a neutralizer specific for the test formulation is added to the sampling solution. For waterless formulations, a similar procedure is used. TFM criteria for efficacy are as follows: a 2- \log_{10} reduction of the indicator organism on each hand within 5 minutes after the first use, and a 3- \log_{10} reduction of the indicator organism on each hand within 5 minutes after the tenth use (19).

Products intended for use as surgical hand scrubs have been evaluated also by using a standardized method (19). Volunteers clean under fingernails with a nail stick and clip their fingernails. All jewelry is removed from hands and arms. Hands and two thirds of forearms are rinsed with tap water (38°C–42°C) for 30 seconds, and then they are washed with a non-antimicrobial soap for 30 seconds and are rinsed for 30 seconds under tap water. Baseline microbial hand counts can then be determined. Next, a surgical scrub is performed with the test formulation using directions provided by the manufacturer. If no instructions are provided with the formulation, two 5-minute scrubs of hands and forearms followed by rinsing are performed. Reduction from baseline microbial hand counts is determined in a series of 11 scrubs conducted during 5 days. Hands are sampled at 1 minute, 3 hours, and 6 hours after the first scrubs on day 1, day 2, and day 5. After washing, volunteers wear rubber gloves; 75 mL of sampling solution are then added to one glove, and all surfaces of the hands are massaged

for 1 minute. Samples are then taken aseptically and cultured quantitatively. The other glove remains on the other hand for 6 hours and is sampled in the same manner. TFM requires that formulations reduce the number of bacteria 1 \log_{10} on each hand within 1 minute of product application and that the bacterial cell count on each hand does not subsequently exceed baseline within 6 hours on day 1; the formulation must produce a 2- \log_{10} reduction in microbial flora on each hand within 1 minute of product application by the end of the second day of enumeration and a 3- \log_{10} reduction of microbial flora on each hand within 1 minute of product use by the end of the fifth day when compared with the established baseline (19).

The method most widely used in Europe to evaluate the efficacy of hand-hygiene agents is European Standard 1500–1997 (EN 1500—Chemical disinfectants and antiseptics. Hygienic hand-rub test method and requirements) (79). This method requires 12–15 test volunteers and an 18- to 24-hour growth of broth culture of *E. coli* K12. Hands are washed with a soft soap, dried, and then immersed halfway to the metacarpals in the broth culture for 5 seconds. Hands are removed from the broth culture, excess fluid is drained off, and hands are dried in the air for 3 minutes. Bacterial recovery for the initial value is obtained by kneading the fingertips of each hand separately for 60 seconds in 10 mL of tryptic soy broth (TSB) without neutralizers. The hands are removed from the broth and disinfected with 3 mL of the hand-rub agent for 30 seconds in a set design. The same operation is repeated with total disinfection time not exceeding 60 seconds. Both hands are rinsed in running water for 5 seconds and water is drained off. Fingertips of each hand are kneaded separately in 10 mL of TSB with added neutralizers. These broths are used to obtain the final value. \log_{10} dilutions of recovery medium are prepared and plated out. Within 3 hours, the same volunteers are tested with the reference disinfectant (60% 2-propanol [isopropanol]) and the test product. Colony counts are performed after 24 and 48 hours of incubation at 36°C. The average colony count of both left and right hand is used for evaluation. The log-reduction factor is calculated and compared with the initial and final values. The reduction factor of the test product should be superior or the same as the reference alcohol-based rub for acceptance. If a difference exists, then the results are analyzed statistically using the Wilcoxon test. Products that have log reductions substantially less than that observed with the reference alcohol-based hand rub (i.e., approximately 4 \log_{10} reduction) are classified as not meeting the standard.

Because of different standards for efficacy, criteria cited in FDA TFM and the European EN 1500 document for establishing alcohol-based hand rubs vary (1, 19, 79). Alcohol-based

hand rubs that meet TFM criteria for efficacy may not necessarily meet the EN 1500 criteria for efficacy (80). In addition, scientific studies have not established the extent to which counts of bacteria or other microorganisms on the hands need to be reduced to minimize transmission of pathogens in health-care facilities (1,8); whether bacterial counts on the hands must be reduced by 1 log₁₀ (90% reduction), 2 log₁₀ (99%), 3 log₁₀ (99.9%), or 4 log₁₀ (99.99%) is unknown. Several other methods also have been used to measure the efficacy of antiseptic agents against various viral pathogens (81–83).

Shortcomings of Traditional Methodologies

Accepted methods of evaluating hand-hygiene products intended for use by HCWs require that test volunteers wash their hands with a plain or antimicrobial soap for 30 seconds or 1 minute, despite the observation in the majority of studies that the average duration of handwashing by hospital personnel is <15 seconds (52,84–89). A limited number of investigators have used 15-second handwashing or hygienic hand-wash protocols (90–94). Therefore, almost no data exist regarding the efficacy of plain or antimicrobial soaps under conditions in which they are actually used by HCWs. Similarly, certain accepted methods for evaluating waterless antiseptic agents for use as antiseptic hand rubs require that 3 mL of alcohol be rubbed into the hands for 30 seconds, followed by a repeat application for the same duration. This type of protocol also does not reflect actual usage patterns among HCWs. Furthermore, volunteers used in evaluations of products are usually surrogates for HCWs, and their hand flora may not reflect flora found on the hands of personnel working in health-care settings. Further studies should be conducted among practicing HCWs using standardized protocols to obtain more realistic views of microbial colonization and risk of bacterial transfer and cross-transmission (51).

Review of Preparations Used for Hand Hygiene

Plain (Non-Antimicrobial) Soap

Soaps are detergent-based products that contain esterified fatty acids and sodium or potassium hydroxide. They are available in various forms including bar soap, tissue, leaflet, and liquid preparations. Their cleaning activity can be attributed to their detergent properties, which result in removal of dirt, soil, and various organic substances from the hands. Plain soaps have minimal, if any, antimicrobial activity. However, handwashing with plain soap can remove loosely adherent transient flora. For example, handwashing with plain soap and water for 15 seconds reduces bacterial counts on the skin by 0.6–1.1 log₁₀, whereas washing for 30 seconds reduces counts

by 1.8–2.8 log₁₀ (1). However, in several studies, handwashing with plain soap failed to remove pathogens from the hands of hospital personnel (25,45). Handwashing with plain soap can result in paradoxical increases in bacterial counts on the skin (92,95–97). Non-antimicrobial soaps may be associated with considerable skin irritation and dryness (92,96,98), although adding emollients to soap preparations may reduce their propensity to cause irritation. Occasionally, plain soaps have become contaminated, which may lead to colonization of hands of personnel with gram-negative bacilli (99).

Alcohols

The majority of alcohol-based hand antiseptics contain either isopropanol, ethanol, n-propanol, or a combination of two of these products. Although n-propanol has been used in alcohol-based hand rubs in parts of Europe for many years, it is not listed in TFM as an approved active agent for HCW handwashes or surgical hand-scrub preparations in the United States. The majority of studies of alcohols have evaluated individual alcohols in varying concentrations. Other studies have focused on combinations of two alcohols or alcohol solutions containing limited amounts of hexachlorophene, quaternary ammonium compounds, povidone-iodine, triclosan, or chlorhexidine gluconate (61,93,100–119).

The antimicrobial activity of alcohols can be attributed to their ability to denature proteins (120). Alcohol solutions containing 60%–95% alcohol are most effective, and higher concentrations are less potent (120–122) because proteins are not denatured easily in the absence of water (120). The alcohol content of solutions may be expressed as percent by weight (w/w), which is not affected by temperature or other variables, or as percent by volume (vol/vol), which can be affected by temperature, specific gravity, and reaction concentration (123). For example, 70% alcohol by weight is equivalent to 76.8% by volume if prepared at 15°C, or 80.5% if prepared at 25°C (123). Alcohol concentrations in antiseptic hand rubs are often expressed as percent by volume (19).

Alcohols have excellent in vitro germicidal activity against gram-positive and gram-negative vegetative bacteria, including multidrug-resistant pathogens (e.g., MRSA and VRE), *Mycobacterium tuberculosis*, and various fungi (120–122,124–129). Certain enveloped (lipophilic) viruses (e.g., herpes simplex virus, human immunodeficiency virus [HIV], influenza virus, respiratory syncytial virus, and vaccinia virus) are susceptible to alcohols when tested in vitro (120,130,131) (Table 1). Hepatitis B virus is an enveloped virus that is somewhat less susceptible but is killed by 60%–70% alcohol; hepatitis C virus also is likely killed by this percentage of alcohol (132). In a porcine tissue carrier model used to study antiseptic activity, 70% ethanol and 70% isopropanol were found to

TABLE 1. Virucidal activity of antiseptic agents against enveloped viruses

Ref. no.	Test method	Viruses	Agent	Results
(379)	Suspension	HIV	19% EA	LR = 2.0 in 5 minutes
(380)	Suspension	HIV	50% EA 35% IPA	LR > 3.5 LR > 3.7
(381)	Suspension	HIV	70% EA	LR = 7.0 in 1 minute
(382)	Suspension	HIV	70% EA	LR = 3.2B 5.5 in 30 seconds
(383)	Suspension	HIV	70% IPA/0.5% CHG 4% CHG	LR = 6.0 in 15 seconds LR = 6.0 in 15 seconds
(384)	Suspension	HIV	Chloroxylenol Benzalkonium chloride	Inactivated in 1 minute Inactivated in 1 minute
(385)	Suspension	HIV	Povidone-iodine Chlorhexidine	Inactivated Inactivated
(386)	Suspension	HIV	Detergent/0.5% PCMX	Inactivated in 30 seconds
(387)	Suspension/dried plasma chimpanzee challenge	HBV	70% IPA	LR = 6.0 in 10 minutes
(388)	Suspension/plasma chimpanzee challenge	HBV	80% EA	LR = 7.0 in 2 minutes
(389)	Suspension	HSV	95% EA 75% EA 95% IPA 70% EA + 0.5% CHG	LR > 5.0 in 1 minute LR > 5.0 LR > 5.0 LR > 5.0
(130)	Suspension	RSV	35% IPA 4% CHG	LR > 4.3 in 1 minute LR > 3.3
(141)	Suspension	Influenza Vaccinia	95% EA 95% EA	Undetectable in 30 seconds Undetectable in 30 seconds
(141)	Hand test	Influenza Vaccinia	95% EA 95% EA	LR > 2.5 LR > 2.5

Note: HIV = human immunodeficiency virus, EA = ethanol, LR = Log₁₀ reduction, IPA = isopropanol, CHG = chlorhexidine gluconate, HBV = hepatitis B virus, RSV = respiratory syncytial virus, HSV = herpes simplex virus, HAV = hepatitis A virus, and PCMX = chloroxylenol.

reduce titers of an enveloped bacteriophage more effectively than an antimicrobial soap containing 4% chlorhexidine gluconate (133). Despite its effectiveness against these organisms, alcohols have very poor activity against bacterial spores, protozoan oocysts, and certain nonenveloped (nonlipophilic) viruses.

Numerous studies have documented the *in vivo* antimicrobial activity of alcohols. Alcohols effectively reduce bacterial counts on the hands (14, 121, 125, 134). Typically, log reductions of the release of test bacteria from artificially contaminated hands average 3.5 log₁₀ after a 30-second application and 4.0–5.0 log₁₀ after a 1-minute application (1). In 1994, the FDA TFM classified ethanol 60%–95% as a Category I agent (i.e., generally safe and effective for use in antiseptic handwash or HCW hand-wash products) (19). Although TFM placed isopropanol 70%–91.3% in category IIIIE (i.e., insufficient data to classify as effective), 60% isopropanol has subse-

quently been adopted in Europe as the reference standard against which alcohol-based hand-rub products are compared (79). Alcohols are rapidly germicidal when applied to the skin, but they have no appreciable persistent (i.e., residual) activity. However, regrowth of bacteria on the skin occurs slowly after use of alcohol-based hand antiseptics, presumably because of the sublethal effect alcohols have on some of the skin bacteria (135, 136). Addition of chlorhexidine, quaternary ammonium compounds, octenidine, or triclosan to alcohol-based solutions can result in persistent activity (1).

Alcohols, when used in concentrations present in alcohol-based hand rubs, also have *in vivo* activity against several nonenveloped viruses (Table 2). For example, 70% isopropanol and 70% ethanol are more effective than medicated soap or nonmedicated soap in reducing rotavirus titers on fingerpads (137, 138). A more recent study using the same test methods evaluated a commercially available product containing 60%

TABLE 2. Virucidal activity of antiseptic agents against nonenveloped viruses

Ref. no.	Test method	Viruses	Antiseptic	Result
(390)	Suspension	Rotavirus	4% CHG 10% Povidone-Iodine 70% IPA/0.1% HCP	LR < 3.0 in 1 minute LR > 3.0 LR > 3.0
(141)	Hand test	Adenovirus Poliovirus Coxsackie	95% EA 95% EA 95% EA	LR > 1.4 LR = 0.2–1.0 LR = 1.1–1.3
	Finger test	Adenovirus Poliovirus Coxsackie	95% EA 95% EA 95% EA	LR > 2.3 LR = 0.7–2.5 LR = 2.9
(389)	Suspension	ECHO virus	95% EA 75% EA 95% IPA 70% IPA + 0.5% CHG	LR > 3.0 in 1 minute LR ≤ 1.0 LR = 0 LR = 0
(140)	Finger pad	HAV	70% EA 62% EA foam plain soap 4% CHG 0.3% Triclosan	87.4% reduction 89.3% reduction 78.0% reduction 89.6% reduction 92.0% reduction
(105)	Finger tips	Bovine Rotavirus	n-propanol + IPA 70% IPA 70% EA 2% triclosan water (control) 7.5% povidone-iodine plain soap 4% CHG	LR = 3.8 in 30 seconds LR = 3.1 LR = 2.9 LR = 2.1 LR = 1.3 LR = 1.3 LR = 1.2 LR = 0.5
(137)	Finger pad	Human Rotavirus	70% IPA plain soap	98.9% decrease in 10 seconds 77.1%
(138)	Finger pad	Human Rotavirus	70% IPA 2% CHG plain soap	99.6% decrease in 10 seconds 80.3% 72.5%
(81)	Finger pad	Rotavirus Rhinovirus Adenovirus	60% EA gel 60% EA gel 60% EA gel	LR > 3.0 in 10 seconds LR > 3.0 LR > 3.0
(139)	Finger pad	Poliovirus	70% EA 70% IPA	LR = 1.6 in 10 seconds LR = 0.8
(200)	Finger tips	Poliovirus	Plain soap 80% EA	LR = 2.1 LR = 0.4

Note: HIV = human immunodeficiency virus, EA = ethanol, LR = Log₁₀ reduction, IPA = isopropanol, CHG = chlorhexidine gluconate, HBV = hepatitis B virus, RSV = respiratory syncytial virus, HSV = herpes simplex virus, and HAV = hepatitis A virus.

ethanol and found that the product reduced the infectivity titers of three nonenveloped viruses (i.e., rotavirus, adenovirus, and rhinovirus) by >3 logs (81). Other nonenveloped viruses such as hepatitis A and enteroviruses (e.g., poliovirus) may require 70%–80% alcohol to be reliably inactivated (82,139). However, both 70% ethanol and a 62% ethanol foam product with emollients reduced hepatitis A virus titers on whole hands or fingertips more than nonmedicated soap; both were equally as effective as antimicrobial soap containing 4% chlorhexidine gluconate in reducing reduced viral counts on hands (140). In the same study, both 70% ethanol and the 62% ethanol foam product demonstrated greater virucidal activity against poliovirus than either non-antimicrobial

soap or a 4% chlorhexidine gluconate-containing soap (140). However, depending on the alcohol concentration, the amount of time that hands are exposed to the alcohol, and viral variant, alcohol may not be effective against hepatitis A and other nonlipophilic viruses. The inactivation of nonenveloped viruses is influenced by temperature, disinfectant-virus volume ratio, and protein load (141). Ethanol has greater activity against viruses than isopropanol. Further in vitro and in vivo studies of both alcohol-based formulations and antimicrobial soaps are warranted to establish the minimal level of virucidal activity that is required to interrupt direct contact transmission of viruses in health-care settings.

Alcohols are not appropriate for use when hands are visibly dirty or contaminated with proteinaceous materials. However, when relatively small amounts of proteinaceous material (e.g., blood) are present, ethanol and isopropanol may reduce viable bacterial counts on hands more than plain soap or antimicrobial soap (142).

Alcohol can prevent the transfer of health-care-associated pathogens (25,63,64). In one study, gram-negative bacilli were transferred from a colonized patient's skin to a piece of catheter material via the hands of nurses in only 17% of experiments after antiseptic hand rub with an alcohol-based hand rinse (25). In contrast, transfer of the organisms occurred in 92% of experiments after handwashing with plain soap and water. This experimental model indicates that when the hands of HCWs are heavily contaminated, an antiseptic hand rub using an alcohol-based rinse can prevent pathogen transmission more effectively than can handwashing with plain soap and water.

Alcohol-based products are more effective for standard handwashing or hand antisepsis by HCWs than soap or antimicrobial soaps (Table 3) (25,53,61,93,106–112,119,143–152). In all but two of the trials that compared alcohol-based solutions with antimicrobial soaps or detergents, alcohol reduced bacterial counts on hands more than washing hands with soaps or detergents containing hexachlorophene, povidone-iodine, 4% chlorhexidine, or triclosan. In studies exam-

ining antimicrobial-resistant organisms, alcohol-based products reduced the number of multidrug-resistant pathogens recovered from the hands of HCWs more effectively than did handwashing with soap and water (153–155).

Alcohols are effective for preoperative cleaning of the hands of surgical personnel (1,101,104,113–119,135,143,147,156–159) (Tables 4 and 5). In multiple studies, bacterial counts on the hands were determined immediately after using the product and again 1–3 hours later; the delayed testing was performed to determine if regrowth of bacteria on the hands is inhibited during operative procedures. Alcohol-based solutions were more effective than washing hands with plain soap in all studies, and they reduced bacterial counts on the hands more than antimicrobial soaps or detergents in the majority of experiments (101,104,113–119,135,143,147,157–159). In addition, the majority of alcohol-based preparations were more effective than povidone-iodine or chlorhexidine.

The efficacy of alcohol-based hand-hygiene products is affected by several factors, including the type of alcohol used, concentration of alcohol, contact time, volume of alcohol used, and whether the hands are wet when the alcohol is applied. Applying small volumes (i.e., 0.2–0.5 mL) of alcohol to the hands is not more effective than washing hands with plain soap and water (63,64). One study documented that 1 mL of alcohol was substantially less effective than 3 mL (91). The ideal volume of product to apply to the hands is not known

TABLE 3. Studies comparing the relative efficacy (based on log₁₀ reductions achieved) of plain soap or antimicrobial soaps versus alcohol-based antiseptics in reducing counts of viable bacteria on hands

Ref. no.	Year	Skin contamination	Assay method	Time (sec)	Relative efficacy
(143)	1965	Existing hand flora	Finger-tip agar culture	60	Plain soap < HCP < 50% EA foam
(119)	1975	Existing hand flora	Hand-rub broth culture	—	Plain soap < 95% EA
(106)	1978	Artificial contamination	Finger-tip broth culture	30	Plain soap < 4% CHG < P-I < 70% EA = alc. CHG
(144)	1978	Artificial contamination	Finger-tip broth culture	30	Plain soap < 4% CHG < 70% EA
(107)	1979	Existing hand flora	Hand-rub broth culture	120	Plain soap < 0.5% aq. CHG < 70% EA < 4% CHG < alc.CHG
(145)	1980	Artificial contamination	Finger-tip broth culture	60–120	4% CHG < P-I < 60% IPA
(53)	1980	Artificial contamination	Finger-tip broth culture	15	Plain soap < 3% HCP < P-I < 4% CHG < 70% EA
(108)	1982	Artificial contamination	Glove juice test	15	P-I < alc. CHG
(109)	1983	Artificial contamination	Finger-tip broth culture	120	0.3–2% triclosan = 60% IPA = alc. CHG < alc. triclosan
(146)	1984	Artificial contamination	Finger-tip agar culture	60	Phenolic < 4% CHG < P-I < EA < IPA < n-P
(147)	1985	Existing hand flora	Finger-tip agar culture	60	Plain soap < 70% EA < 95% EA
(110)	1986	Artificial contamination	Finger-tip broth culture	60	Phenolic = P-I < alc. CHG < n-P
(93)	1986	Existing hand flora	Sterile-broth bag technique	15	Plain soap < IPA < 4% CHG = IPA-E = alc. CHG
(61)	1988	Artificial contamination	Finger-tip broth culture	30	Plain soap < triclosan < P-I < IPA < alc. CHG < n-P
(25)	1991	Patient contact	Glove-juice test	15	Plain soap < IPA-E
(148)	1991	Existing hand flora	Agar-plate/image analysis	30	Plain soap < 1% triclosan < P-I < 4% CHG < IPA
(111)	1992	Artificial contamination	Finger-tip agar culture	60	Plain soap < IPA < EA < alc. CHG
(149)	1992	Artificial contamination	Finger-tip broth culture	60	Plain soap < 60% n-P
(112)	1994	Existing hand flora	Agar-plate/image analysis	30	Plain soap < alc. CHG
(150)	1999	Existing hand flora	Agar-plate culture	N.S.	Plain soap < commercial alcohol mixture
(151)	1999	Artificial contamination	Glove-juice test	20	Plain soap < 0.6% PCMX < 65% EA
(152)	1999	Artificial contamination	Finger-tip broth culture	30	4% CHG < plain soap < P-I < 70% EA

Note: Existing hand flora = without artificially contaminating hands with bacteria, alc. CHG = alcoholic chlorhexidine gluconate, aq. CHG = aqueous chlorhexidine gluconate, 4% CHG = chlorhexidine gluconate detergent, EA = ethanol, HCP = hexachlorophene soap/detergent, IPA = isopropanol, IPA-E = isopropanol + emollients, n-P = n-propanol, PCMX = chloroxyleneol detergent, P-I = povidone-iodine detergent, and N.S. = not stated.

TABLE 4. Studies comparing the relative efficacy of plain soap or antimicrobial soap versus alcohol-containing products in reducing counts of bacteria recovered from hands immediately after use of products for pre-operative cleansing of hands

Ref. no.	Year	Assay method	Relative efficacy
(143)	1965	Finger-tip agar culture	HCP < 50% EA foam + QAC
(157)	1969	Finger-tip agar culture	HCP < P-I < 50% EA foam + QAC
(101)	1973	Finger-tip agar culture	HCP soap < EA foam + 0.23% HCP
(135)	1974	Broth culture	Plain soap < 0.5% CHG < 4% CHG < alc. CHG
(119)	1975	Hand-broth test	Plain soap < 0.5% CHG < 4% CHG < alc. CHG
(118)	1976	Glove-juice test	0.5% CHG < 4% CHG < alc. CHG
(114)	1977	Glove-juice test	P-I < CHG < alc. CHG
(117)	1978	Finger-tip agar culture	P-I = 46% EA + 0.23% HCP
(113)	1979	Broth culture of hands	Plain soap < P-I < alc. CHG < alc. P-I
(116)	1979	Glove-juice test	70% IPA = alc. CHG
(147)	1985	Finger-tip agar culture	Plain soap < 70% - 90% EA
(115)	1990	Glove-juice test, modified	Plain soap < triclosan < CHG < P-I < alc. CHG
(104)	1991	Glove-juice test	Plain soap < 2% triclosan < P-I < 70% IPA
(158)	1998	Finger-tip broth culture	70% IPA < 90% IPA = 60% n-P
(159)	1998	Glove-juice test	P-I < CHG < 70% EA

Note: QAC = quaternary ammonium compound, alc. CHG = alcoholic chlorhexidine gluconate, CHG = chlorhexidine gluconate detergent, EA = ethanol, HCP = hexachlorophene detergent, IPA = isopropanol, and P-I = povidone-iodine detergent.

TABLE 5. Efficacy of surgical hand-rub solutions in reducing the release of resident skin flora from clean hands

Study	Rub	Concentration* (%)	Time (min)	Mean log reduction				
				Immediate	Sustained (3 hr)			
1	n-Propanol	60	5	2.9 [†]	1.6 [†]			
2			5	2.7 [†]	NA			
3			5	2.5 [†]	1.8 [†]			
4			5	2.3 [†]	1.6 [†]			
5			3	2.9 [§]	NA			
4			3	2.0 [†]	1.0 [†]			
4			1	1.1 [†]	0.5 [†]			
6			Isopropanol	90	3	2.4 [§]	1.4 [§]	
6					3	2.3 [§]	1.2 [§]	
7					5	2.4 [†]	2.1 [†]	
4	5	2.1 [†]			1.0 [†]			
6	3	2.0 [§]			0.7 [§]			
5	3	1.7 ^c			NA			
4	3	1.5 [†]			0.8 [†]			
8	2	1.2			0.8			
4	1	0.7 [†]	0.2					
9	1	0.8	NA					
10	Isopropanol + chlorhexidine gluc. (w/v)	60	5	1.7	1.0			
7			70 + 0.5	5	2.5 [†]	2.7 [†]		
8				2	1.0	1.5		
11				Ethanol	95	2.1	NA	
5					85	2.4 [§]	NA	
12					80	1.5	NA	
8					70	1.0	0.6	
13				Ethanol + chlorhexidine gluc. (w/v)	95 + 0.5	2	1.7	NA
14					77 + 0.5	5	2.0	1.5 [¶]
8					70 + 0.5	2	0.7	1.4
8	Chlorhexidine gluc. (aq. Sol., w/v)	0.5		2	0.4	1.2		
15	Povidone-iodine (aq. Sol., w/v)	1.0	5	1.9 [†]	0.8 [†]			
16	Peracetic acid (w/v)	0.5	5	1.9	NA			

Note: NA = not available.

Source: Rotter M. Hand washing and hand disinfection [Chapter 87]. In: Mayhall CG, ed. Hospital epidemiology and infection control. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999. Table 5 is copyrighted by Lippincott Williams & Wilkins; it is reprinted here with their permission and permission from Manfred Rotler, M.D., Professor of Hygiene and Microbiology, Klinisches Institute für Hygiene der Universität Wien, Germany.

* Volume/volume unless otherwise stated.

[†] Tested according to Deutsche Gesellschaft für Hygiene, and Mikrobiologic (DGHM)-German Society of Hygiene and Microbiology method.

[§] Tested according to European Standard prEN.

[¶] After 4 hours.

and may vary for different formulations. However, if hands feel dry after rubbing hands together for 10–15 seconds, an insufficient volume of product likely was applied. Because alcohol-impregnated towelettes contain a limited amount of alcohol, their effectiveness is comparable to that of soap and water (63,160,161).

Alcohol-based hand rubs intended for use in hospitals are available as low viscosity rinses, gels, and foams. Limited data are available regarding the relative efficacy of various formulations. One field trial demonstrated that an ethanol gel was slightly more effective than a comparable ethanol solution at reducing bacterial counts on the hands of HCWs (162). However, a more recent study indicated that rinses reduced bacterial counts on the hands more than the gels tested (80). Further studies are warranted to determine the relative efficacy of alcohol-based rinses and gels in reducing transmission of health-care-associated pathogens.

Frequent use of alcohol-based formulations for hand antisepsis can cause drying of the skin unless emollients, humectants, or other skin-conditioning agents are added to the formulations. The drying effect of alcohol can be reduced or eliminated by adding 1%–3% glycerol or other skin-conditioning agents (90,93,100,101,106,135,143,163,164). Moreover, in several recent prospective trials, alcohol-based rinses or gels containing emollients caused substantially less skin irritation and dryness than the soaps or antimicrobial detergents tested (96,98,165,166). These studies, which were conducted in clinical settings, used various subjective and objective methods for assessing skin irritation and dryness. Further studies are warranted to establish whether products with different formulations yield similar results.

Even well-tolerated alcohol hand rubs containing emollients may cause a transient stinging sensation at the site of any broken skin (e.g., cuts and abrasions). Alcohol-based hand-rub preparations with strong fragrances may be poorly tolerated by HCWs with respiratory allergies. Allergic contact dermatitis or contact urticaria syndrome caused by hypersensitivity to alcohol or to various additives present in certain alcohol hand rubs occurs only rarely (167,168).

Alcohols are flammable. Flash points of alcohol-based hand rubs range from 21°C to 24°C, depending on the type and concentration of alcohol present (169). As a result, alcohol-based hand rubs should be stored away from high temperatures or flames in accordance with National Fire Protection Agency recommendations. In Europe, where alcohol-based hand rubs have been used extensively for years, the incidence of fires associated with such products has been low (169). One recent U.S. report described a flash fire that occurred as a result of an unusual series of events, which included an HCW applying an alcohol gel to her hands, immediately removing a

polyester isolation gown, and then touching a metal door before the alcohol had evaporated (170). Removing the polyester gown created a substantial amount of static electricity that generated an audible static spark when the HCW touched the metal door, igniting the unevaporated alcohol on her hands (170). This incident emphasizes the need to rub hands together after application of alcohol-based products until all the alcohol has evaporated.

Because alcohols are volatile, containers should be designed to minimize evaporation. Contamination of alcohol-based solutions has seldom been reported. One report documented a cluster of pseudoinfections caused by contamination of ethyl alcohol by *Bacillus cereus* spores (171).

Chlorhexidine

Chlorhexidine gluconate, a cationic bisbiguanide, was developed in England in the early 1950s and was introduced into the United States in the 1970s (8,172). Chlorhexidine base is only minimally soluble in water, but the digluconate form is water-soluble. The antimicrobial activity of chlorhexidine is likely attributable to attachment to, and subsequent disruption of, cytoplasmic membranes, resulting in precipitation of cellular contents (1,8). Chlorhexidine's immediate antimicrobial activity occurs more slowly than that of alcohols. Chlorhexidine has good activity against gram-positive bacteria, somewhat less activity against gram-negative bacteria and fungi, and only minimal activity against tubercle bacilli (1,8,172). Chlorhexidine is not sporicidal (1,172). It has in vitro activity against enveloped viruses (e.g., herpes simplex virus, HIV, cytomegalovirus, influenza, and RSV) but substantially less activity against nonenveloped viruses (e.g., rotavirus, adenovirus, and enteroviruses) (130,131,173). The antimicrobial activity of chlorhexidine is only minimally affected by the presence of organic material, including blood. Because chlorhexidine is a cationic molecule, its activity can be reduced by natural soaps, various inorganic anions, nonionic surfactants, and hand creams containing anionic emulsifying agents (8,172,174). Chlorhexidine gluconate has been incorporated into a number of hand-hygiene preparations. Aqueous or detergent formulations containing 0.5% or 0.75% chlorhexidine are more effective than plain soap, but they are less effective than antiseptic detergent preparations containing 4% chlorhexidine gluconate (135,175). Preparations with 2% chlorhexidine gluconate are slightly less effective than those containing 4% chlorhexidine (176).

Chlorhexidine has substantial residual activity (106,114–116,118,135,146,175). Addition of low concentrations (0.5%–1.0%) of chlorhexidine to alcohol-based preparations results in greater residual activity than alcohol alone (116,135). When used as recommended, chlorhexidine has a good safety

record (172). Minimal, if any, absorption of the compound occurs through the skin. Care must be taken to avoid contact with the eyes when using preparations with $\geq 1\%$ chlorhexidine, because the agent can cause conjunctivitis and severe corneal damage. Ototoxicity precludes its use in surgery involving the inner or middle ear. Direct contact with brain tissue and the meninges should be avoided. The frequency of skin irritation is concentration-dependent, with products containing 4% most likely to cause dermatitis when used frequently for antiseptic handwashing (177); allergic reactions to chlorhexidine gluconate are uncommon (118,172). Occasional outbreaks of nosocomial infections have been traced to contaminated solutions of chlorhexidine (178–181).

Chloroxylenol

Chloroxylenol, also known as parachlorometaxylenol (PCMX), is a halogen-substituted phenolic compound that has been used as a preservative in cosmetics and other products and as an active agent in antimicrobial soaps. It was developed in Europe in the late 1920s and has been used in the United States since the 1950s (182).

The antimicrobial activity of PCMX likely is attributable to inactivation of bacterial enzymes and alteration of cell walls (1). It has good in vitro activity against gram-positive organisms and fair activity against gram-negative bacteria, mycobacteria, and certain viruses (1,7,182). PCMX is less active against *P. aeruginosa*, but addition of ethylenediaminetetraacetic acid (EDTA) increases its activity against *Pseudomonas* spp. and other pathogens.

A limited number of articles focusing on the efficacy of PCMX-containing preparations intended for use by HCWs have been published in the last 25 years, and the results of studies have sometimes been contradictory. For example, in studies in which antiseptics were applied to abdominal skin, PCMX had the weakest immediate and residual activity of any of the agents studied (183). However, when 30-second handwashes were performed using 0.6% PCMX, 2% chlorhexidine gluconate, or 0.3% triclosan, the immediate effect of PCMX was similar to that of the other agents. When used 18 times per day for 5 consecutive days, PCMX had less cumulative activity than did chlorhexidine gluconate (184). When PCMX was used as a surgical scrub, one report indicated that 3% PCMX had immediate and residual activity comparable to 4% chlorhexidine gluconate (185), whereas two other studies demonstrated that the immediate and residual activity of PCMX was inferior to both chlorhexidine gluconate and povidone-iodine (176,186). The disparity between published studies may be associated with the various concentrations of PCMX included in the preparations evaluated and with other aspects of the formulations tested, including the

presence or absence of EDTA (7,182). PCMX is not as rapidly active as chlorhexidine gluconate or iodophors, and its residual activity is less pronounced than that observed with chlorhexidine gluconate (7,182). In 1994, FDA TFM tentatively classified PCMX as a Category IIISE active agent (i.e., insufficient data are available to classify this agent as safe and effective) (19). Further evaluation of this agent by the FDA is ongoing.

The antimicrobial activity of PCMX is minimally affected by the presence of organic matter, but it is neutralized by non-ionic surfactants. PCMX, which is absorbed through the skin (7,182), is usually well-tolerated, and allergic reactions associated with its use are uncommon. PCMX is available in concentrations of 0.3%–3.75%. In-use contamination of a PCMX-containing preparation has been reported (187).

Hexachlorophene

Hexachlorophene is a bisphenol composed of two phenolic groups and three chlorine moieties. In the 1950s and early 1960s, emulsions containing 3% hexachlorophene were widely used for hygienic handwashing, as surgical scrubs, and for routine bathing of infants in hospital nurseries. The antimicrobial activity of hexachlorophene results from its ability to inactivate essential enzyme systems in microorganisms. Hexachlorophene is bacteriostatic, with good activity against *S. aureus* and relatively weak activity against gram-negative bacteria, fungi, and mycobacteria (7).

Studies of hexachlorophene as a hygienic handwash and surgical scrub demonstrated only modest efficacy after a single handwash (53,143,188). Hexachlorophene has residual activity for several hours after use and gradually reduces bacterial counts on hands after multiple uses (i.e., it has a cumulative effect) (1,101,188,189). With repeated use of 3% hexachlorophene preparations, the drug is absorbed through the skin. Infants bathed with hexachlorophene and personnel regularly using a 3% hexachlorophene preparation for handwashing have blood levels of 0.1–0.6 ppm hexachlorophene (190). In the early 1970s, certain infants bathed with hexachlorophene developed neurotoxicity (vacuolar degeneration) (191). As a result, in 1972, the FDA warned that hexachlorophene should no longer be used routinely for bathing infants. However, after routine use of hexachlorophene for bathing infants in nurseries was discontinued, investigators noted that the incidence of health-care-associated *S. aureus* infections in hospital nurseries increased substantially (192,193). In several instances, the frequency of infections decreased when hexachlorophene bathing of infants was reinstated. However, current guidelines still recommend against the routine bathing of neonates with hexachlorophene because of its potential neurotoxic effects (194). The agent is classified by FDA TFM as not

generally recognized as safe and effective for use as an antiseptic handwash (19). Hexachlorophene should not be used to bathe patients with burns or extensive areas of susceptible, sensitive skin. Soaps containing 3% hexachlorophene are available by prescription only (7).

Iodine and Iodophors

Iodine has been recognized as an effective antiseptic since the 1800s. However, because iodine often causes irritation and discoloring of skin, iodophors have largely replaced iodine as the active ingredient in antiseptics.

Iodine molecules rapidly penetrate the cell wall of microorganisms and inactivate cells by forming complexes with amino acids and unsaturated fatty acids, resulting in impaired protein synthesis and alteration of cell membranes (195). Iodophors are composed of elemental iodine, iodide or triiodide, and a polymer carrier (i.e., the complexing agent) of high molecular weight. The amount of molecular iodine present (so-called “free” iodine) determines the level of antimicrobial activity of iodophors. “Available” iodine refers to the total amount of iodine that can be titrated with sodium thiosulfate (196). Typical 10% povidone-iodine formulations contain 1% available iodine and yield free iodine concentrations of 1 ppm (196). Combining iodine with various polymers increases the solubility of iodine, promotes sustained release of iodine, and reduces skin irritation. The most common polymers incorporated into iodophors are polyvinyl pyrrolidone (i.e., povidone) and ethoxylated nonionic detergents (i.e., poloxamers) (195,196). The antimicrobial activity of iodophors also can be affected by pH, temperature, exposure time, concentration of total available iodine, and the amount and type of organic and inorganic compounds present (e.g., alcohols and detergents).

Iodine and iodophors have bactericidal activity against gram-positive, gram-negative, and certain spore-forming bacteria (e.g., clostridia and *Bacillus* spp.) and are active against mycobacteria, viruses, and fungi (8,195,197–200). However, in concentrations used in antiseptics, iodophors are not usually sporicidal (201). In vivo studies have demonstrated that iodophors reduce the number of viable organisms that are recovered from the hands of personnel (113,145,148,152,155). Povidone-iodine 5%–10% has been tentatively classified by FDA TFM as a Category I agent (i.e., a safe and effective agent for use as an antiseptic handwash and an HCW handwash) (19). The extent to which iodophors exhibit persistent antimicrobial activity after they have been washed off the skin is unclear. In one study, persistent activity was noted for 6 hours (176); however, several other studies demonstrated persistent activity for only 30–60 minutes after washing hands with an iodophor (61,117,202). In studies in which bacterial counts

were obtained after gloves were worn for 1–4 hours after washing, iodophors have demonstrated poor persistent activity (1,104,115,189,203–208). The in vivo antimicrobial activity of iodophors is substantially reduced in the presence of organic substances (e.g., blood or sputum) (8).

The majority of iodophor preparations used for hand hygiene contain 7.5%–10% povidone-iodine. Formulations with lower concentrations also have good antimicrobial activity because dilution can increase free iodine concentrations (209). However, as the amount of free iodine increases, the degree of skin irritation also may increase (209). Iodophors cause less skin irritation and fewer allergic reactions than iodine, but more irritant contact dermatitis than other antiseptics commonly used for hand hygiene (92). Occasionally, iodophor antiseptics have become contaminated with gram-negative bacilli as a result of poor manufacturing processes and have caused outbreaks or pseudo-outbreaks of infection (196).

Quaternary Ammonium Compounds

Quaternary ammonium compounds are composed of a nitrogen atom linked directly to four alkyl groups, which may vary in their structure and complexity (210). Of this large group of compounds, alkyl benzalkonium chlorides are the most widely used as antiseptics. Other compounds that have been used as antiseptics include benzethonium chloride, cetrимide, and cetylpyridium chloride (1). The antimicrobial activity of these compounds was first studied in the early 1900s, and a quaternary ammonium compound for preoperative cleaning of surgeons' hands was used as early as 1935 (210). The antimicrobial activity of this group of compounds likely is attributable to adsorption to the cytoplasmic membrane, with subsequent leakage of low molecular weight cytoplasmic constituents (210).

Quaternary ammonium compounds are primarily bacteriostatic and fungistatic, although they are microbicidal against certain organisms at high concentrations (1); they are more active against gram-positive bacteria than against gram-negative bacilli. Quaternary ammonium compounds have relatively weak activity against mycobacteria and fungi and have greater activity against lipophilic viruses. Their antimicrobial activity is adversely affected by the presence of organic material, and they are not compatible with anionic detergents (1,210). In 1994, FDA TFM tentatively classified benzalkonium chloride and benzethonium chloride as Category IIISE active agents (i.e., insufficient data exists to classify them as safe and effective for use as an antiseptic handwash) (19). Further evaluation of these agents by FDA is in progress.

Quaternary ammonium compounds are usually well tolerated. However, because of weak activity against

gram-negative bacteria, benzalkonium chloride is prone to contamination by these organisms. Several outbreaks of infection or pseudoinfection have been traced to quaternary ammonium compounds contaminated with gram-negative bacilli (211–213). For this reason, in the United States, these compounds have been seldom used for hand antisepsis during the last 15–20 years. However, newer handwashing products containing benzalkonium chloride or benzethonium chloride have recently been introduced for use by HCWs. A recent study of surgical intensive-care unit personnel found that cleaning hands with antimicrobial wipes containing a quaternary ammonium compound was about as effective as using plain soap and water for handwashing; both were less effective than decontaminating hands with an alcohol-based hand rub (214). One laboratory-based study reported that an alcohol-free hand-rub product containing a quaternary ammonium compound was efficacious in reducing microbial counts on the hands of volunteers (215). Further studies of such products are needed to determine if newer formulations are effective in health-care settings.

Triclosan

Triclosan (chemical name: 2,4,4'-trichloro-2'-hydroxydiphenyl ether) is a nonionic, colorless substance that was developed in the 1960s. It has been incorporated into soaps for use by HCWs and the public and into other consumer products. Concentrations of 0.2%–2% have antimicrobial activity. Triclosan enters bacterial cells and affects the cytoplasmic membrane and synthesis of RNA, fatty acids, and proteins (216). Recent studies indicate this agent's antibacterial activity is attributable to binding to the active site of enoyl-acyl carrier protein reductase (217,218).

Triclosan has a broad range of antimicrobial activity, but it is often bacteriostatic (1). Minimum inhibitory concentrations (MICs) range from 0.1 to 10 µg/mL, whereas minimum bactericidal concentrations are 25–500 µg/mL. Triclosan's activity against gram-positive organisms (including MRSA) is greater than against gram-negative bacilli, particularly *P. aeruginosa* (1,216). The agent possesses reasonable activity against mycobacterial and *Candida* spp., but it has limited activity against filamentous fungi. Triclosan (0.1%) reduces bacterial counts on hands by 2.8 log₁₀ after a 1-minute hygienic handwash (1). In several studies, log reductions have been lower after triclosan is used than when chlorhexidine, iodophors, or alcohol-based products are applied (1,61,149,184,219). In 1994, FDA TFM tentatively classified triclosan ≤1.0% as a Category IIISE active agent (i.e., insufficient data exist to classify this agent as safe and effective for use as an antiseptic handwash) (19). Further evaluation of this agent by the FDA is underway. Like chlorhexidine, triclosan has persistent activity on the skin. Its activity in

hand-care products is affected by pH, the presence of surfactants, emollients, or humectants and by the ionic nature of the particular formulation (1,216). Triclosan's activity is not substantially affected by organic matter, but it can be inhibited by sequestration of the agent in micelle structures formed by surfactants present in certain formulations. The majority of formulations containing <2% triclosan are well-tolerated and seldom cause allergic reactions. Certain reports indicate that providing hospital personnel with a triclosan-containing preparation for hand antisepsis has led to decreased MRSA infections (72,73). Triclosan's lack of potent activity against gram-negative bacilli has resulted in occasional reports of contamination (220).

Other Agents

Approximately 150 years after puerperal-fever-related maternal mortality rates were demonstrated by Semmelweis to be reduced by use of a hypochlorite hand rinse, the efficacy of rubbing hands for 30 seconds with an aqueous hypochlorite solution was studied once again (221). The solution was demonstrated to be no more effective than distilled water. The regimen used by Semmelweis, which called for rubbing hands with a 4% [w/w] hypochlorite solution until the hands were slippery (approximately 5 minutes), has been revisited by other researchers (222). This more current study indicated that the regimen was 30 times more effective than a 1-minute rub using 60% isopropanol. However, because hypochlorite solutions are often irritating to the skin when used repeatedly and have a strong odor, they are seldom used for hand hygiene.

Certain other agents are being evaluated by FDA for use in health-care-related antiseptics (19). However, the efficacy of these agents has not been evaluated adequately for use in handwashing preparations intended for use by HCWs. Further evaluation of these agents is warranted. Products that use different concentrations of traditional antiseptics (e.g., low concentrations of iodophor) or contain novel compounds with antiseptic properties are likely to be introduced for use by HCWs. For example, preliminary studies have demonstrated that adding silver-containing polymers to an ethanol carrier (i.e., Surfacine®) results in a preparation that has persistent antimicrobial activity on animal and human skin (223). New compounds with good in vitro activity must be tested in vivo to determine their abilities to reduce transient and resident skin flora on the hands of HCWs.

Activity of Antiseptic Agents Against Spore-Forming Bacteria

The widespread prevalence of health-care-associated diarrhea caused by *Clostridium difficile* and the recent occurrence

in the United States of human *Bacillus anthracis* infections associated with contaminated items sent through the postal system has raised concern regarding the activity of antiseptic agents against spore-forming bacteria. None of the agents (including alcohols, chlorhexidine, hexachlorophene, iodophors, PCMX, and triclosan) used in antiseptic handwash or antiseptic hand-rub preparations are reliably sporicidal against *Clostridium* spp. or *Bacillus* spp. (120,172,224,225). Washing hands with non-antimicrobial or antimicrobial soap and water may help to physically remove spores from the surface of contaminated hands. HCWs should be encouraged to wear gloves when caring for patients with *C. difficile*-associated diarrhea (226). After gloves are removed, hands should be washed with a non-antimicrobial or an antimicrobial soap and water or disinfected with an alcohol-based hand rub. During outbreaks of *C. difficile*-related infections, washing hands with a non-antimicrobial or antimicrobial soap and water after removing gloves is prudent. HCWs with suspected or documented exposure to *B. anthracis*-contaminated items also should be encouraged to wash their hands with a non-antimicrobial or antimicrobial soap and water.

Reduced Susceptibility of Bacteria to Antiseptics

Reduced susceptibility of bacteria to antiseptic agents can either be an intrinsic characteristic of a species or can be an acquired trait (227). Several reports have described strains of bacteria that appear to have acquired reduced susceptibility (when defined by MICs established in vitro) to certain antiseptics (e.g., chlorhexidine, quaternary ammonium compounds, and triclosan) (227–230). However, because the antiseptic concentrations that are actually used by HCWs are often substantially higher than the MICs of strains with reduced antiseptic susceptibility, the clinical relevance of the in vitro findings is questionable. For example, certain strains of MRSA have chlorhexidine and quaternary ammonium compound MICs that are several-fold higher than methicillin-susceptible strains, and certain strains of *S. aureus* have elevated MICs to triclosan (227,228). However, such strains were readily inhibited by the concentrations of these antiseptics that are actually used by practicing HCWs (227,228). The description of a triclosan-resistant bacterial enzyme has raised the question of whether resistance to this agent may develop more readily than to other antiseptic agents (218). In addition, exposing *Pseudomonas* strains containing the MexAB-OprM efflux system to triclosan may select for mutants that are resistant to multiple antibiotics, including fluoroquinolones (230). Further studies are needed to determine whether reduced susceptibility to antiseptic agents is of epidemiologic

significance and whether resistance to antiseptics has any influence on the prevalence of antibiotic-resistant strains (227).

Surgical Hand Antisepsis

Since the late 1800s, when Lister promoted the application of carbolic acid to the hands of surgeons before procedures, preoperative cleansing of hands and forearms with an antiseptic agent has been an accepted practice (231). Although no randomized, controlled trials have been conducted to indicate that surgical-site infection rates are substantially lower when preoperative scrubbing is performed with an antiseptic agent rather than a non-antimicrobial soap, certain other factors provide a strong rationale for this practice. Bacteria on the hands of surgeons can cause wound infections if introduced into the operative field during surgery (232); rapid multiplication of bacteria occurs under surgical gloves if hands are washed with a non-antimicrobial soap. However, bacterial growth is slowed after preoperative scrubbing with an antiseptic agent (14,233). Reducing resident skin flora on the hands of the surgical team for the duration of a procedure reduces the risk of bacteria being released into the surgical field if gloves become punctured or torn during surgery (1,156,169). Finally, at least one outbreak of surgical-site infections occurred when surgeons who normally used an antiseptic surgical scrub preparation began using a non-antimicrobial product (234).

Antiseptic preparations intended for use as surgical hand scrubs are evaluated for their ability to reduce the number of bacteria released from hands at different times, including 1) immediately after scrubbing, 2) after wearing surgical gloves for 6 hours (i.e., persistent activity), and 3) after multiple applications over 5 days (i.e., cumulative activity). Immediate and persistent activity are considered the most important in determining the efficacy of the product. U.S. guidelines recommend that agents used for surgical hand scrubs should substantially reduce microorganisms on intact skin, contain a nonirritating antimicrobial preparation, have broad-spectrum activity, and be fast-acting and persistent (19,235).

Studies have demonstrated that formulations containing 60%–95% alcohol alone or 50%–95% when combined with limited amounts of a quaternary ammonium compound, hexachlorophene, or chlorhexidine gluconate, lower bacterial counts on the skin immediately postscrub more effectively than do other agents (Table 4). The next most active agents (in order of decreasing activity) are chlorhexidine gluconate, iodophors, triclosan, and plain soap (104,119,186,188,203,204,206,208,236). Because studies of PCMX as a surgical scrub have yielded contradictory results, further studies are needed to establish how the efficacy of this compound compares with the other agents (176,185,186).

Although alcohols are not considered to have persistent antimicrobial activity, bacteria appear to reproduce slowly on the hands after a surgical scrub with alcohol, and bacterial counts on hands after wearing gloves for 1–3 hours seldom exceed baseline (i.e., prescrub) values (1). However, a recent study demonstrated that a formulation containing 61% ethanol alone did not achieve adequate persistent activity at 6 hours postscrub (237). Alcohol-based preparations containing 0.5% or 1% chlorhexidine gluconate have persistent activity that, in certain studies, has equaled or exceeded that of chlorhexidine gluconate-containing detergents (1,118,135,237).*

Persistent antimicrobial activity of detergent-based surgical scrub formulations is greatest for those containing 2% or 4% chlorhexidine gluconate, followed by hexachlorophene, triclosan, and iodophors (1,102,113–115,159,189,203,204,206–208,236). Because hexachlorophene is absorbed into the blood after repeated use, it is seldom used as a surgical scrub.

Surgical staff have been traditionally required to scrub their hands for 10 minutes preoperatively, which frequently leads to skin damage. Several studies have demonstrated that scrubbing for 5 minutes reduces bacterial counts as effectively as a 10-minute scrub (117,238,239). In other studies, scrubbing for 2 or 3 minutes reduced bacterial counts to acceptable levels (156,205,207,240,241).

Studies have indicated that a two-stage surgical scrub using an antiseptic detergent, followed by application of an alcohol-containing preparation, is effective. For example, an initial 1- or 2-minute scrub with 4% chlorhexidine gluconate or povidone-iodine followed by application of an alcohol-based product has been as effective as a 5-minute scrub with an antiseptic detergent (114,242).

Surgical hand-antiseptic protocols have required personnel to scrub with a brush. But this practice can damage the skin of personnel and result in increased shedding of bacteria from the hands (95,243). Scrubbing with a disposable sponge or combination sponge-brush has reduced bacterial counts on the hands as effectively as scrubbing with a brush (244–246). However, several studies indicate that neither a brush nor a

sponge is necessary to reduce bacterial counts on the hands of surgical personnel to acceptable levels, especially when alcohol-based products are used (102,117,159,165,233,237,247,248). Several of these studies performed cultures immediately or at 45–60 minutes postscrub (102,117,233,247,248), whereas in other studies, cultures were obtained 3 and 6 hours postscrub (159,237). For example, a recent laboratory-based study using volunteers demonstrated that brushless application of a preparation containing 1% chlorhexidine gluconate plus 61% ethanol yielded lower bacterial counts on the hands of participants than using a sponge/brush to apply a 4% chlorhexidine-containing detergent preparation (237).

Relative Efficacy of Plain Soap, Antiseptic Soap/Detergent, and Alcohols

Comparing studies related to the in vivo efficacy of plain soap, antimicrobial soaps, and alcohol-based hand rubs is problematic, because certain studies express efficacy as the percentage reduction in bacterial counts achieved, whereas others give log₁₀ reductions in counts achieved. However, summarizing the relative efficacy of agents tested in each study can provide an overview of the in vivo activity of various formulations intended for handwashing, hygienic handwash, antiseptic hand rub, or surgical hand antiseptics (Tables 2–4).

Irritant Contact Dermatitis Resulting from Hand-Hygiene Measures

Frequency and Pathophysiology of Irritant Contact Dermatitis

In certain surveys, approximately 25% of nurses report symptoms or signs of dermatitis involving their hands, and as many as 85% give a history of having skin problems (249). Frequent and repeated use of hand-hygiene products, particularly soaps and other detergents, is a primary cause of chronic irritant contact dermatitis among HCWs (250). The potential of detergents to cause skin irritation can vary considerably and can be ameliorated by the addition of emollients and humectants. Irritation associated with antimicrobial soaps may be caused by the antimicrobial agent or by other ingredients of the formulation. Affected persons often complain of a feeling of dryness or burning; skin that feels “rough;” and erythema, scaling, or fissures. Detergents damage the skin by causing denaturation of stratum corneum proteins, changes in intercellular lipids (either depletion or reorganization of lipid moieties), decreased corneocyte cohesion, and decreased stratum corneum water-binding capacity (250,251). Damage

* In a recent randomized clinical trial, surgical site infection rates were monitored among patients who were operated on by surgical personnel who cleaned their hands preoperatively either by performing a traditional 5-minute surgical hand scrub using 4% povidone-iodine or 4% antiseptic antimicrobial soap, or by washing their hands for 1 minute with a non-antimicrobial soap followed by a 5-minute hand-rubbing technique using an alcohol-based hand rinse containing 0.2% mectronium etilsulfate. The incidence of surgical site infections was virtually identical in the two groups of patients. (Source: Parienti JJ, Thibon P, Heller R, et al. for Members of the Antiseptics Chirurgicales des Mains Study Group. Hand-rubbing with an aqueous alcoholic solution vs traditional surgical hand-scrubbing and 30-day surgical site infection rates: a randomized equivalence study. JAMA 2002;288:722–7).

to the skin also changes skin flora, resulting in more frequent colonization by staphylococci and gram-negative bacilli (17,90). Although alcohols are among the safest antiseptics available, they can cause dryness and irritation of the skin (1,252). Ethanol is usually less irritating than n-propanol or isopropanol (252).

Irritant contact dermatitis is more commonly reported with iodophors (92). Other antiseptic agents that can cause irritant contact dermatitis (in order of decreasing frequency) include chlorhexidine, PCMX, triclosan, and alcohol-based products. Skin that is damaged by repeated exposure to detergents may be more susceptible to irritation by alcohol-based preparations (253). The irritancy potential of commercially prepared hand-hygiene products, which is often determined by measuring transepidermal water loss, may be available from the manufacturer. Other factors that can contribute to dermatitis associated with frequent handwashing include using hot water for handwashing, low relative humidity (most common in winter months), failure to use supplementary hand lotion or cream, and the quality of paper towels (254,255). Shear forces associated with wearing or removing gloves and allergy to latex proteins may also contribute to dermatitis of the hands of HCWs.

Allergic Contact Dermatitis Associated with Hand-Hygiene Products

Allergic reactions to products applied to the skin (i.e., contact allergies) may present as delayed type reactions (i.e., allergic contact dermatitis) or less commonly as immediate reactions (i.e., contact urticaria). The most common causes of contact allergies are fragrances and preservatives; emulsifiers are less common causes (256–259). Liquid soaps, hand lotions or creams, and “udder ointments” may contain ingredients that cause contact allergies among HCWs (257,258).

Allergic reactions to antiseptic agents, including quaternary ammonium compounds, iodine or iodophors, chlorhexidine, triclosan, PCMX, and alcohols have been reported (118,167,172,256,260–265). Allergic contact dermatitis associated with alcohol-based hand rubs is uncommon. Surveillance at a large hospital in Switzerland, where a commercial alcohol hand rub has been used for >10 years, failed to identify a single case of documented allergy to the product (169). In late 2001, a Freedom of Information Request for data in the FDA’s Adverse Event Reporting System regarding adverse reactions to popular alcohol hand rubs in the United States yielded only one reported case of an erythematous rash reaction attributed to such a product (John M. Boyce, M.D., Hospital of St. Raphael, New Haven, Connecticut, personal communication, 2001). However, with increasing use of such products by HCWs, true allergic reactions to such products likely will be encountered.

Allergic reactions to alcohol-based products may represent true allergy to alcohol, allergy to an impurity or aldehyde metabolite, or allergy to another constituent of the product (167). Allergic contact dermatitis or immediate contact urticarial reactions may be caused by ethanol or isopropanol (167). Allergic reactions can be caused by compounds that may be present as inactive ingredients in alcohol-based hand rubs, including fragrances, benzyl alcohol, stearyl or isostearyl alcohol, phenoxyethanol, myristyl alcohol, propylene glycol, parabens, and benzalkonium chloride (167,256,266–270).

Proposed Methods for Reducing Adverse Effects of Agents

Potential strategies for minimizing hand-hygiene-related irritant contact dermatitis among HCWs include reducing the frequency of exposure to irritating agents (particularly anionic detergents), replacing products with high irritation potential with preparations that cause less damage to the skin, educating personnel regarding the risks of irritant contact dermatitis, and providing caregivers with moisturizing skin-care products or barrier creams (96,98,251,271–273). Reducing the frequency of exposure of HCWs to hand-hygiene products would prove difficult and is not desirable because of the low levels of adherence to hand-hygiene policies in the majority of institutions. Although hospitals have provided personnel with non-antimicrobial soaps in hopes of minimizing dermatitis, frequent use of such products may cause greater skin damage, dryness, and irritation than antiseptic preparations (92,96,98). One strategy for reducing the exposure of personnel to irritating soaps and detergents is to promote the use of alcohol-based hand rubs containing various emollients. Several recent prospective, randomized trials have demonstrated that alcohol-based hand rubs containing emollients were better tolerated by HCWs than washing hands with non-antimicrobial soaps or antimicrobial soaps (96,98,166). Routinely washing hands with soap and water immediately after using an alcohol hand rub may lead to dermatitis. Therefore, personnel should be reminded that it is neither necessary nor recommended to routinely wash hands after each application of an alcohol hand rub.

Hand lotions and creams often contain humectants and various fats and oils that can increase skin hydration and replace altered or depleted skin lipids that contribute to the barrier function of normal skin (251,271). Several controlled trials have demonstrated that regular use (e.g., twice a day) of such products can help prevent and treat irritant contact dermatitis caused by hand-hygiene products (272,273). In one study, frequent and scheduled use of an oil-containing lotion improved skin condition, and thus led to a 50% increase in

handwashing frequency among HCWs (273). Reports from these studies emphasize the need to educate personnel regarding the value of regular, frequent use of hand-care products.

Recently, barrier creams have been marketed for the prevention of hand-hygiene-related irritant contact dermatitis. Such products are absorbed to the superficial layers of the epidermis and are designed to form a protective layer that is not removed by standard handwashing. Two recent randomized, controlled trials that evaluated the skin condition of caregivers demonstrated that barrier creams did not yield better results than did the control lotion or vehicle used (272,273). As a result, whether barrier creams are effective in preventing irritant contact dermatitis among HCWs remains unknown.

In addition to evaluating the efficacy and acceptability of hand-care products, product-selection committees should inquire about the potential deleterious effects that oil-containing products may have on the integrity of rubber gloves and on the efficacy of antiseptic agents used in the facility (8,236).

Factors To Consider When Selecting Hand-Hygiene Products

When evaluating hand-hygiene products for potential use in health-care facilities, administrators or product-selection committees must consider factors that can affect the overall efficacy of such products, including the relative efficacy of antiseptic agents against various pathogens (Appendix) and acceptance of hand-hygiene products by personnel (274,275). Soap products that are not well-accepted by HCWs can be a deterrent to frequent handwashing (276). Characteristics of a product (either soap or alcohol-based hand rub) that can affect acceptance by personnel include its smell, consistency (i.e., “feel”), and color (92,277,278). For soaps, ease of lathering also may affect user preference.

Because HCWs may wash their hands from a limited number of times per shift to as many as 30 times per shift, the tendency of products to cause skin irritation and dryness is a substantial factor that influences acceptance, and ultimate usage (61,98,274,275,277,279). For example, concern regarding the drying effects of alcohol was a primary cause of poor acceptance of alcohol-based hand-hygiene products in hospitals in the United States (5,143). However, several studies have demonstrated that alcohol-based hand rubs containing emollients are acceptable to HCWs (90,93,98,100,101,106,143,163,164,166). With alcohol-based products, the time required for drying may also affect user acceptance.

Studies indicate that the frequency of handwashing or antiseptic handwashing by personnel is affected by the accessibility of hand-hygiene facilities (280–283). In certain health-care

facilities, only one sink is available in rooms housing several patients, or sinks are located far away from the door of the room, which may discourage handwashing by personnel leaving the room. In intensive-care units, access to sinks may be blocked by bedside equipment (e.g., ventilators or intravenous infusion pumps). In contrast to sinks used for handwashing or antiseptic handwash, dispensers for alcohol-based hand rubs do not require plumbing and can be made available adjacent to each patient’s bed and at many other locations in patient-care areas. Pocket carriage of alcohol-based hand-rub solutions, combined with availability of bedside dispensers, has been associated with substantial improvement in adherence to hand-hygiene protocols (74,284). To avoid any confusion between soap and alcohol hand rubs, alcohol hand-rub dispensers should not be placed adjacent to sinks. HCWs should be informed that washing hands with soap and water after each use of an alcohol hand rub is not necessary and is not recommended, because it may lead to dermatitis. However, because personnel feel a “build-up” of emollients on their hands after repeated use of alcohol hand gels, washing hands with soap and water after 5–10 applications of a gel has been recommended by certain manufacturers.

Automated handwashing machines have not been demonstrated to improve the quality or frequency of handwashing (88,285). Although technologically advanced automated handwashing devices and monitoring systems have been developed recently, only a minimal number of studies have been published that demonstrate that use of such devices results in enduring improvements in hand-hygiene adherence among HCWs. Further evaluation of automated handwashing facilities and monitoring systems is warranted.

Dispenser systems provided by manufacturers or vendors also must be considered when evaluating hand-hygiene products. Dispensers may discourage use by HCWs when they 1) become blocked or partially blocked and do not deliver the product when accessed by personnel, and 2) do not deliver the product appropriately onto the hands. In one hospital where a viscous alcohol-based hand rinse was available, only 65% of functioning dispensers delivered product onto the caregivers’ hands with one press of the dispenser lever, and 9% of dispensers were totally occluded (286). In addition, the volume delivered was often suboptimal, and the product was sometimes squirted onto the wall instead of the caregiver’s hand.

Only limited information is available regarding the cost of hand-hygiene products used in health-care facilities (165,287). These costs were evaluated in patient-care areas at a 450-bed community teaching hospital (287); the hospital spent \$22,000 (\$0.72 per patient-day) on 2% chlorhexidine-containing preparations, plain soap, and an alcohol hand rinse. (287) When

hand-hygiene supplies for clinics and nonpatient care areas were included, the total annual budget for soaps and hand antiseptic agents was \$30,000 (approximately \$1 per patient-day). Annual hand-hygiene product budgets at other institutions vary considerably because of differences in usage patterns and varying product prices. One researcher (287) determined that if non-antimicrobial liquid soap were assigned an arbitrary relative cost of 1.0, the cost per liter would be 1.7 times as much for 2% chlorhexidine gluconate detergent, 1.6–2.0 times higher for alcohol-based hand-rub products, and 4.5 times higher for an alcohol-based foam product. A recent cost comparison of surgical scrubbing with an antimicrobial soap versus brushless scrubbing with an alcohol-based hand rub revealed that costs and time required for preoperative scrubbing were less with the alcohol-based product (165). In a trial conducted in two critical-care units, the cost of using an alcohol hand rub was half as much as using an antimicrobial soap for handwashing (\$0.025 versus \$0.05 per application, respectively) (166).

To put expenditures for hand-hygiene products into perspective, health-care facilities should consider comparing their budget for hand-hygiene products to estimated excess hospital costs resulting from health-care-associated infections. The excess hospital costs associated with only four or five health-care-associated infections of average severity may equal the entire annual budget for hand-hygiene products used in inpatient-care areas. Just one severe surgical site infection, lower respiratory tract infection, or bloodstream infection may cost the hospital more than the entire annual budget for antiseptic agents used for hand hygiene (287). Two studies provided certain quantitative estimates of the benefit of hand-hygiene-promotion programs (72,74). One study demonstrated a cost saving of approximately \$17,000 resulting from reduced use of vancomycin after the observed decrease in MRSA incidence in a 7-month period (72). In another study that examined both direct costs associated with the hand-hygiene promotion program (increased use of hand-rub solution and poster production) and indirect costs associated with health-care-personnel time (74), costs of the program were an estimated \$57,000 or less per year (an average of \$1.42 per patient admitted). Supplementary costs associated with the increased use of alcohol-based hand-rub solution averaged \$6.07 per 100 patient-days. Based on conservative estimates of \$2,100 saved per infection averted and on the assumption that only 25% of the observed reduction in the infection rate was associated with improved hand-hygiene practice, the program was substantially cost-effective. Thus, hospital administrators must consider that by purchasing more effective or more acceptable hand-hygiene products to improve hand-hygiene practices, they

will avoid the occurrence of nosocomial infections; preventing only a limited number of additional health-care-associated infections per year will lead to savings that will exceed any incremental costs of improved hand-hygiene products.

Hand-Hygiene Practices Among HCWs

In observational studies conducted in hospitals, HCWs washed their hands an average of five times per shift to as many as 30 times per shift (Table 6) (17,61,90,98,274,288); certain nurses washed their hands ≤ 100 times per shift (90). Hospitalwide surveillance of hand hygiene reveals that the average number of handwashing opportunities varies markedly between hospital wards. For example, nurses in pediatric wards had an average of eight opportunities for hand hygiene per hour of patient care compared with an average of 20 for nurses in intensive-care units (11). The duration of handwashing or hygienic handwash episodes by HCWs has averaged 6.6–24.0 seconds in observational studies (Table 7) (17,52,59,84–87,89,249,279). In addition to washing their

TABLE 6. Handwashing frequency among health-care workers

Ref. no.	Year	Avg. no./time period	Range	Avg. no./hr
(61)	1988	5/8 hour	N.S.	
(89)	1984	5–10/shift	N.S.	
(96)	2000	10/shift	N.S.	
(273)	2000	12–18/day	2–60	
(98)	2000	13–15/8 hours	5–27	1.6–1.8/hr
(90)	1977	20–42/8 hours	10–100	
(391)	2000	21/12 hours	N.S.	
(272)	2000	22/day	0–70	
(88)	1991			1.7–2.1/hr
(17)	1998			2.1/hr
(279)	1978			3/hr
(303)	1994			3.3/hr

Note: N.S. = Not Stated.

TABLE 7. Average duration of handwashing by health-care workers

Ref. no.	Year	Mean/median time
(392)	1997	4.7–5.3 seconds
(303)	1994	6.6 seconds
(52)	1974	8–9.3 seconds
(85)	1984	8.6 seconds
(86)	1994	<9 seconds
(87)	1994	9.5 seconds
(88)	1991	<10 seconds
(294)	1990	10 seconds
(89)	1984	11.6 seconds
(300)	1992	12.5 seconds
(59)	1988	15.6–24.4 seconds
(17)	1998	20.6 seconds
(279)	1978	21 seconds
(293)	1989	24 seconds

hands for limited time periods, personnel often fail to cover all surfaces of their hands and fingers (288).

Adherence of HCWs to Recommended Hand-Hygiene Practices

Observational Studies of Hand-Hygiene Adherence. Adherence of HCWs to recommended hand-hygiene procedures has been poor, with mean baseline rates of 5%–81% (overall average: 40%) (Table 8) (71,74,86,87,276,280,281,283,285,289–313). The methods used for defining adherence (or non-adherence) and those used for conducting observations vary considerably among studies, and reports do not provide

detailed information concerning the methods and criteria used. The majority of studies were conducted with hand-hygiene adherence as the major outcome measure, whereas a limited number measured adherence as part of a broader investigation. Several investigators reported improved adherence after implementing various interventions, but the majority of studies had short follow-up periods and did not confirm whether behavioral improvements were long-lasting. Other studies established that sustained improvements in handwashing behavior occurred during a long-term program to improve adherence to hand-hygiene policies (74,75).

TABLE 8. Hand-hygiene adherence by health-care workers (1981–2000)

Ref. no.	Year	Setting	Before/after	Adherence baseline	Adherence after intervention	Intervention
(280)	1981	ICU	A	16%	30%	More convenient sink locations
(289)	1981	ICU	A	41%	—	
		ICU	A	28%	—	
(290)	1983	All wards	A	45%	—	
(281)	1986	SICU	A	51%	—	
		MICU	A	76%	—	
(276)	1986	ICU	A	63%	92%	Performance feedback
(291)	1987	PICU	A	31%	30%	Wearing overgown
(292)	1989	MICU	B/A	14%/28%*	73%/81%	Feedback, policy reviews, memo, and posters
		MICU	B/A	26%/23%	38%/60%	
(293)	1989	NICU	A/B	75%/50%	—	
(294)	1990	ICU	A	32%	45%	Alcohol rub introduced
(295)	1990	ICU	A	81%	92%	Inservices first, then group feedback
(296)	1990	ICU	B/A	22%	30%	
(297)	1991	SICU	A	51%	—	
(298)	1991	Pedi OPDs	B	49%	49%	Signs, feedback, and verbal reminders to physicians
(299)	1991	Nursery and NICU	B/A†	28%	63%	Feedback, dissemination of literature, and results of environmental cultures
(300)	1992	NICU/others	A	29%	—	
(71)	1992	ICU	N.S.	40%	—	
(301)	1993	ICUs	A	40%	—	
(87)	1994	Emergency Room	A	32%	—	
(86)	1994	All wards	A	32%	—	
(285)	1994	SICU	A	22%	38%	Automated handwashing machines available
(302)	1994	NICU	A	62%	60%	No gowning required
(303)	1994	ICU Wards	AA	30%/29%	—	
(304)	1995	ICU Oncol Ward	A	56%	—	
(305)	1995	ICU	N.S.	5%	63%	Lectures, feedback, and demonstrations
(306)	1996	PICU	B/A	12%/11%	68%/65%	Overt observation, followed by feedback
(307)	1996	MICU	A	41%	58%	Routine wearing of gowns and gloves
(308)	1996	Emergency Dept	A	54%	64%	Signs/distributed review paper
(309)	1998	All wards	A	30%	—	
(310)	1998	Pediatric wards	B/A	52%/49%	74%/69%	Feedback, movies, posters, and brochures
(311)	1999	MICU	B/A	12%/55%	—	
(74)	2000	All wards	B/A	48%	67%	Posters, feedback, administrative support, and alcohol rub
(312)	2000	MICU	A	42%	61%	Alcohol hand rub made available
(283)	2000	MICU	B/A	10%/22%	23%/48%	Education, feedback, and alcohol gel made available
		CTICU	B/A	4%/13%	7%/14%	
(313)	2000	Medical wards	A	60%	52%	Education, reminders, and alcohol gel made available

Note: ICU = intensive care unit, SICU = surgical ICU, MICU = medical ICU, PICU = pediatric ICU, NICU = neonatal ICU, Emerg = emergency, Oncol = oncology, CTICU = cardiothoracic ICU, and N.S. = not stated.

* Percentage compliance before/after patient contact.

† After contact with inanimate objects.

Factors Affecting Adherence. Factors that may influence hand hygiene include those identified in epidemiologic studies and factors reported by HCWs as being reasons for lack of adherence to hand-hygiene recommendations. Risk factors for poor adherence to hand hygiene have been determined objectively in several observational studies or interventions to improve adherence (11,12,274,292,295,314–317). Among these, being a physician or a nursing assistant, rather than a nurse, was consistently associated with reduced adherence (Box 1).

In the largest hospitalwide survey of hand-hygiene practices among HCWs (11), predictors of poor adherence to recommended hand-hygiene measures were identified. Predictor variables included professional category, hospital ward, time of day/week, and type and intensity of patient care, defined as the number of opportunities for hand hygiene per hour of patient care. In 2,834 observed opportunities for hand hygiene, average adherence was 48%. In multivariate analysis, nonadherence was lowest among nurses and during weekends

BOX 1. Factors influencing adherence to hand-hygiene practices*

Observed risk factors for poor adherence to recommended hand-hygiene practices

- Physician status (rather than a nurse)
- Nursing assistant status (rather than a nurse)
- Male sex
- Working in an intensive-care unit
- Working during the week (versus the weekend)
- Wearing gowns/gloves
- Automated sink
- Activities with high risk of cross-transmission
- High number of opportunities for hand hygiene per hour of patient care

Self-reported factors for poor adherence with hand hygiene

- Handwashing agents cause irritation and dryness
- Sinks are inconveniently located/shortage of sinks
- Lack of soap and paper towels
- Often too busy/insufficient time
- Understaffing/overcrowding
- Patient needs take priority
- Hand hygiene interferes with health-care worker relationships with patients
- Low risk of acquiring infection from patients
- Wearing of gloves/beliefs that glove use obviates the need for hand hygiene
- Lack of knowledge of guidelines/protocols
- Not thinking about it/forgetfulness
- No role model from colleagues or superiors
- Skepticism regarding the value of hand hygiene
- Disagreement with the recommendations
- Lack of scientific information of definitive impact of improved hand hygiene on health-care-associated infection rates

Additional perceived barriers to appropriate hand hygiene

- Lack of active participation in hand-hygiene promotion at individual or institutional level
- Lack of role model for hand hygiene
- Lack of institutional priority for hand hygiene
- Lack of administrative sanction of noncompliers/rewarding compliers
- Lack of institutional safety climate

* Source: Adapted from Pittet D. Improving compliance with hand hygiene in hospitals. *Infect Control Hosp Epidemiol* 2000;21:381–6.

(Odds Ratio [OR]: 0.6; 95% confidence interval [CI] = 0.4–0.8). Nonadherence was higher in intensive-care units compared with internal medicine wards (OR: 2.0; 95% CI = 1.3–3.1), during procedures that carried a high risk of bacterial contamination (OR: 1.8; 95% CI = 1.4–2.4), and when intensity of patient care was high (21–40 handwashing opportunities — OR: 1.3; 95% CI = 1.0–1.7; 41–60 opportunities — OR: 2.1; 95% CI = 1.5–2.9; >60 opportunities — OR: 2.1; 95% CI = 1.3–3.5). The higher the demand for hand hygiene, the lower the adherence; on average, adherence decreased by 5% (\pm 2%) for each increase of 10 opportunities per hour when the intensity of patient care exceeded 10 opportunities per hour. Similarly, the lowest adherence rate (36%) was found in intensive-care units, where indications for hand hygiene were typically more frequent (on average, 20 opportunities per patient-hour). The highest adherence rate (59%) was observed in pediatrics wards, where the average intensity of patient care was lower than in other hospital areas (an average of eight opportunities per patient-hour). The results of this study indicate that full adherence to previous guidelines may be unrealistic, and that facilitated access to hand hygiene could help improve adherence (11,12,318).

Perceived barriers to adherence with hand-hygiene practice recommendations include skin irritation caused by hand-hygiene agents, inaccessible hand-hygiene supplies, interference with HCW-patient relationships, priority of care (i.e., the patients' needs are given priority over hand hygiene), wearing of gloves, forgetfulness, lack of knowledge of the guidelines, insufficient time for hand hygiene, high workload and understaffing, and the lack of scientific information indicating a definitive impact of improved hand hygiene on health-care-associated infection rates (11,274,292,295,315–317). Certain perceived barriers to adherence with hand-hygiene guidelines have been assessed or quantified in observational studies (12,274,292,295,314–317) (Box 1).

Skin irritation by hand-hygiene agents constitutes a substantial barrier to appropriate adherence (319). Because soaps and detergents can damage skin when applied on a regular basis, HCWs must be better informed regarding the possible adverse effects associated with hand-hygiene agents. Lack of knowledge and education regarding this subject is a barrier to motivation. In several studies, alcohol-based hand rubs containing emollients (either isopropanol, ethanol, or n-propanol in 60%–90% vol/vol) were less irritating to the skin than the soaps or detergents tested. In addition, the alcohol-based products containing emollients that were tested were at least as tolerable and efficacious as the detergents tested. Also, studies demonstrate that several hand lotions have reduced skin scaling and cracking, which may reduce microbial shedding from the hands (67,272,273).

Easy access to hand-hygiene supplies, whether sink, soap, medicated detergent, or alcohol-based hand-rub solution, is essential for optimal adherence to hand-hygiene recommendations. The time required for nurses to leave a patient's bedside, go to a sink, and wash and dry their hands before attending the next patient is a deterrent to frequent handwashing or hand antisepsis (11,318). Engineering controls could facilitate adherence, but careful monitoring of hand-hygiene behavior should be conducted to exclude the possible negative effect of newly introduced handwashing devices (88).

The impact of wearing gloves on adherence to hand-hygiene policies has not been definitively established, because published studies have yielded contradictory results (87,290,301,320). Hand hygiene is required regardless of whether gloves are used or changed. Failure to remove gloves after patient contact or between "dirty" and "clean" body-site care on the same patient must be regarded as nonadherence to hand-hygiene recommendations (11). In a study in which experimental conditions approximated those occurring in clinical practice (321), washing and reusing gloves between patient contacts resulted in observed bacterial counts of 0–4.7 log on the hands after glove removal. Therefore, this practice should be discouraged; handwashing or disinfection should be performed after glove removal.

Lack of 1) knowledge of guidelines for hand hygiene, 2) recognition of hand-hygiene opportunities during patient care, and 3) awareness of the risk of cross-transmission of pathogens are barriers to good hand-hygiene practices. Furthermore, certain HCWs believe they have washed their hands when necessary, even when observations indicate they have not (89,92,295,296,322).

Perceived barriers to hand-hygiene behavior are linked not only to the institution, but also to HCWs' colleagues. Therefore, both institutional and small-group dynamics need to be considered when implementing a system change to secure an improvement in HCWs' hand-hygiene practice.

Possible Targets for Hand-Hygiene Promotion

Targets for the promotion of hand hygiene are derived from studies assessing risk factors for nonadherence, reported reasons for the lack of adherence to recommendations, and additional factors perceived as being important to facilitate appropriate HCW behavior. Although certain factors cannot be modified (Box 1), others can be changed.

One factor that must be addressed is the time required for HCWs to clean their hands. The time required for traditional handwashing may render full adherence to previous guidelines unrealistic (11,12,318) and more rapid access to hand-hygiene materials could help improve adherence. One study conducted in an intensive-care unit demonstrated that it took

nurses an average of 62 seconds to leave a patient's bedside, walk to a sink, wash their hands, and return to patient care (318). In contrast, an estimated one fourth as much time is required when using alcohol-based hand rub placed at each patient's bedside. Providing easy access to hand-hygiene materials is mandatory for appropriate hand-hygiene behavior and is achievable in the majority of health-care facilities (323). In particular, in high-demand situations (e.g., the majority of critical-care units), under hectic working conditions, and at times of overcrowding or understaffing, HCWs may be more likely to use an alcohol-based hand rub than to wash their hands (323). Further, using alcohol-based hand rubs may be a better option than traditional handwashing with plain soap and water or antiseptic handwash, because they not only require less time (166,318) but act faster (1) and irritate hands less often (1,67,96,98,166). They also were used in the only program that reported a sustained improvement in hand-hygiene adherence associated with decreased infection rates (74). However, making an alcohol-based hand rub available to personnel without providing ongoing educational and motivational activities may not result in long-lasting improvement in hand-hygiene practices (313). Because increased use of hand-hygiene agents might be associated with skin dryness, the availability of free skin-care lotion is recommended.

Education is a cornerstone for improvement with hand-hygiene practices. Topics that must be addressed by educational programs include the lack of 1) scientific information for the definitive impact of improved hand hygiene on health-care-associated infection and resistant organism transmission rates; 2) awareness of guidelines for hand hygiene and insufficient knowledge concerning indications for hand hygiene during daily patient care; 3) knowledge concerning the low average adherence rate to hand hygiene by the majority of HCWs; and 4) knowledge concerning the appropriateness, efficacy, and understanding of the use of hand-hygiene and skin-care-protection agents.

HCWs necessarily evolve within a group that functions within an institution. Possible targets for improvement in hand-hygiene behavior not only include factors linked to individual HCWs, but also those related to the group(s) and the institution as a whole (317,323). Examples of possible targets for hand-hygiene promotion at the group level include education and performance feedback on hand-hygiene adherence; efforts to prevent high workload, downsizing, and understaffing; and encouragement and provision of role models from key members in the work unit. At the institutional level, targets for improvement include 1) written guidelines, hand-hygiene agents, skin-care promotions and agents, or hand-hygiene facilities; 2) culture or tradition of adherence; and 3)

administrative leadership, sanction, support, and rewards. Several studies, conducted in various types of institutions, reported modest and even low levels of adherence to recommended hand-hygiene practices, indicating that such adherence varied by hospital ward and by type of HCW. These results indicate educational sessions may need to be designed specifically for certain types of personnel (11,289,290,294,317,323).

Lessons Learned from Behavioral Theories

In 1998, the prevailing behavioral theories and their applications with regard to the health professions were reviewed by researchers in an attempt to better understand how to target more successful interventions (317). The researchers proposed a hypothetical framework to enhance hand-hygiene practices and stressed the importance of considering the complexity of individual and institutional factors when designing behavioral interventions.

Although behavioral theories and secondary interventions have primarily targeted individual workers, this practice might be insufficient to produce sustained change (317,324,325). Interventions aimed at improving hand-hygiene practices must account for different levels of behavior interaction (12,317,326). Thus, the interdependence of individual factors, environmental constraints, and the institutional climate must be taken into account in the strategic planning and development of hand-hygiene campaigns. Interventions to promote hand hygiene in hospitals should consider variables at all these levels. Various factors involved in hand-hygiene behavior include intention, attitude towards the behavior, perceived social norm, perceived behavioral control, perceived risk for infection, hand-hygiene practices, perceived role model, perceived knowledge, and motivation (317). The factors necessary for change include 1) dissatisfaction with the current situation, 2) perception of alternatives, and 3) recognition, both at the individual and institutional level, of the ability and potential to change. Although the latter implies education and motivation, the former two necessitate a system change.

Among the reported reasons for poor adherence with hand-hygiene recommendations (Box 1), certain ones are clearly associated with the institution or system (e.g., lack of institutional priority for hand hygiene, administrative sanctions, and a safety climate). Although all of these reasons would require a system change in the majority of institutions, the third requires management commitment, visible safety programs, an acceptable level of work stress, a tolerant and supportive attitude toward reported problems, and belief in the efficacy

of preventive strategies (12,317,325,327). Most importantly, an improvement in infection-control practices requires 1) questioning basic beliefs, 2) continuous assessment of the group (or individual) stage of behavioral change, 3) intervention(s) with an appropriate process of change, and 4) supporting individual and group creativity (317). Because of the complexity of the process of change, single interventions often fail. Thus, a multimodal, multidisciplinary strategy is likely necessary (74,75,317,323,326).

Methods Used To Promote Improved Hand Hygiene

Hand-hygiene promotion has been challenging for >150 years. In-service education, information leaflets, workshops and lectures, automated dispensers, and performance feedback on hand-hygiene adherence rates have been associated with transient improvement (291,294–296,306,314).

Several strategies for promotion of hand hygiene in hospitals have been published (Table 9). These strategies require education, motivation, or system change. Certain strategies are based on epidemiologic evidence, others on the authors' and other investigators' experience and review of current knowledge. Some strategies may be unnecessary in certain circumstances, but may be helpful in others. In particular, changing the hand-hygiene agent could be beneficial in institutions or hospital wards with a high workload and a high demand for hand hygiene when alcohol-based hand rubs are not available (11,73,78,328). However, a change in the recommended hand-hygiene agent could be deleterious if introduced during winter, at a time of higher hand-skin irritability, and if not accompanied by the provision of skin-care products (e.g., pro-

TECTIVE creams and lotions). Additional specific elements should be considered for inclusion in educational and motivational programs (Box 2).

Several strategies that could potentially be associated with successful promotion of hand hygiene require a system change (Box 1). Hand-hygiene adherence and promotion involve factors at both the individual and system level. Enhancing individual and institutional attitudes regarding the feasibility of making changes (self-efficacy), obtaining active participation of personnel at both levels, and promoting an institutional safety climate represent challenges that exceed the current perception of the role of infection-control professionals.

Whether increased education, individual reinforcement technique, appropriate rewarding, administrative sanction, enhanced self-participation, active involvement of a larger number of organizational leaders, enhanced perception of health threat, self-efficacy, and perceived social pressure (12,317,329,330), or combinations of these factors can improve HCWs' adherence with hand hygiene needs further investigation. Ultimately, adherence to recommended hand-hygiene practices should become part of a culture of patient safety where a set of interdependent quality elements interact to achieve a shared objective (331).

On the basis of both these hypothetical considerations and successful, actual experiences in certain institutions, strategies to improve adherence to hand-hygiene practices should be both multimodal and multidisciplinary. However, strategies must be further researched before they are implemented.

TABLE 9. Strategies for successful promotion of hand hygiene in hospitals

Strategy	Tool for change*	Selected references†
Education	E (M, S)	(74,295,306,326,393)
Routine observation and feedback	S (E, M)	(74,294,306,326,393)
Engineering control		
Make hand hygiene possible, easy, and convenient	S	(74,281,326,393)
Make alcohol-based hand rub available	S	(74)
(at least in high-demand situations)	S	(74,283,312)
Patient education	S (M)	(283,394)
Reminders in the workplace	S	(74,395)
Administrative sanction/rewarding	S	(12,317)
Change in hand-hygiene agent	S (E)	(11,67,71,283,312)
Promote/facilitate skin care for health-care-workers' hands	S (E)	(67,74,274,275)
Obtain active participation at individual and institutional level	E, M, S	(74,75,317)
Improve institutional safety climate	S (M)	(74,75,317)
Enhance individual and institutional self-efficacy	S (E, M)	(74,75,317)
Avoid overcrowding, understaffing, and excessive workload	S	(11,74,78,297,396)
Combine several of above strategies	E, M, S	(74,75,295,306,317,326)

* The dynamic of behavioral change is complex and involves a combination of education (E), motivation (M), and system change (S).

† Only selected references have been listed; readers should refer to more extensive reviews for exhaustive reference lists (1,8,317,323,397).

BOX 2. Elements of health-care worker educational and motivational programs**Rationale for hand hygiene**

- Potential risks of transmission of microorganisms to patients
- Potential risks of health-care worker colonization or infection caused by organisms acquired from the patient
- Morbidity, mortality, and costs associated with health-care–associated infections

Indications for hand hygiene

- Contact with a patient's intact skin (e.g., taking a pulse or blood pressure, performing physical examinations, lifting the patient in bed) (25,26,45,48,51,53)
- Contact with environmental surfaces in the immediate vicinity of patients (46,51,53,54)
- After glove removal (50,58,71)

Techniques for hand hygiene

- Amount of hand-hygiene solution
- Duration of hand-hygiene procedure
- Selection of hand-hygiene agents
 - Alcohol-based hand rubs are the most efficacious agents for reducing the number of bacteria on the hands of personnel. Antiseptic soaps and detergents are the next most effective, and non-antimicrobial soaps are the least effective (1,398).
 - Soap and water are recommended for visibly soiled hands.
 - Alcohol-based hand rubs are recommended for routine decontamination of hands for all clinical indications (except when hands are visibly soiled) and as one of the options for surgical hand hygiene.

Methods to maintain hand skin health

- Lotions and creams can prevent or minimize skin dryness and irritation caused by irritant contact dermatitis
- Acceptable lotions or creams to use
- Recommended schedule for applying lotions or creams

Expectations of patient care managers/administrators

- Written statements regarding the value of, and support for, adherence to recommended hand-hygiene practices
- Role models demonstrating adherence to recommended hand hygiene practices (399)

Indications for, and limitations of, glove use

- Hand contamination may occur as a result of small, undetected holes in examination gloves (321,361)
- Contamination may occur during glove removal (50)
- Wearing gloves does not replace the need for hand hygiene (58)
- Failure to remove gloves after caring for a patient may lead to transmission of microorganisms from one patient to another (373).

Efficacy of Promotion and Impact of Improved Hand Hygiene

The lack of scientific information of the definitive impact of improved hand hygiene on health-care–associated infection rates is a possible barrier to appropriate adherence with hand-hygiene recommendations (Box 1). However, evidence supports the belief that improved hand hygiene can reduce health-care–associated infection rates. Failure to perform appropriate hand hygiene is considered the leading cause of

health-care–associated infections and spread of multiresistant organisms and has been recognized as a substantial contributor to outbreaks.

Of nine hospital-based studies of the impact of hand hygiene on the risk of health-care–associated infections (Table 10) (48,69–75,296), the majority demonstrated a temporal relationship between improved hand-hygiene practices and reduced infection rates.

In one of these studies, endemic MRSA in a neonatal intensive-care unit was eliminated 7 months after introduction of a new

TABLE 10. Association between improved adherence with hand-hygiene practice and health-care–associated infection rates

Year	Ref. no.	Hospital setting	Results	Duration of follow-up
1977	(48)	Adult ICU	Reduction in health-care–associated infections caused by endemic <i>Klebsiella</i> spp.	2 years
1982	(69)	Adult ICU	Reduction in health-care-associated infection rates	N.S.
1984	(70)	Adult ICU	Reduction in health-care–associated infection rates	N.S.
1990	(296)	Adult ICU	No effect (average hand hygiene adherence improvement did not reach statistical significance)	11 months
1992	(71)	Adult ICU	Substantial difference between rates of health-care–associated infection between two different hand-hygiene agents	8 months
1994	(72)	NICU	Elimination of MRSA, when combined with multiple other infection-control measures. Reduction of vancomycin use	9 months
1995	(73)	Newborn nursery	Elimination of MRSA, when combined with multiple other infection-control measures	3.5 years
2000	(75)	MICU/NICU	85% relative reduction of VRE rate in the intervention hospital; 44% relative reduction in control hospital; no change in MRSA	8 months
2000	(74)	Hospitalwide	Substantial reduction in the annual overall prevalence of health-care–associated infections and MRSA cross-transmission rates. Active surveillance cultures and contact precautions were implemented during same period	5 years

Note: ICU = intensive care unit, NICU = neonatal ICU, MRSA = methicillin-resistant *Staphylococcus aureus*, MICU = medical ICU, and N.S. = not stated.

hand antiseptic (1% triclosan); all other infection-control measures remained in place, including the practice of conducting weekly active surveillance by obtaining cultures (72). Another study reported an MRSA outbreak involving 22 infants in a neonatal unit (73). Despite intensive efforts, the outbreak could not be controlled until a new antiseptic was added (i.e., 0.3% triclosan); all previously used control measures remained in place, including gloves and gowns, cohorting, and obtaining cultures for active surveillance.

The effectiveness of a longstanding, hospitalwide program to promote hand hygiene at the University of Geneva hospitals was recently reported (74). Overall adherence to hand-hygiene guidelines during routine patient care was monitored during hospitalwide observational surveys. These surveys were conducted biannually during December 1994–December 1997, before and during implementation of a hand-hygiene campaign that specifically emphasized the practice of bedside, alcohol-based hand disinfection. Individual-sized bottles of hand-rub solution were distributed to all wards, and custom-made holders were mounted on all beds to facilitate access to hand disinfection. HCWs were also encouraged to carry bottles in their pockets, and in 1996, a newly designed flat (instead of round) bottle was made available to further facilitate pocket carriage. The promotional strategy was multimodal and involved a multidisciplinary team of HCWs, the use of wall posters, the promotion of antiseptic hand rubs located at bed-sides throughout the institution, and regular performance feedback to all HCWs (see <http://www.hopisafe.ch> for further

details on methodology). Health-care–associated infection rates, attack rates of MRSA cross-transmission, and consumption of hand-rub disinfectant were measured. Adherence to recommended hand-hygiene practices improved progressively from 48% in 1994 to 66% in 1997 ($p < 0.001$). Whereas recourse to handwashing with soap and water remained stable, frequency of hand disinfection markedly increased during the study period ($p < 0.001$), and the consumption of alcohol-based hand-rub solution increased from 3.5 to 15.4 liters per 1,000 patient-days during 1993–1998 ($p < 0.001$). The increased frequency of hand disinfection was unchanged after adjustment for known risk factors of poor adherence. During the same period, both overall health-care–associated infection and MRSA transmission rates decreased (both $p < 0.05$). The observed reduction in MRSA transmission may have been affected by both improved hand-hygiene adherence and the simultaneous implementation of active surveillance cultures for detecting and isolating patients colonized with MRSA (332). The experience from the University of Geneva hospitals constitutes the first report of a hand-hygiene campaign with a sustained improvement over several years. An additional multimodal program also yielded sustained improvements in hand-hygiene practices over an extended period (75); the majority of studies have been limited to a 6- to 9-month observation period.

Although these studies were not designed to assess the independent contribution of hand hygiene on the prevention of health-care–associated infections, the results indicate that

improved hand-hygiene practices reduce the risk of transmission of pathogenic microorganisms. The beneficial effects of hand-hygiene promotion on the risk of cross-transmission also have been reported in surveys conducted in schools and day care centers (333–338), as well as in a community setting (339–341).

Other Policies Related to Hand Hygiene

Fingernails and Artificial Nails

Studies have documented that subungual areas of the hand harbor high concentrations of bacteria, most frequently coagulase-negative staphylococci, gram-negative rods (including *Pseudomonas* spp.), Corynebacteria, and yeasts (14,342,343). Freshly applied nail polish does not increase the number of bacteria recovered from periungual skin, but chipped nail polish may support the growth of larger numbers of organisms on fingernails (344,345). Even after careful handwashing or the use of surgical scrubs, personnel often harbor substantial numbers of potential pathogens in the subungual spaces (346–348).

Whether artificial nails contribute to transmission of health-care-associated infections is unknown. However, HCWs who wear artificial nails are more likely to harbor gram-negative pathogens on their fingertips than are those who have natural nails, both before and after handwashing (347–349). Whether the length of natural or artificial nails is a substantial risk factor is unknown, because the majority of bacterial growth occurs along the proximal 1 mm of the nail adjacent to subungual skin (345,347,348). Recently, an outbreak of *P. aeruginosa* in a neonatal intensive care unit was attributed to two nurses (one with long natural nails and one with long artificial nails) who carried the implicated strains of *Pseudomonas* spp. on their hands (350). Patients were substantially more likely than controls to have been cared for by the two nurses during the exposure period, indicating that colonization of long or artificial nails with *Pseudomonas* spp. may have contributed to causing the outbreak. Personnel wearing artificial nails also have been epidemiologically implicated in several other outbreaks of infection caused by gram-negative bacilli and yeast (351–353). Although these studies provide evidence that wearing artificial nails poses an infection hazard, additional studies are warranted.

Gloving Policies

CDC has recommended that HCWs wear gloves to 1) reduce the risk of personnel acquiring infections from patients, 2) prevent health-care worker flora from being transmitted to patients, and 3) reduce transient contamination of the hands

of personnel by flora that can be transmitted from one patient to another (354). Before the emergence of the acquired immunodeficiency syndrome (AIDS) epidemic, gloves were worn primarily by personnel caring for patients colonized or infected with certain pathogens or by personnel exposed to patients with a high risk of hepatitis B. Since 1987, a dramatic increase in glove use has occurred in an effort to prevent transmission of HIV and other bloodborne pathogens from patients to HCWs (355). The Occupational Safety and Health Administration (OSHA) mandates that gloves be worn during all patient-care activities that may involve exposure to blood or body fluids that may be contaminated with blood (356).

The effectiveness of gloves in preventing contamination of HCWs' hands has been confirmed in several clinical studies (45,51,58). One study found that HCWs who wore gloves during patient contact contaminated their hands with an average of only 3 CFUs per minute of patient care, compared with 16 CFUs per minute for those not wearing gloves (51). Two other studies, involving personnel caring for patients with *C. difficile* or VRE, revealed that wearing gloves prevented hand contamination among the majority of personnel having direct contact with patients (45,58). Wearing gloves also prevented personnel from acquiring VRE on their hands when touching contaminated environmental surfaces (58). Preventing heavy contamination of the hands is considered important, because handwashing or hand antisepsis may not remove all potential pathogens when hands are heavily contaminated (25,111).

Several studies provide evidence that wearing gloves can help reduce transmission of pathogens in health-care settings. In a prospective controlled trial that required personnel to routinely wear vinyl gloves when handling any body substances, the incidence of *C. difficile* diarrhea among patients decreased from 7.7 cases/1,000 patient discharges before the intervention to 1.5 cases/1,000 discharges during the intervention (226). The prevalence of asymptomatic *C. difficile* carriage also decreased substantially on "glove" wards, but not on control wards. In intensive-care units where VRE or MRSA have been epidemic, requiring all HCWs to wear gloves to care for all patients in the unit (i.e., universal glove use) likely has helped control outbreaks (357,358).

The influence of glove use on the hand-hygiene habits of personnel is not clear. Several studies found that personnel who wore gloves were less likely to wash their hands upon leaving a patient's room (290,320). In contrast, two other studies found that personnel who wore gloves were substantially more likely to wash their hands after patient care (87,301).

The following caveats regarding use of gloves by HCWs must be considered. Personnel should be informed that gloves

do not provide complete protection against hand contamination. Bacterial flora colonizing patients may be recovered from the hands of $\leq 30\%$ of HCWs who wear gloves during patient contact (50,58). Further, wearing gloves does not provide complete protection against acquisition of infections caused by hepatitis B virus and herpes simplex virus (359,360). In such instances, pathogens presumably gain access to the caregiver's hands via small defects in gloves or by contamination of the hands during glove removal (50,321,359,361).

Gloves used by HCWs are usually made of natural rubber latex and synthetic nonlatex materials (e.g., vinyl, nitrile, and neoprene [polymers and copolymers of chloroprene]). Because of the increasing prevalence of latex sensitivity among HCWs and patients, FDA has approved several powdered and powder-free latex gloves with reduced protein contents, as well as synthetic gloves that can be made available by health-care institutions for use by latex-sensitive employees. In published studies, the barrier integrity of gloves varies on the basis of type and quality of glove material, intensity of use, length of time used, manufacturer, whether gloves were tested before or after use, and method used to detect glove leaks (359,361–366). In published studies, vinyl gloves have had defects more frequently than latex gloves, the difference in defect frequency being greatest after use (359,361,364,367). However, intact vinyl gloves provide protection comparable to that of latex gloves (359). Limited studies indicate that nitrile gloves have leakage rates that approximate those of latex gloves (368–371). Having more than one type of glove available is desirable, because it allows personnel to select the type that best suits their patient-care activities. Although recent studies indicate that improvements have been made in the quality of gloves (366), hands should be decontaminated or washed after removing gloves (8,50,58,321,361). Gloves should not be washed or reused (321,361). Use of petroleum-based hand lotions or creams may adversely affect the integrity of latex gloves (372). After use of powdered gloves, certain alcohol hand rubs may interact with residual powder on the hands of personnel, resulting in a gritty feeling on the hands. In facilities where powdered gloves are commonly used, various alcohol-based hand rubs should be tested after removal of powdered gloves to avoid selecting a product that causes this undesirable reaction. Personnel should be reminded that failure to remove gloves between patients may contribute to transmission of organisms (358,373).

Jewelry

Several studies have demonstrated that skin underneath rings is more heavily colonized than comparable areas of skin on fingers without rings (374–376). One study found that 40% of nurses harbored gram-negative bacilli (e.g., *E. cloacae*, *Klebsiella*, and *Acinetobacter*) on skin under rings and that certain nurses carried the same organism under their rings for several months (375). In a more recent study involving >60 intensive care unit nurses, multivariable analysis revealed that rings were the only substantial risk factor for carriage of gram-negative bacilli and *S. aureus* and that the concentration of organisms recovered correlated with the number of rings worn (377). Whether the wearing of rings results in greater transmission of pathogens is unknown. Two studies determined that mean bacterial colony counts on hands after handwashing were similar among persons wearing rings and those not wearing rings (376,378). Further studies are needed to establish if wearing rings results in greater transmission of pathogens in health-care settings.

Hand-Hygiene Research Agenda

Although the number of published studies concerning hand hygiene has increased considerably in recent years, many questions regarding hand-hygiene products and strategies for improving adherence of personnel to recommended policies remain unanswered. Several concerns must still be addressed by researchers in industry and by clinical investigators (Box 3).

Web-Based Hand-Hygiene Resources

Additional information regarding improving hand hygiene is available at <http://www.hopisafe.ch>

University of Geneva Hospitals, Geneva, Switzerland

<http://www.cdc.gov/ncidod/hip>

CDC, Atlanta, Georgia

<http://www.jr2.ox.ac.uk/bandolier/band88/b88-8.html>

Bandolier journal, United Kingdom

<http://www.med.upenn.edu>

University of Pennsylvania, Philadelphia, Pennsylvania

BOX 3. Hand-hygiene research agenda**Education and promotion**

- Provide health-care workers (HCWs) with better education regarding the types of patient care activities that can result in hand contamination and cross-transmission of microorganisms.
- Develop and implement promotion hand-hygiene programs in pregraduate courses.
- Study the impact of population-based education on hand-hygiene behavior.
- Design and conduct studies to determine if frequent glove use should be encouraged or discouraged.
- Determine evidence-based indications for hand cleansing (considering that it might be unrealistic to expect HCWs to clean their hands after every contact with the patient).
- Assess the key determinants of hand-hygiene behavior and promotion among the different populations of HCWs.
- Develop methods to obtain management support.
- Implement and evaluate the impact of the different components of multimodal programs to promote hand hygiene.

Hand-hygiene agents and hand care

- Determine the most suitable formulations for hand-hygiene products.
- Determine if preparations with persistent antimicrobial activity reduce infection rates more effectively than do preparations whose activity is limited to an immediate effect.
- Study the systematic replacement of conventional handwashing by the use of hand disinfection.
- Develop devices to facilitate the use and optimal application of hand-hygiene agents.
- Develop hand-hygiene agents with low irritancy potential.
- Study the possible advantages and eventual interaction of hand-care lotions, creams, and other barriers to help minimize the potential irritation associated with hand-hygiene agents.

Laboratory-based and epidemiologic research and development

- Develop experimental models for the study of cross-contamination from patient to patient and from environment to patient.
- Develop new protocols for evaluating the in vivo efficacy of agents, considering in particular short application times and volumes that reflect actual use in health-care facilities.
- Monitor hand-hygiene adherence by using new devices or adequate surrogate markers, allowing frequent individual feedback on performance.
- Determine the percentage increase in hand-hygiene adherence required to achieve a predictable risk reduction in infection rates.
- Generate more definitive evidence for the impact on infection rates of improved adherence to recommended hand-hygiene practices.
- Provide cost-effectiveness evaluation of successful and unsuccessful promotion campaigns.

Part II. Recommendations**Categories**

These recommendations are designed to improve hand-hygiene practices of HCWs and to reduce transmission of pathogenic microorganisms to patients and personnel in health-care settings. This guideline and its recommendations are not intended for use in food processing or food-service establishments, and are not meant to replace guidance provided by FDA's Model Food Code.

As in previous CDC/HICPAC guidelines, each recommendation is categorized on the basis of existing scientific data, theoretical rationale, applicability, and economic impact. The CDC/HICPAC system for categorizing recommendations is as follows:

Category IA. Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiologic studies.

Category IB. Strongly recommended for implementation and supported by certain experimental, clinical, or epidemiologic studies and a strong theoretical rationale.

Category IC. Required for implementation, as mandated by federal or state regulation or standard.

Category II. Suggested for implementation and supported by suggestive clinical or epidemiologic studies or a theoretical rationale.

No recommendation. Unresolved issue. Practices for which insufficient evidence or no consensus regarding efficacy exist.

Recommendations

1. Indications for handwashing and hand antisepsis
 - A. When hands are visibly dirty or contaminated with proteinaceous material or are visibly soiled with blood or other body fluids, wash hands with either a non-antimicrobial soap and water or an antimicrobial soap and water (IA) (66).
 - B. If hands are not visibly soiled, use an alcohol-based hand rub for routinely decontaminating hands in all other clinical situations described in items 1C–J (IA) (74,93,166,169,283,294,312,398). Alternatively, wash hands with an antimicrobial soap and water in all clinical situations described in items 1C–J (IB) (69-71,74).
 - C. Decontaminate hands before having direct contact with patients (IB) (68,400).
 - D. Decontaminate hands before donning sterile gloves when inserting a central intravascular catheter (IB) (401,402).
 - E. Decontaminate hands before inserting indwelling urinary catheters, peripheral vascular catheters, or other invasive devices that do not require a surgical procedure (IB) (25,403).
 - F. Decontaminate hands after contact with a patient's intact skin (e.g., when taking a pulse or blood pressure, and lifting a patient) (IB) (25,45,48,68).
 - G. Decontaminate hands after contact with body fluids or excretions, mucous membranes, nonintact skin, and wound dressings if hands are not visibly soiled (IA) (400).
 - H. Decontaminate hands if moving from a contaminated-body site to a clean-body site during patient care (II) (25,53).
 - I. Decontaminate hands after contact with inanimate objects (including medical equipment) in the immediate vicinity of the patient (II) (46,53,54).
 - J. Decontaminate hands after removing gloves (IB) (50,58,321).
 - K. Before eating and after using a restroom, wash hands with a non-antimicrobial soap and water or with an antimicrobial soap and water (IB) (404-409).
 - L. Antimicrobial-impregnated wipes (i.e., towelettes) may be considered as an alternative to washing hands with non-antimicrobial soap and water. Because they are not as effective as alcohol-based hand rubs or washing hands with an antimicrobial soap and water for reducing bacterial counts on the hands of HCWs, they are not a substitute for using an alcohol-based hand rub or antimicrobial soap (IB) (160,161).
 - M. Wash hands with non-antimicrobial soap and water or with antimicrobial soap and water if exposure to *Bacillus anthracis* is suspected or proven. The physical action of washing and rinsing hands under such circumstances is recommended because alcohols, chlorhexidine, iodophors, and other antiseptic agents have poor activity against spores (II) (120,172,224,225).
 - N. No recommendation can be made regarding the routine use of nonalcohol-based hand rubs for hand hygiene in health-care settings. Unresolved issue.
2. Hand-hygiene technique
 - A. When decontaminating hands with an alcohol-based hand rub, apply product to palm of one hand and rub hands together, covering all surfaces of hands and fingers, until hands are dry (IB) (288,410). Follow the manufacturer's recommendations regarding the volume of product to use.
 - B. When washing hands with soap and water, wet hands first with water, apply an amount of product recommended by the manufacturer to hands, and rub hands together vigorously for at least 15 seconds, covering all surfaces of the hands and fingers. Rinse hands with water and dry thoroughly with a disposable towel. Use towel to turn off the faucet (IB) (90-92,94,411). Avoid using hot water, because repeated exposure to hot water may increase the risk of dermatitis (IB) (254,255).
 - C. Liquid, bar, leaflet or powdered forms of plain soap are acceptable when washing hands with a non-antimicrobial soap and water. When bar soap is used, soap racks that facilitate drainage and small bars of soap should be used (II) (412-415).
 - D. Multiple-use cloth towels of the hanging or roll type are not recommended for use in health-care settings (II) (137,300).
3. Surgical hand antisepsis
 - A. Remove rings, watches, and bracelets before beginning the surgical hand scrub (II) (375,378,416).
 - B. Remove debris from underneath fingernails using a nail cleaner under running water (II) (14,417).

- C. Surgical hand antisepsis using either an antimicrobial soap or an alcohol-based hand rub with persistent activity is recommended before donning sterile gloves when performing surgical procedures (IB) (115,159,232,234,237,418).
 - D. When performing surgical hand antisepsis using an antimicrobial soap, scrub hands and forearms for the length of time recommended by the manufacturer, usually 2–6 minutes. Long scrub times (e.g., 10 minutes) are not necessary (IB) (117,156,205,207,238-241).
 - E. When using an alcohol-based surgical hand-scrub product with persistent activity, follow the manufacturer's instructions. Before applying the alcohol solution, prewash hands and forearms with a non-antimicrobial soap and dry hands and forearms completely. After application of the alcohol-based product as recommended, allow hands and forearms to dry thoroughly before donning sterile gloves (IB) (159,237).
4. Selection of hand-hygiene agents
 - A. Provide personnel with efficacious hand-hygiene products that have low irritancy potential, particularly when these products are used multiple times per shift (IB) (90,92,98,166,249). This recommendation applies to products used for hand antisepsis before and after patient care in clinical areas and to products used for surgical hand antisepsis by surgical personnel.
 - B. To maximize acceptance of hand-hygiene products by HCWs, solicit input from these employees regarding the feel, fragrance, and skin tolerance of any products under consideration. The cost of hand-hygiene products should not be the primary factor influencing product selection (IB) (92,93,166,274,276-278).
 - C. When selecting non-antimicrobial soaps, antimicrobial soaps, or alcohol-based hand rubs, solicit information from manufacturers regarding any known interactions between products used to clean hands, skin care products, and the types of gloves used in the institution (II) (174,372).
 - D. Before making purchasing decisions, evaluate the dispenser systems of various product manufacturers or distributors to ensure that dispensers function adequately and deliver an appropriate volume of product (II) (286).
 - E. Do not add soap to a partially empty soap dispenser. This practice of "topping off" dispensers can lead to bacterial contamination of soap (IA) (187,419).
 5. Skin care
 - A. Provide HCWs with hand lotions or creams to minimize the occurrence of irritant contact dermatitis associated with hand antisepsis or handwashing (IA) (272,273).
 - B. Solicit information from manufacturers regarding any effects that hand lotions, creams, or alcohol-based hand antiseptics may have on the persistent effects of antimicrobial soaps being used in the institution (IB) (174,420,421).
 6. Other Aspects of Hand Hygiene
 - A. Do not wear artificial fingernails or extenders when having direct contact with patients at high risk (e.g., those in intensive-care units or operating rooms) (IA) (350–353).
 - B. Keep natural nails tips less than 1/4-inch long (II) (350).
 - C. Wear gloves when contact with blood or other potentially infectious materials, mucous membranes, and nonintact skin could occur (IC) (356).
 - D. Remove gloves after caring for a patient. Do not wear the same pair of gloves for the care of more than one patient, and do not wash gloves between uses with different patients (IB) (50,58,321,373).
 - E. Change gloves during patient care if moving from a contaminated body site to a clean body site (II) (50,51,58).
 - F. No recommendation can be made regarding wearing rings in health-care settings. Unresolved issue.
 7. Health-care worker educational and motivational programs
 - A. As part of an overall program to improve hand-hygiene practices of HCWs, educate personnel regarding the types of patient-care activities that can result in hand contamination and the advantages and disadvantages of various methods used to clean their hands (II) (74,292,295,299).
 - B. Monitor HCWs' adherence with recommended hand-hygiene practices and provide personnel with information regarding their performance (IA) (74,276,292,295,299,306,310).
 - C. Encourage patients and their families to remind HCWs to decontaminate their hands (II) (394,422).
 8. Administrative measures
 - A. Make improved hand-hygiene adherence an institutional priority and provide appropriate

- administrative support and financial resources (IB) (74,75).
- B. Implement a multidisciplinary program designed to improve adherence of health personnel to recommended hand-hygiene practices (IB) (74,75).
 - C. As part of a multidisciplinary program to improve hand-hygiene adherence, provide HCWs with a readily accessible alcohol-based hand-rub product (IA) (74,166,283,294,312).
 - D. To improve hand-hygiene adherence among personnel who work in areas in which high workloads and high intensity of patient care are anticipated, make an alcohol-based hand rub available at the entrance to the patient's room or at the bedside, in other convenient locations, and in individual pocket-sized containers to be carried by HCWs (IA) (11,74,166,283,284,312,318,423).
 - E. Store supplies of alcohol-based hand rubs in cabinets or areas approved for flammable materials (IC).

Part III. Performance Indicators

1. The following performance indicators are recommended for measuring improvements in HCWs' hand-hygiene adherence:
 - A. Periodically monitor and record adherence as the number of hand-hygiene episodes performed by personnel/number of hand-hygiene opportunities, by ward or by service. Provide feedback to personnel regarding their performance.
 - B. Monitor the volume of alcohol-based hand rub (or detergent used for handwashing or hand antisepsis) used per 1,000 patient-days.
 - C. Monitor adherence to policies dealing with wearing of artificial nails.
 - D. When outbreaks of infection occur, assess the adequacy of health-care worker hand hygiene.

References

1. Rotter M. Hand washing and hand disinfection [Chapter 87]. In: Mayhall CG, ed. Hospital epidemiology and infection control. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999.
2. Labarraque AG. Instructions and observations regarding the use of the chlorides of soda and lime. Porter J, ed. [French] New Haven, CT: Baldwin and Treadway, 1829.
3. Semmelweis I. Etiology, concept, and prophylaxis of childbed fever. Carter KC, ed. 1st ed. Madison, WI: The University of Wisconsin Press, 1983.
4. Coppage CM. Hand washing in patient care [Motion picture]. Washington, DC: US Public Health Service, 1961.
5. Steere AC, Mallison GF. Handwashing practices for the prevention of nosocomial infections. *Ann Intern Med* 1975;83:683–90.
6. Garner JS, Favero MS. CDC guideline for handwashing and hospital environmental control, 1985. *Infect Control* 1986;7:231–43.
7. Larson E. Guideline for use of topical antimicrobial agents. *Am J Infect Control* 1988;16:253–66.
8. Larson EL, APIC Guidelines Committee. APIC guideline for handwashing and hand antisepsis in health care settings. *Am J Infect Control* 1995;23:251–69.
9. Hospital Infection Control Practices Advisory Committee (HICPAC). Recommendations for preventing the spread of vancomycin resistance. *Infect Control Hosp Epidemiol* 1995;16:105–13.
10. Garner JS, Hospital Infection Control Practices Advisory Committee. Guideline for isolation precautions in hospitals. *Infect Control Hosp Epidemiol* 1996;17:53–80.
11. Pittet D, Mourouga P, Perneger TV, Members of the Infection Control Program. Compliance with handwashing in a teaching hospital. *Ann Intern Med* 1999;130:126–30.
12. Boyce JM. It is time for action: improving hand hygiene in hospitals. *Ann Intern Med* 1999;130:153–5.
13. Selwyn S. Microbiology and ecology of human skin. *Practitioner* 1980;224:1059–62.
14. Price PB. Bacteriology of normal skin: a new quantitative test applied to a study of the bacterial flora and the disinfectant action of mechanical cleansing. *J Infect Dis* 1938;63:301–18.
15. Larson E. Effects of handwashing agent, handwashing frequency, and clinical area on hand flora. *Am J Infect Control* 1984;11:76–82.
16. Maki D. Control of colonization and transmission of pathogenic bacteria in the hospital. *Ann Intern Med* 1978;89(Pt 2):777–80.
17. Larson EL, Norton Hughes CA, Pyrak JD, Sparks SM, Cagatay EU, Bartkus JM. Changes in bacterial flora associated with skin damage on hands of health care personnel. *Am J Infect Control* 1998;26:513–21.
18. Sprunt K, Redman W, Leidy G. Antibacterial effectiveness of routine hand washing. *Pediatrics* 1973;52:264–71.
19. Food and Drug Administration. Tentative final monograph for healthcare antiseptic drug products; proposed rule. *Federal Register* 1994;59:31441–52.
20. Lowbury EJJ. Gram-negative bacilli on the skin. *Br J Dermatol* 1969;81(suppl 1):55–61.
21. Noble WC. Distribution of the Micrococccaceae. *Br J Dermatol* 1969;81(suppl 1):27–31.
22. McBride ME, Duncan WC, Bodey GP, McBride CM. Microbial skin flora of selected cancer patients and hospital personnel. *J Clin Microbiol* 1976;3:14–20.
23. Casewell MW. Role of hands in nosocomial gram-negative infection. In: Maibach HI, Aly R, eds. Skin microbiology: relevance to clinical infection. New York, NY: Springer-Verlag, 1981.
24. Larson EL, McGinley KJ, Foglia AR, Talbot GH, Leyden JJ. Composition and antimicrobial resistance of skin flora in hospitalized and healthy adults. *J Clin Microbiol* 1986;23:604–8.
25. Ehrenkranz NJ, Alfonso BC. Failure of bland soap handwash to prevent hand transfer of patient bacteria to urethral catheters. *Infect Control Hosp Epidemiol* 1991;12:654–62.
26. Sanderson PJ, Weissler S. Recovery of coliforms from the hands of nurses and patients: activities leading to contamination. *J Hosp Infect* 1992;21:85–93.
27. Coello R, Jiménez J, García M, et al. Prospective study of infection, colonization and carriage of methicillin-resistant *Staphylococcus aureus* in an outbreak affecting 990 patients. *Eur J Clin Microbiol Infect Dis* 1994;13:74–81.

28. Sanford MD, Widmer AF, Bale MJ, Jones RN, Wenzel RP. Efficient detection and long-term persistence of the carriage of methicillin-resistant *Staphylococcus aureus*. Clin Infect Dis 1994;19:1123–8.
29. Bertone SA, Fisher MC, Mortensen JE. Quantitative skin cultures at potential catheter sites in neonates. Infect Control Hosp Epidemiol 1994;15:315–8.
30. Bonten MJM, Hayden MK, Nathan C, VanVoorhis J, et al. Epidemiology of colonisation of patients and environment with vancomycin-resistant enterococci. Lancet 1996;348:1615–9.
31. Larson EL, Cronquist AB, Whittier S, Lai L, Lyle CT, Della Latta P. Differences in skin flora between inpatients and chronically ill patients. Heart Lung 2000;29:298–305.
32. Polakoff S, Richards IDG, Parker MT, Lidwell OM. Nasal and skin carriage of *Staphylococcus aureus* by patients undergoing surgical operation. J Hyg (Lond) 1967;65:559–66.
33. Leyden JJ, McGinley KJ, Nordstrom KM, Webster GF. Skin microflora. J Invest Dermatol 1987;88:65s–72s.
34. Tuazon CU, Perez A, Kishaba T, Sheagren JN. *Staphylococcus aureus* among insulin-injecting diabetic patients. JAMA 1975;231:1272.
35. Kaplowitz LG, Comstock JA, Landwehr DM, Dalton HP, Mayhall CG. Prospective study of microbial colonization of the nose and skin and infection of the vascular access site in hemodialysis patients. J Clin Microbiol 1988;26:1257–62.
36. Aly R, Maibach HI, Shinefield HR. Microbial flora of atopic dermatitis. Arch Dermatol 1977;113:780–2.
37. Kirmani N, Tuazon CU, Murray HW, Parrish AE, Sheagren JN. *Staphylococcus aureus* carriage rate of patients receiving long-term hemodialysis. Arch Intern Med 1978;138:1657–9.
38. Goldblum SE, Ulrich JA, Goldman RS, Reed WP. Nasal and cutaneous flora among hemodialysis patients and personnel: quantitative and qualitative characterization and patterns of staphylococcal carriage. Am J Kidney Dis 1982;11:281–6.
39. Boelaert JR, Van Landuyt HW, Gordts BZ, De Baere YA, Messer SA, Herwaldt LA. Nasal and cutaneous carriage of *Staphylococcus aureus* in hemodialysis patients: the effect of nasal mupirocin. Infect Control Hosp Epidemiol 1996;17:809–11.
40. Zimakoff J, Pedersen FB, Bergen L, et al. *Staphylococcus aureus* carriage and infections among patients in four haemo- and peritoneal-dialysis centres in Denmark. J Hosp Infect 1996;33:289–300.
41. Bibel DJ, Greenbert JH, Cook JL. *Staphylococcus aureus* and the microbial ecology of atopic dermatitis. Can J Microbiol 1997;23:1062–8.
42. Noble WC. Dispersal of skin microorganisms. Br J Dermatol 1975;93:477–85.
43. Walter CW, Kundsins RB, Shilkret MA, Day MM. Spread of staphylococci to the environment. Antibiotics Annual 1959:952–7.
44. Boyce JM, Opal SM, Chow JW, et al. Outbreak of multidrug-resistant *Enterococcus faecium* with transferable *vanB* class vancomycin resistance. J Clin Microbiol 1994;32:1148–53.
45. McFarland LV, Mulligan ME, Kwok RYY, Stamm WE. Nosocomial acquisition of *Clostridium difficile* infection. N Engl J Med 1989;320:204–10.
46. Samore MH, Venkataraman L, DeGirolami PC, Levin E, Arbeit RD, Karchmer AW. Clinical and molecular epidemiology of sporadic and clustered cases of nosocomial *Clostridium difficile* diarrhea. Am J Med 1996;100:32–40.
47. Lidwell OM, Towers AG, Ballard J, Gladstone B. Transfer of microorganisms between nurses and patients in a clean air environment. J Appl Bacteriol 1974;37:649–56.
48. Casewell M, Phillips I. Hands as route of transmission for *Klebsiella* species. Br Med J 1977;2:1315–7.
49. Hall CB, Douglas RG. Modes of transmission of respiratory syncytial virus. J Pediatr 1981;99:100–2.
50. Olsen RJ, Lynch P, Coyle MB, Cummings J, Bokete T, Stamm WE. Examination gloves as barriers to hand contamination in clinical practice. JAMA 1993;270:350–3.
51. Pittet D, Dharan S, Touveneau S, Sauvan V, Perneger TV. Bacterial contamination of the hands of hospital staff during routine patient care. Arch Intern Med 1999;159:821–6.
52. Fox MK, Langner SB, Wells RW. How good are hand washing practices? Am J Nursing 1974;74:1676–8.
53. Ojajärvi J. Effectiveness of hand washing and disinfection methods in removing transient bacteria after patient nursing. J Hyg (Lond) 1980;85:193–203.
54. Boyce JM, Potter-Bynoe G, Chenevert C, King T. Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. Infect Control Hosp Epidemiol 1997;18:622–7.
55. Hayden MK, Blom, DW, Lyle, EA, et al. The risk of hand and glove contamination by healthcare workers (HCWs) after contact with a VRE (+) patient (pt) or the pts environment (env) [Abstract K-1334]. Presented at the 41st Interscience Conference on Antimicrobial Agents and Chemotherapy Chicago: American Society for Microbiology, 2001.
56. Scott E, Bloomfield SF. The survival and transfer of microbial contamination via cloths, hands and utensils. J Appl Bacteriol 1990;68:271–8.
57. Bauer TM, Ofner E, Just HM, Just H, Daschner FD. An epidemiological study assessing the relative importance of airborne and direct contact transmission of microorganisms in a medical intensive care unit. J Hosp Infect 1990;15:301–9.
58. Tenorio AR, Badri SM, Sahgal NB, et al. Effectiveness of gloves in the prevention of hand carriage of vancomycin-resistant *Enterococcus* species by health care workers after patient care. Clin Infect Dis 2001;32:826–9.
59. Daschner FD. How cost-effective is the present use of antiseptics? J Hosp Infect 1988;11 (suppl A):227–35.
60. Knittle MA, Eitzman DV, Baer H. Role of hand contamination of personnel in the epidemiology of gram-negative nosocomial infections. J Pediatr 1975;86:433–7.
61. Ayliffe GAJ, Babb JR, Davies JG, Lilly HA. Hand disinfection: a comparison of various agents in laboratory and ward studies. J Hosp Infect 1988;11:226–43.
62. Strausbaugh LJ, Sewell DL, Ward TT, Pfaller MA, Heitzman T, Tjoelker R. High frequency of yeast carriage on hands of hospital personnel. J Clin Microbiol 1994;32:2299–300.
63. Marples RR, Towers AG. A laboratory model for the investigation of contact transfer of micro-organisms. J Hyg (Lond) 1979;82:237–48.
64. Mackintosh CA, Hoffman PN. An extended model for transfer of micro-organisms via the hands: differences between organisms and the effect of alcohol disinfection. J Hyg (Lond) 1984;92:345–55.
65. Patrick DR, Findon G, Miller TE. Residual moisture determines the level of touch-contact-associated bacterial transfer following hand washing. Epidemiol Infect 1997;119:319–25.
66. Larson E. A causal link between handwashing and risk of infection? Examination of the evidence. Infect Control Hosp Epidemiol 1988;9:28–36.
67. Larson E. Skin hygiene and infection prevention: more of the same or different approaches? Clin Infect Dis 1999;29:1287–94.

68. Mortimer EA Jr, Lipsitz PJ, Wolinsky E, Gonzaga AJ, Rammelkamp CH Jr. Transmission of staphylococci between newborns. *Am J Dis Child* 1962;104:289–95.
69. Maki DG. The use of antiseptics for handwashing by medical personnel. *J Chemother* 1989;1(suppl 1):3–11.
70. Massanari RM, Hierholzer WJ Jr. A crossover comparison of antiseptic soaps on nosocomial infection rates in intensive care units. *Am J Infect Control* 1984;12:247–8.
71. Doebbeling BN, Stanley GL, Sheetz CT, et al. Comparative efficacy of alternative hand-washing agents in reducing nosocomial infections in intensive care units. *N Engl J Med* 1992;327:88–93.
72. Webster J, Faoagali JL, Cartwright D. Elimination of methicillin-resistant *Staphylococcus aureus* from a neonatal intensive care unit after hand washing with triclosan. *J Paediatr Child Health* 1994;30:59–64.
73. Zafar AB, Butler RC, Reese DJ, Gaydos LA, Mennonona PA. Use of 0.3% triclosan (Bacti-Stat*) to eradicate an outbreak of methicillin-resistant *Staphylococcus aureus* in a neonatal nursery. *Am J Infect Control* 1995;23:200–8.
74. Pittet D, Hugonnet S, Harbarth S, Mourouga P, Sauvan V, Touveneau S. Effectiveness of a hospital-wide programme to improve compliance with hand hygiene. *Lancet* 2000;356:1307–12.
75. Larson EL, Early E, Cloonan P, Sugrue S, Parides M. An organizational climate intervention associated with increased handwashing and decreased nosocomial infections. *Behav Med* 2000;26:14–22.
76. Fridkin SK, Pear SM, Williamson TH, Galgiani JN, Jarvis WR. The role of understaffing in central venous catheter-associated bloodstream infections. *Infect Control Hosp Epidemiol* 1996;17:150–8.
77. Vicca AF. Nursing staff workload as a determinant of methicillin-resistant *Staphylococcus aureus* spread in an adult intensive therapy unit. *J Hosp Infect* 1999;43:109–13.
78. Harbarth S, Sudre P, Dharan S, Cadenas M, Pittet D. Outbreak of *Enterobacter cloacae* related to understaffing, overcrowding, and poor hygiene practices. *Infect Control Hosp Epidemiol* 1999;20:598–603.
79. European Committee for Standardization. Chemical disinfectants and antiseptics—hygienic handrub—test method and requirements (phase 2/step 2) [European standard EN 1500]. Brussels, Belgium: Central Secretariat; 1997.
80. Kramer A, Rudolph P, Kampf G, Pittet D. Limited efficacy of alcohol-based hand gels. *Lancet* 2002;359:1489–90.
81. Sattar SA, Abebe M, Bueti AJ, Jampani H, Newman J, Hua S. Activity of an alcohol-based hand gel against human adeno-, rhino-, and rotaviruses using the fingerpad method. *Infect Control Hosp Epidemiol* 2000;21:516–9.
82. Wolff MH, Schmitt J, Rahaus M, König A. Hepatitis A virus: a test method for virucidal activity. *J Hosp Infect* 2001;48(suppl A):S18–S22.
83. Steinmann J. Some principles of virucidal testing. *J Hosp Infect* 2001;48(suppl A):S15–S17.
84. Gould D, Ream E. Assessing nurses' hand decontamination performance. *Nursing Times* 1993;89:47–50.
85. Quraishi ZA, McGuckin M, Blais FX. Duration of handwashing in intensive care units: a descriptive study. *Am J Infect Control* 1984;11:83–7.
86. Lund S, Jackson J, Leggett J, Hales L, Dworkin R, Gilbert D. Reality of glove use and handwashing in a community hospital. *Am J Infect Control* 1994;22:352–7.
87. Meengs MR, Giles BK, Chisholm CD, Cordell WH, Nelson DR. Hand washing frequency in an emergency department. *Ann Emerg Med* 1994;23:1307–12.
88. Larson E, McGeer A, Quraishi ZA, et al. Effect of an automated sink on handwashing practices and attitudes in high-risk units. *Infect Control Hosp Epidemiol* 1991;12:422–8.
89. Broughall JM, Marshman C, Jackson B, Bird P. An automatic monitoring system for measuring handwashing frequency. *J Hosp Infect* 1984;5:447–53.
90. Ojajärvi J, Mäkelä P, Rantasalo I. Failure of hand disinfection with frequent hand washing: a need for prolonged field studies. *J Hyg (Lond)* 1977;79:107–19.
91. Larson EL, Eke PI, Wilder MP, Laughon BE. Quantity of soap as a variable in handwashing. *Infect Control* 1987;8:371–5.
92. Larson E, Leyden JJ, McGinley KJ, Grove GL, Talbot GH. Physiologic and microbiologic changes in skin related to frequent handwashing. *Infect Control* 1986;7:59–63.
93. Larson EL, Eke PI, Laughon BE. Efficacy of alcohol-based hand rinses under frequent-use conditions. *Antimicrob Agents Chemother* 1986;30:542–4.
94. Larson EL, Laughon BE. Comparison of four antiseptic products containing chlorhexidine gluconate. *Antimicrob Agents Chemother* 1987;31:1572–4.
95. Meers PD, Yeo GA. Shedding of bacteria and skin squames after handwashing. *J Hyg (Lond)* 1978;81:99–105.
96. Winnefeld M, Richard MA, Drancourt M, Grobb JJ. Skin tolerance and effectiveness of two hand decontamination procedures in everyday hospital use. *Br J Dermatol* 2000;143:546–50.
97. Maki DG, Zilz MA, Alvarado CJ. Evaluation of the antibacterial efficacy of four agents for handwashing. In: Nelson JC, Grassi C, eds. Current chemotherapy and infectious disease proceedings of the 11th International Congress on Chemotherapy and the 19th ICACC. Washington, DC: American Society for Microbiology, 1979.
98. Boyce JM, Kelliher S, Vallande N. Skin irritation and dryness associated with two hand-hygiene regimens: soap-and-water handwashing versus hand antiseptics with an alcoholic hand gel. *Infect Control Hosp Epidemiol* 2000;21:442–8.
99. Sartor C, Jacomo V, Duvivier C, Tissot-Dupont H, Sambuc R, Drancourt M. Nosocomial *Serratia marcescens* infections associated with extrinsic contamination of a liquid nonmedicated soap. *Infect Control Hosp Epidemiol* 2000;21:196–9.
100. Walter CW. Editorial: disinfection of hands. *Am J Surg* 1965;109:691–3.
101. Gravens DL, Butcher HR Jr, Ballinger WF, Dewar NE. Septisol antiseptic foam for hands of operating room personnel: an effective antibacterial agent. *Surgery* 1973;73:360–7.
102. Eitzen HE, Ritter MA, French MLV, Gioe TJ. A microbiological in-use comparison of surgical hand-washing agents. *J Bone Joint Surg Am* 1979;61-A:403–6.
103. Minakuchi K, Yamamoto Y, Matsunaga K, et al. The antiseptic effect of a quick drying rubbing type povidone-iodine alcoholic disinfectant solution. *Postgrad Med J* 1993;69(suppl 3):S23–S26.
104. Babb JR, Davies JG, Ayliffe GAJ. A test procedure for evaluating surgical hand disinfection. *J Hosp Infect* 1991;18(suppl B):41–9.
105. Bellamy K, Alcock R, Babb JR, Davies JG, Ayliffe GA. A test for the assessment of 'hygienic' hand disinfection using rotavirus. *J Hosp Infect* 1993;24:201–10.
106. Ayliffe GAJ, Babb JR, Quraishi AH. A test for 'hygienic' hand disinfection. *J Clin Pathol* 1978;31:923–8.
107. Lilly HA, Lowbury EJJ, Wilkins MD. Detergents compared with each other and with antiseptics as skin 'degerming' agents. *J Hyg (Lond)* 1979;82:89–93.

108. Ulrich JA. Clinical study comparing hibistat (0.5% chlorhexidine gluconate in 70% isopropyl alcohol) and betadine surgical scrub (7.5% povidone-iodine) for efficacy against experimental contamination of human skin. *Curr Ther Res* 1982;31:27–30.
109. Bartzokas CA, Gibson MF, Graham R, Pinder DC. A comparison of triclosan and chlorhexidine preparations with 60 per cent isopropyl alcohol for hygienic hand disinfection. *J Hosp Infect* 1983;4:245–55.
110. Rotter ML, Koller W, Wewalka G, Werner HP, Ayliffe GAJ, Babb JR. Evaluation of procedures for hygienic hand-disinfection: controlled parallel experiments on the Vienna test model. *J Hyg (Lond)* 1986;96:27–37.
111. Kjrlen H, Andersen BM. Handwashing and disinfection of heavily contaminated hands—effective or ineffective? *J Hosp Infect* 1992;21:61–71.
112. Namura S, Nishijima S, Asada Y. An evaluation of the residual activity of antiseptic handrub lotions: an ‘in use’ setting study. *J Dermatol* 1994;21:481–5.
113. Jarvis JD, Wynne CD, Enwright L, Williams JD. Handwashing and antiseptic-containing soaps in hospital. *J Clin Path* 1979;32:732–7.
114. Pereira LJ, Lee GM, Wade KJ. An evaluation of five protocols for surgical handwashing in relation to skin condition and microbial counts. *J Hosp Infect* 1997;36:49–65.
115. Larson EL, Butz AM, Gullette DL, Laughon BA. Alcohol for surgical scrubbing? *Infect Control Hosp Epidemiol* 1990;11:139–43.
116. Aly R, Maibach HI. Comparative study on the antimicrobial effect of 0.5% chlorhexidine gluconate and 70% isopropyl alcohol on the normal flora of hands. *Appl Environ Microbiol* 1979;37:610–3.
117. Galle PC, Homesley HD, Rhyne AL. Reassessment of the surgical scrub. *Surg Gynecol Obstet* 1978;147:215–8.
118. Rosenberg A, Alatary SD, Peterson AF. Safety and efficacy of the antiseptic chlorhexidine gluconate. *Surg Gynecol Obstet* 1976;143:789–92.
119. Ayliffe GAJ, Babb JR, Bridges K, et al. Comparison of two methods for assessing the removal of total organisms and pathogens from the skin. *J Hyg (Lond)* 1975;75:259–74.
120. Larson EL, Morton HE. Alcohols [Chapter 11]. In: Block SS, ed. *Disinfection, sterilization and preservation*. 4th ed. Philadelphia, PA: Lea and Febiger, 1991:642–54.
121. Price PB. Ethyl alcohol as a germicide. *Arch Surg* 1939;38:528–42.
122. Harrington C, Walker H. The germicidal action of alcohol. *Boston Medical and Surgical Journal* 1903;148:548–52.
123. Price PB. New studies in surgical bacteriology and surgical technic. *JAMA* 1938;111:1993–6.
124. Coulthard CE, Sykes G. The germicidal effect of alcohol with special reference to its action on bacterial spores. *Pharmaceutical Journal* 1936;137:79–81.
125. Pohle WD, Stuart LS. The germicidal action of cleaning agents—a study of a modification of Price’s procedure. *J Infect Dis* 1940;67:275–81.
126. Gardner AD. Rapid disinfection of clean unwashed skin: further experiments. *Lancet* 1948;760–3.
127. Sakuragi T, Yanagisawa K, Dan K. Bactericidal activity of skin disinfectants on methicillin-resistant *Staphylococcus aureus*. *Anesth Analg* 1995;81:555–8.
128. Kampf G, Jarosch R, Rüden H. Limited effectiveness of chlorhexidine based hand disinfectants against methicillin-resistant *Staphylococcus aureus* (MRSA). *J Hosp Infect* 1998;38:297–303.
129. Kampf G, Höfer M, Wendt C. Efficacy of hand disinfectants against vancomycin-resistant enterococci in vitro. *J Hosp Infect* 1999;42:143–50.
130. Platt J, Bucknall RA. The disinfection of respiratory syncytial virus by isopropanol and a chlorhexidine-detergent handwash. *J Hosp Infect* 1985;6:89–94.
131. Krilov LR, Harkness SH. Inactivation of respiratory syncytial virus by detergents and disinfectants. *Pediatr Infect Dis* 1993;12:582–4.
132. Sattar SA, Tetro J, Springthorpe VS, Giulivi A. Preventing the spread of hepatitis B and C viruses: where are germicides relevant? *Am J Infect Control* 2001;29:187–97.
133. Woolwine JD, Gerberding JL. Effect of testing method on apparent activities of antiviral disinfectants and antiseptics. *Antimicrob Agents Chemother* 1995;39:921–3.
134. Pillsbury DM, Livingood CS, Nichols AC. Bacterial flora of the normal skin: report on the effect of various ointment and solutions, with comments on the clinical significance of this study. *Arch Dermatol* 1942;45:61–80.
135. Lowbury EJJ, Lilly HA, Ayliffe GAJ. Preoperative disinfection of surgeons’ hands: use of alcoholic solutions and effects of gloves on skin flora. *Br Med J* 1974;4:369–72.
136. Lilly HA, Lowbury EJJ, Wilkins MD, Zaggy A. Delayed antimicrobial effects of skin disinfection by alcohol. *J Hyg (Lond)* 1979;82:497–500.
137. Ansari SA, Springthorpe VS, Sattar SA, Tostowaryk W, Wells GA. Comparison of cloth, paper, and warm air drying in eliminating viruses and bacteria from washed hands. *Am J Infect Control* 1991;19:243–9.
138. Ansari SA, Sattar SA, Springthorpe VS, Wells GA, Tostowaryk W. In vivo protocol for testing efficacy of hand-washing agents against viruses and bacteria: experiments with rotavirus and *Escherichia coli*. *Appl Environ Microbiol* 1989;55:3113–8.
139. Steinmann J, Nehrkorn R, Meyer A, Becker K. Two in-vivo protocols for testing virucidal efficacy of handwashing and hand disinfection. *Zentralbl Hyg Umweltmed.* 1995;196:425–36.
140. Mbithi JN, Springthorpe VS, Sattar SA. Comparative in vivo efficiencies of hand-washing agents against hepatitis A virus (HM-175) and poliovirus type 1 (Sabin). *Appl Environ Microbiol* 1993;59:3463–9.
141. Schurmann W, Eggers HJ. Antiviral activity of an alcoholic hand disinfectant: comparison of the in vitro suspension test with in vivo experiments on hands, and on individual fingertips. *Antiviral Res* 1983;3:25–41.
142. Larson E, Bobo L. Effective hand degerming in the presence of blood. *J Emerg Med* 1992;10:7–11.
143. Dineen P, Hildick-Smith G. Antiseptic care of the hands [Chapter 21]. In: Maibach HI, Hildick-Smith G, eds. *Skin bacteria and their role in infection*. New York: McGraw-Hill, 1965.
144. Lilly HA, Lowbury EJJ. Transient skin flora: their removal by cleansing or disinfection in relation to their mode of deposition. *J Clin Path* 1978;31:919–22.
145. Rotter M, Koller W, Wewalka G. Povidone-iodine and chlorhexidine gluconate-containing detergents for disinfection of hands. *J Hosp Infect* 1980;1:149–58.
146. Rotter ML. Hygienic hand disinfection. *Infect Control* 1984;1:18–22.
147. Blech MF, Hartemann P, Paquin JL. Activity of non antiseptic soaps and ethanol for hand disinfection. *Zentralbl Bakteriolog Hyg [B]* 1985;181:496–512.

148. Leyden JJ, McGinley KJ, Kaminer MS, et al. Computerized image analysis of full-hand touch plates: a method for quantification of surface bacteria on hands and the effect of antimicrobial agents. *J Hosp Infect* 1991;18(suppl B):13–22.
149. Rotter ML, Koller W. Test models for hygienic handrub and hygienic handwash: the effects of two different contamination and sampling techniques. *J Hosp Infect* 1992;20:163–71.
150. Zaragoza M, Sallés M, Gomez J, Bayas JM, Trilla A. Handwashing with soap or alcoholic solutions? A randomized clinical trial of its effectiveness. *Am J Infect Control* 1999;27:258–61.
151. Paulson DS, Fendler EJ, Dolan MJ, Williams RA. A close look at alcohol gel as an antimicrobial sanitizing agent. *Am J Infect Control* 1999;27:332–8.
152. Cardoso CL, Pereira HH, Zequim JC, Guilhermetti M. Effectiveness of hand-cleansing agents for removing *Acinetobacter baumannii* strain from contaminated hands. *Am J Infect Control* 1999;27:327–31.
153. Casewell MW, Law MM, Desai N. A laboratory model for testing agents for hygienic hand disinfection: handwashing and chlorhexidine for the removal of klebsiella. *J Hosp Infect* 1988;12:163–75.
154. Wade JJ, Desai N, Casewell MW. Hygienic hand disinfection for the removal of epidemic vancomycin-resistant *Enterococcus faecium* and gentamicin-resistant *Enterobacter cloacae*. *J Hosp Infect* 1991;18:211–8.
155. Huang Y, Oie S, Kamiya A. Comparative effectiveness of hand-cleansing agents for removing methicillin-resistant *Staphylococcus aureus* from experimentally contaminated fingertips. *Am J Infect Control* 1994;22:224–7.
156. Lowbury EJJ, Lilly HA. Disinfection of the hands of surgeons and nurses. *Br Med J* 1960;1:5184.
157. Berman RE, Knight RA. Evaluation of hand antisepsis. *Arch Environ Health* 1969;18:781–3.
158. Rotter ML, Simpson RA, Koller W. Surgical hand disinfection with alcohols at various concentrations: parallel experiments using the new proposed European standards method. *Infect Control Hosp Epidemiol* 1998;19:778–81.
159. Hobson DW, Woller W, Anderson L, Guthery E. Development and evaluation of a new alcohol-based surgical hand scrub formulation with persistent antimicrobial characteristics and brushless application. *Am J Infect Control* 1998;26:507–12.
160. Jones MV, Rowe GB, Jackson B, Pritchard NJ. The use of alcoholic paper wipes for routine hand cleansing: results of trials in two hospitals. *J Hosp Infect* 1986;8:268–74.
161. Butz AM, Laughon BE, Gullette DL, Larson EL. Alcohol-impregnated wipes as an alternative in hand hygiene. *Am J Infect Control* 1990;18:70–6.
162. Ojajarvi J. Handwashing in Finland. *J Hosp Infect* 1991;18(suppl B):35–40.
163. Newman JL, Seitz JC. Intermittent use of an antimicrobial hand gel for reducing soap-induced irritation of health care personnel. *Am J Infect Control* 1990;18:194–200.
164. Rotter ML, Koller W, Neumann R. The influence of cosmetic additives on the acceptability of alcohol-based hand disinfectants. *J Hosp Infect* 1991;18 (suppl B):57–63.
165. Larson EL, Aiello AE, Heilman JM, et al. Comparison of different regimens for surgical hand preparation. *AORN J* 2001;73:412–20.
166. Larson EL, Aiello AE, Bastyr J, et al. Assessment of two hand hygiene regimens for intensive care unit personnel. *Crit Care Med* 2001;29:944–51.
167. Ophaswongse S, Maibach HI. Alcohol dermatitis: allergic contact dermatitis and contact urticaria syndrome: a review. *Contact Dermatitis* 1994;30:1–6.
168. Rilliet A, Hunziker N, Brun R. Alcohol contact urticaria syndrome (immediate-type hypersensitivity): case report. *Dermatologica* 1980;161:361–4.
169. Widmer AF. Replace hand washing with use of a waterless alcohol hand rub? *Clin Infect Dis* 2000;31:136–43.
170. Bryant KA, Pearce J, Stover B. Flash fire associated with the use of alcohol-based antiseptic agent [Letter]. *Am J Infect Control* 2002;30:256–7.
171. Hsueh PR, Teng LJ, Yang PC, Pan HL, Ho SW, Luh KT. Nosocomial pseudoepidemic caused by *Bacillus cereus* traced to contaminated ethyl alcohol from a liquor factory. *J Clin Microbiol* 1999;37:2280–4.
172. Denton GW. Chlorhexidine [Chapter 16]. In: Block SS, ed. *Disinfection, sterilization and reservation*. 4th ed. Philadelphia, PA: Lea and Febiger, 1991.
173. Narang HK, Codd AA. Action of commonly used disinfectants against enteroviruses. *J Hosp Infect* 1983;4:209–12.
174. Walsh B, Blakemore PH, Drabu YJ. The effect of handcream on the antibacterial activity of chlorhexidine gluconate. *J Hosp Infect* 1987;9:30–3.
175. Lowbury EJJ, Lilly HA. Use of 4% chlorhexidine detergent solution (Hibiscrub) and other methods of skin disinfection. *Br Med J* 1973;1:510–5.
176. Paulson DS. Comparative evaluation of five surgical hand scrub preparations. *AORN J* 1994;60:246–56.
177. Stringeni L, Lapomarda V, Lisi P. Occupational hand dermatitis in hospital environments. *Contact Dermatitis* 1995;33:172–6.
178. Marrie TJ, Costerton JW. Prolonged survival of *Serratia marcescens* in chlorhexidine. *Appl Environ Microbiol* 1981;42:1093–102.
179. McAllister TA, Lucas CE, Mocan H, et al. *Serratia marcescens* outbreak in a paediatric oncology unit traced to contaminated chlorhexidine. *Scott Med J* 1989;34:525–8.
180. Vigeant P, Loo VG, Bertrand C, et al. An outbreak of *Serratia marcescens* infections related to contaminated chlorhexidine. *Infect Control Hosp Epidemiol* 1998;19:791–4.
181. Vu-Thien H, Darbord JC, Moissenet D, et al. Investigation of an outbreak of wound infections due to *Alcaligenes xylosoxidans* transmitted by chlorhexidine in a burns unit. *Eur J Clin Microbiol* 1998;17:724–6.
182. Larson E, Talbot GH. An approach for selection of health care personnel handwashing agents. *Infect Control* 1986;7:419–24.
183. Davies J, Babb JR, Ayliffe GAJ, Wilkins MD. Disinfection of the skin of the abdomen. *Br J Surg* 1978;65:855–8.
184. Larson E, Mayur K, Laughon BA. Influence of two handwashing frequencies on reduction in colonizing flora with three handwashing products used by health care personnel. *Am J Infect Control* 1988;17:83–8.
185. Soulsby ME, Barnett JB, Maddox S. Brief report: the antiseptic efficacy of chlorxylenol-containing vs. chlorhexidine gluconate-containing surgical scrub preparations. *Infect Control* 1986;7:223–6.
186. Aly R, Maibach HI. Comparative antibacterial efficacy of a 2-minute surgical scrub with chlorhexidine gluconate, povidone-iodine, and chloroxylenol sponge-brushes. *Am J Infect Control* 1988;16:173–7.
187. Archibald LK, Corl A, Shah B, et al. *Serratia marcescens* outbreak associated with extrinsic contamination of 1% chlorxylenol soap. *Infect Control Hosp Epidemiol* 1997;18:704–9.
188. Lowbury EJJ, Lilly HA, Bull JP. Disinfection of hands: removal of resident bacteria. *Br Med J* 1963;1:1251–6.

189. Kundsins RB, Walter CW. The surgical scrub—practical consideration. *Arch Surg* 1973;107:75–7.
190. Lockhart J. How toxic is hexachlorophene? *Pediatrics* 1972;50:229–35.
191. Shuman RM, Leech RW, Alvord EC Jr. Neurotoxicity of hexachlorophene in humans: II. a clinicopathological study of 46 premature infants. *Arch Neurol* 1975;32:320–5.
192. Dixon RE, Kaslow RA, Mallison GF, Bennett JV. Staphylococcal disease outbreaks in hospital nurseries in the United States—December 1971 through March 1972. *Pediatrics* 1973;51:413–6.
193. Kaslow RA, Dixon RE, Martin SM, et al. Staphylococcal disease related to hospital nursery bathing practices—a nationwide epidemiologic investigation. *Pediatrics* 1973;51:418–29.
194. American Academy of Pediatrics, American College of Obstetricians and Gynecologists. Guidelines for perinatal care. 4th ed. Elk Grove Village, IL: American Academy of Pediatrics; Washington, DC: American Academy of Obstetricians and Gynecologists, 1997.
195. Gottardi W. Iodine and iodine compounds [Chapter 8]. In: Block SS, ed. *Disinfection, sterilization and preservation*. 4th ed. Philadelphia, PA: Lea & Febiger; 1991.
196. Anderson RL. Iodophor antiseptics: intrinsic microbial contamination with resistant bacteria. *Infect Control Hosp Epidemiol* 1989;10:443–6.
197. Goldenheim PD. In vitro efficacy of povidone-iodine solution and cream against methicillin-resistant *Staphylococcus aureus*. *Postgrad Med J* 1993;69(suppl 3):S62–S65.
198. Traoré O, Fayard SF, Laveran H. An in-vitro evaluation of the activity of povidone-iodine against nosocomial bacterial strains. *J Hosp Infect* 1996;34:217–22.
199. McLure AR, Gordon J. In-vitro evaluation of povidone-iodine and chlorhexidine against methicillin-resistant *Staphylococcus aureus*. *J Hosp Infect* 1992;21:291–9.
200. Davies JG, Babb JR, Bradley CR, Ayliffe GAJ. Preliminary study of test methods to assess the virucidal activity of skin disinfectants using poliovirus and bacteriophages. *J Hosp Infect* 1993;25:125–31.
201. Rotter ML. Chapter 79/Hand washing and hand disinfection. In: Mayhall CG, ed. *Hospital Epidemiology and Infection Control*. Baltimore, MD: Williams & Wilkins, 1996.
202. Wade JJ, Casewell MW. The evaluation of residual antimicrobial activity on hands and its clinical relevance. *J Hosp Infect* 1991;18 (suppl B):23–8.
203. Aly R, Maibach HI. Comparative evaluation of chlorhexidine gluconate (Hibiclens[®]) and povidone-iodine (E-Z Scrub[®]) sponge/brushes for presurgical hand scrubbing. *Curr Ther Res* 1983;34:740–5.
204. Herruzo-Cabrera R, Vizcaino-Alcaide MJ, Fdez-AciZero MJ. Usefulness of an alcohol solution of N-duopropenide for the surgical antisepsis of the hands compared with handwashing with iodine-povidone and chlorhexidine: clinical essay. *J Surgical Research* 2000;94:6–12.
205. Hingst V, Juditzki I, Heeg P, Sonntag HG. Evaluation of the efficacy of surgical hand disinfection following a reduced application time of 3 instead of 5 min. *J Hosp Infect* 1992;20:79–86.
206. Faoagali J, Fong J, George N, Mahoney P, O'Rourke V. Comparison of the immediate, residual, and cumulative antibacterial effects of Novaderm R, Novascrub R, Betadine Surgical Scrub, Hibiclens, and liquid soap. *Am J Infect Control* 1995;23:337–43.
207. Pereira LJ, Lee GM, Wade KJ. The effect of surgical handwashing routines on the microbial counts of operating room nurses. *Am J Infect Control* 1990;18:354–64.
208. Peterson AF, Rosenberg A. Comparative evaluation of surgical scrub preparations. *Surg Gynecol Obstet* 1978;146:63–5.
209. Berkelman RL, Holland BW, Anderson RL. Increased bactericidal activity of dilute preparations of povidone-iodine solutions. *J Clin Microbiol* 1982;15:635–9.
210. Merianos JJ. Quaternary ammonium antimicrobial compounds [Chapter 13]. In: Block SS, ed. *Disinfection, Sterilization, and Preservation*. 4th ed. Philadelphia, PA: Lea and Febiger; 1991.
211. Dixon RE, Kaslow RA, Mackel DC, Fulkerson CC, Mallison GF. Aqueous quaternary ammonium antiseptics and disinfectants: use and misuse. *JAMA* 1976;236:2415–7.
212. Sautter RL, Mattman LH, Legaspi RC. *Serratia marcescens* meningitis associated with a contaminated benzalkonium chloride solution. *Infect Control* 1984;5:223–5.
213. Oie S, Kamiya A. Microbial contamination of antiseptics and disinfectants. *Am J Infect Control* 1996;24:389–95.
214. Hayes RA, Trick WE, Vernon MO, et al. Comparison of three hand hygiene (HH) methods in a surgical intensive care unit (SICU) [Abstract K-1337]. Presented at the 41st Interscience Conference on Antimicrobial Agents and Chemotherapy Chicago, IL: American Society for Microbiology, 2001.
215. Dyer DL, Gerenraich KB, Wadhams PS. Testing a new alcohol-free hand sanitizer to combat infection. *AORN J* 1998;68:239–51.
216. Jones RD, Jampani HB, Newman JL, Lee AS. Triclosan: a review of effectiveness and safety in health care settings. *Am J Infect Control* 2000;28:184–96.
217. Ward WH, Holdgate GA, Rowsell S, et al. Kinetic and structural characteristics of the inhibition of enoyl (acyl carrier protein) reductase by triclosan. *Biochemistry* 1999;38:12514–25.
218. Heath RJ, Li J, Roland GE. Inhibition of the *Staphylococcus aureus* NADPH-dependent enoyl-acyl carrier protein reductase by triclosan and hexachlorophene. *J Biol Chem* 2000;275:4654–9.
219. Faoagali JL, George N, Fong J, Davy J, Dowser M. Comparison of the antibacterial efficacy of 4% chlorhexidine gluconate and 1% triclosan handwash products in an acute clinical ward. *Am J Infect Control* 1999;27:320–6.
220. Barry MA, Craven DE, Goularte TA, Lichtenberg DA. *Serratia marcescens* contamination of antiseptic soap containing triclosan: implications for nosocomial infection. *Infect Control* 1984;5:427–30.
221. Lowbury EJL, Lilly HA, Bull JP. Disinfection of hands: removal of transient organisms. *Br Med J* 1964;2:230–3.
222. Rotter ML. Semmelweis' sesquicentennial: a little-noted anniversary of handwashing. *Current Opinion in Infectious Diseases* 1998;11:457–60.
223. Manivannan G, Brady MJ, Cahalan PT, et al. Immediate, persistent and residual antimicrobial efficiency of Surfacine[™] hand sanitizer [Abstract]. *Infection Control Hosp Epidemiol* 2000;21:105.
224. Gershenfeld L. Povidone-iodine as a sporicide. *Am J Pharm* 1962;134:79–81.
225. Russell AD. Chemical sporicidal and sporostatic agents [Chapter 22]. In: Block SS, ed. *Disinfection, sterilization and preservation*. 4th ed. Philadelphia, PA: Lea and Febiger, 1991.
226. Johnson S, Gerding DN, Olson MM, et al. Prospective, controlled study of vinyl glove use to interrupt *Clostridium difficile* nosocomial transmission. *Am J Med* 1990;88:137–40.
227. Russell AD. Mechanisms of bacterial insusceptibility to biocides. *Am J Infect Control* 2001;29:259–61.
228. Cookson BD, Bolton MC, Platt JH. Chlorhexidine resistance in methicillin-resistant *Staphylococcus aureus* or just an elevated MIC? An in vitro and in vivo assessment. *Antimicrob Agents Chemother* 1991;35:1997–2002.

229. McMurry LM, Oethinger M, Levy SB. Overexpression of *marA*, *soxS*, or *acrAB* produces resistance to triclosan in laboratory and clinical strains of *Escherichia coli*. *FEMS Microbiol Lett* 1998;166:305–9.
230. Chuanchuen R, Beinlich K, Hoang TT, et al. Cross-resistance between triclosan and antibiotics in *Pseudomonas aeruginosa* is mediated by multidrug efflux pumps: exposure of a susceptible mutant strain to triclosan selects *nfxB* mutants overexpressing MexCD-OprJ. *Antimicrob Agents Chemother* 2001;45:428–32.
231. Gröschel DHM, Pruett TL. Surgical antisepsis [Chapter 36]. In: Block SS, ed. *Disinfection, sterilization and preservation*. 4th ed. Philadelphia, PA: Lea and Febiger, 1991.
232. Boyce JM, Potter-Bynoe G, Opal SM, Dziobek L, Medeiros AA. A common-source outbreak of *Staphylococcus epidermidis* infections among patients undergoing cardiac surgery. *J Infect Dis* 1990;161:493–9.
233. Dewar NE, Gravens DL. Effectiveness of seprisol antiseptic foam as a surgical scrub agent. *Appl Microbiol* 1973;26:544–9.
234. Grinbaum RS, de Mendonça JS, Cardo DM. An outbreak of handscrubbing-related surgical site infections in vascular surgical procedures. *Infect Control Hosp Epidemiol* 1995;16:198–202.
235. AORN Recommended Practices Committee. Recommended practices for surgical hand scrubs. In: Fogg D, Parker N, Shevlin D, eds. *Standards, Recommended Practices, and Guidelines*. Denver, CO: AORN, 2001.
236. Larson E, Anderson JK, Baxendale L, Bobo L. Effects of a protective foam on scrubbing and gloving. *Am J Infect Control* 1993;21:297–301.
237. Mulberry G, Snyder AT, Heilman J, Pyrek J, Stahl J. Evaluation of a waterless, scrubless chlorhexidine gluconate/ethanol surgical scrub for antimicrobial efficacy. *Am J Infect Control* 2001;29:377–82.
238. Dineen P. An evaluation of the duration of the surgical scrub. *Surg Gynecol Obstet* 1969;129:1181–4.
239. O'Farrell DA, Kenny G, O'Sullivan M, Nicholson P, Stephens M, Hone R. Evaluation of the optimal hand-scrub duration prior to total hip arthroplasty. *J Hosp Infect* 1994;26:93–8.
240. O'Shaughnessy M, O'Malley VP, Corbett G, Given HF. Optimum duration of surgical scrub-time [Short note]. *Br J Surg* 1991;78:685–6.
241. Wheelock SM, Lookinland S. Effect of surgical hand scrub time on subsequent bacterial growth. *AORN J* 1997;65:1087–98.
242. Deshmukh N, Kjellberg SI, Kramer JW. A comparison of 5-minute povidone-iodine scrub and 1-minute povidone-iodine scrub followed by alcohol foam. *Military Medicine* 1998;163:145–7.
243. Kikuchi-Numagami K, Saishu T, Fukaya M, Kanazawa E, Tagami H. Irritancy of scrubbing up for surgery with or without a brush. *Acta Derm Venereol* 1999;79:230–2.
244. Dineen P. The use of a polyurethane sponge in surgical scrubbing. *Surg Gynecol Obstet* 1966;123:595–8.
245. Bornside GH, Crowder VH Jr, Cohn I Jr. A bacteriological evaluation of surgical scrubbing with disposable iodophor-soap impregnated polyurethane scrub sponges. *Surgery* 1968;64:743–51.
246. McBride ME, Duncan WC, Knox JM. An evaluation of surgical scrub brushes. *Surg Gynecol Obstet* 1973;137:934–6.
247. Berman RE, Knight RA. Evaluation of hand antisepsis. *Arch Environ Health* 1969;18:781–3.
248. Loeb MB, Wilcox L, Smaill F, Walter S, Duff Z. A randomized trial of surgical scrubbing with a brush compared to antiseptic soap alone. *Am J Infect Control* 1997;25:11–5.
249. Larson E, Friedman C, Cohran J, Treston-Aurand J, Green S. Prevalence and correlates of skin damage on the hands of nurses. *Heart Lung* 1997;26:404–12.
250. Tupker RA. Detergents and cleansers [Chapter 7]. In: van der Valk PGM, Maibach HI, eds. *The Irritant Contact Dermatitis Syndrome*. New York, NY: CRC Press, 1996.
251. Wilhelm KP. Prevention of surfactant-induced irritant contact dermatitis. *Curr Probl Dermatol* 1996;25:78–85.
252. de Haan P, Meester HHM, Bruynzeel DP. Irritancy of alcohols [Chapter 6]. In: van der Valk PGM, Maibach HI, eds. *The Irritant Contact Dermatitis Syndrome*. New York, NY: CRC Press, 1996.
253. Lübke J, Ruffieux C, van Melle G, Perrenoud D. Irritancy of the skin disinfectant n-propanol. *Contact Dermatitis* 2001;45:226–31.
254. qhlenschlaeger J, Friberg J, Ramsing D, Agner T. Temperature dependency of skin susceptibility to water and detergents. *Acta Derm Venereol* 1996;76:274–6.
255. Emilson A, Lindberg M, Forslind B. The temperature effect of in vitro penetration of sodium lauryl sulfate and nickel chloride through human skin. *Acta Derm Venereol* 1993;73:203–7.
256. de Groot AC. Contact allergy to cosmetics: causative ingredients. *Contact Dermatitis* 1987;17:26–34.
257. Schnuch A, Uter W, Geier J, Frosch PJ, Rustemeyer T. Contact allergies in healthcare workers—results from the IVDK. *Acta Derm Venereol* 1998;78:358–63.
258. Rastogi SC, Heydorn S, Johansen JD, Basketter DA. Fragrance chemicals in domestic and occupational products. *Contact Dermatitis* 2001;45:221–5.
259. Uter W, Schnuch A, Geier J, Pfahlberg A, Gefeller O. Association between occupation and contact allergy to the fragrance mix: a multifactorial analysis of national surveillance data. *Occup Environ Med* 2001;58:392–8.
260. Perrenoud D, Bircher A, Hunziker T, et al. Frequency of sensitization to 13 common preservatives in Switzerland. *Contact Dermatitis* 1994;30:276–9.
261. Kiec-Swierczynska M, Krecisz B. Occupational skin diseases among the nurses in the region of Lodz. *Int J Occup Med Environ Health* 2000;13:179–84.
262. Garvey LH, Roed-Petersen J, and Husum B. Anaphylactic reactions in anaesthetised patients—four cases of chlorhexidine allergy. *Acta Anaesthesiol Scand* 2001;45:1290–4.
263. Pham, NH, Weiner JM, Reisner GS, and Baldo BA. Anaphylaxis to chlorhexidine. Case report. Implication of immunoglobulin E antibodies and identification of an allergenic determinant. *Clin Exp Allergy* 2000;30:1001–7.
264. Nishioka K, Seguchi T, Yasuno H, Yamamoto T, Tominaga K. The results of ingredient patch testing in contact dermatitis elicited by povidone-iodine preparations. *Contact Dermatitis* 2000;42:90–4.
265. Wong CSM, Beck MH. Allergic contact dermatitis from triclosan in antibacterial handwashes. *Contact Dermatitis* 2001;45:307.
266. Guin JD, Goodman J. Contact urticaria from benzyl alcohol presenting as intolerance to saline soaks. *Contact Dermatitis* 2001;45:182–3.
267. Podda M, Zollner T, Grundmann-Kollmann M, Kaufmann R, Boehncke WF. Allergic contact dermatitis from benzyl alcohol during topical antimycotic treatment. *Contact Dermatitis* 1999;41:302–3.
268. Yesudian PD, King CM. Allergic contact dermatitis from stearyl alcohol in Efudix[®] cream. *Contact Dermatitis* 2001;45:313–4.

269. Aust LB, Maibach HI. Incidence of human skin sensitization to isostearyl alcohol in two separate groups of panelists. *Contact Dermatitis* 1980;6:269–71.
270. Funk JO, Maibach HI. Propylene glycol dermatitis: re-evaluation of an old problem. *Contact Dermatitis* 1994;31:236–41.
271. Hannuksela M. Moisturizers in the prevention of contact dermatitis. *Curr Probl Dermatol* 1996;25:214–20.
272. Berndt U, Wigger-Alberti W, Gabard B, Elsner P. Efficacy of a barrier cream and its vehicle as protective measures against occupational irritant contact dermatitis. *Contact Dermatitis* 2000;42:77–80.
273. McCormick RD, Buchman TL, Maki DG. Double-blind, randomized trial of scheduled use of a novel barrier cream and an oil-containing lotion for protecting the hands of health care workers. *Am J Infect Control* 2000;28:302–10.
274. Larson E, Killien M. Factors influencing handwashing behavior of patient care personnel. *Am J Infect Control* 1982;10:93–9.
275. Zimakoff J, Kjelsberg AB, Larsen SO, Holstein B. A multicenter questionnaire investigation of attitudes toward hand hygiene, assessed by the staff in fifteen hospitals in Denmark and Norway. *Am J Infect Control* 1992;20:58–64.
276. Mayer JA, Dubbert PM, Miller M, Burkett PA, Chapman SW. Increasing handwashing in an intensive care unit. *Infect Control* 1986;7:259–62.
277. Ojajärvi J. The importance of soap selection for routine hand hygiene in hospital. *J Hyg (Lond)* 1981;86:275–83.
278. Scott D, Barnes A, Lister M, Arkell P. An evaluation of the user acceptability of chlorhexidine handwash formulations. *J Hosp Infect* 1991;18(suppl B):51–5.
279. Taylor LJ. An evaluation of handwashing techniques—2. *Nursing Times* 1978;74:108–10.
280. Preston GA, Larson EL, Stamm WE. The effect of private isolation rooms on patient care practices, colonization and infection in an intensive care unit. *Am J Med* 1981;70:641–5.
281. Kaplan LM, McGuckin M. Increasing handwashing compliance with more accessible sinks. *Infect Control* 1986;7:408–10.
282. Freeman, J. Prevention of nosocomial infections by location of sinks for hand washing adjacent to the bedside [Abstract 60]. In: Program and abstracts of the 33rd Interscience Conference on Antimicrobial Agents and Chemotherapy. Washington, DC: American Society for Microbiology, 1993:130.
283. Bischoff WE, Reynolds TM, Sessler CN, Edmond MB, Wenzel RP. Handwashing compliance by health care workers. The impact of introducing an accessible, alcohol-based hand antiseptic. *Arch Intern Med* 2000;160:1017–21.
284. Pittet D. Compliance with hand disinfection and its impact on hospital-acquired infections. *J Hosp Infect* 2001;48(suppl A):S40–S46.
285. Wurtz R, Moye G, Jovanovic B. Handwashing machines, handwashing compliance, and potential for cross-contamination. *Am J Infect Control* 1994;22:228–30.
286. Kohan C, Ligi C, Dumigan DG, Boyce JM. The importance of evaluating product dispensers when selecting alcohol-based handrubs. *Am J Infect Control* 2002 (in press).
287. Boyce JM. Antiseptic technology: access, affordability, and acceptance. *Emerg Infect Diseases* 2001;7:231–3.
288. Taylor LJ. An evaluation of handwashing techniques—1. *Nursing Times* 1978:54–5.
289. Albert RK, Condie F. Hand-washing patterns in medical intensive-care units. *N Engl J Med* 1981;304:1465–6.
290. Larson E. Compliance with isolation technique. *Am J Infect Control* 1983;11:221–5.
291. Donowitz LG. Handwashing technique in a pediatric intensive care unit. *Am J Dis Child* 1987;141:683–5.
292. Conly JM, Hill S, Ross J, Lertzman J, Loule TJ. Handwashing practices in an intensive care unit: the effects of an educational program and its relationship to infection rates. *Am J Infect Control* 1989;17:330–9.
293. DeCarvalho M, Lopes JMA, Pellitteri M. Frequency and duration of handwashing in a neonatal intensive care unit. *Pediatr Infect Dis J* 1989;8:179–80.
294. Graham M. Frequency and duration of handwashing in an intensive care unit. *Am J Infect Control* 1990;18:77–80.
295. Dubbert PM, Dolce J, Richter W, Miller M, Chapman SW. Increasing ICU staff handwashing: effects of education and group feedback. *Infect Control Hosp Epidemiol* 1990;11:191–3.
296. Simmons B, Bryant J, Neiman K, Spencer L, Arheart K. The role of handwashing in prevention of endemic intensive care unit infections. *Infect Control Hosp Epidemiol* 1990;11:589–94.
297. Pettinger A, Nettleman MD. Epidemiology of isolation precautions. *Infect Control Hosp Epidemiol* 1991;12:303–7.
298. Lohr JA, Ingram DL, Dudley SM, Lawton EL, Donowitz LG. Hand washing in pediatric ambulatory settings: an inconsistent practice. *Am J Dis Child* 1991;145:1198–9.
299. Raju TNK, Kobler C. Improving handwashing habits in the newborn nurseries. *Am J Med.Sci* 1991;302:355–8.
300. Larson EL, McGinley KJ, Foglia A, et al. Handwashing practices and resistance and density of bacterial hand flora on two pediatric units in Lima, Peru. *Am J Infect Control* 1992;20:65–72.
301. Zimakoff J, Stormark M, Larsen SO. Use of gloves and handwashing behaviour among health care workers in intensive care units. A multicentre investigation in four hospitals in Denmark and Norway. *J Hosp Infect* 1993;24:63–7.
302. Pelke S, Ching D, Easa D, Melish ME. Gowning does not affect colonization or infection rates in a neonatal intensive care unit. *Arch Pediatr Adolesc Med* 1994;148:1016–20.
303. Gould D. Nurses' hand decontamination practice: results of a local study. *J Hosp Infect* 1994;28:15–30.
304. Shay DK, Maloney SA, Montecalvo M, et al. Epidemiology and mortality risk of vancomycin-resistant enterococcal bloodstream infections. *J Infect Dis* 1995;172:993–1000.
305. Berg DE, Hershov RC, Ramirez CA. Control of nosocomial infections in an intensive care unit in Guatemala City. *Clin Infect Dis* 1995;21:588–93.
306. Tibballs J. Teaching hospital medical staff to handwash. *Med J Aust* 1996;164:395–8.
307. Slaughter S, Hayden MK, Nathan C, et al. A comparison of the effect of universal use of gloves and gowns with that of glove use alone on acquisition of vancomycin-resistant enterococci in a medical intensive care unit. *Ann Intern Med* 1996;125:448–56.
308. Dorsey ST, Cydulka RK, Emerman CL. Is handwashing teachable?: failure to improve handwashing behavior in an urban emergency department. *Acad Emerg Med* 1996;3:360–5.
309. Watanakunakorn C, Wang C, Hazy J. An observational study of hand washing and infection control practices by healthcare workers. *Infect Control Hosp Epidemiol* 1998;19:858–60.

310. Avila-Agüero ML, UmaZa MA, Jiménez AL, Faingezicht I, París MM. Handwashing practices in a tertiary-care, pediatric hospital and the effect on an educational program. *Clin Perform Qual Health Care* 1998;6:70–2.
311. Kirkland KB, Weinstein JM. Adverse effects of contact isolation. *Lancet* 1999;354:1177–8.
312. Maury E, Alzieu M, Baudel JL, et al. Availability of an alcohol solution can improve hand disinfection compliance in an intensive care unit. *Am J Respir Crit Care Med* 2000;162:324–7.
313. Muto CA, Siström MG, Farr BM. Hand hygiene rates unaffected by installation of dispensers of a rapidly acting hand antiseptic. *Am J Infect Control* 2000;28:273–6.
314. Jarvis WR. Handwashing—the Semmelweis lesson forgotten? *Lancet* 1994;344:1311–2.
315. Larson E, Kretzer EK. Compliance with handwashing and barrier precautions. *J Hosp Infect* 1995;30(suppl):88–106.
316. Sproat LJ, Inglis TJJ. A multicentre survey of hand hygiene practice in intensive care units. *J Hosp Infect* 1994;26:137–48.
317. Kretzer EK, Larson EL. Behavioral interventions to improve infection control practices. *Am J Infect Control* 1998;26:245–53.
318. Voss A, Widmer AF. No time for handwashing!? Handwashing versus alcoholic rub: can we afford 100% compliance? *Infect Control Hosp Epidemiol* 1997;18:205–8.
319. Larson E. Handwashing and skin physiologic and bacteriologic aspects. *Infect Control* 1985;6:14–23.
320. Thompson BL, Dwyer DM, Ussery XT, Denman S, Vacek P, Schwartz B. Handwashing and glove use in a long-term care facility. *Infect Control Hosp Epidemiol* 1997;18:97–103.
321. Doebbeling BN, Pfaller MA, Houston AK, Wenzel RP. Removal of nosocomial pathogens from the contaminated glove. *Ann Intern Med* 1988;109:394–8.
322. McLane C, Chenelly S, Sylwestrak ML, Kirchoff KT. A nursing practice problem: failure to observe aseptic technique. *Am J Infect Control* 1983;11:178–82.
323. Pittet D. Improving compliance with hand hygiene in hospitals. *Infect Control Hosp Epidemiol* 2000;21:381–6.
324. Teare L, Handwashing Liaison Group. Hand washing: a modest measure—with big effects. *Br Med J* 1999;318:686.
325. Teare EL, Cookson B, French GL, et al. UK handwashing initiative. *J Hosp Infect* 1999;43:1–3.
326. Larson EL, Bryan JL, Adler LM, Blane C. A multifaceted approach to changing handwashing behavior. *Am J Infect Control* 1997;25:3–10.
327. Weeks A. Why I don't wash my hands between each patient contact [Letter]. *Br Med J* 1999;319:518.
328. Webster J. Handwashing in a neonatal intensive care nursery: product acceptability and effectiveness of chlorhexidine gluconate 4% and triclosan 1%. *J Hosp Infect* 1992;21:137–41.
329. Kelen GD, Green GB, Hexter DA, et al. Substantial improvement in compliance with universal precautions in an emergency department following institution of policy. *Arch Intern Med* 1991;151:2051–6.
330. Lundberg GD. Changing physician behavior in ordering diagnostic tests [Editorial]. *JAMA* 1998;280:2036.
331. Phillips DF. “New look” reflects changing style of patient safety enhancement. *JAMA* 1999;281:217–9.
332. Harbarth S, Martin Y, Rohner P, Henry N, Auckenthaler R, Pittet D. Effect of delayed infection control measures on a hospital outbreak of methicillin-resistant *Staphylococcus aureus*. *J Hosp Infect* 2000;46:43–9.
333. Early E, Battle K, Cantwell E, English J, Lavin JE, Larson E. Effect of several interventions on the frequency of handwashing among elementary public school children. *Am J Infect Control* 1998;26:263–9.
334. Butz AM, Larson E, Fosarelli P, Yolken R. Occurrence of infectious symptoms in children in day care homes. *Am J Infect Control* 1990;18:347–53.
335. Kimel LS. Handwashing education can decrease illness absenteeism. *J Sch Nurs* 1996;12:14–6, 18.
336. Master D, Hess Longe S, Dickson H. Scheduled hand washing in an elementary school population. *Fam Med* 1997;29:336–9.
337. Roberts L, Smith W, Jorm L, Patel M, Douglas RM, McGilchrist C. Effect of infection control measures on the frequency of upper respiratory infection in child care: a randomized, controlled trial. *Pediatrics* 2000;105:738–42.
338. Roberts L, Jorm L, Patel M, Smith W, Douglas RM, McGilchrist C. Effect of infection control measures on the frequency of diarrheal episodes in child care: a randomized, controlled trial. *Pediatrics* 2000;105:743–6.
339. Khan MU. Interruption of shigellosis by handwashing. *Trans R Soc Trop Med Hyg* 1982;76:164–8.
340. Shahid NS, Greenough WB, Samadi AR, Huq MI, Rahman N. Hand washing with soap reduces diarrhoea and spread of bacterial pathogens in a Bangladesh village. *J Diarrhoeal Dis Res* 1996;14:85–9.
341. Stanton BE, Clemens JD. An educational intervention for altering water-sanitation behaviors to reduce childhood diarrhea in urban Bangladesh. *Am J Epidemiol* 1987;125:292–301.
342. McGinley KJ, Larson EL, Leyden JJ. Composition and density of microflora in the subungual space of the hand. *J Clin Microbiol* 1988;26:950–3.
343. Hedderwick SA, McNeil SA, Lyons MJ, Kauffman CA. Pathogenic organisms associated with artificial fingernails worn by healthcare workers. *Infect Control Hosp Epidemiol* 2000;21:505–9.
344. Baumgardner CA, Maragos CS, Larson EL. Effects of nail polish on microbial growth of fingernails: dispelling sacred cows. *AORN J* 1993;58:84–8.
345. Wynd CA, Samstag DE, Lapp AM. Bacterial carriage on the fingernails of OR nurses. *AORN J* 1994;60:796–805.
346. Gross A, Cutright DE, D'Allessandro SM. Effect of surgical scrub on microbial population under the fingernails. *Am J Surg* 1979;138:463–7.
347. Pottinger J, Burns S, Manske C. Bacterial carriage by artificial versus natural nails. *Am J Infect Control* 1989;17:340–4.
348. McNeil SA, Foster CL, Hedderwick SA, Kauffman CA. Effect of hand cleansing with antimicrobial soap or alcohol-based gel on microbial colonization of artificial fingernails worn by health care workers. *Clin Infect Dis* 2001;32:367–72.
349. Rubin DM. Prosthetic fingernails in the OR. *AORN J* 1988;47:944–5, 948.
350. Moolenaar RL, Crutcher M, San Joaquin VH, et al. A prolonged outbreak of *Pseudomonas aeruginosa* in a neonatal intensive care unit: did staff fingernails play a role in disease transmission? *Infect Control Hosp Epidemiol* 2000;21:80–5.
351. Passaro DJ, Waring L, Armstrong R, et al. Postoperative *Serratia marcescens* wound infections traced to an out-of-hospital source. *J Infect Dis* 1997;175:992–5.
352. Foca M, Jakob K, Whittier S, et al. Endemic *Pseudomonas aeruginosa* infection in a neonatal intensive care unit. *N Engl J Med* 2000;343:695–700.

353. Parry MF, Grant B, Yukna M, et al. *Candida* osteomyelitis and diskitis after spinal surgery: an outbreak that implicates artificial nail use. *Clin Infect Dis* 2001;32:352–7.
354. Garner JS, Simmons BP. Guideline for isolation precautions in hospitals. *Infect Control* 1983;4(suppl 4):245–325.
355. CDC. Recommendations for prevention of HIV transmission in health-care settings. *MMWR* 1987;36(suppl 2S):3S–18S.
356. Occupational Safety and Health Administration. 29 CFR Part 1910.1030. Occupational exposure to bloodborne pathogens: final rule. *Federal Register* 1991;29CFR Part 1910:1030.
357. Hartstein AI, Denny MA, Morthland VH, LeMonte AM, Pfaller MA. Control of methicillin-resistant *Staphylococcus aureus* in a hospital and an intensive care unit. *Infect Control Hosp Epidemiol* 1995;16:405–11.
358. Maki DG, McCormick RD, Zilz MA, Stolz SM, Alvarado CJ. An MRSA outbreak in a SICU during universal precautions: new epidemiology for nosocomial MRSA: downside for universal precautions [Abstract 473]. In: Program and abstracts of the 30th Interscience Conference on Antimicrobial Agents and Chemotherapy. Washington, DC: American Society for Microbiology, 1990.
359. Kotilainen HR, Brinker JP, Avato JL, Gantz NM. Latex and vinyl examination gloves: quality control procedures and implications for health care workers. *Arch Intern Med* 1989;149:2749–53.
360. Reingold AL, Kane MA, Hightower AW. Failure of gloves and other protective devices to prevent transmission of hepatitis B virus to oral surgeons. *JAMA* 1988;259:2558–60.
361. Korniewicz DM, Laughon BE, Butz A, Larson E. Integrity of vinyl and latex procedures gloves. *Nurs Res* 1989;38:144–6.
362. DeGroot-Kosolcharoen J, Jones JM. Permeability of latex and vinyl gloves to water and blood. *Am J Infect Control* 1989;17:196–201.
363. Korniewicz DM, Kirwin M, Cresci K, Markut C, Larson E. In-use comparison of latex gloves in two high-risk units: surgical intensive care and acquired immunodeficiency syndrome. *Heart Lung* 1992;21:81–4.
364. Korniewicz DM, Kirwin M, Cresci K, et al. Barrier protection with examination gloves: double versus single. *Am J Infect Control* 1994;22:12–5.
365. Siström MG, Muto CA, Neal J, Strain BA, Farr BM. Glove leakage rates as a function of latex content and brand: caveat emptor [Abstract 24]. In: Program and abstracts of the 10th Annual Meeting of Society of Healthcare Epidemiology of America, Orlando, Florida, 1998.
366. Flanagan H, Farr B. Continued evaluation of glove leakage rates at the University of Virginia. Presented at the 11th Annual Meeting of the Society for Healthcare Epidemiology of America, Toronto, Canada, April 1, 2001.
367. Korniewicz DM, Laughon BE, Cyr WH, Lytle CD, Larson E. Leakage of virus through used vinyl and latex examination gloves. *J Clin Microbiol* 1990;28:787–8.
368. Rego A, Roley L. In-use barrier integrity of gloves: latex and nitrile superior to vinyl. *Am J Infect Control* 1999;27:405–10.
369. Fisher MD, Reddy VR, Williams FM, Lin KY, Thacker JG, Edlich RF. Biomechanical performance of powder-free examination gloves. *J Emerg Med* 1999;17:1011–8.
370. Edlich RF, Suber F, Neal JG, Jackson EM, Williams FM. Integrity of powder-free examination gloves to bacteriophage penetration. *J Biomed Mater Res* 1999;48:755–8.
371. Murray CA, Burke FJT, McHugh S. An assessment of the incidence of punctures in latex and non-latex dental examination gloves in routine clinical practice. *Br Dental Journal* 2001;190:377–80.
372. Jones RD, Jampani H, Mulberry G, Rizer RL. Moisturizing alcohol hand gels for surgical hand preparation. *AORN J* 2000;71:584–99.
373. Patterson JE, Vecchio J, Pantelick EL, et al. Association of contaminated gloves with transmission of *Acinetobacter calcoaceticus* var. *antitratius* in an intensive care unit. *Am J Med* 1991;91:479–83.
374. Lowbury EJJ. Aseptic methods in the operating suite. *Lancet* 1968;1:705–9.
375. Hoffman PN, Cooke EM, McCarville MR, Emmerson AM. Microorganisms isolated from skin under wedding rings worn by hospital staff. *Br Med J* 1985;290:206–7.
376. Jacobson G, Thiele JE, McCune JH, Farrell LD. Handwashing: ring-wearing and number of microorganisms. *Nurs Res* 1985;34:186–8.
377. Hayes RA, Trick WE, Vernon MO, et al. Ring use as a risk factor (RF) for hand colonization in a surgical intensive care unit (SICU) [Abstract K-1333]. In: Program and abstracts of the 41st Interscience Conference on Antimicrobial Agents and Chemotherapy. Washington, DC: American Society for Microbiology, 2001.
378. Salisbury DM, Hutfilz P, Treen LM, Bollin GE, Gautam S. The effect of rings on microbial load of health care workers' hands. *Am J Infect Control* 1997;25:24–7.
379. Spire B, Barré-Sinoussi F, Montagnier L, Chermann JC. Inactivation of lymphadenopathy associated virus by chemical disinfectants. *Lancet* 1984;2:899–901.
380. Martin LS, McDougal JS, Loskoski SL. Disinfection and inactivation of the human T lymphotropic virus type III/lymphadenopathy-associated virus. *J Infect Dis* 1985;152:400–3.
381. Resnick L, Veren K, Salahuddin SZ, Tondreau S, Markham PD. Stability and inactivation of HTLV-III/LAV under clinical and laboratory environments. *JAMA* 1986;255:1887–91.
382. van Bueren J, Larkin DP, Simpson RA. Inactivation of human immunodeficiency virus type 1 by alcohols. *J Hosp Infect* 1994;28:137–48.
383. Montefiori DC, Robinson WE Jr, Modliszewski A, Mitchell WM. Effective inactivation of human immunodeficiency virus with chlorhexidine antiseptics containing detergents and alcohol. *J Hosp Infect* 1990;15:279–82.
384. Wood A, Payne D. The action of three antiseptics/disinfectants against enveloped and non-enveloped viruses. *J Hosp Infect* 1998;38:283–95.
385. Harbison MA, Hammer SM. Inactivation of human immunodeficiency virus by Betadine products and chlorhexidine. *J Acquir Immune Defic Syndr* 1989;2:16–20.
386. Lavelle GC, Gubbe SL, Neveaux JL, Bowden BJ. Evaluation of an antimicrobial soap formula for virucidal efficacy in vitro against human immunodeficiency virus in a blood-virus mixture. *Antimicrob Agents Chemother* 1989;33:2034–6.
387. Bond WW, Favero MS, Petersen NJ, Ebert JW. Inactivation of hepatitis B virus by intermediate-to-high level disinfectant chemicals. *J Clin Microbiol* 1983;18:535–8.
388. Kobayashi H, Tsuzuki M, Koshimizu K, et al. Susceptibility of hepatitis B virus to disinfectants or heat. *J Clin Microbiol* 1984;20:214–6.
389. Kurtz JB. Virucidal effect of alcohols against echovirus 11 [Letter]. *Lancet* 1979;1:496–7.
390. Sattar SA, Raphael RA, Lochnan H, Springthorpe VS. Rotavirus inactivation by chemical disinfectants and antiseptics used in hospitals. *Can J Microbiol* 1983;29:1464–9.
391. Larson E, Silberger M, Jakob K, et al. Assessment of alternative hand hygiene regimens to improve skin health among neonatal intensive care unit nurses. *Heart Lung* 2000;29:136–42.

392. Gould D, Chamberlain A. The use of a ward-based educational teaching package to enhance nurses' compliance with infection control procedures. *J Clin Nursing* 1997;6:55-67.
393. Aspöck C, Koller W. A simple hand hygiene exercise. *Am J Infect Control* 1999;27:370-2.
394. McGuckin M, Waterman R, Porten L, et al. Patient education model for increasing handwashing compliance [Practice forum]. *Am J Infect Control* 1999;27:309-14.
395. Khatib M, Jamaledine G, Abdallah A, Ibrahim Y. Hand washing and use of gloves while managing patients receiving mechanical ventilation in the ICU. *Chest* 1999;116:172-5.
396. Haley RW, Bregman DA. The role of understaffing and overcrowding in recurrent outbreaks of staphylococcal infection in a neonatal special-care unit. *J Infect Dis* 1982;145:875-85.
397. Pittet D, Boyce JM. Hand hygiene and patient care: pursuing the Semmelweis legacy. *Lancet Infectious Diseases* 2001;April:9-20.
398. Boyce JM. Scientific basis for handwashing with alcohol and other waterless antiseptic agents. In: Rutala WA, ed. *Disinfection, sterilization and antiseptics: principles and practices in healthcare facilities*. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc, 2001.
399. O'Boyle CA, Henly SJ, Duckett LJ. Nurses' motivation to wash their hands: a standardized measurement approach. *Applied Nursing Research* 2001;14:136-45.
400. Semmelweis IP. *Die aetiologie, der begriff und die prophylaxis des kindbettfiebers*. Pest, Wien und Leipzig: CA Hartleben's Verlags-Expedition 1861.
401. Eggmann P, Harbarth S, Constantin MN, Touvneau S, Chevrolet JC, Pittet D. Impact of a prevention strategy targeted at vascular-access care on incidence of infections acquired in intensive care. *Lancet* 2000;355:1864-8.
402. Bull DA, Neumayer LA, Hunter GC, et al. Improved sterile technique diminishes the incidence of positive line cultures in cardiovascular patients. *J Surgical Research* 1992;52:106-10.
403. Hirschmann H, Fux L, Podusel J, et al. The influence of hand hygiene prior to insertion of peripheral venous catheters on the frequency of complications. *J Hosp Infect* 2001;49:199-203.
404. Drusin LM, Sohmer M, Groshen SL, Spiritos MD, Senterfit LB, Christenson WN. Nosocomial hepatitis A infection in a paediatric intensive care unit. *Arch Dis Child* 1987;62:690-5.
405. Doebbeling BN, Li N, Wenzel RP. An outbreak of hepatitis A among health care workers: risk factors for transmission. *Am J Public Health* 1993;83:1679-84.
406. Standaert SM, Hutcheson RH, Schaffner W. Nosocomial transmission of *Salmonella gastroenteritis* to laundry workers in a nursing home. *Infect Control Hosp Epidemiol* 1994;15:22-6.
407. Rodriguez EM, Parrott C, Rolka H, Monroe SS, Dwyer DM. An outbreak of viral gastroenteritis in a nursing home: importance of excluding ill employees. *Infect Control Hosp Epidemiol* 1996;17:587-92.
408. Schaffner W, Lefkowitz LB Jr, Goodman JS, Koenig MG. Hospital outbreak of infections with group A streptococci traced to an asymptomatic anal carrier. *N Engl J Med* 1969;280:1224-5.
409. Viglionese A, Nottebart VF, Bodman HA, Platt R. Recurrent group A streptococcal carriage in a health care worker associated with widely separated nosocomial outbreaks. *Am J Med* 1991;91(suppl 3B):329S-33S.
410. Ojajarvi J. An evaluation of antiseptics used for hand disinfection in wards. *J Hyg (Lond)* 1976;76:75-82.
411. Mermel LA, Josephson SL, Dempsey J, Parenteau S, Perry C, Magill N. Outbreak of *Shigella sonnei* in a clinical microbiology laboratory. *J Clin Microbiol* 1997;35:3163-5.
412. McBride ME. Microbial flora of in-use soap products. *Appl Environ Microbiol* 1984;48:338-41.
413. Kabara JJ, Brady MB. Contamination of bar soaps under "in use" condition. *J Environ Pathol Toxicol Oncol* 1984;5:1-14.
414. Heinze JE, Yackovich F. Washing with contaminated bar soap is unlikely to transfer bacteria. *Epidem Inf* 1988;101:135-42.
415. Bannan EA, Judge LF. Bacteriological studies relating to handwashing: 1. the inability of soap bars to transmit bacteria. *Am J Public Health* 1965;55:915-21.
416. Field EA, McGowan P, Pearce PK, Martin MV. Rings and watches: should they be removed prior to operative dental procedures? *J Dent* 1996;24:65-9.
417. Lowbury EJJ, Lilly HA. Gloved hand as applicator of antiseptic to operation sites. *Lancet* 1975;2:153-6.
418. AORN Recommended Practices Committee. Recommended practices for surgical hand scrubs. *AORN J* 1999;69:842-50.
419. Grohskopf LA, Roth VR, Feikin DR, et al. *Serratia liquefaciens* bloodstream infections from contamination of epoetin alfa at a hemodialysis center. *N Engl J Med* 2001;344:1491-7.
420. Dharan S, Hugonnet S, Sax H, Pittet D. Evaluation of interference of a hand care cream with alcohol-based hand disinfection. *Occup Environ Dermatol* 2001;49:81-4.
421. Heeg P. Does hand care ruin hand disinfection? *J Hosp Infect* 2001;48(suppl A):S37-S39.
422. McGuckin M, Waterman R, Storr J, et al. Evaluation of a patient-empowering hand hygiene programme in the U.K. *J Hosp Infect* 2001;48:222-7.
423. Girou E, Oppein F. Handwashing compliance in a French university hospital: new perspective with the introduction of hand-rubbing with a waterless alcohol-based solution. *J Hosp Infect* 2001;48(suppl A):S55-S57.

Appendix

Antimicrobial Spectrum and Characteristics of Hand-Hygiene Antiseptic Agents*

Group	Gram-positive bacteria	Gram-negative bacteria	Mycobacteria	Fungi	Viruses	Speed of action	Comments
Alcohols	+++	+++	+++	+++	+++	Fast	Optimum concentration 60%–95%; no persistent activity
Chlorhexidine (2% and 4% aqueous)	+++	++	+	+	+++	Intermediate	Persistent activity; rare allergic reactions
Iodine compounds	+++	+++	+++	++	+++	Intermediate	Causes skin burns; usually too irritating for hand hygiene
Iodophors	+++	+++	+	++	++	Intermediate	Less irritating than iodine; acceptance varies
Phenol derivatives	+++	+	+	+	+	Intermediate	Activity neutralized by nonionic surfactants
Tricolsan	+++	++	+	—	+++	Intermediate	Acceptability on hands varies
Quaternary ammonium compounds	+	++	—	—	+	Slow	Used only in combination with alcohols; ecologic concerns

Note: +++ = excellent; ++ = good, but does not include the entire bacterial spectrum; + = fair; — = no activity or not sufficient.

*Hexachlorophene is not included because it is no longer an accepted ingredient of hand disinfectants.



MMWR™

Morbidity and Mortality Weekly Report

Recommendations and Reports

October 25, 2002 / Vol. 51 / No. RR-16

Continuing Education Activity Sponsored by CDC Guideline for Hand Hygiene in Health-Care Settings

Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force

EXPIRATION — October 25, 2004

You must complete and return the response form electronically or by mail by **October 25, 2004**, to receive continuing education credit. If you answer all of the questions, you will receive an award letter for 1.75 hours Continuing Medical Education (CME) credit; 0.15 Continuing Education Units (CEUs); 1.5 hours Certified Health Education Specialist (CHES) credit; or 1.9 contact

hours Continuing Nursing Education (CNE) credit. If you return the form electronically, you will receive educational credit immediately. If you mail the form, you will receive educational credit in approximately 30 days. No fees are charged for participating in this continuing education activity.

INSTRUCTIONS

By Internet

1. Read this *MMWR* (Vol. 51, RR-16), which contains the correct answers to the questions beginning on the next page.
2. Go to the *MMWR* Continuing Education Internet site at <<http://www.cdc.gov/mmwr/cme/conted.html>>.
3. Select which exam you want to take and select whether you want to register for CME, CEU, CHES, or CNE credit.
4. Fill out and submit the registration form.
5. Select exam questions. To receive continuing education credit, you must answer all of the questions. Questions with more than one correct answer will instruct you to "Indicate all that apply."
6. Submit your answers no later than **October 25, 2004**.
7. Immediately print your Certificate of Completion for your records.

By Mail or Fax

1. Read this *MMWR* (Vol. 50, RR-16), which contains the correct answers to the questions beginning on the next page.
2. Complete all registration information on the response form, including your name, mailing address, phone number, and e-mail address, if available.
3. Indicate whether you are registering for CME, CEU, CHES, or CNE credit.
4. Select your answers to the questions, and mark the corresponding letters on the response form. To receive continuing education credit, you must answer all of the questions. Questions with more than one correct answer will instruct you to "Indicate all that apply."
5. Sign and date the response form or a photocopy of the form and send no later than **October 25, 2004**, to
Fax: 404-639-4198 Mail: MMWR CE Credit
Office of Scientific and Health Communications
Epidemiology Program Office, MS C-08
Centers for Disease Control and Prevention
1600 Clifton Rd, N.E.
Atlanta, GA 30333
6. Your Certificate of Completion will be mailed to you within 30 days.

ACCREDITATION

Continuing Medical Education (CME). CDC is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. CDC designates this educational activity for a maximum of 1.75 hours in category 1 credit toward the AMA Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

Continuing Education Unit (CEU). CDC has been approved as an authorized provider of continuing education and training programs by the International Association for Continuing Education and Training and awards 0.15 Continuing Education Units (CEUs).

Certified Health Education Specialist (CHES). CDC is a designated provider of continuing education contact hours in health education by the National Commission for Health Education Credentialing, Inc. This program is a designated event for CHES to receive 1.5 hours in category 1 credit in health education. CDC provider number GA0082.

Continuing Nursing Education (CNE). This activity for 1.9 contact hours is provided by CDC, which is accredited as a provider of continuing education in nursing by the American Nurses Credentialing Center's Commission on Accreditation.

CENTERS FOR DISEASE CONTROL AND PREVENTION

SAFER • HEALTHIER • PEOPLE™

Goal and Objectives

This *MMWR* provides evidence-based recommendations for hand hygiene in health-care settings. These recommendations were developed by the Healthcare Infection Control Practices Advisory Committee (HICPAC), the Society for Healthcare Epidemiology of America, the Association for Professionals in Infection Control and Epidemiology, and the Infectious Diseases Society of America Hand Hygiene Task Force. The goal of this report is to provide guidance for clinicians and other health-care practitioners regarding strategies to improve hand-hygiene practices and reduce transmission of microorganisms in health-care settings. Upon completion of this educational activity, the reader should be able to 1) describe the indications for hand hygiene in health-care settings; 2) list the advantages of alcohol-based hand rubs; and 3) describe the barriers to hand hygiene in health-care settings.

To receive continuing education credit, please answer all of the following questions.

1. **Hand hygiene refers to . . .**
 - A. handwashing using plain soap and water.
 - B. using an antiseptic hand rub (e.g alcohol, chlorhexidine, iodine).
 - C. handwashing using antimicrobial soap and water.
 - D. all of the above.
2. **Hand hygiene adherence in health-care facilities might be improved by . . .**
 - A. providing personnel with individual containers of alcohol-based hand rubs.
 - B. providing personnel with hand lotions or creams.
 - C. providing personnel with feedback regarding hand-hygiene adherence/performance.
 - D. all of the above.
3. **Alcohol-based hand rubs have good or excellent antimicrobial activity against all of the following except . . .**
 - A. viruses.
 - B. fungi.
 - C. mycobacteria.
 - D. bacterial spores.
 - E. gram-positive and gram-negative bacteria.
4. **Alcohol-based hand rubs are indicated for all of the following clinical situations except . . .**
 - A. when the hands are visibly soiled.
 - B. preoperative cleaning of hands by surgical personnel.
 - C. before inserting urinary catheters, intravascular catheters, or other invasive devices.
 - D. after removing gloves.
5. **Each of the following statements regarding alcohol-based hand rubs is true except . . .**
 - A. alcohol-based hand rubs reduce bacterial counts on the hands of health-care personnel more effectively than plain soaps.
 - B. alcohol-based hand rubs can be made more accessible than sinks or other handwashing facilities.
 - C. alcohol-based hand rubs require less time to use than traditional handwashing.
 - D. alcohol-based hand rubs have been demonstrated to cause less skin irritation and dryness than handwashing using soap and water.
 - E. alcohol-based hand rubs are only effective if they are applied for ≥ 60 seconds.
6. **Which of the following statements regarding preoperative surgical hand antisepsis is true?**
 - A. Antimicrobial counts on hands are reduced as effectively with a 5-minute scrub as with a 10-minute scrub.
 - B. A brush or sponge must be used when applying the antiseptic agent to adequately reduce bacterial counts on hands.
 - C. Alcohol-based hand rubs for preoperative surgical scrub have been associated with increased surgical site infection rates.
 - D. A and B are true.
 - E. A and C are true.
7. **Antimicrobial-impregnated wipes (i.e., towelettes) . . .**
 - A. might be considered as an alternative to handwashing with plain soap and water.
 - B. are as effective as alcohol-based hands rubs.
 - C. are as effective as washing hands with antimicrobial soap and water.
 - D. A and C.
8. **The following statements regarding hand hygiene in health-care settings are true except . . .**
 - A. Overall adherence among health-care personnel is approximately 40%.
 - B. Poor adherence to hand-hygiene practice is a primary contributor to health-care-associated infection and transmission of antimicrobial-resistant pathogens.
 - C. Personnel wearing artificial nails or extenders have been linked to nosocomial outbreaks.
 - D. Hand hygiene is not necessary if gloves are worn.
9. **Indicate your work setting.**
 - A. State/local health department.
 - B. Other public health setting.
 - C. Hospital clinic/private practice.
 - D. Managed care organization.
 - E. Academic institution.
 - F. Other.
10. **Which best describes your professional activities?**
 - A. Patient care — emergency/urgent care department.
 - B. Patient care — inpatient.
 - C. Patient care — primary-care clinic or office.
 - D. Laboratory/pharmacy.
 - E. Public health.
 - F. Other.
11. **I plan to use these recommendations as the basis for . . . (Indicate all that apply.)**
 - A. health education materials.
 - B. insurance reimbursement policies.
 - C. local practice guidelines.
 - D. public policy.
 - E. other.
12. **Each month, approximately how many patients do you examine?**
 - A. None.
 - B. 1–5.
 - C. 6–20.
 - D. 21–50.
 - E. 51–100.
 - F. >100.
13. **How much time did you spend reading this report and completing the exam?**
 - A. 1–1.5 hours.
 - B. More than 1.5 hours but fewer than 2 hours.
 - C. 2–2.5 hours.
 - D. More than 2.5 hours.

- 14. After reading this report, I am confident I can describe the guidance for clinicians and other health-care practitioners regarding strategies to improve hand-hygiene practices and reduce transmission of microorganisms in health-care settings.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.
- 15. After reading this report, I am confident I can describe the indications for hand hygiene in health-care settings.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.
- 16. After reading this report, I am confident I can list the advantages of alcohol-based hand rubs.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.

- 17. After reading this report, I am confident I can describe the barriers to hand hygiene in health-care settings.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.
- 18. The objectives are relevant to the goal of this report.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.
- 19. The tables and text boxes are useful.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.
- 20. Overall, the presentation of the report enhanced my ability to understand the material.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.

**MMWR Response Form for Continuing Education Credit
October 25, 2002/Vol. 51/No. RR-16
Guideline for Hand Hygiene in Health-Care Settings**

Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force

To receive continuing education credit, you must

1. provide your contact information;
2. indicate your choice of CME, CEU, CHES, or CNE credit;
3. answer all of the test questions;
4. sign and date this form or a photocopy;
5. submit your answer form by October 25, 2004.

Failure to complete these items can result in a delay or rejection of your application for continuing education credit.

Detach or photocopy.

- Check One*
- CME Credit
 - CEU Credit
 - CHES Credit
 - CNE Credit

Last Name _____ First Name _____

Street Address or P.O. Box _____

Apartment _____ or _____ Suite _____

City _____ State _____ ZIP Code _____

Phone Number _____ Fax Number _____

E-Mail Address _____

Fill in the appropriate blocks to indicate your answers. Remember, you must answer all of the questions to receive continuing education credit!

1. [] A [] B [] C [] D	14. [] A [] B [] C [] D [] E
2. [] A [] B [] C [] D	15. [] A [] B [] C [] D [] E
3. [] A [] B [] C [] D [] E	16. [] A [] B [] C [] D [] E
4. [] A [] B [] C [] D	17. [] A [] B [] C [] D [] E
5. [] A [] B [] C [] D [] E	18. [] A [] B [] C [] D [] E
6. [] A [] B [] C [] D [] E	19. [] A [] B [] C [] D [] E
7. [] A [] B [] C [] D	20. [] A [] B [] C [] D [] E
8. [] A [] B [] C [] D	21. [] A [] B [] C [] D [] E
9. [] A [] B [] C [] D [] E [] F	22. [] A [] B [] C [] D [] E
10. [] A [] B [] C [] D [] E [] F	23. [] A [] B [] C [] D [] E [] F
11. [] A [] B [] C [] D [] E	
12. [] A [] B [] C [] D [] E [] F	
13. [] A [] B [] C [] D	

Signature _____ Date I Completed Exam _____

21. These recommendations will affect my practice.

- A. Strongly agree.
- B. Agree.
- C. Neither agree nor disagree.
- D. Disagree.
- E. Strongly disagree.

22. The availability of continuing education credit influenced my decision to read this report.

- A. Strongly agree.
- B. Agree.
- C. Neither agree nor disagree.
- D. Disagree.
- E. Strongly disagree.

23. How did you learn about this continuing education activity?

- A. Internet.
- B. Advertisement (e.g., fact sheet, *MMWR* cover, newsletter, or journal).
- C. Coworker/supervisor.
- D. Conference presentation.
- E. *MMWR* subscription.
- F. Other.

Correct answers for questions 1–8:
1. D; 2. D; 3. D; 4. A; 5. E; 6. A; 7. A; 8. D.

Hospital Infection Control Practices Advisory Committee Members

Chair: Robert A. Weinstein, M.D., Cook County Hospital, Chicago, Illinois.

Co-Chair: Jane D. Siegel, M.D., University of Texas Southwestern Medical Center, Dallas, Texas.

Executive Secretary: Michele L. Pearson, M.D. CDC, Atlanta, Georgia.

Members: Raymond Y.W. Chinn, M.D., Sharp Memorial Hospital, San Diego, California; Alfred DeMaria, Jr, M.D., Massachusetts Department of Public Health, Jamaica Plain, Massachusetts; Elaine L. Larson, Ph.D., Columbia University School of Nursing, New York, New York; James T. Lee, M.D., Ph.D., University of Minnesota, Minneapolis, Minnesota; Ramon E. Moncada, M.D., Coronado Physician's Medical Center, Coronado, California; William A. Rutala, Ph.D., University of North Carolina, Chapel Hill, North Carolina; William E. Scheckler, M.D., University of Wisconsin, Madison, Wisconsin; and Marjorie A. Underwood, Mt. Diablo Medical Center, Concord, California.

Hand Hygiene Task Force Members

Chair: John M. Boyce, M.D., Hospital of Saint Raphael, New Haven, Connecticut.

Members: Barry M. Farr, M.D., University of Virginia, Charlottesville, Virginia; William R. Jarvis, M.D., CDC, Atlanta, Georgia; Elaine L. Larson, Ph.D., Columbia School of Nursing, New York, New York; Maryanne McGuckin, DrScEd, University of Pennsylvania, Philadelphia, Pennsylvania; Carol O'Boyle, Ph.D., Minnesota Department of Health, Minneapolis, Minnesota; Didier Pittet, M.D., University of Geneva, Geneva, Switzerland; Jane D. Siegel, M.D., University of Texas Southwestern Medical Center, Dallas, Texas; Marjorie A. Underwood, Mt. Diablo Medical Center, Concord, California; Andreas F. Widmer, M.D., University Hospitals, Basel, Switzerland; Jolynn Zeller, Avera St. Lukes Hospital, Aberdeen, South Dakota; and Beth H. Stover, Kosair Children's Hospital, Louisville, Kentucky.

Disclosure of Financial Interests and Relationships

John Boyce: Research and educational grants from Gojo Industries; honorarium from Procter and Gamble; and consultant to Bode Chemical.

Barry Farr: Research support from Gojo Industries.

Elaine Larson: Received products for testing in research studies from Procter and Gamble, 3M Corporation, and Steris.

Carol O'Boyle: Honorarium from 3M Corporation.

All *MMWR* references are available on the Internet at <http://www.cdc.gov/mmwr>. Use the search function to find specific articles.

Use of trade names and commercial sources is for identification only and does not imply endorsement by the U.S. Department of Health and Human Services.

References to non-CDC sites on the Internet are provided as a service to *MMWR* readers and do not constitute or imply endorsement of these organizations or their programs by CDC or the U.S. Department of Health and Human Services. CDC is not responsible for the content of these sites. URL addresses listed in *MMWR* were current as of the date of publication.

The *Morbidity and Mortality Weekly Report (MMWR)* series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format and on a paid subscription basis for paper copy. To receive an electronic copy each week, send an e-mail message to listserv@listserv.cdc.gov. The body content should read *SUBscribe mmwr-toc*. Electronic copy also is available from CDC's Internet server at <http://www.cdc.gov/mmwr> or from CDC's file transfer protocol server at <ftp://ftp.cdc.gov/pub/publications/mmwr>. To subscribe for paper copy, contact Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone 202-512-1800.

Data in the weekly *MMWR* are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the following Friday. Address inquiries about the *MMWR* series, including material to be considered for publication, to Editor, *MMWR* Series, Mailstop C-08, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30333; telephone 888-232-3228.

All material in the *MMWR* series is in the public domain and may be used and reprinted without permission; however, citation of the source is appreciated.

HICPAC

HEALTHCARE INFECTION CONTROL
PRACTICES ADVISORY COMMITTEE

GUIDELINE FOR PREVENTION OF CATHETER- ASSOCIATED URINARY TRACT INFECTIONS 2009

Carolyn V. Gould, MD, MSCR ¹; Craig A. Umscheid, MD, MSCE ²; Rajender K. Agarwal, MD, MPH ²; Gretchen Kuntz, MSW, MSLIS ²; David A. Pegues, MD ³ and the Healthcare Infection Control Practices Advisory Committee (HICPAC) ⁴

¹ Division of Healthcare Quality Promotion
Centers for Disease Control and Prevention
Atlanta, GA

² Center for Evidence-based Practice
University of Pennsylvania Health System
Philadelphia, PA

³ Division of Infectious Diseases
David Geffen School of Medicine at UCLA
Los Angeles, CA



4 Healthcare Infection Control Practices Advisory Committee

(HICPAC)

Chair

BRENNAN, Patrick J., MD
Chief Medical Officer
Division of Infectious Diseases
University of Pennsylvania Health System

Department of Pathology
University of Florida Health Science
Center-Jacksonville

Executive Secretary

BELL, Michael R., MD
Associate Director for Infection Control
Division of Healthcare Quality Promotion
National Center for Infectious Diseases
Centers for Disease Control and
Prevention

MURPHY, Denise M., MPH, RN, CIC
Vice President, Safety and Quality
Barnes-Jewish Hospital at Washington
University Medical Center

OLMSTED, Russell N., MPH
Epidemiologist
Infection Control Services
St. Joseph Mercy Health System

Members

BURNS, Lillian A., MPH
Infection Control Coordinator
Infectious Diseases Department,
Greenwich Hospital

PEGUES, David Alexander, MD
Professor of Medicine, Hospital Epidemiologist
David Geffen School of Medicine at
UCLA

ELWARD, Alexis, MD, MPH
Medical Director of Infection Control
Assistant Professor, Pediatric Infectious
Diseases
Washington University School of Medicine

RAMSEY, Keith M., MD
Professor of Medicine
Medical Director of Infection Control
Pitt County Memorial

ENGEL, Jeffrey, MD
Chief, Epidemiology Section, North
Carolina Division of Public Health
North Carolina State Epidemiologist

SINGH, Nalini, MD, MPH
Professor of Pediatrics
Epidemiology and International Health
George Washington University
Children's National Medical Center

LUNDSTROM, Tammy, MD, JD
Chief Medical Officer
Providence Hospital

SOULE, Barbara M., RN, MPA, CIC
Practice Leader
Infection Prevention Services
Joint Commission Resources/Joint
Commission International

GORDON, Steven M., MD
Chairman, Department of Infectious
Diseases
Hospital Epidemiologist
Cleveland Clinic Foundation

SCHECTER, William, P., MD
Department of Surgery, Ward 3A 17
San Francisco General Hospital

MCCARTER, Yvette S., PhD
Director, Clinical Microbiology
Laboratory

STEVENSON, Kurt Brown, MD, MPH
Division of Infectious Diseases
Department of Internal Medicine
The Ohio State University Medical Center

Ex-officio Members

Agency for Healthcare Research and Quality

Ex-Officio

BAINE, William B., MD
Senior Medical Advisor
Center for Outcomes and Evidence
Agency for Healthcare Research and Quality

National Institute of Health

Ex-Officio

HENDERSON, David, MD
Deputy Director for Clinical Care
National Institute of Health

Health Resources and Services

Administration Ex-Officio

JAY, Lorine J., MPH, RN, CPHQ
Regional Coordinator

Food and Drug Administration

Ex-Officio

MURPHEY, Sheila A., MD
Chief, Infection Control Devices Branch
Division of Anesthesiology, General Hospital
Infection Control and Dental Devices
Center for Devices and Radiology Health
Food and Drug Administration

Center for Medicare & Medicaid Services

(CMS) Ex-Officio

MILLER, Jeannie RN, MPH
Deputy Director, Office of Clinical
Standards and Quality/ Clinical Standards
Group

Department of Veterans Affairs (VA)

ROSELLE, Gary A., MD
National Program Director, Infectious
Diseases
VA Central Office
Cincinnati VA Medical Center

Liaisons

Association of Professionals of Infection Control and Epidemiology, Inc.

BJERKE, Nancy BSN, RN, MPH, CIC

Council of State and Territorial Epidemiologists

KAINER, Marion MD, MPH
Medical Epidemiologist/Infections
Diseases Physician Director, Hospital
Infections and Antimicrobial Resistance
Program, Tennessee Department of Health

Infection Control Consultant
Infection Control Associates

American Health Care Association

FITZLER, Sandra L., RN
Senior Director of Clinical Services
American Health Care Association

American College of Occupational and Environmental Medicine

RUSSI, Mark, MD, MPH
American College of Occupational and
Environmental Medicine

Advisory Council for the Elimination of

Tuberculosis STRICOF, Rachel L., MPH
New York State Department of Health

American Hospital Association

SCHULMAN, Roslyne, MHA, MBA
Senior Associate Director, Policy
Development

Association of periOperative Registered Nurses

BLANCHARD, Joan C., RN, BSN,
MSS, CNOR, CIC
Association of periOperative Registered
Nurses

Society for Healthcare Epidemiology of America

MARAGAKIS, Lisa, MD
Assistant Professor of Medicine
Johns Hopkins Medical Institutions

Joint Commission on Accreditation of Healthcare Organizations

WISE, Robert A., MD
Division of Standards & Survey
Methods
Joint Commission on Accreditation of
Healthcare Organizations

Consumers Union

Senior Policy Analyst on
MCGIFFERT, Lisa
Health Issues, Project Director
Stop Hospital Infections Organization

Acknowledgement

HICPAC thanks the following members who served on the HICPAC CAUTI Guideline subcommittee during the guideline development process: Russell N. Olmsted, MPH, Yvette S. McCarter, PhD, Barbara M. Soule, RN, MPA, CIC, and Nalini Singh, MD, MPH.

HICPAC thanks the following outside experts for reviewing a draft of this guideline: Edward S. Wong, MD, Lindsay E. Nicolle, MD, Anthony J. Schaeffer, MD, and Harriett M. Pitt, RN, MS, CIC. The opinions of the reviewers might not be reflected in all the recommendations contained in this document.

HICPAC would also like to thank the many individuals and organizations who provided valuable feedback on the guideline during the public comment period.

Disclosure of Financial Interests and Relationships

The authors C.V.G., C.A.U., R.K.A., and G.K. report no actual or potential conflicts of interest. D.A.P. is on the Speakers Bureau of Merck, Pfizer, Schering, and Cubist and is a consultant for Dow Pharmaceuticals, DaVita, and Vasonova. C.A.U. and R.K.A. received funding from the CDC to support the guideline development process.

Table of Contents

<i>Abbreviations</i>	6
I. Executive Summary	8
II. Summary of Recommendations.....	10
III. Implementation and Audit	18
IV. Recommendations for Further Research	20
V. Background.....	22
VI. Scope and Purpose	25
VII. Methods	26
VIII. Evidence Review	34
References.....	49

Abbreviations

ADL	Activities of daily living
APACHE II	Acute Physiology and Chronic Health Evaluation II
ASA	American Society of Anesthesiologists
ASB	Asymptomatic bacteriuria
BUN	Blood urea nitrogen
CAUTI	Catheter-associated urinary tract infection
CDC	Centers for Disease Control and Prevention
CFU	Colony-forming units
CI	Confidence interval
CIC	Clean intermittent catheterization
CICU	Coronary intensive care unit
COPD	Chronic obstructive pulmonary disease
ED	Emergency department
F/U	Follow-up
GRADE	Grading of Recommendations Assessment, Development, and Evaluation system
Hb	Hemoglobin concentration
HICPAC	Healthcare Infection Control Practices Advisory Committee
H/O	History of
HPF	High power field
HR	Hazard ratio
ICU	Intensive care unit
IDR	Incidence-density ratio
LOS	Length of stay
MDR	Multi-drug resistant
MICU	Medical intensive care unit
NHSN	National Healthcare Safety Network
NIH	National Institutes of Health
NS	Not significant
OBS	Observational controlled study
OR	Odds ratio
P	P value
PACU	Post-anesthesia care unit
PVC	Polyvinyl chloride

RCT	Randomized controlled trial
RD	Risk difference
RH	Relative hazard
RR	Relative risk
SAPS II	Simplified Acute Physiology Score II
SICU	Surgical intensive care unit
SR	Systematic review
SUTI	Symptomatic urinary tract infection
TMP/SMX	Trimethoprim/sulfamethoxazole
TURP	Transurethral resection of prostate
UTI	Urinary tract infection
VAS	Visual analog scale
WMD	Weighted mean difference

I. Executive Summary

This guideline updates and expands the original Centers for Disease Control and Prevention (CDC) Guideline for Prevention of Catheter-associated Urinary Tract Infections (CAUTI) published in 1981. Several developments necessitated revision of the 1981 guideline, including new research and technological advancements for preventing CAUTI, increasing need to address patients in non-acute care settings and patients requiring long-term urinary catheterization, and greater emphasis on prevention initiatives as well as better defined goals and metrics for outcomes and process measures. In addition to updating the previous guideline, this revised guideline reviews the available evidence on CAUTI prevention for patients requiring chronic indwelling catheters and individuals who can be managed with alternative methods of urinary drainage (e.g., intermittent catheterization). The revised guideline also includes specific recommendations for implementation, performance measurement, and surveillance. Although the general principles of CAUTI prevention have not changed from the previous version, the revised guideline provides clarification and more specific guidance based on a defined, systematic review of the literature through July 2007. For areas where knowledge gaps exist, recommendations for further research are listed. Finally, the revised guideline outlines high-priority recommendations for CAUTI prevention in order to offer guidance for implementation.

This document is intended for use by infection prevention staff, healthcare epidemiologists, healthcare administrators, nurses, other healthcare providers, and persons responsible for developing, implementing, and evaluating infection prevention and control programs for healthcare settings across the continuum of care. The guideline can also be used as a resource for societies or organizations that wish to develop more detailed implementation guidance for prevention of CAUTI.

Our goal was to develop a guideline based on a targeted systematic review of the best available evidence, with explicit links between the evidence and recommendations. To accomplish this, we used an adapted GRADE system approach for evaluating quality of evidence and determining strength of recommendations. The methodology, structure, and components of this guideline are approved by HICPAC and will be used for subsequent guidelines issued by HICPAC. A more detailed description of our approach is available in the [Methods](#) section.

To evaluate the evidence on preventing CAUTI, we examined data addressing three key questions and related subquestions:

1. Who should receive urinary catheters?
 - A. When is urinary catheterization necessary?
 - B. What are the risk factors for CAUTI?
 - C. What populations are at highest risk of mortality related to urinary catheters?
2. For those who may require urinary catheters, what are the best practices?
Specifically, what are the risks and benefits associated with:
 - A. Different approaches to catheterization?
 - B. Different catheters or collecting systems?
 - C. Different catheter management techniques?
 - D. Different systems interventions (i.e., quality improvement programs)?
3. What are the best practices for preventing CAUTI associated with obstructed urinary catheters?

Evidence addressing the key questions was used to formulate recommendations, and explicit links between the evidence and recommendations are available in the Evidence Review in the body of the guideline, and Evidence Tables and GRADE Tables in the Appendices. **It is important to note that Category I recommendations are all considered strong recommendations and should be equally implemented;** it is only the *quality* of the evidence underlying the recommendation that distinguishes between levels A and B. Category IC recommendations are required by state or federal regulation and may have any level of supporting evidence.

The categorization scheme used in this guideline is presented in Table 1 in the Summary of Recommendations and described further in the Methods section.

The Summary of Recommendations is organized as follows: 1) recommendations for who should receive indwelling urinary catheters (or, for certain populations, alternatives to indwelling catheters); 2) recommendations for catheter insertion; 3) recommendations for catheter maintenance; 4) quality improvement programs to achieve appropriate placement, care, and removal of catheters; 5) administrative infrastructure required; and 6) surveillance strategies.

The Implementation and Audit section includes a prioritization of recommendations (i.e., high-priority recommendations that are essential for every healthcare facility), organized by modules, in order to provide facilities more guidance on implementation of these guidelines. A list of recommended performance measures that can potentially be used for internal reporting purposes is also included.

Areas in need of further research identified during the evidence review are outlined in the Recommendations for Further Research. This section includes guidance for specific methodological approaches that should be used in future studies.

Readers who wish to examine the primary evidence underlying the recommendations are referred to the Evidence Review in the body of the guideline, and the Evidence Tables and GRADE Tables in the Appendices. The Evidence Review includes narrative summaries of the data presented in the Evidence Tables and GRADE Tables. The Evidence Tables include all study-level data used in the guideline, and the GRADE Tables assess the overall quality of evidence for each question. The Appendices also contain a clearly delineated search strategy that will be used for periodic updates to ensure that the guideline remains a timely resource as new information becomes available.

II. Summary of Recommendations

Category IA	A strong recommendation supported by high to moderate quality† evidence suggesting net clinical benefits or harms
Category IB	A strong recommendation supported by low quality evidence suggesting net clinical benefits or harms or an accepted practice (e.g., aseptic technique) supported by low to very low quality evidence
Category IC	A strong recommendation required by state or federal regulation.
Category II	A weak recommendation supported by any quality evidence suggesting a trade off between clinical benefits and harms
No recommendation/ unresolved issue	Unresolved issue for which there is low to very low quality evidence with uncertain trade offs between benefits and harms

* Please refer to Methods (p.32) for implications of Category designations

†Please refer to Methods (p. 29-30) for process used to grade quality of evidence

I. Appropriate Urinary Catheter Use

- A. Insert catheters only for appropriate indications (see Table 2 for guidance), and leave in place only as long as needed. **(Category IB)** (Key Questions 1B and 2C)
 1. Minimize urinary catheter use and duration of use in all patients, particularly those at higher risk for CAUTI or mortality from catheterization such as women, the elderly, and patients with impaired immunity. **(Category IB)** (Key Questions 1B and 1C)
 2. Avoid use of urinary catheters in patients and nursing home residents for management of incontinence. **(Category IB)** (Key Question 1A)
 - a. Further research is needed on periodic (e.g., nighttime) use of external catheters (e.g., condom catheters) in incontinent patients or residents and the use of catheters to prevent skin breakdown. **(No recommendation/unresolved issue)** (Key Question 1A)
 3. Use urinary catheters in operative patients only as necessary, rather than routinely. **(Category IB)** (Key Question 1A)
 4. For operative patients who have an indication for an indwelling catheter, remove the catheter as soon as possible postoperatively, preferably within 24 hours, unless there are appropriate indications for continued use. **(Category IB)** (Key Questions 2A and 2C)

Table 2.
A. Examples of Appropriate Indications for Indwelling Urethral Catheter Use ¹⁻⁴
Patient has acute urinary retention or bladder outlet obstruction
Need for accurate measurements of urinary output in critically ill patients
Perioperative use for selected surgical procedures: <ul style="list-style-type: none"> • Patients undergoing urologic surgery or other surgery on contiguous structures of the genitourinary tract • Anticipated prolonged duration of surgery (catheters inserted for this reason should be removed in PACU) • Patients anticipated to receive large-volume infusions or diuretics during surgery • Need for intraoperative monitoring of urinary output
To assist in healing of open sacral or perineal wounds in incontinent patients
Patient requires prolonged immobilization (e.g., potentially unstable thoracic or lumbar spine, multiple traumatic injuries such as pelvic fractures)
To improve comfort for end of life care if needed
B. Examples of Inappropriate Uses of Indwelling Catheters
As a substitute for nursing care of the patient or resident with incontinence
As a means of obtaining urine for culture or other diagnostic tests when the patient can voluntarily void
For prolonged postoperative duration without appropriate indications (e.g., structural repair of urethra or contiguous structures, prolonged effect of epidural anaesthesia, etc.)

Note: These indications are based primarily on expert consensus.

B. Consider using alternatives to indwelling urethral catheterization in selected patients when appropriate.

1. Consider using external catheters as an alternative to indwelling urethral catheters in cooperative male patients without urinary retention or bladder outlet obstruction. **(Category II)** (Key Question 2A)
2. Consider alternatives to chronic indwelling catheters, such as intermittent catheterization, in spinal cord injury patients. **(Category II)** (Key Question 1A)
3. Intermittent catheterization is preferable to indwelling urethral or suprapubic catheters in patients with bladder emptying dysfunction. **(Category II)** (Key Question 2A)
4. Consider intermittent catheterization in children with myelomeningocele and neurogenic bladder to reduce the risk of urinary tract deterioration. **(Category II)** (Key Question 1A)
5. Further research is needed on the benefit of using a urethral stent as an alternative to an indwelling catheter in selected patients with bladder outlet obstruction. **(No recommendation/unresolved issue)** (Key Question 1A)
6. Further research is needed on the risks and benefits of suprapubic catheters as an alternative to indwelling urethral catheters in selected patients requiring short- or long-term catheterization, particularly with respect to complications related to catheter insertion or the catheter site. **(No recommendation/unresolved issue)** (Key Question 2A)

II. Proper Techniques for Urinary Catheter Insertion

- A. Perform hand hygiene immediately before and after insertion or any manipulation of the catheter device or site. **(Category IB)** (Key Question 2D)
- B. Ensure that only properly trained persons (e.g., hospital personnel, family members, or patients themselves) who know the correct technique of aseptic catheter insertion and maintenance are given this responsibility. **(Category IB)** (Key Question 1B)
- C. In the acute care hospital setting, insert urinary catheters using aseptic technique and sterile equipment. **(Category IB)**
 - 1. Use sterile gloves, drape, sponges, an appropriate antiseptic or sterile solution for periurethral cleaning, and a single-use packet of lubricant jelly for insertion. **(Category IB)**
 - 2. Routine use of antiseptic lubricants is not necessary. **(Category II)** (Key Question 2C)
 - 3. Further research is needed on the use of antiseptic solutions vs. sterile water or saline for periurethral cleaning prior to catheter insertion. **(No recommendation/unresolved issue)** (Key Question 2C)
- D. In the non-acute care setting, clean (i.e., non-sterile) technique for intermittent catheterization is an acceptable and more practical alternative to sterile technique for patients requiring chronic intermittent catheterization. **(Category IA)** (Key Question 2A)
 - 1. Further research is needed on optimal cleaning and storage methods for catheters used for clean intermittent catheterization. **(No recommendation/unresolved issue)** (Key Question 2C)
- E. Properly secure indwelling catheters after insertion to prevent movement and urethral traction. **(Category IB)**
- F. Unless otherwise clinically indicated, consider using the smallest bore catheter possible, consistent with good drainage, to minimize bladder neck and urethral trauma. **(Category II)**
- G. If intermittent catheterization is used, perform it at regular intervals to prevent bladder overdistension. **(Category IB)** (Key Question 2A)
- H. Consider using a portable ultrasound device to assess urine volume in patients undergoing intermittent catheterization to assess urine volume and reduce unnecessary catheter insertions. **(Category II)** (Key Question 2C)
 - 1. If ultrasound bladder scanners are used, ensure that indications for use are clearly stated, nursing staff are trained in their use, and equipment is adequately cleaned and disinfected in between patients. **(Category IB)**

III. Proper Techniques for Urinary Catheter Maintenance

- A. Following aseptic insertion of the urinary catheter, maintain a closed drainage system **(Category IB)** (Key Question 1B and 2B)
 - 1. If breaks in aseptic technique, disconnection, or leakage occur, replace the catheter and collecting system using aseptic technique and sterile equipment. **(Category IB)**
 - 2. Consider using urinary catheter systems with preconnected, sealed catheter-tubing junctions. **(Category II)** (Key Question 2B)
- B. Maintain unobstructed urine flow. **(Category IB)** (Key Questions 1B and 2D)
 - 1. Keep the catheter and collecting tube free from kinking. **(Category IB)**
 - 2. Keep the collecting bag below the level of the bladder at all times. Do not rest the bag on the floor. **(Category IB)**
 - 3. Empty the collecting bag regularly using a separate, clean collecting container for each patient; avoid splashing, and prevent contact of the drainage spigot with the nonsterile collecting container. **(Category IB)**
- C. Use Standard Precautions, including the use of gloves and gown as appropriate, during any manipulation of the catheter or collecting system. **(Category IB)**
- D. Complex urinary drainage systems (utilizing mechanisms for reducing bacterial entry such as antiseptic-release cartridges in the drain port) are not necessary for routine use. **(Category II)** (Key Question 2B)
- E. Changing indwelling catheters or drainage bags at routine, fixed intervals is not recommended. Rather, it is suggested to change catheters and drainage bags based on clinical indications such as infection, obstruction, or when the closed system is compromised. **(Category II)** (Key Question 2C)
- F. Unless clinical indications exist (e.g., in patients with bacteriuria upon catheter removal post urologic surgery), do not use systemic antimicrobials routinely to prevent CAUTI in patients requiring either short or long-term catheterization. **(Category IB)** (Key Question 2C)
 - 1. Further research is needed on the use of urinary antiseptics (e.g., methenamine) to prevent UTI in patients requiring short-term catheterization. **(No recommendation/unresolved issue)** (Key Question 2C)
- G. Do not clean the periurethral area with antiseptics to prevent CAUTI while the catheter is in place. Routine hygiene (e.g., cleansing of the meatal surface during daily bathing or showering) is appropriate. **(Category IB)** (Key Question 2C)
- H. Unless obstruction is anticipated (e.g., as might occur with bleeding after prostatic or bladder surgery) bladder irrigation is not recommended. **(Category II)** (Key Question 2C)

1. If obstruction is anticipated, closed continuous irrigation is suggested to prevent obstruction. **(Category II)**
- I. Routine irrigation of the bladder with antimicrobials is not recommended. **(Category II)** (Key Question 2C)
- J. Routine instillation of antiseptic or antimicrobial solutions into urinary drainage bags is not recommended. **(Category II)** (Key Question 2C)
- K. Clamping indwelling catheters prior to removal is not necessary. **(Category II)** (Key Question 2C)
- L. Further research is needed on the use of bacterial interference (i.e., bladder inoculation with a nonpathogenic bacterial strain) to prevent UTI in patients requiring chronic urinary catheterization. **(No recommendation/unresolved issue)** (Key Question 2C)

Catheter Materials

- M. If the CAUTI rate is not decreasing after implementing a comprehensive strategy to reduce rates of CAUTI, consider using antimicrobial/antiseptic-impregnated catheters. The comprehensive strategy should include, at a minimum, the high priority recommendations for urinary catheter use, aseptic insertion, and maintenance (see Section III. Implementation and Audit). **(Category IB)** (Key Question 2B)
 1. Further research is needed on the effect of antimicrobial/antiseptic-impregnated catheters in reducing the risk of symptomatic UTI, their inclusion among the primary interventions, and the patient populations most likely to benefit from these catheters. **(No recommendation/unresolved issue)** (Key Question 2B)
- N. Hydrophilic catheters might be preferable to standard catheters for patients requiring intermittent catheterization. **(Category II)** (Key Question 2B)
- O. Silicone might be preferable to other catheter materials to reduce the risk of encrustation in long-term catheterized patients who have frequent obstruction. **(Category II)** (Key Question 3)
- P. Further research is needed to clarify the benefit of catheter valves in reducing the risk of CAUTI and other urinary complications. **(No recommendation/unresolved issue)** (Key Question 2B)

Management of Obstruction

- Q. If obstruction occurs and it is likely that the catheter material is contributing to obstruction, change the catheter. **(Category IB)**
- R. Further research is needed on the benefit of irrigating the catheter with acidifying solutions or use of oral urease inhibitors in long-term catheterized patients who have frequent catheter obstruction. **(No recommendation/unresolved issue)** (Key Question 3)

- S. Further research is needed on the use of a portable ultrasound device to evaluate for obstruction in patients with indwelling catheters and low urine output. **(No recommendation/unresolved issue)** (Key Question 2C)
- T. Further research is needed on the use of methenamine to prevent encrustation in patients requiring chronic indwelling catheters who are at high risk for obstruction. **(No recommendation/unresolved issue)** (Key Question 2C)

Specimen Collection

- U. Obtain urine samples aseptically. **(Category IB)**
 - 1. If a small volume of fresh urine is needed for examination (i.e., urinalysis or culture), aspirate the urine from the needleless sampling port with a sterile syringe/cannula adapter after cleansing the port with a disinfectant. **(Category IB)**
 - 2. Obtain large volumes of urine for special analyses (not culture) aseptically from the drainage bag. **(Category IB)**

Spatial Separation of Catheterized Patients

- V. Further research is needed on the benefit of spatial separation of patients with urinary catheters to prevent transmission of pathogens colonizing urinary drainage systems. **(No recommendation/unresolved issue)** (Key Question 2D)

IV. Quality Improvement Programs

- A. Implement quality improvement (QI) programs or strategies to enhance appropriate use of indwelling catheters and to reduce the risk of CAUTI based on a facility risk assessment. **(Category IB)** (Key Question 2D)

The purposes of QI programs should be: 1) to assure appropriate utilization of catheters 2) to identify and remove catheters that are no longer needed (e.g., daily review of their continued need) and 3) to ensure adherence to hand hygiene and proper care of catheters. Examples of programs that have been demonstrated to be effective include:

- 1. A system of alerts or reminders to identify all patients with urinary catheters and assess the need for continued catheterization
- 2. Guidelines and protocols for nurse-directed removal of unnecessary urinary catheters
- 3. Education and performance feedback regarding appropriate use, hand hygiene, and catheter care
- 4. Guidelines and algorithms for appropriate peri-operative catheter management, such as:

- a. Procedure-specific guidelines for catheter placement and postoperative catheter removal
- b. Protocols for management of postoperative urinary retention, such as nurse-directed use of intermittent catheterization and use of bladder ultrasound scanners

V. Administrative Infrastructure

A. Provision of guidelines

1. Provide and implement evidence-based guidelines that address catheter use, insertion, and maintenance. **(Category IB)**
 - a. Consider monitoring adherence to facility-based criteria for acceptable indications for indwelling urinary catheter use. **(Category II)**

B. Education and Training

1. Ensure that healthcare personnel and others who take care of catheters are given periodic in-service training regarding techniques and procedures for urinary catheter insertion, maintenance, and removal. Provide education about CAUTI, other complications of urinary catheterization, and alternatives to indwelling catheters. **(Category IB)**
2. When feasible, consider providing performance feedback to these personnel on what proportion of catheters they have placed meet facility-based criteria and other aspects related to catheter care and maintenance. **(Category II)**

C. Supplies

1. Ensure that supplies necessary for aseptic technique for catheter insertion are readily available. **(Category IB)**

D. System of documentation

1. Consider implementing a system for documenting the following in the patient record: indications for catheter insertion, date and time of catheter insertion, individual who inserted catheter, and date and time of catheter removal. **(Category II)**
 - a. Ensuring that documentation is accessible in the patient record and recorded in a standard format for data collection and quality improvement purposes is suggested. Electronic documentation that is searchable is preferable. **(Category II)**

E. Surveillance resources

1. If surveillance for CAUTI is performed, ensure that there are sufficient trained personnel and technology resources to support surveillance for urinary catheter use and outcomes. **(Category IB)**

VI. Surveillance

- A. Consider surveillance for CAUTI when indicated by facility-based risk assessment. **(Category II)**
 - 1. Identify the patient groups or units on which to conduct surveillance based on frequency of catheter use and potential risk of CAUTI.
- B. Use standardized methodology for performing CAUTI surveillance. **(Category IB)**
 - 1. Examples of metrics that should be used for CAUTI surveillance include:
 - a. Number of CAUTI per 1000 catheter-days
 - b. Number of bloodstream infections secondary to CAUTI per 1000 catheter-days
 - c. Catheter utilization ratio: (urinary catheter days/patient days) x 100
 - 2. Use CDC/NHSN criteria for identifying patients who have symptomatic UTI (SUTI) (numerator data) (see NHSN Patient Safety Manual: <http://www.cdc.gov/nhsn/library.html>).
 - 3. For more information on metrics, please see the U.S. Department of Health & Human Services (HHS) Action Plan to Prevent Healthcare-Associated Infections: <http://www.hhs.gov/ophs/initiatives/hai/infection.html>.
- C. Routine screening of catheterized patients for asymptomatic bacteriuria (ASB) is not recommended. **(Category II)** (Key Question 2D)
- D. When performing surveillance for CAUTI, consider providing regular (e.g., quarterly) feedback of unit-specific CAUTI rates to nursing staff and other appropriate clinical care staff. **(Category II)** (Key Question 2D)

III. Implementation and Audit

Prioritization of Recommendations

In this section, the recommendations considered essential for *all* healthcare facilities caring for patients requiring urinary catheterization are organized into modules in order to provide more guidance to facilities on implementation of these guidelines. The high-priority recommendations were chosen by a consensus of experts based on strength of recommendation as well as on the likely impact of the strategy in preventing CAUTI. The administrative functions and infrastructure listed above in the summary of recommendations are necessary to accomplish the high priority recommendations and are therefore critical to the success of a prevention program. In addition, quality improvement programs should be implemented as an active approach to accomplishing these recommendations and when process and outcome measure goals are not being met based on internal reporting.

Priority Recommendations for Appropriate Urinary Catheter Use (Module 1)

- Insert catheters only for appropriate indications (see Table 2), and leave in place only as long as needed. **(Category IB)**
 - Avoid use of urinary catheters in patients and nursing home residents for management of incontinence. **(Category IB)**
 - For operative patients who have an indication for an indwelling catheter, remove the catheter as soon as possible postoperatively, preferably within 24 hours, unless there are appropriate indications for continued use. **(Category IB)**

Priority Recommendations for Aseptic Insertion of Urinary Catheters (Module 2)

- Ensure that only properly trained persons (e.g., hospital personnel, family members, or patients themselves) who know the correct technique of aseptic catheter insertion and maintenance are given this responsibility. **(Category IB)**
- In the acute care hospital setting, insert catheters using aseptic technique and sterile equipment. **(Category IB)**

Priority Recommendations for Proper Urinary Catheter Maintenance (Module 3)

- Following aseptic insertion of the urinary catheter, maintain a closed drainage system **(Category IB)**
- Maintain unobstructed urine flow. **(Category IB)**

Performance Measures

- A. Internal Reporting. Consider reporting both process and outcome measures to senior administrative, medical, and nursing leadership and clinicians who care for patients at risk for CAUTI. **(Category II)**
 1. Examples of process measures:
 - a) Compliance with educational program: Calculate percent of personnel who have proper training:
 - Numerator: number of personnel who insert urinary catheters and who have proper training
 - Denominator: number of personnel who insert urinary catheters
 - Standardization factor: 100 (i.e., multiply by 100 so that measure is expressed as a percentage)

- b) Compliance with documentation of catheter insertion and removal dates: Conduct random audits of selected units and calculate compliance rate:
 - Numerator: number of patients on unit with catheters with proper documentation of insertion and removal dates
 - Denominator: number of patients on the unit with a catheter in place at some point during admission
 - Standardization factor: 100 (i.e., multiply by 100 so that measure is expressed as a percentage)
 - c) Compliance with documentation of indication for catheter placement: Conduct random audits of selected units and calculate compliance rate
 - Numerator: number of patients on unit with catheters with proper documentation of indication
 - Denominator: number of patients on the unit with catheter in place
 - Standardization factor: 100 (i.e., multiply by 100 so that measure is expressed as a percentage)
2. Recommended outcome measures:
- a) Rates of CAUTI: Use NHSN definitions (see <http://www.cdc.gov/nhsn/library.html>). Measurement of rates allows an individual facility to gauge the longitudinal impact of implementation of prevention strategies:
 - Numerator: number of CAUTIs in each location monitored
 - Denominator: total number of urinary catheter-days for all patients that have an indwelling urinary catheter in each location monitored
 - Standardization factor: Multiply by 1000 so that the measure is expressed as cases per 1000 catheter-days
 - b) Rate of bloodstream infections secondary to CAUTI: Use NHSN definitions for laboratory-confirmed bloodstream infection, available at <http://www.cdc.gov/nhsn/library.html>.
 - Numerator: number of episodes of bloodstream infections secondary to CAUTI
 - Denominator: total number of urinary catheter-days for all patients that have an indwelling urinary catheter in each location monitored
 - Standardization factor: Multiply by 1000 so that the measure is expressed as cases per 1000 catheter-days
- B. External Reporting. Current NHSN definitions for CAUTI were developed for monitoring of rates within a facility; however, reporting of CAUTI rates for facility-to-facility comparison might be requested by state requirements and external quality initiatives.

IV. Recommendations for Further Research

Our literature review revealed that many of the studies addressing strategies to prevent CAUTI were not of sufficient quality to allow firm conclusions regarding the benefit of certain interventions. Future studies of CAUTI prevention should:

- 1) Be primary analytic research (i.e. systematic reviews, meta-analyses, interventional studies, and observational studies [cohort, case-control, analytic cross-sectional studies])
- 2) Evaluate clinically relevant outcomes (e.g., SUTI, bloodstream infections secondary to CAUTI)
- 3) Adjust for confounders as needed using multivariable analyses
- 4) Stratify outcomes by patient populations at risk for CAUTI
- 5) Ensure adequate statistical power to detect differences

The following is a compilation of recommendations for further research:

1. Catheter materials
 - a. Antimicrobial and antiseptic-impregnated catheters
 - i. Effect of catheters on reducing the risk of SUTI and other clinically significant outcomes
 - ii. Patient populations most likely to benefit
 - iii. Incidence of antimicrobial resistance in urinary pathogens
 - iv. Role of bacterial biofilms in the pathogenesis of CAUTI
 - b. Standard catheters
 - i. Optimal materials for reducing the risk of CAUTI and other urethral complications
2. Appropriate urinary catheter use
 - a. Incontinent patients
 - i. Risks and benefits of periodic (e.g., nighttime) use of external catheters
 - ii. Risk of local complications (e.g., skin maceration, phimosis) with the use of external catheters
 - iii. Appropriate use of urinary catheters to manage sacral or perineal wounds
 - b. Appropriate indications for continued use in postoperative patients and associated risks
3. Antiseptics
 - a. Use of antiseptic vs. sterile solutions for periurethral cleaning prior to catheter insertion
 - b. Use of antiseptics (e.g., methenamine) to prevent CAUTI
4. Alternatives to indwelling urethral catheters and bag drainage
 - a. Risks and benefits of suprapubic catheters as an alternative to chronic indwelling urethral catheters
 - b. Use of a urethral stent as an alternative to an indwelling catheter in selected patients with bladder outlet obstruction
 - c. Use of catheter valves in reducing the risk of CAUTI and other urinary complications
 - d. Other alternative methods of urinary drainage

5. Optimal methods for preventing encrustation in long-term catheterized patients who have frequent obstruction
 - a. Optimal catheter materials
 - b. Irrigation with acidifying solutions or oral urease inhibitors
 - c. Use of methenamine

6. Other prevention measures
 - a. Use of portable ultrasound in patients with low-urine output to reduce unnecessary catheter insertions or irrigations (in catheterized patients)
 - b. Use of new prevention strategies such as bacterial interference in patients requiring chronic catheterization
 - c. Optimal cleaning and storage procedures (e.g., wet vs. dry storage) for catheters used for clean intermittent catheterization

7. Prevention of transmission
 - a. Spatial separation of patients with urinary catheters (in the absence of epidemic spread or frequent cross-infection) to prevent transmission of pathogens colonizing urinary drainage systems

V. Background

Urinary tract infections are the most common type of healthcare-associated infection, accounting for more than 30% of infections reported by acute care hospitals.¹⁹ Virtually all healthcare-associated UTIs are caused by instrumentation of the urinary tract. Catheter-associated urinary tract infection (CAUTI) has been associated with increased morbidity, mortality, hospital cost, and length of stay.⁶⁻⁹ In addition, bacteriuria commonly leads to unnecessary antimicrobial use, and urinary drainage systems are often reservoirs for multidrug-resistant bacteria and a source of transmission to other patients.^{10,11}

Definitions

An indwelling urinary catheter is a drainage tube that is inserted into the urinary bladder through the urethra, is left in place, and is connected to a closed collection system. Alternative methods of urinary drainage may be employed in some patients. Intermittent (“in-and-out”) catheterization involves brief insertion of a catheter into the bladder through the urethra to drain urine at intervals. An external catheter is a urine containment device that fits over or adheres to the genitalia and is attached to a urinary drainage bag. The most commonly used external catheter is a soft flexible sheath that fits over the penis (“condom” catheter). A suprapubic catheter is surgically inserted into the bladder through an incision above the pubis.

Although UTIs associated with alternative urinary drainage systems are considered device-associated, CAUTI rates reported to the National Healthcare Safety Network (NHSN) only refer to those associated with indwelling urinary catheters. NHSN has recently revised the UTI surveillance definition criteria. Among the changes are removal of the asymptomatic bacteriuria (ASB) criterion and refinement of the criteria for defining symptomatic UTI (SUTI). The time period for follow-up surveillance after catheter removal also has been shortened from 7 days to 48 hours to align with other device-associated infections. The new UTI criteria, which took effect in January 2009, can be found in the NHSN Patient Safety Manual (<http://www.cdc.gov/nhsn/library.html>).

The limitations and heterogeneity of definitions of CAUTI used in various studies present major challenges in appraising the quality of evidence in the CAUTI literature. Study investigators have used numerous different definitions for CAUTI outcomes, ranging from simple bacteriuria at a range of concentrations to, less commonly, symptomatic infection defined by combinations of bacteriuria and various signs and symptoms. Furthermore, most studies that used CDC/NHSN definitions for CAUTI did not distinguish between SUTI and ASB in their analyses.³⁰ The heterogeneity of definitions used for CAUTI may reduce the quality of evidence for a given intervention and often precludes meta-analyses.

The clinical significance of ASB in catheterized patients is undefined. Approximately 75% to 90% of patients with ASB do not develop a systemic inflammatory response or other signs or symptoms to suggest infection.^{6,31} Monitoring and treatment of ASB is also not an effective prevention measure for SUTI, as most cases of SUTI are not preceded by bacteriuria for more than a day.²⁵ Treatment of ASB has not been shown to be clinically beneficial and is associated with the selection of antimicrobial-resistant organisms.

Epidemiology

Between 15% and 25% of hospitalized patients may receive short-term indwelling urinary catheters.^{12,13} In many cases, catheters are placed for inappropriate indications, and healthcare providers are often unaware that their patients have catheters, leading to prolonged, unnecessary use.¹⁴⁻¹⁶ In acute care hospitals reporting to NHSN in 2006, pooled mean urinary catheter utilization ratios in ICU and non-ICU areas ranged from 0.23-0.91 urinary catheter-days/patient-days.¹⁷ While the numbers of units reporting were small, the highest ratios were in trauma ICUs and the lowest in inpatient medical/surgical wards. The overall prevalence of long-term indwelling urethral catheterization use is unknown. The prevalence of urinary catheter use in residents in long-term care facilities in the United States is on the order of 5%, representing approximately 50,000 residents with catheters at any given time.¹⁸ This number appears to be declining over time, likely because of federally mandated nursing home quality measures. However, the high prevalence of urinary catheters in patients transferred to skilled nursing facilities suggests that acute care hospitals should focus more efforts on removing unnecessary catheters prior to transfer.¹⁸

Reported rates of UTI among patients with urinary catheters vary substantially. National data from NHSN acute care hospitals in 2006 showed a range of pooled mean CAUTI rates of 3.1-7.5 infections per 1000 catheter-days.¹⁷ The highest rates were in burn ICUs, followed by inpatient medical wards and neurosurgical ICUs, although these sites also had the fewest numbers of locations reporting. The lowest rates were in medical/surgical ICUs.

Although morbidity and mortality from CAUTI is considered to be relatively low compared to other HAIs, the high prevalence of urinary catheter use leads to a large cumulative burden of infections with resulting infectious complications and deaths. An estimate of annual incidence of HAIs and mortality in 2002, based on a broad survey of US hospitals, found that urinary tract infections made up the highest number of infections (> 560,000) compared to other HAIs, and attributable deaths from UTI were estimated to be over 13,000 (mortality rate 2.3%).¹⁹ And while fewer than 5% of bacteriuric cases develop bacteremia,⁶ CAUTI is the leading cause of secondary nosocomial bloodstream infections; about 17% of hospital-acquired bacteremias are from a urinary source, with an associated mortality of approximately 10%.²⁰ In the nursing home setting, bacteremias are most commonly caused by UTIs, the majority of which are catheter-related.²¹

An estimated 17% to 69% of CAUTI may be preventable with recommended infection control measures, which means that up to 380,000 infections and 9000 deaths related to CAUTI per year could be prevented.²²

Pathogenesis and Microbiology

The source of microorganisms causing CAUTI can be endogenous, typically via meatal, rectal, or vaginal colonization, or exogenous, such as via contaminated hands of healthcare personnel or equipment. Microbial pathogens can enter the urinary tract either by the extraluminal route, via migration along the outside of the catheter in the periurethral mucous sheath, or by the intraluminal route, via movement along the internal lumen of the catheter from a contaminated collection bag or catheter-drainage tube junction. The relative contribution of each route in the pathogenesis of CAUTI is not well known. The marked reduction in risk of bacteriuria with the introduction of the sterile, closed urinary drainage system in the 1960's²³ suggests the importance of the intraluminal route. However, even with the closed drainage system,

bacteriuria inevitably occurs over time either via breaks in the sterile system or via the extraluminal route.²⁴ The daily risk of bacteriuria with catheterization is 3% to 10%,^{25,26} approaching 100% after 30 days, which is considered the delineation between short and long-term catheterization.²⁷

Formation of biofilms by urinary pathogens on the surface of the catheter and drainage system occurs universally with prolonged duration of catheterization.²⁸ Over time, the urinary catheter becomes colonized with microorganisms living in a sessile state within the biofilm, rendering them resistant to antimicrobials and host defenses and virtually impossible to eradicate without removing the catheter. The role of bacteria within biofilms in the pathogenesis of CAUTI is unknown and is an area requiring further research.

The most frequent pathogens associated with CAUTI (combining both ASB and SUTI) in hospitals reporting to NHSN between 2006-2007 were *Escherichia coli* (21.4%) and *Candida* spp (21.0%), followed by *Enterococcus* spp (14.9%), *Pseudomonas aeruginosa* (10.0%), *Klebsiella pneumoniae* (7.7%), and *Enterobacter* spp (4.1%). A smaller proportion was caused by other gram-negative bacteria and *Staphylococcus* spp⁵.

Antimicrobial resistance among urinary pathogens is an ever increasing problem. About a quarter of *E. coli* isolates and one third of *P. aeruginosa* isolates from CAUTI cases were fluoroquinolone-resistant. Resistance of gram-negative pathogens to other agents, including third-generation cephalosporins and carbapenems, was also substantial⁵. The proportion of organisms that were multidrug-resistant, defined by non-susceptibility to all agents in 4 classes, was 4% of *P. aeruginosa*, 9% of *K. pneumoniae*, and 21% of *Acinetobacter baumannii*.²⁹

VI. Scope and Purpose

This guideline updates and expands the original CDC Guideline for Prevention of CAUTI published in 1981. The revised guideline addresses the prevention of CAUTI for patients in need of either short- or long-term (i.e., > 30 days) urinary catheterization in any type of healthcare facility and evaluates evidence for alternative methods of urinary drainage, including intermittent catheterization, external catheters, and suprapubic catheters. The guideline also includes specific recommendations for implementation, performance measurement, and surveillance. Recommendations for further research are also provided to address the knowledge gaps in CAUTI prevention identified during the literature review.

To evaluate the evidence on preventing CAUTI, we examined data addressing three key questions and related subquestions:

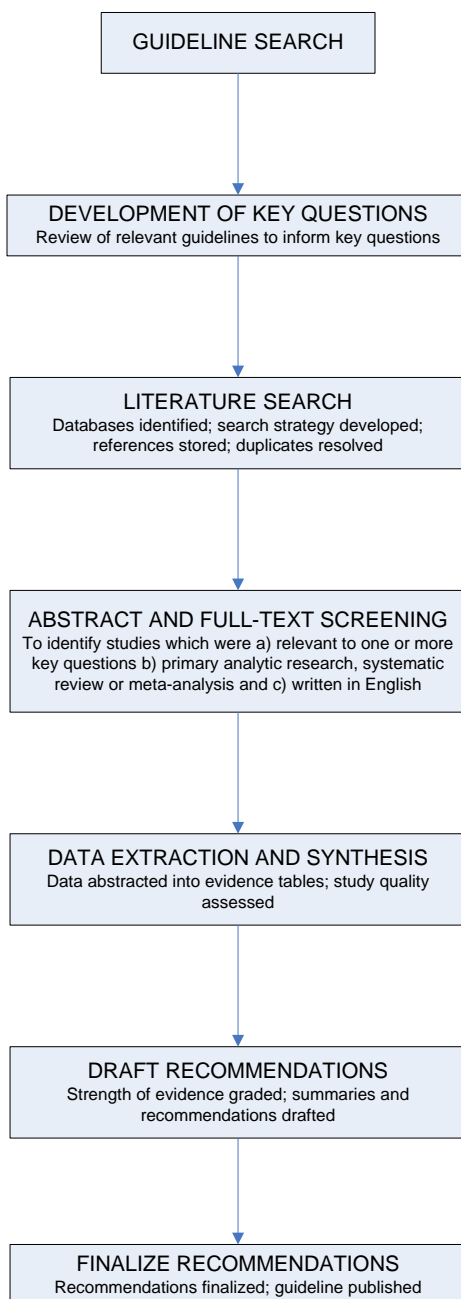
1. Who should receive urinary catheters?
 - A. When is urinary catheterization necessary?
 - B. What are the risk factors for CAUTI?
 - C. What populations are at highest risk of mortality from catheters?
2. For those who may require urinary catheters, what are the best practices?
Specifically, what are the risks and benefits associated with:
 - A. Different approaches to catheterization?
 - B. Different catheters or collecting systems?
 - C. Different catheter management techniques?
 - D. Different systems interventions (i.e., quality improvement programs)?
3. What are the best practices for preventing UTI associated with obstructed urinary catheters?

This document is intended for use by infection prevention staff, healthcare epidemiologists, healthcare administrators, nurses, other healthcare providers, and persons responsible for developing, implementing, and evaluating infection prevention and control programs for healthcare settings across the continuum of care. The guideline can also be used as a resource for societies or organizations that wish to develop more detailed implementation guidance for prevention of CAUTI.

VII. Methods

This guideline was based on a targeted systematic review of the best available evidence on CAUTI prevention. We used the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach³²⁻³⁴ to provide explicit links between the available evidence and the resulting recommendations. Our guideline development process is outlined in *Figure 1*.

Figure 1. The Guideline Development Process



Development of Key Questions

We first conducted an electronic search of the National Guideline Clearinghouse® (Agency for Healthcare Research and Quality), Medline® (National Library of Medicine) using the Ovid® Platform (Ovid Technologies, Wolters Kluwer, New York, NY), the Cochrane® Health Technology Assessment Database (Cochrane Collaboration, Oxford, UK), the NIH Consensus Development Program, and the United States Preventive Services Task Force database for existing national and international guidelines relevant to CAUTI. The strategy used for the guideline search and the search results can be found in [Appendix 1A](#). A preliminary list of key questions was developed from a review of the relevant guidelines identified in the search.^{1,35,36} Key questions were finalized after vetting them with a panel of content experts and HICPAC members.

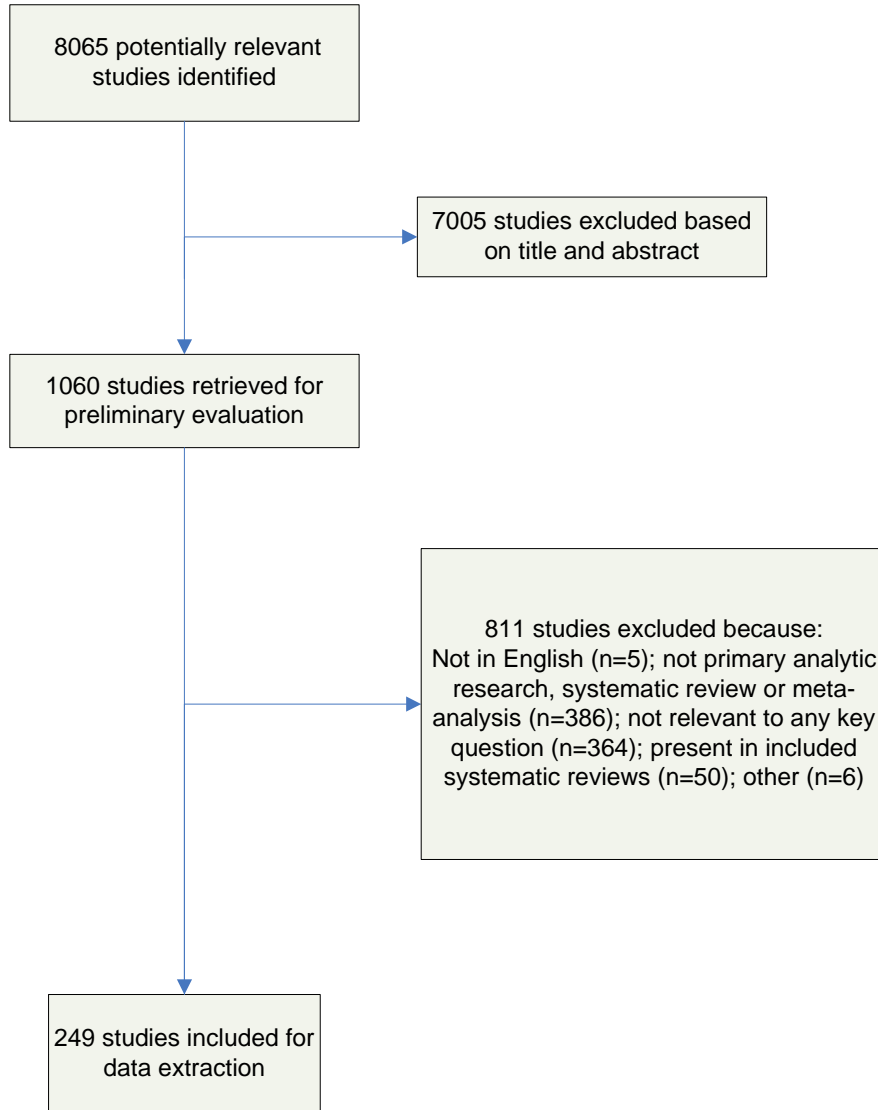
Literature Search

Following the development of the key questions, search terms were developed for identifying literature relevant to the key questions. For the purposes of quality assurance, we compared these terms to those used in relevant seminal studies and guidelines. These search terms were then incorporated into search strategies for the relevant electronic databases. Searches were performed in Medline® (National Library of Medicine) using the Ovid® Platform (Ovid Technologies, Wolters Kluwer, New York, NY), EMBASE® (Elsevier BV, Amsterdam, Netherlands), CINAHL® (Ebsco Publishing, Ipswich, MA) and Cochrane® (Cochrane Collaboration, Oxford, UK) (all databases were searched in July 2007), and the resulting references were imported into a reference manager, where duplicates were resolved. For Cochrane reviews ultimately included in our guideline, we checked for updates in July 2008. The detailed search strategy used for identifying primary literature and the results of the search can be found in [Appendix 1B](#).

Study Selection

Titles and abstracts from references were screened by a single author (C.V.G, R.K.A., or D.A.P.) and the full text articles were retrieved if they were 1) relevant to one or more key questions, 2) primary analytic research, systematic reviews or meta-analyses, and 3) written in English. Likewise, the full-text articles were screened by a single author (C.V.G. or D.A.P.) using the same criteria, and included studies underwent a second review for inclusion by another author (R.K.A.). Disagreements were resolved by the remaining authors. The results of this process are depicted in *Figure 2*.

Figure 2: Results of the Study Selection Process



Data Extraction and Synthesis

Data on the study author, year, design, objective, population, setting, sample size, power, follow-up, and definitions and results of clinically relevant outcomes were extracted into evidence tables ([Appendix 2](#)). Three evidence tables were developed, each of which represented one of our key questions. Studies were extracted into the most relevant evidence table. Then, studies were organized by the common themes that emerged within each evidence table. Data were extracted by one author (R.K.A.) and cross-checked by another (C.V.G.). Disagreements were resolved by the remaining authors. Data and analyses were extracted as originally presented in the included studies. Meta-analyses were performed only where their use was deemed critical to a recommendation, and only in circumstances where multiple studies with sufficiently homogenous populations, interventions, and outcomes could be analyzed. Systematic reviews were included in our review. To avoid duplication of data, we excluded primary studies if they were also included in a systematic review captured by our search. The only exception to this was if the primary study also addressed a relevant question that was outside the scope of the included systematic review. Before exclusion, data from the primary studies that we originally captured were abstracted into the evidence tables and reviewed. We also excluded systematic reviews that analyzed primary studies that were fully captured in a more recent systematic review. The only exception to this was if the older systematic review also addressed a relevant question that was outside the scope of the newer systematic review. To ensure that all relevant studies were captured in the search, the bibliography was vetted by a panel of clinical experts.

Grading of Evidence

First, the quality of each study was assessed using scales adapted from existing methodology checklists, and scores were recorded in the evidence tables. [Appendix 3](#) includes the sets of questions we used to assess the quality of each of the major study designs. Next, the quality of the evidence base was assessed using methods adapted from the GRADE Working Group.³² Briefly, GRADE tables were developed for each of the interventions or questions addressed within the evidence tables. Included in the GRADE tables were the intervention of interest, any outcomes listed in the evidence tables that were judged to be clinically important, the quantity and type of evidence for each outcome, the relevant findings, and the GRADE of evidence for each outcome, as well as an overall GRADE of the evidence base for the given intervention or question. The initial GRADE of evidence for each outcome was deemed high if the evidence base included a randomized controlled trial (RCT) or a systematic review of RCTs, low if the evidence base included only observational studies, or very low if the evidence base consisted only of uncontrolled studies. The initial GRADE could then be modified by eight criteria.³⁴ Criteria which could decrease the GRADE of an evidence base included quality, consistency, directness, precision, and publication bias. Criteria that could increase the GRADE included a large magnitude of effect, a dose-response gradient, or inclusion of unmeasured confounders that would increase the magnitude of effect ([Table 3](#)). GRADE definitions are as follows:

1. High - further research is very unlikely to change confidence in the estimate of effect
2. Moderate - further research is likely to affect confidence in the estimate of effect and may change the estimate
3. Low - further research is very likely to affect confidence in the estimate of effect and is likely to change the estimate
4. Very low - any estimate of effect is very uncertain

After determining the GRADE of the evidence base for each outcome of a given intervention or question, we calculated the overall GRADE of the evidence base for that intervention or question. The overall GRADE was based on the lowest GRADE for the outcomes deemed critical to making a recommendation.

Table 3. Rating the Quality of Evidence Using the GRADE Approach

Type of Evidence	Initial Grade	Criteria to Decrease Grade	Criteria to Increase Grade	Overall Quality Grade
RCT	High	<u>Quality</u> Serious (-1 grade) or very serious (-2 grades) limitation to study quality	<u>Strong association</u> Strong (+1 grade) or very strong evidence of association (+2 grades)	High
				Moderate
Observational study	Low	<u>Consistency</u> Important inconsistency (-1 grade)	<u>Dose-response</u> Evidence of a dose-response gradient (+1 grade)	Low
Any other evidence (e.g., expert opinion)	Very low	<u>Directness</u> Some (-1 grade) or major (-2 grades) uncertainty about directness	<u>Unmeasured Confounders</u> Inclusion of unmeasured confounders increases the magnitude of effect (+1 grade)	Very low
		<u>Precision</u> Imprecise or sparse data (-1 grade)		
		<u>Publication bias</u> High risk of bias (-1 grade)		

Formulating Recommendations

Narrative evidence summaries were then drafted by the working group using the evidence and GRADE tables. One summary was written for each theme that emerged under each key question. The working group then used the narrative evidence summaries to develop guideline recommendations. Factors determining the strength of a recommendation included 1) the values and preferences used to determine which outcomes were "critical," 2) the harms and benefits that result from weighing the "critical" outcomes, and 3) the overall GRADE of the evidence base for the given intervention or question (Table 4).³³ If weighing the "critical outcomes" for a given intervention or question resulted in a "net benefit" or a "net harm," then a "Category I Recommendation" was formulated to strongly recommend for or against the given intervention respectively. If weighing the "critical outcomes" for a given intervention or question resulted in a "trade off" between benefits and harms, then a "Category II Recommendation" was formulated to recommend that providers or institutions consider the intervention when deemed appropriate. If weighing the "critical outcomes" for a given intervention or question resulted in

an "uncertain trade off" between benefits and harms, then a "No Recommendation" was formulated to reflect this uncertainty.

HICPAC Recommendation	Weighing Benefits and Harms for Critical Outcomes	Quality of Evidence
STRONG (I)	Interventions with net benefits or net harms	IA – High to Moderate IB – Low or Very Low (Accepted Practice) IC – High to Very Low (Regulatory)
WEAK (II)	Interventions with trade offs between benefits and harms	High to Very Low
No recommendation/ unresolved issue	Uncertain trade offs between benefits and harms	Low to Very Low

For Category I recommendations, levels A and B represent the quality of the evidence underlying the recommendation, with A representing high to moderate quality evidence and B representing low quality evidence or, in the case of an established standard (e.g., aseptic technique, education and training), very low quality to no evidence based on our literature review. For IB recommendations, although there may be low to very low quality or even no available evidence directly supporting the benefits of the intervention, the theoretical benefits are clear, and the theoretical risks are marginal. Level C represents practices required by state or federal regulation, regardless of the quality of evidence. It is important to note that the strength of a Category IA recommendation is equivalent to that of a Category IB or IC recommendation; it is only the quality of the evidence underlying the IA recommendation that makes it different from a IB.

In some instances, multiple recommendations emerged from a single narrative evidence summary. The new HICPAC categorization scheme for recommendations is provided in [Table 1](#), which is reproduced below.

Category IA	A strong recommendation supported by high to moderate quality evidence suggesting net clinical benefits or harms
Category IB	A strong recommendation supported by low quality evidence suggesting net clinical benefits or harms or an accepted practice (e.g., aseptic technique) supported by low to very low quality evidence
Category IC	A strong recommendation required by state or federal regulation.
Category II	A weak recommendation supported by any quality evidence suggesting a trade off between clinical benefits and harms
No recommendation/ unresolved issue	Unresolved issue for which there is low to very low quality evidence with uncertain trade offs between benefits and harms

Category I recommendations are defined as strong recommendations with the following implications:

1. For patients: Most people in the patient's situation would want the recommended course of action and only a small proportion would not; request discussion if the intervention is not offered.
2. For clinicians: Most patients should receive the recommended course of action.
3. For policymakers: The recommendation may be adopted as a policy.

Category II recommendations are defined as weak recommendations with the following implications:

1. For patients: Most people in the patient's situation would want the recommended course of action, but many would not.
2. For clinicians: Different choices will be appropriate for different patients, and clinicians must help each patient to arrive at a management decision consistent with her or his values and preferences.
3. For policymakers: Policy making will require substantial debate and involvement of many stakeholders.

It should be noted that Category II recommendations are discretionary for the individual institution and are not intended to be enforced.

The wording of each recommendation was carefully selected to reflect the recommendation's strength. In most cases, we used the active voice when writing Category I recommendations - the strong recommendations. Phrases like "do" or "do not" and verbs without auxiliaries or conditionals were used to convey certainty. We used a more passive voice when writing Category II recommendations - the weak recommendations. Words like "consider" and phrases like "is preferable," "is suggested," "is not suggested," or "is not recommended" were chosen to reflect the lesser certainty of the Category II recommendations. Rather than a simple statement of fact, each recommendation is actionable, describing precisely a proposed action to take.

The category "No recommendation/unresolved issue" was most commonly applied to situations where either 1) the overall quality of the evidence base for a given intervention was low to very low and there was no consensus on the benefit of the intervention or 2) there was no published evidence on outcomes deemed critical to weighing the risks and benefits of a given intervention. If the latter was the case, those critical outcomes will be noted at the end of the relevant evidence summary.

Our evidence-based recommendations were cross-checked with those from guidelines identified in our original systematic search. Recommendations from previous guidelines for topics not directly addressed by our systematic review of the evidence were included in our "Summary of Recommendations" if they were deemed critical to the target users of this guideline. Unlike recommendations informed by our literature search, these recommendations are not linked to a key question. These recommendations were agreed upon by expert consensus and are designated either IB if they represent a strong recommendation based on accepted practices (e.g., aseptic technique) or II if they are a suggestion based on a probable net benefit despite limited evidence.

All recommendations were approved by HICPAC. Recommendations focused only on efficacy, effectiveness, and safety. The optimal use of these guidelines should include a consideration of the costs relevant to the local setting of guideline users.

Reviewing and Finalizing the Guideline

After a draft of the tables, narrative summaries, and recommendations was completed, the working group shared the draft with the expert panel for in-depth review. While the expert panel was reviewing this draft, the working group completed the remaining sections of the guideline, including the executive summary, background, scope and purpose, methods, summary of recommendations, and recommendations for guideline implementation, audit, and further research. The working group then made revisions to the draft based on feedback from members of the expert panel and presented the entire draft guideline to HICPAC for review. The guideline was then posted on the Federal Register for public comment. After a period of public comment, the guideline was revised accordingly, and the changes were reviewed and voted on by HICPAC. The final guideline was cleared internally by CDC and published and posted on the HICPAC website.

Updating the Guideline

Future revisions to this guideline will be dictated by new research and technological advancements for preventing CAUTI and will occur at the request of HICPAC.

VIII. Evidence Review

Q1. Who should receive urinary catheters?

To answer this question, we focused on three subquestions: A) When is urinary catheterization necessary? B) What are the risk factors for CAUTI? and C) What populations are at highest risk of mortality from urinary catheters?

Q1A. When is urinary catheterization necessary?

The available data examined five main populations. In all populations, we considered CAUTI outcomes as well as other outcomes we deemed critical to weighing the risks and benefits of catheterization. The evidence for this question consists of 1 systematic review,³⁷ 9 RCTs,³⁸⁻⁴⁶ and 12 observational studies.⁴⁷⁻⁵⁸ The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 1A.

For *operative patients*, low-quality evidence suggested a benefit of avoiding urinary catheterization.^{37-44,47-49} This was based on a decreased risk of bacteriuria/unspecified UTI, no effect on bladder injury, and increased risk of urinary retention in patients without catheters. Urinary retention in patients without catheters was specifically seen following urogenital surgeries. The most common surgeries studied were urogenital, gynecological, laparoscopic, and orthopedic surgeries. Our search did not reveal data on the impact of catheterization on peri-operative hemodynamic management.

For *incontinent patients*, low-quality evidence suggested a benefit of avoiding urinary catheterization.^{45,50-52} This was based on a decreased risk of both SUTI and bacteriuria/unspecified UTI in male nursing home residents without urinary catheters compared to those with continuous condom catheters. We found no difference in the risk of UTI between having a condom catheter only at night and having no catheter. Our search did not reveal data on the impact of catheterization on skin breakdown.

For *patients with bladder outlet obstruction*, very low-quality evidence suggested a benefit of a urethral stent over an indwelling catheter.⁵³ This was based on a reduced risk of bacteriuria in those receiving a urethral stent. Our search did not reveal data on the impact of catheterization versus stent placement on urinary complications.

For *patients with spinal cord injury*, very low-quality evidence suggested a benefit of avoiding indwelling urinary catheters.^{54,56} This was based on a decreased risk of SUTI and bacteriuria in those without indwelling catheters (including patients managed with spontaneous voiding, clean intermittent catheterization [CIC], and external striated sphincterotomy with condom catheter drainage), as well as a lower risk of urinary complications, including hematuria, stones, and urethral injury (fistula, erosion, stricture).

For *children with myelomeningocele and neurogenic bladder*, very low-quality evidence suggested a benefit of CIC compared to urinary diversion or self voiding.^{46,57,58} This was based on a decreased risk of bacteriuria/unspecified UTI in patients receiving CIC compared to urinary diversion, and a lower risk of urinary tract deterioration (defined by febrile urinary tract infection, vesicoureteral reflux, hydronephrosis, or increases in BUN or serum creatinine) compared to self-voiding and in those receiving CIC early (< 1 year of age) versus late (> 3 years of age).

Evidence Review Table 1A. When is urinary catheterization necessary?

1A.1. Use urinary catheters in operative patients only as necessary, rather than routinely. **(Category IB)**

1A.2. Avoid use of urinary catheters in patients and nursing home residents for management of incontinence. **(Category IB)**

1A.2.a. Further research is needed on periodic (e.g., nighttime) use of external catheters in incontinent patients or residents and the use of catheters to prevent skin breakdown. **(No recommendation/unresolved issue)**

1A.3. Further research is needed on the benefit of using a urethral stent as an alternative to an indwelling catheter in selected patients with bladder outlet obstruction. **(No recommendation/unresolved issue)**

1A.4. Consider alternatives to chronic indwelling catheters, such as intermittent catheterization, in spinal cord injury patients. **(Category II)**

1A.5. Consider intermittent catheterization in children with myelomeningocele and neurogenic bladder to reduce the risk of urinary tract deterioration. **(Category II)**

Q1B. What are the risk factors for CAUTI?

To answer this question, we reviewed the quality of evidence for those risk factors examined in more than one study. We considered the critical outcomes for decision-making to be SUTI and bacteriuria. The evidence for this question consists of 11 RCTs⁵⁹⁻⁶⁹ and 37 observational studies.^{9,50,54,70-103} The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 1B.

For *SUTI*,^{50,54,61,62,74,75,79,83,102,103} low-quality evidence suggested that female sex, older age, prolonged catheterization, impaired immunity, and lack of antimicrobial exposure are risk factors. Very low quality evidence suggested that catheter blockage and low albumin level are also risk factors. For *bacteriuria*,^{9,59-61,63-68,72,73,76-78,82,84-86,89-94,96-100} multiple risk factors were identified; there was high quality evidence for prolonged catheterization and moderate quality evidence for female sex, positive meatal cultures, and lack of antimicrobial exposure. Low-quality evidence also implicated the following risk factors for bacteriuria: older age, disconnection of the drainage system, diabetes, renal dysfunction, higher severity of illness, impaired immunity, placement of the catheter outside of the operating room, lower professional training of the person inserting the catheter, incontinence, and being on an orthopaedic or neurology service. Our search did not reveal data on adverse events and antimicrobial resistance associated with antimicrobial use, although one observational study found that the protective effect of antimicrobials lasted only for the first four days of catheterization, and that antimicrobial exposure led to changes in the epidemiology of bacterial flora in the urine.

Evidence Review Table 1B. What are the risk factors for CAUTI?

1B.1. Following aseptic insertion of the urinary catheter, maintain a closed drainage system. **(Category IB)^a**

1B.2. Insert catheters only for appropriate indications, and leave in place only as long as needed. **(Category IB)^b**

1B.3. Minimize urinary catheter use and duration of use in all patients, particularly those at higher risk for CAUTI such as women, the elderly, and patients with impaired immunity. **(Category IB)**

1B.4. Ensure that only properly trained persons (e.g., hospital personnel, family members, or patients themselves) who know the correct technique of aseptic catheter insertion and maintenance are given this responsibility. **(Category IB)**

1B.5. Maintain unobstructed urine flow. **(Category IB)^c**

^a More data are available under Question 2B.

^b More data are available under Question 2C.

^c More data are available under Question 2D.

Q1C. What populations are at highest risk of mortality from urinary catheters?

To answer this question, we reviewed the quality of evidence for those risk factors examined in more than one study. The evidence for this question consists of 2 observational studies.^{7,74} The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 1C.

Low-quality evidence suggested that older age, higher severity of illness, and being on an internal medicine service compared to a surgical service were independent risk factors for mortality in patients with indwelling urinary catheters. Both studies evaluating these risk factors found the highest risk of mortality in patients over 70 years of age. Low-quality evidence also suggested that CAUTI was a risk factor for mortality in patients with catheters.

Evidence Review Table 1C. What populations are at highest risk of mortality from catheters?

1C.1. Minimize urinary catheter use and duration in all patients, particularly those who may be at higher risk for mortality due to catheterization, such as the elderly and patients with severe illness. **(Category IB)**

Q2. For those who may require urinary catheters, what are the best practices?

To answer this question, we focused on four subquestions: A) What are the risks and benefits associated with different approaches to catheterization?, B) What are the risks and benefits associated with different types of catheters or collecting systems?, C) What are the risks and benefits associated with different catheter management techniques, and D) What are the risks and benefits associated with different systems interventions?

Q2A. What are the risks and benefits associated with different approaches to catheterization?

The available data examined the following comparisons of different catheterization approaches:

- 1) External versus indwelling urethral
- 2) Intermittent versus indwelling urethral
- 3) Intermittent versus suprapubic
- 4) Suprapubic versus indwelling urethral
- 5) Clean intermittent versus sterile intermittent

For all comparisons, we considered SUTI, bacteriuria/unspecified UTI, or combinations of these outcomes depending on availability, as well as other outcomes critical to weighing the risks and benefits of different catheterization approaches. The evidence for this question consists of 6 systematic reviews,^{37,104-108} 16 RCTs,^{62,63,109-122} and 18 observational studies.^{54,73,81,84,123-136} The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 2A

Q2A.1. External versus indwelling urethral

Low-quality evidence suggested a benefit of using external catheters over indwelling urethral catheters in male patients who require a urinary collection device but do not have an indication for an indwelling catheter such as urinary retention or bladder outlet obstruction.^{81,109,123} This was based on a decreased risk of a composite outcome of SUTI, bacteriuria, or death as well as increased patient satisfaction with condom catheters. Differences were most pronounced in men without dementia. Statistically significant differences were not found or reported for the individual CAUTI outcomes or death. Our search did not reveal data on differences in local complications such as skin maceration or phimosis.

Q2A.2. Intermittent versus indwelling urethral

Low-quality evidence suggested a benefit of using intermittent catheterization over indwelling urethral catheters in selected populations.^{84,104-106,110-114,124-126,135,136} This was based on a decreased risk of SUTI and bacteriuria/unspecified UTI but an increased risk of urinary retention in postoperative patients with intermittent catheterization. In one study, urinary retention and bladder distension were avoided by performing catheterization at regular intervals (every 6-8 hrs) until return of voiding. Studies of patients with neurogenic bladder most consistently found a decreased risk of CAUTI with intermittent catheterization. Studies in operative patients whose catheters were removed within 24 hrs of surgery found no differences in bacteriuria with intermittent vs. indwelling catheterization, while studies where catheters were left in for longer durations had mixed results. Our search did not reveal data on differences in patient satisfaction.

Q2A.3. Intermittent versus suprapubic

Very low-quality evidence suggested a benefit of intermittent over suprapubic catheterization in selected populations^{115,116,134-136} based on increased patient acceptability and decreased risk of urinary complications (bladder calculi, vesicoureteral reflux, and upper tract abnormalities). Although we found a decreased risk of bacteriuria/unspecified UTI with suprapubic catheterization, there were no differences in SUTI. The populations studied included women undergoing urogynecologic surgery and spinal cord injury patients.

Q2A.4. Suprapubic versus indwelling urethral

Low-quality evidence suggested a benefit of suprapubic catheters over indwelling urethral catheters in selected populations.^{37,62,104,107,108,128-133,135,136} This was based on a decreased risk of bacteriuria/unspecified UTI, recatheterization, and urethral stricture, and increased patient comfort and satisfaction. However, there were no differences in SUTI and an increased risk of longer duration of catheterization with suprapubic catheters. Studies involved primarily postoperative and spinal cord injury patients. Our search did not reveal data on differences in complications related to catheter insertion or the catheter site.

Q2A.5. Clean intermittent versus sterile intermittent

Moderate-quality evidence suggested no benefit of using sterile over clean technique for intermittent catheterization.^{63,73,105,117-122} No differences were found in the risk of SUTI or bacteriuria/unspecified UTI. Study populations included nursing home residents and adults and children with neurogenic bladder/spinal cord injury.

Evidence Review Table 2A. What are the risks and benefits associated with different approaches to catheterization?

2A.1. Consider using external catheters as an alternative to indwelling urethral catheters in cooperative male patients without urinary retention or bladder outlet obstruction. **(Category II)**

2A.2. Intermittent catheterization is preferable to indwelling urethral or suprapubic catheters in patients with bladder emptying dysfunction. **(Category II)**

2A.3. If intermittent catheterization is used, perform it at regular intervals to prevent bladder overdistension. **(Category IB)**

2A.4. For operative patients who have an indication for an indwelling catheter, remove the catheter as soon as possible postoperatively, preferably within 24 hours, unless there are appropriate indications for continued use. **(Category IB)***

2A.5. Further research is needed on the risks and benefits of suprapubic catheters as an alternative to indwelling urethral catheters in selected patients requiring short- or long-term catheterization, particularly with respect to complications related to catheter insertion or the catheter site. **(No recommendation/unresolved issue)**

2A.6. In the non-acute care setting, clean (i.e., non-sterile) technique for intermittent catheterization is an acceptable and more practical alternative to sterile technique for patients requiring chronic intermittent catheterization. **(Category IA)**

* More data are available under Question 2C

Q2B. What are the risks and benefits associated with different catheters or collecting systems?

The available data examined the following comparisons between different types of catheters and drainage systems:

1. Antimicrobial/antiseptic catheters vs. standard catheters
 - a. Silver-coated catheters vs. standard catheters
 - b. Nitrofurazone-impregnated catheters vs. standard catheters
2. Hydrophilic catheters vs. standard catheters
3. Closed vs. open drainage systems
4. Complex vs. simple drainage systems
5. Preconnected/sealed junction catheters vs. standard catheters
6. Catheter valves vs. catheter bags

For all comparisons, we considered CAUTI outcomes as well as other outcomes critical to weighing the risks and benefits of different types of catheters or collecting systems. The evidence for this question consists of 5 systematic reviews,^{37,137-140} 17 RCTs,^{64,143-158} 23 observational studies,^{82,86,89,97,159-163, 165-178} and 3 economic analyses.^{179,180,181} The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 2B.

Q2B.1.a. Silver-coated catheters vs. standard catheters

Low-quality evidence suggested a benefit of silver-coated catheters over standard latex catheters.^{37,82,86,137-139,143,159-163, 165,166} This was based on a decreased risk of bacteriuria/unspecified UTI with silver-coated catheters and no evidence of increased urethral irritation or antimicrobial resistance in studies that reported data on microbiological outcomes. Differences were significant for silver alloy-coated catheters but not silver oxide-coated catheters. In a meta-analysis of randomized controlled trials (see [Appendix](#)), silver alloy-coated catheters reduced the risk of asymptomatic bacteriuria compared to standard latex catheters (control latex catheters were either uncoated or coated with hydrogel, Teflon®, or silicone), whereas there were no differences when compared to standard, all silicone catheters. The effect of silver alloy catheters compared to latex catheters was more pronounced when used in patients catheterized <1 week. The results were robust to inclusion or exclusion of non peer-reviewed studies. Only one observational study found a decrease in SUTI with silver alloy-coated catheters.¹⁶⁶ The setting was a burn referral center, where the control catheters were latex, and patients in the intervention group had new catheters placed on admission, whereas the control group did not. Recent observational studies in hospitalized patients found mixed results for bacteriuria/unspecified UTI.

Q2B.1.b. Nitrofurazone-impregnated catheters vs. standard catheters

Low-quality evidence suggested a benefit of nitrofurazone-impregnated catheters in patients catheterized for short periods of time.^{137,138} This was based on a decreased risk of bacteriuria and no evidence of increased antimicrobial resistance in studies that reported microbiological outcomes. Differences were significant in a meta-analysis of three studies examining nitrofurazone-impregnated catheters (only one individual study significant) when duration of catheterization was <1 week. No differences were seen when duration of catheterization was >1 week, although the meta-analysis was borderline significant.

Q2B.2. Hydrophilic catheters vs. standard catheters

Very low-quality evidence suggested a benefit of hydrophilic catheters over standard non-hydrophilic catheters in specific populations undergoing clean intermittent catheterization.^{137,144-148,169} This was based on a decreased risk of SUTI, bacteriuria, hematuria, and pain during insertion, and increased patient satisfaction. Differences in CAUTI outcomes were limited to one study of spinal cord injury patients and one study of patients receiving intravesical immunochemoprophylaxis for bladder cancer, while multiple other studies found no significant differences.

Q2B.3. Closed vs. open drainage systems

Very low-quality evidence suggested a benefit of using a closed rather than open urinary drainage system.^{89,171} This was based on a decreased risk of bacteriuria with a closed drainage system. One study also found a suggestion of a decreased risk of SUTI, bacteremia, and UTI-related mortality associated with closed drainage systems, but differences were not statistically significant. Sterile, continuously closed drainage systems became the standard of care based on an uncontrolled study published in 1966 demonstrating a dramatic reduction in the risk of infection in short-term catheterized patients with the use of a closed system.²³ Recent data also include the finding that disconnection of the drainage system is a risk factor for bacteriuria (Q1B).

Q2B.4. Complex vs. simple drainage systems

Low-quality evidence suggested no benefit of complex closed urinary drainage systems over simple closed urinary drainage systems.^{150-152,154,172,176,177} Although there was a decreased risk of bacteriuria with the complex systems, differences were found only in studies published before 1990, and not in more recent studies. The complex drainage systems studied included various mechanisms for reducing bacterial entry, such as antiseptic-releasing cartridges at the drain port of the urine collection bag; see evidence table for systems evaluated.

Q2B.5. Preconnected/sealed junction catheters vs. standard catheters

Low-quality evidence suggested a benefit of using preconnected catheters with junction seals over catheters with unsealed junctions to reduce the risk of disconnections.^{64,153,156,175} This was based on a decreased risk of SUTI and bacteriuria with preconnected sealed catheters. Studies that found differences had higher rates of CAUTI in the control group than studies that did not find an effect.

Q2B.6. Catheter valves vs. drainage bags

Moderate-quality evidence suggested a benefit of catheter valves over drainage bags in selected patients with indwelling urinary catheters.¹⁴⁰ Catheter valves led to greater patient satisfaction but no differences in bacteriuria/unspecified UTI or pain/bladder spasms. Details regarding the setting for recruitment and follow-up of the patients in the studies were unclear, and the majority of subjects were men. Our search did not reveal data on the effect of catheter valves on bladder function, bladder/urethral trauma, or catheter blockage.

Evidence Review Table 2B. What are the risks and benefits associated with different catheters or collecting systems?

2B.1. If the CAUTI rate is not decreasing after implementing a comprehensive strategy to reduce rates of CAUTI, consider using antimicrobial/antiseptic-impregnated catheters. The comprehensive strategy should include, at a minimum, the high priority recommendations for urinary catheter use, aseptic insertion, and maintenance (see Section III. Implementation and Audit). **(Category IB)**

2B.1.a. Further research is needed on the effect of antimicrobial/antiseptic-impregnated catheters in reducing the risk of symptomatic UTI, their inclusion among the primary interventions, and the patient populations most likely to benefit from these catheters. **(No recommendation/unresolved issue)**

2B.2. Hydrophilic catheters might be preferable to standard catheters for patients requiring intermittent catheterization. **(Category II)**

2B.3. Following aseptic insertion of the urinary catheter, maintain a closed drainage system. **(Category IB)**

2B.4. Complex urinary drainage systems (utilizing mechanisms for reducing bacterial entry such as antiseptic-release cartridges in the drain port) are not necessary for routine use. **(Category II)**

2B.5. Urinary catheter systems with preconnected, sealed catheter-tubing junctions are suggested for use. **(Category II)**

2B.6. Further research is needed to clarify the benefit of catheter valves in reducing the risk of CAUTI and other urinary complications. **(No recommendation/unresolved issue)**

Q2C. What are the risks and benefits associated with different catheter management techniques?

The available data examined the following catheter management techniques:

1. Antimicrobial prophylaxis
2. Urinary antiseptics (i.e., methanamine)
3. Bladder irrigation
4. Antiseptic instillation in the drainage bag
5. Periurethral care
6. Routine catheter or bag change
7. Catheter lubricants
8. Securing devices
9. Bacterial interference
10. Catheter cleansing
11. Catheter removal strategies (clamping vs. free drainage prior to removal, postoperative duration of catheterization)
12. Assessment of urine volumes

For all comparisons, we considered CAUTI outcomes as well as other outcomes critical to weighing the risks and benefits of different catheter management techniques. The evidence for this question consists of 6 systematic reviews,^{37,105,106,182-184} 56 RCTs,^{60,61,65-69,143,158,158,185-231} 34 observational studies,^{83,85,88,90,96,102,133,167,178,232-258} and 1 economic analysis.¹⁸⁰ The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 2C.

Q2C.1. Antimicrobial prophylaxis

Low-quality evidence suggested no benefit of antimicrobial prophylaxis in patients undergoing short-term catheterization.^{37,60,61,83,85,133,158,178,182,185,186,189-191,232-234} This was based on heterogeneous results for SUTI and bacteriuria/unspecified UTI and no adverse events related to antimicrobials. Lack of consistency in specific factors, such as patient population, antimicrobial agents, timing of administration, and duration of follow-up, did not allow for a summary of evidence of the effect of antimicrobial prophylaxis on CAUTI in patients undergoing short term catheterization. Only two studies evaluated adverse events related to antimicrobials. Our search did not reveal data on antimicrobial resistance or *Clostridium difficile* infection.

Low-quality evidence suggested no benefit of antimicrobial prophylaxis in patients undergoing long-term catheterization (indwelling and clean intermittent catheterization).^{106,183,192,194,235,238} This was based on a decreased risk of bacteriuria, heterogeneous results for SUTI, and no differences reported for catheter encrustation or adverse events, although data were sparse. One systematic review suggested an increase in antimicrobial resistance with antimicrobial use.

Q2C.2. Urinary antiseptics

Low-quality evidence suggested a benefit of methenamine for short-term catheterized patients.^{196,197} This was based on a reduced risk of SUTI and bacteriuria and no differences in adverse events. Evidence was limited to two studies of patients following gynecological surgery in Norway and Sweden.

Very low-quality evidence suggested a benefit of methanamine for long-term catheterized patients.^{106,236-239} This was based on a reduced risk of encrustation but no differences in risk of SUTI or bacteriuria. Data on encrustation was limited to one study. Studies involved primarily elderly and spinal cord injury patients with chronic indwelling catheters

Q2C.3. Bladder irrigation

Low-quality evidence suggested no benefit of bladder irrigation in patients with indwelling or intermittent catheters.^{66,69,199-206,240-242} This was based on no differences in SUTI and heterogeneous findings for bacteriuria.

Q2C.4. Antiseptic instillation in the drainage bag

Low-quality evidence suggested no benefit of antiseptic instillation in urinary drainage bags.^{90,207-211,243-245} This was based on no differences in SUTI and heterogeneous results for bacteriuria.

Q2C.5. Periurethral care

Low-quality evidence suggested no benefit of antiseptic meatal cleaning regimens before or during catheterization to prevent CAUTI.^{65,67,68,88,158,212-216,246,247} This was based on no difference in the risk of bacteriuria in patients receiving periurethral care regimens compared to those not receiving them. One study found a higher risk of bacteriuria with cleaning of the urethral meatus-catheter junction (either twice daily application of povidine-iodine or once daily cleaning with a non-antiseptic solution of green soap and water) in a subgroup of women with positive meatal cultures and in patients not receiving antimicrobials. Periurethral cleaning with chlorhexidine before catheter insertion did not have an effect in two studies.

Q2C.6. Routine catheter or bag change

Low-quality evidence suggested no benefit of routine catheter or drainage bag changes to prevent CAUTI.^{102,217-219,248,249} This was based on no difference or an increased risk of SUTI and no difference in bacteriuria with routine compared to as-needed changes or with more frequent changing intervals. One study in nursing home residents found no differences in SUTI with routine monthly catheter changes compared to changing only for obstruction or infection, but the study was underpowered to detect a difference. Another study in home care patients found an increased risk of SUTI when catheters were changed more frequently than monthly.

Q2C.7. Catheter lubricants

Very low-quality evidence suggested a benefit of using lubricants for catheter insertion.^{167,220-223,250-254} This was based on a decreased risk of SUTI and bacteriuria with the use of a pre-lubricated catheter compared to a catheter lubricated by the patient and a decreased risk of bacteriuria with use of a lubricant versus no lubricant. Studies were heterogeneous both in the interventions and outcomes studied. Several studies comparing antiseptic lubricants to non-antiseptic lubricants found no significant differences.

Q2C.8. Securing devices

Low-quality evidence suggested no benefit of using catheter securing devices to prevent CAUTI.²²⁴ This was based on no significant difference in the risk of SUTI or meatal erosion. The only study in this category looked at one particular product.

Q2C.9. Bacterial interference

Moderate-quality evidence suggested a benefit of using bacterial interference in catheterized patients.²²⁵ In the one study evaluating this intervention, urinary colonization with a non-pathogenic *Escherichia coli* was associated with a decreased risk of SUTI in adults with spinal cord injury and a history of frequent CAUTI.

Q2C.10. Catheter cleansing

Very low-quality evidence suggested a benefit of wet versus dry storage procedures for catheters used in clean intermittent catheterization.²⁵⁵ This was based on a decreased risk of SUTI with a wet storage procedure in one study of spinal cord injury patients undergoing clean intermittent catheterization compared to a dry storage procedure where the catheter was left to air dry after washing. In the wet procedure, the catheter was stored in a dilute povidone-iodine solution after washing with soap and water.

Q2C.11. Catheter removal strategies

a. Clamping vs. free drainage prior to removal

Low-quality evidence suggested no benefit of clamping versus free drainage before catheter removal.^{37,184} This was based on no difference in risk of bacteriuria, urinary retention, or recatheterization between the two strategies. One study comparing a clamp and release strategy to free drainage over 72 hours found a greater risk of bacteriuria in the clamping group.

b. Postoperative duration of catheterization

Moderate-quality evidence suggested a benefit of shorter versus longer postoperative durations of catheterization.^{37,184,227,228} This was based on a decreased risk of bacteriuria/unspecified UTI, decreased time to ambulation and length of stay, no differences in urinary retention and SUTI, and increased risk of recatheterization. Significant decreases in bacteriuria/unspecified UTI were found specifically for comparisons of 1 day versus 3 or 5 days of postoperative catheterization. Recatheterization risk was greater in only one study comparing immediate removal to removal 6 or 12 hours after hysterectomy.

Q2C.12. Assessment of urine volumes

Low-quality evidence suggested a benefit of using portable ultrasound to assess urine volume in patients undergoing intermittent catheterization.^{229,230} This was based on fewer catheterizations but no reported differences in risk of unspecified UTI. Patients studied were adults with neurogenic bladder in inpatient rehabilitation centers. Our search did not reveal data on the use of ultrasound in catheterized patients in other settings.

Evidence Review Table 2C. What are the risks and benefits associated with different catheter management techniques?

2C.1. Unless clinical indications exist (e.g., in patients with bacteriuria upon catheter removal post urologic surgery), do not use systemic antimicrobials routinely as prophylaxis for UTI in patients requiring either short or long-term catheterization. **(Category IB)**

2C.2.a. Further research is needed on the use of urinary antiseptics (e.g., methanamine) to prevent UTI in patients requiring short-term catheterization. **(No recommendation/unresolved issue)**

2C.2.b. Further research is needed on the use of methanamine to prevent encrustation in patients requiring chronic indwelling catheters who are at high risk for obstruction. **(No recommendation/unresolved issue)**

2C.3.a. Unless obstruction is anticipated (e.g., as might occur with bleeding after prostatic or bladder surgery), bladder irrigation is not recommended. **(Category II)**

2C.3.b. Routine irrigation of the bladder with antimicrobials is not recommended. **(Category II)**

2C.4. Routine instillation of antiseptic or antimicrobial solutions into urinary drainage bags is not recommended. **(Category II)**

2C.5.a. Do not clean the periurethral area with antiseptics to prevent CAUTI while the catheter is in place. Routine hygiene (e.g., cleansing of the meatal surface during daily bathing) is

appropriate. **(Category IB)**

2C.5.b. Further research is needed on the use of antiseptic solutions vs. sterile water or saline for periurethral cleaning prior to catheter insertion. **(No recommendation/unresolved issue)**

2C.6. Changing indwelling catheters or drainage bags at routine, fixed intervals is not recommended. Rather, catheters and drainage bags should be changed based on clinical indications such as infection, obstruction, or when the closed system is compromised. **(Category II)**

2C.7.a. Use a sterile, single-use packet of lubricant jelly for catheter insertion. **(Category IB)**

2C.7.b. Routine use of antiseptic lubricants is not necessary. **(Category II)**

2C.8. Further research is needed on the use of bacterial interference to prevent UTI in patients requiring chronic urinary catheterization. **(No recommendation/unresolved issue)**

2C.9. Further research is needed on optimal cleaning and storage methods for catheters used for clean intermittent catheterization. **(No recommendation/unresolved issue)**

2C.10.a. Clamping indwelling catheters prior to removal is not necessary. **(Category II)**

2C.10.b. Insert catheters only for appropriate indications, and leave in place only as long as needed. **(Category IB)**

2C.10.c. For operative patients who have an indication for an indwelling catheter, remove the catheter as soon as possible postoperatively, preferably within 24 hours, unless there are appropriate indications for continued use. **(Category IB)**

2C.11.a. Consider using a portable ultrasound device to assess urine volume in patients undergoing intermittent catheterization to assess urine volume and reduce unnecessary catheter insertions. **(Category II)**

2C.11.b. Further research is needed on the use of a portable ultrasound device to evaluate for obstruction in patients with indwelling catheters and low urine output. **(No recommendation/unresolved issue)**

Q2D. What are the risks and benefits associated with different systems interventions?

The available data examined the following systems interventions:

1. Infection control/quality improvement programs (multifaceted)
2. Catheter reminders
3. Bacteriologic monitoring
4. Hand hygiene
5. Patient placement
6. Catheter team versus self-catheterization
7. Feedback
8. Nurse-directed catheter removal

We considered CAUTI outcomes, duration of catheterization, recatheterization, and transmission of pathogens when weighing the risks and benefits of different systems interventions. The evidence for this question consists of 1 RCT²⁵⁹ and 19 observational

studies.^{3,25,260-276} The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 2D.

Q2D.1. Multifaceted infection control/quality improvement programs

Low-quality evidence suggested a benefit of multifaceted infection control/quality improvement programs to reduce the risk of CAUTI.^{3,260-267} This was based on a decreased risk of SUTI, bacteriuria/unspecified UTI, and duration of catheter use with implementation of such programs. Studies evaluated various multifaceted interventions. The studies with significant findings included: 1) education and performance feedback regarding compliance with catheter care, emphasizing hand hygiene, and maintaining unobstructed urine flow; 2) computerized alerts to physicians, nurse-driven protocols to remove catheters, and use of handheld bladder scanners to assess for urinary retention; 3) guidelines and education focusing on perioperative catheter management; and 4) a multifaceted infection control program including guidelines for catheter insertion and maintenance. A program using a checklist and algorithm for appropriate catheter use also suggested a decrease in unspecified UTI and catheter duration, but statistical differences were not reported.

Q2D.2. Reminders

Very low-quality evidence suggested a benefit of using urinary catheter reminders to prevent CAUTI.²⁶⁸⁻²⁷⁰ This was based on a decreased risk of bacteriuria and duration of catheterization and no differences in recatheterization or SUTI when reminders were used. Reminders to physicians included both computerized and non-computerized alerts about the presence of urinary catheters and the need to remove unnecessary catheters.

Q2D.3. Bacteriologic monitoring

Very low-quality evidence suggested no benefit of bacteriologic monitoring to prevent CAUTI.^{25,271} Although one study found a decreased risk of bacteriuria during a period of bacteriologic monitoring and feedback, only 2% of SUTI episodes were considered potentially preventable with the use of bacteriologic monitoring.

Q2D.4. Hand hygiene

Very low-quality evidence suggested a benefit of using alcohol hand sanitizer in reducing CAUTI. This was based on one study in a rehabilitation facility that found a decrease in unspecified UTI, although no statistical differences were reported.²⁷² A separate multifaceted study that included education and performance feedback on compliance with catheter care and hand hygiene showed a decrease in risk of SUTI.²⁶⁵

Q2D.5. Patient placement

Very low-quality evidence suggested a benefit of spatially separating patients to prevent transmission of urinary pathogens.²⁷³ This was based on a decreased risk of transmission of urinary bacterial pathogens in nursing home residents in separate rooms compared to residents in the same rooms.

Q2D.6. Catheter team versus self-catheterization

Very low-quality evidence suggested no benefit of a catheter team to prevent CAUTI among patients requiring intermittent catheterization.²⁷⁴ This was based on one study showing no difference in unspecified UTI between use of a catheter care team and self-catheterization for intermittent catheterization in paraplegic patients.

Q2D.7. Feedback

Very low-quality evidence suggested a benefit of using nursing feedback to prevent CAUTI.²⁷⁵ This was based on a decreased risk of unspecified UTI during an intervention where nursing staff were provided with regular reports of unit-specific rates of CAUTI.

Q2D.8. Nurse-directed catheter removal

Very low-quality evidence suggested a benefit of a nurse-directed catheter removal program to prevent CAUTI.²⁷⁶ This was based on a decreased risk of unspecified UTI during an intervention where criteria were developed that allowed a registered nurse to remove a catheter without a physician's order when no longer medically necessary. Of the three intensive care units where the intervention was implemented, differences were significant only in the coronary intensive care unit.

Evidence Review Table 2D. What are the risks and benefits associated with different systems interventions?

2D.1.a. Ensure that healthcare personnel and others who take care of catheters are given periodic in-service training stressing the correct techniques and procedures for urinary catheter insertion, maintenance, and removal. **(Category IB)**

2D.1.b. Implement quality improvement (QI) programs or strategies to enhance appropriate use of indwelling catheters and to reduce the risk of CAUTI based on a facility risk assessment. **(Category IB)**

Examples of programs that have been demonstrated to be effective include:

1. A system of alerts or reminders to identify all patients with urinary catheters and assess the need for continued catheterization
2. Guidelines and protocols for nurse-directed removal of unnecessary urinary catheters
3. Education and performance feedback regarding appropriate use, hand hygiene, and catheter care
4. Guidelines and algorithms for appropriate peri-operative catheter management, such as:
 - a. Procedure-specific guidelines for catheter placement and postoperative catheter removal
 - b. Protocols for management of postoperative urinary retention, such as nurse-directed use of intermittent catheterization and use of ultrasound bladder scanners

2D.2. Routine screening of catheterized patients for asymptomatic bacteriuria is not recommended. **(Category II)**

2D.3. Perform hand hygiene immediately before and after insertion or any manipulation of the catheter site or device. **(Category IB)**

2D.5. Maintain unobstructed urine flow. **(Category IB)**

2D.6. Further research is needed on the benefit of spatial separation of patients with urinary catheters to prevent transmission of pathogens colonizing urinary drainage systems. **(No recommendation/unresolved issue)**

2D.7. When performing surveillance for CAUTI, consider providing regular (e.g., quarterly) feedback of unit-specific CAUTI rates to nursing staff and other appropriate clinical care staff. **(Category II)**

Q3: What are the best practices for preventing UTI associated with obstructed urinary catheters?

The available data examined the following practices:

1. Methods to prevent/reduce encrustations or blockage
2. Catheter materials preventing blockage

For this question, available relevant outcomes included blockage/encrustation. We did not find data on the outcomes of CAUTI. The evidence for this question consists of 1 systematic review,²⁷⁷ 2 RCTs,^{278,279} and 2 observational studies.^{280,281} The findings of the evidence review and the grades for all important outcomes are shown in Evidence Review Table 3.

Q3.1. Methods to prevent/reduce encrustations or blockage

Low-quality evidence suggested a benefit of acidifying solutions or oral acetohydroxamic acid in preventing or reducing catheter encrustations and blockage in long-term catheterized patients.^{277,278,280,281} No differences were seen with daily catheter irrigation with normal saline.

Q3.2. Catheter materials preventing blockage

Low-quality evidence suggested a benefit of silicone over latex or Teflon-coated catheters in prevention or reducing catheter encrustations in long-term catheterized patients who were prone to blockage. No differences were seen with different materials in patients considered “non-blockers.”²⁷⁹

Evidence Review Table 3. What are the best practices for preventing UTI associated with obstructed urinary catheters?

3.1.a. Further research is needed on the benefit of irrigating the catheter with acidifying solutions or use of oral urease inhibitors in long-term catheterized patients who have frequent catheter obstruction. **(No recommendation/unresolved issue)**

3.2.a. Silicone might be preferable to other materials to reduce the risk of encrustation in long-term catheterized patients who have frequent obstruction. **(Category II)**

References

1. Wong ES. Guideline for prevention of catheter-associated urinary tract infections. *Am J Infect Control*. 1983;11(1):28-36.
2. Lo E, Nicolle L, Classen D, et al. Strategies to prevent catheter-associated urinary tract infections in acute care hospitals. *Infect Control Hosp Epidemiol*. 2008;29:S41-S50.
3. Stephan F, Sax H, Wachsmuth M, Hoffmeyer P, Clergue F, Pittet D. Reduction of urinary tract infection and antibiotic use after surgery: A controlled, prospective, before-after intervention study. *Clin Infect Dis*. 2006;42(11):1544-1551.
4. Dolin SJ, Cashman JN. Tolerability of acute postoperative pain management: Nausea, vomiting, sedation, pruritis, and urinary retention. evidence from published data. *Br J Anaesth*. 2005;95(5):584-591.
5. Hidron AI, Edwards JR, Patel J, et al. NHSN annual update: Antimicrobial-resistant pathogens associated with healthcare-associated infections: Annual summary of data reported to the national healthcare safety network at the centers for disease control and prevention, 2006-2007. *Infect Control Hosp Epidemiol*. 2008;29(11):996-1011.
6. Saint S. Clinical and economic consequences of nosocomial catheter-related bacteriuria. *Am J Infect Control*. 2000;28(1):68-75.
7. Platt R, Polk BF, Murdock B, Rosner B. Mortality associated with nosocomial urinary-tract infection. *N Engl J Med*. 1982;307(11):637-642.
8. Givens CD, Wenzel RP. Catheter-associated urinary tract infections in surgical patients: A controlled study on the excess morbidity and costs. *J Urol*. 1980;124(5):646-648.
9. Tambyah PA, Knasinski V, Maki DG. The direct costs of nosocomial catheter-associated urinary tract infection in the era of managed care. *Infect Control Hosp Epidemiol*. 2002;23(1):27-31.
10. Schaberg DR, Weinstein RA, Stamm WE. Epidemics of nosocomial urinary tract infection caused by multiply resistant gram-negative bacilli: Epidemiology and control. *J Infect Dis*. 1976;133(3):363-366.
11. Yoon HJ, Choi JY, Park YS, et al. Outbreaks of *Serratia marcescens* bacteriuria in a neurosurgical intensive care unit of a tertiary care teaching hospital: A clinical, epidemiologic, and laboratory perspective. *Am J Infect Control*. 2005;33(10):595-601.
12. Warren JW. Catheter-associated urinary tract infections. *Int J Antimicrob Agents*. 2001;17(4):299-303.
13. Weinstein JW, Mazon D, Pantelick E, Reagan-Cirincione P, Dembry LM, Hierholzer WJ, Jr. A decade of prevalence surveys in a tertiary-care center: Trends in nosocomial infection rates, device utilization, and patient acuity. *Infect Control Hosp Epidemiol*. 1999;20(8):543-548.
14. Munasinghe RL, Yazdani H, Siddique M, Hafeez W. Appropriateness of use of indwelling urinary catheters in patients admitted to the medical service. *Infect Control Hosp Epidemiol*. 2001;22(10):647-649.

15. Jain P, Parada JP, David A, Smith LG. Overuse of the indwelling urinary tract catheter in hospitalized medical patients. *Arch Intern Med.* 1995;155(13):1425-1429.
16. Saint S, Wiese J, Amory JK, et al. Are physicians aware of which of their patients have indwelling urinary catheters? *Am J Med.* 2000;109(6):476-480.
17. Edwards JR, Peterson KD, Andrus ML, et al. National healthcare safety network (NHSN) report, data summary for 2006, issued june 2007. *Am J Infect Control.* 2007;35(5):290-301.
18. Rogers MA, Mody L, Kaufman SR, Fries BE, McMahon LF, Jr, Saint S. Use of urinary collection devices in skilled nursing facilities in five states. *J Am Geriatr Soc.* 2008;56(5):854-861.
19. Klevens RM, Edwards JR, Richards CL, Jr, et al. Estimating health care-associated infections and deaths in U.S. hospitals, 2002. *Public Health Rep.* 2007;122(2):160-166.
20. Weinstein MP, Towns ML, Quartey SM, et al. The clinical significance of positive blood cultures in the 1990s: A prospective comprehensive evaluation of the microbiology, epidemiology, and outcome of bacteremia and fungemia in adults. *Clin Infect Dis.* 1997;24(4):584-602.
21. Muder RR, Brennen C, Wagener MM, Goetz AM. Bacteremia in a long-term-care facility: A five-year prospective study of 163 consecutive episodes. *Clin Infect Dis.* 1992;14(3):647-654.
22. Umscheid C, Mitchell M, Agarwal R, Williams K, Brennan P. Mortality from reasonably-preventable hospital acquired infections. included in written testimony by the society of healthcare epidemiology of america for the committee on oversight and government reform hearing on healthcare-associated infections: A preventable epidemic, chaired by henry A. waxman, april 16, 2008, washington, DC. [congressional testimony]. .
23. Kunin CM, McCormack RC. Prevention of catheter-induced urinary-tract infections by sterile closed drainage. *N Engl J Med.* 1966;274(21):1155-1161.
24. Tambyah PA, Halvorson KT, Maki DG. A prospective study of pathogenesis of catheter-associated urinary tract infections. *Mayo Clin Proc.* 1999;74(2):131-136.
25. Garibaldi RA, Mooney BR, Epstein BJ, Britt MR. An evaluation of daily bacteriologic monitoring to identify preventable episodes of catheter-associated urinary tract infection. *Infect Control.* 1982;3(6):466-470.
26. Saint S, Lipsky BA, Goold SD. Indwelling urinary catheters: A one-point restraint? *Ann Intern Med.* 2002;137(2):125-127.
27. Warren JW, Tenney JH, Hoopes JM, Muncie HL, Anthony WC. A prospective microbiologic study of bacteriuria in patients with chronic indwelling urethral catheters. *J Infect Dis.* 1982;146(6):719-723.
28. Saint S, Chenoweth CE. Biofilms and catheter-associated urinary tract infections. *Infect Dis Clin North Am.* 2003;17(2):411-432.

29. Kallen AJ, Hidron AI, Edwards JR, Patel J, Srinivasan A. Antibiotic resistance among important gram-negative pathogens causing healthcare-associated infections reported to the national healthcare safety network, 2006-2007 [abstract 376] in: Program and abstracts of the 18th annual scientific meeting of the society for healthcare epidemiology of america, orlando, FL, apr 5-8, 2008. .
30. Horan TC, Gaynes RP. Surveillance of nosocomial infections. In: Mayhall CG, ed. *Hospital Epidemiology and Infection Control*. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2004:1659-1702.
31. Tambyah PA, Maki DG. Catheter-associated urinary tract infection is rarely symptomatic: A prospective study of 1,497 catheterized patients. *Arch Intern Med*. 2000;160(5):678-682.
32. Atkins D, Best D, Briss PA, et al. Grading quality of evidence and strength of recommendations. *BMJ*. 2004;328(7454):1490.
33. Guyatt GH, Oxman AD, Kunz R, et al. Going from evidence to recommendations. *BMJ*. 2008;336(7652):1049-1051.
34. Guyatt GH, Oxman AD, Kunz R, et al. What is "quality of evidence" and why is it important to clinicians? *BMJ*. 2008;336(7651):995-998.
35. Pratt RJ, Pellowe CM, Wilson JA, et al. epic2: National evidence-based guidelines for preventing healthcare-associated infections in NHS hospitals in england. *J Hosp Infect*. 2007;65(Suppl 1):S1-64.
36. Pellowe CM, Pratt RJ, Harper P, et al. Evidence-based guidelines for preventing healthcare-associated infections in primary and community care in england. *J Hosp Infect*. 2003;55(Suppl 2):S2-127.
37. Phipps S, Lim YN, McClinton S, Barry C, Rane A, N'Dow J. Short term urinary catheter policies following urogenital surgery in adults. *Cochrane Database Syst Rev*. 2006(2):CD004374.
38. Tang KK, Wong CK, Lo SF, Ng TK. Is it necessary to catheterise the bladder routinely before gynaecological laparoscopic surgery? *Aust N Z J Obstet Gynaecol*. 2005;45(5):380-383.
39. Iorio R, Healy WL, Patch DA, Appleby D. The role of bladder catheterization in total knee arthroplasty. *Clin Orthop*. 2000(380):80-84.
40. Liu SK, Rassai H, Krasner C, Braun J, Matolo NM. Urinary catheter in laparoscopic cholecystectomy: Is it necessary? *Surg Laparosc Endosc Percutan Tech*. 1999;9(3):184-186.
41. Normelli H, Aaro S, Hedlund R, Svensson O, Stromberg L. Urethral catheterization in spinal surgery: A randomized prospective study. *Eur Spine J*. 1993;2(3):132-135.
42. Carpinello VL, Cendron M, Altman HG, Malloy TR, Booth R. Treatment of urinary complications after total joint replacement in elderly females. *Urology*. 1988;32(3):186-188.
43. Akhtar MS, Beere DM, Wright JT, MacRae KD. Is bladder catheterization really necessary before laparoscopy? *Br J Obstet Gynaecol*. 1985;92(11):1176-1178.

44. Chaudhuri P, Vengadasalam D. Urinary tract infection following abdominal delivery--second thought on indwelling catheter. *Singapore Med J.* 1983;24(4):224-226.
45. McMurdo ME, Davey PG, Elder MA, Miller RM, Old DC, Malek M. A cost-effectiveness study of the management of intractable urinary incontinence by urinary catheterisation or incontinence pads. *J Epidemiol Community Health.* 1992;46(3):222-226.
46. Geraniotis E, Koff SA, Enrile B. The prophylactic use of clean intermittent catheterization in the treatment of infants and young children with myelomeningocele and neurogenic bladder dysfunction. *J Urol.* 1988;139(1):85-86.
47. Kumar P, Mannan K, Chowdhury AM, Kong KC, Pati J. Urinary retention and the role of indwelling catheterization following total knee arthroplasty. *Int Braz J Urol.* 2006;32(1):31-34.
48. Miskry T, Cumbis A, Apostolidis P, Eleftheriou M, Magos A. Do not catheterize the bladder at operative laparoscopy. *Gynaecol Endosc.* 2001;10(1):29-32.
49. Barnes JS. Is it better to avoid urethral catheterization at hysterectomy and caesarean section? *Aust N Z J Obstet Gynaecol.* 1998;38(3):315-316.
50. Ouslander JG, Greengold B, Chen S. External catheter use and urinary tract infections among incontinent male nursing home patients. *J Am Geriatr Soc.* 1987;35(12):1063-1070.
51. Rannikko S, Kyllastinen M, Granqvist B. Comparison of long-term indwelling catheters and bed-pads in the treatment of urinary incontinence in elderly patients. *J Infect.* 1986;12(3):221-227.
52. Nordqvist P, Ekelund P, Edouard L, Svensson ML, Brandberg A, Seeberg S. Catheter-free geriatric care. routines and consequences for clinical infection, care and economy. *J Hosp Infect.* 1984;5(3):298-304.
53. Egilmez T, Aridogan IA, Yachia D, Hassin D. Comparison of nitinol urethral stent infections with indwelling catheter-associated urinary-tract infections. *J Endourol.* 2006;20(4):272-277.
54. Ruz ED, Garcia Leoni E, Herruzo Cabrera R. Epidemiology and risk factors for urinary tract infection in patients with spinal cord injury. *J Urol.* 2000;164(4):1285-1289.
55. Donovan WH, Stolov WC, Clowers DE, et al. Bacteriuria during intermittent catheterization following spinal cord injury. *Arch Phys Med Rehabil.* 1978;59(8):351-357.
56. Larsen LD, Chamberlin DA, Khonsari F, Ahlering TE. Retrospective analysis of urologic complications in male patients with spinal cord injury managed with and without indwelling urinary catheters. *Urology.* 1997;50(3):418-422.
57. Kochakarn W, Ratana-Olarn K, Lertsithichai P, Roongreungsilp U. Follow-up of long-term treatment with clean intermittent catheterization for neurogenic bladder in children. *Asian J.* 2004;27(2):134-136.

58. Ehrlich O, Brem AS. A prospective comparison of urinary tract infections in patients treated with either clean intermittent catheterization or urinary diversion. *Pediatrics*. 1982;70(5):665-669.
59. Darouiche RO, Smith JA, Jr., Hanna H, et al. Efficacy of antimicrobial-impregnated bladder catheters in reducing catheter-associated bacteriuria: A prospective, randomized, multicenter clinical trial. *Urology*. 1999;54(6):976-981.
60. Stricker PD, Grant AB. Relative value of antibiotics and catheter care in the prevention of urinary tract infection after transurethral prostatic resection. *Br J Urol*. 1988;61(6):494-497.
61. Rogers RG, Kammerer-Doak D, Olsen A, et al. A randomized, double-blind, placebo-controlled comparison of the effect of nitrofurantoin monohydrate macrocrystals on the development of urinary tract infections after surgery for pelvic organ prolapse and/or stress urinary incontinence with suprapubic catheterization. *Obstet Gynecol*. 2004;191(1):182-187.
62. Baan AH, Vermeulen H, van der Meulen, J., Bossuyt P, Olszyna D, Gouma DJ. The effect of suprapubic catheterization versus transurethral catheterization after abdominal surgery on urinary tract infection: A randomized controlled trial. *Dig Surg*. 2003;20(4):290-295.
63. Carapeti EA, Andrews SM, Bentley PG. Randomised study of sterile versus non-sterile urethral catheterisation.[erratum appears in ann R coll surg engl 1996 may;78(3 pt 1):167]. *Ann R Coll Surg Engl*. 1996;78(1):59-60.
64. Huth TS, Burke JP, Larsen RA, Classen DC, Stevens LE. Clinical trial of junction seals for the prevention of urinary catheter-associated bacteriuria. *Arch Intern Med*. 1992;152(4):807-812.
65. Huth TS, Burke JP, Larsen RA, Classen DC, Stevens LE. Randomized trial of meatal care with silver sulfadiazine cream for the prevention of catheter-associated bacteriuria. *J Infect Dis*. 1992;165(1):14-18.
66. Schneeberger PM, Vreede RW, Bogdanowicz JF, van Dijk, W. C. A randomized study on the effect of bladder irrigation with povidone-iodine before removal of an indwelling catheter. *J Hosp Infect*. 1992;21(3):223-229.
67. Burke JP, Jacobson JA, Garibaldi RA, Conti MT, Alling DW. Evaluation of daily meatal care with poly-antibiotic ointment in prevention of urinary catheter-associated bacteriuria. *J Urol*. 1983;129(2):331-334.
68. Classen DC, Larsen RA, Burke JP, Alling DW, Stevens LE. Daily meatal care for prevention of catheter-associated bacteriuria: Results using frequent applications of polyantibiotic cream. *Infect Control Hosp Epidemiol*. 1991;12(3):157-162.
69. Warren JW, Platt R, Thomas RJ, Rosner B, Kass EH. Antibiotic irrigation and catheter-associated urinary-tract infections. *N Engl J Med*. 1978;299(11):570-573.
70. Seki N, Masuda K, Kinukawa N, Senoh K, Naito S. Risk factors for febrile urinary tract infection in children with myelodysplasia treated by clean intermittent catheterization. *Int J Urol*. 2004;11(11):973-977.

71. Kelleher RE, Meeropol E, Parks D. Factors influencing the development of symptomatic bacteriuria in children on clean intermittent catheterization. *J Urol Nurs.* 1996;15(2):1256-1265.
72. Waites KB, Canupp KC, DeVivo MJ. Epidemiology and risk factors for urinary tract infection following spinal cord injury. *Arch Phys Med Rehabil.* 1993;74(7):691-695.
73. Anderson RU. Non-sterile intermittent catheterization with antibiotic prophylaxis in the acute spinal cord injured male patient. *J Urol.* 1980;124(3):392-394.
74. Van Der Kooi, T. I. I., De Boer, A. S., Mannien J, et al. Incidence and risk factors of device-associated infections and associated mortality at the intensive care in the dutch surveillance system. *Intensive Care Med.* 2007;33(2):271-278.
75. Bochicchio GV, Joshi M, Shih D, Bochicchio K, Tracy K, Scalea TM. Reclassification of urinary tract infections in critically ill trauma patients: A time-dependent analysis. *Surg Infect.* 2003;4(4):379-385.
76. Leone M, Albanese J, Garnier F, et al. Risk factors of nosocomial catheter-associated urinary tract infection in a polyvalent intensive care unit. *Intensive Care Med.* 2003;29(7):1077-1080.
77. Tissot E, Limat S, Cornette C, Capellier G. Risk factors for catheter-associated bacteriuria in a medical intensive care unit. *Eur J Clin Microbiol Infect Dis.* 2001;20(4):260-262.
78. Colau A, Lucet JC, Rufat P, Botto H, Benoit G, Jardin A. Incidence and risk factors of bacteriuria after transurethral resection of the prostate. *Eur Urol.* 2001;39(3):272-276.
79. Ouslander JG, Greengold B, Chen S. Complications of chronic indwelling urinary catheters among male nursing home patients: A prospective study. *J Urol.* 1987;138(5):1191-1195.
80. Hazelett SE, Tsai M, Gareri M, Allen K. The association between indwelling urinary catheter use in the elderly and urinary tract infection in acute care. *BMC geriatr.* 2006;6:15.
81. Saint S, Kaufman SR, Rogers MAM, Bakers PD, Boyko EJ, Lipsky BA. Risk factors for nosocomial urinary tract-related bacteremia: A case-control study. *Am J Infect Control.* 2006;34(7):401-407.
82. Srinivasan A, Karchmer T, Richards A, Song X, Perl TM. A prospective trial of a novel, silicone-based, silver-coated foley catheter for the prevention of nosocomial urinary tract infections. *Infect Control Hosp Epidemiol.* 2006;27(1):38-43.
83. Cardosi RJ, Cardosi RP, Grendys EC, Jr., Fiorica JV, Hoffman MS. Infectious urinary tract morbidity with prolonged bladder catheterization after radical hysterectomy... includes discussion. *Obstet Gynecol.* 2003;189(2):380-384.
84. Johansson I, Athlin E, Frykholm L, Bolinder H, Larsson G. Intermittent versus indwelling catheters for older patients with hip fractures. *J Clin Nurs.* 2002;11(5):651-656.

85. Hustinx WN, Mintjes-de Groot, Verkooyen RP, Verbrugh HA. Impact of concurrent antimicrobial therapy on catheter-associated urinary tract infection.[see comment]. *J Hosp Infect.* 1991;18(1):45-56.
86. Johnson JR, Roberts PL, Olsen RJ, Moyer KA, Stamm WE. Prevention of catheter-associated urinary tract infection with a silver oxide-coated urinary catheter: Clinical and microbiologic correlates. *J Infect Dis.* 1990;162(5):1145-1150.
87. Lima NL, Guerrant RL, Kaiser DL, Germanson T, Farr BM. A retrospective cohort study of nosocomial diarrhea as a risk factor for nosocomial infection. *J Infect Dis.* 1990;161(5):948-952.
88. Jacono JJ, Talamelli CL. Nosocomial urinary tract infection in catheterized patients. *Infect Control Can.* 1988;3(1):16-20.
89. Lanara V, Plati C, Paniara O, et al. The prevalence of urinary tract infection in patients related to type of drainage bag. *Scand J Caring Sci.* 1988;2(4):163-170.
90. Holliman R, Seal DV, Archer H, Doman S. Controlled trial of chemical disinfection of urinary drainage bags. reduction in hospital-acquired catheter-associated infection. *Br J Urol.* 1987;60(5):419-422.
91. Mulhall AB, Chapman RG, Crow RA. Bacteriuria during indwelling urethral catheterization. *J Hosp Infect.* 1988;11(3):253-262.
92. Pien FD, Landers JQ, Jr. Indwelling urinary catheter infections in small community hospital. role of urinary drainage bag. *Urology.* 1983;22(3):255-258.
93. Shapiro M, Simchen E, Izraeli S, Sacks TG. A multivariate analysis of risk factors for acquiring bacteriuria in patients with indwelling urinary catheters for longer than 24 hours. *Infect Control.* 1984;5(11):525-532.
94. Platt R, Polk BF, Murdock B, Rosner B. Risk factors for nosocomial urinary tract infection. *Am J Epidemiol.* 1986;124(6):977-985.
95. Burke JP, Larsen RA, Stevens LE. Nosocomial bacteriuria: Estimating the potential for prevention by closed sterile urinary drainage. *Infect Control.* 1986;7(2):96-99.
96. Saramma PP. Catheter-associated urinary tract infection: A follow-up study in prevention. *Nurs J India.* 1987;78(10):257-259.
97. Islam AK, Chapman J. Closed catheter drainage and urinary infection--a comparison of two methods of catheter drainage. *Br J Urol.* 1977;49(3):215-220.
98. Hartstein AI, Garber SB, Ward TT, Jones SR, Morthland VH. Nosocomial urinary tract infection: A prospective evaluation of 108 catheterized patients. *Infect Control.* 1981;2(5):380-386.
99. Garibaldi RA, Burke JP, Britt MR, Miller MA, Smith CB. Meatal colonization and catheter-associated bacteriuria. *N Engl J Med.* 1980;303(6):316-318.
100. Garibaldi RA, Burke JP, Dickman ML, Smith CB. Factors predisposing to bacteriuria during indwelling urethral catheterization. *N Engl J Med.* 1974;291(5):215-219.

101. Hirsh DD, Fainstein V, Musher DM. Do condom catheter collecting systems cause urinary tract infection? *JAMA*. 1979;242(4):340-341.
102. White MC, Ragland KE. Urinary catheter-related infections among home care patients. *J Wound Ostomy Continence Nurs*. 1995;22(6):286-290.
103. Wilde MH, Carrigan MJ. A chart audit of factors related to urine flow and urinary tract infection. *J Adv Nurs*. 2003;43(3):254-262.
104. Niel-Weise BS, van den Broek, P. J. Urinary catheter policies for short-term bladder drainage in adults. *Cochrane Libr*. 2006;4.
105. Shekelle PG, Morton SC, Clark KA, Pathak M, Vickrey BG. Systematic review of risk factors for urinary tract infection in adults with spinal cord dysfunction. *J Spinal Cord Med*. 1999;22(4):258-272.
106. Vickrey BG, Shekelle P, Morton S, Clark K, Pathak M, Kamberg C. Prevention and management of urinary tract infections in paralyzed persons. *Evid Rep Technol Assess*. 1999(6):1-3.
107. Branagan GW, Moran BJ. Published evidence favors the use of suprapubic catheters in pelvic colorectal surgery. *Dis Colon Rectum*. 2002;45(8):1104-1108.
108. McPhail MJ, Abu-Hilal M, Johnson CD. A meta-analysis comparing suprapubic and transurethral catheterization for bladder drainage after abdominal surgery. *Br J Surg*. 2006;93(9):1038-1044.
109. Saint S, Kaufman SR, Rogers MA, Baker PD, Ossenkop K, Lipsky BA. Condom versus indwelling urinary catheters: A randomized trial. *J Am Geriatr Soc*. 2006;54(7):1055-1061.
110. Michelson JD, Lotke PA, Steinberg ME. Urinary-bladder management after total joint-replacement surgery. *N Engl J Med*. 1988;319(6):321-326.
111. Tang MW, Kwok TC, Hui E, Woo J. Intermittent versus indwelling urinary catheterization in older female patients. *Maturitas*. 2006;53(3):274-281.
112. Turi MH, Hanif S, Fasih Q, Shaikh MA. Proportion of complications in patients practicing clean intermittent self-catheterization (CISC) vs indwelling catheter. *J Pak Med Assoc*. 2006;56(9):401-404.
113. Tangtrakul S, Taechaiya S, Suthutvoravut S, Linasmita V. Post-cesarean section urinary tract infection: A comparison between intermittent and indwelling catheterization. *J Med Assoc Thai*. 1994;77(5):244-248.
114. Skelly JM, Guyatt GH, Kalbfleisch R, Singer J, Winter L. Management of urinary retention after surgical repair of hip fracture. *Can Med Assoc J*. 1992;146(7):1185-1189.
115. Jannelli ML, Wu JM, Plunkett LW, Williams KS, Visco AG. A randomized controlled trial of clean intermittent self-catheterization versus suprapubic catheterization after urogynecologic surgery. *Am J Obstet Gynecol*. 2007;197(1).
116. Roberts K, Naik R. Catheterization options following radical surgery for cervical cancer. *Br J Nurs*. 2006;15(19):1038-1044.

117. Duffy LM, Cleary J, Ahern S, et al. Clean intermittent catheterization: Safe, cost-effective bladder management for male residents of VA nursing homes. *J Am Geriatr Soc.* 1995;43(8):865-870.
118. Moore KN, Burt J, Voaklander DC. Intermittent catheterization in the rehabilitation setting: A comparison of clean and sterile technique. *Clin Rehabil.* 2006;20(6):461-468.
119. Moore KN, Kelm M, Sinclair O, Cadrain G. Bacteriuria in intermittent catheterization users: The effect of sterile versus clean reused catheters. *Rehabil Nurs.* 1993;18(5):306-309.
120. Prieto-Fingerhut T, Banovac K, Lynne CM. A study comparing sterile and nonsterile urethral catheterization in patients with spinal cord injury. *Rehabil Nurs.* 1997;22(6):299-302.
121. Schlager TA, Clark M, Anderson S. Effect of a single-use sterile catheter for each void on the frequency of bacteriuria in children with neurogenic bladder on intermittent catheterization for bladder emptying. *Pediatrics.* 2001;108(4):E71.
122. Joseph C, Jacobson C, Strausbaugh L, Maxwell M, French M, Colling J. Sterile vs clean urinary catheterization. *J Am Geriatr Soc.* 1991;39(10):1042-1043.
123. Saint S, Lipsky BA, Baker PD, McDonald LL, Ossenkop K. Urinary catheters: What type do men and their nurses prefer? *J Am Geriatr Soc.* 1999;47(12):1453-1457.
124. Ritter MA, Faris PM, Keating EM. Urinary tract catheterization protocols following total joint arthroplasty. *Orthopedics.* 1989;12(8):1085-1087.
125. Oishi CS, Williams VJ, Hanson PB, Schneider JE, Colwell CW, Jr., Walker RH. Perioperative bladder management after primary total hip arthroplasty. *J Arthroplasty.* 1995;10(6):732-736.
126. Furuhashi A, Ogawa K, Saito K, Yamaguchi T. Preoperative intermittent catheterization in patients with prostatic hypertrophy. *Clin Ther.* 1988;10 Spec No:47-51.
127. Alli MO, Singh B, Moodley J, Shaik AS. Prospective evaluation of combined suprapubic and urethral catheterization to urethral drainage alone for intraperitoneal bladder injuries. *Journal of Trauma-Injury Infection & Critical Care.* 2003;55(6):1152-1154.
128. Dunn TS, Figge J, Wolf D. A comparison of outcomes of transurethral versus suprapubic catheterization after burch cystourethropexy. *Int Urogynecol J Pelvic Floor Dysfunct.* 2005;16(1):60-2; discussion 62.
129. Dinneen MD, Wetter LA, May AR. Urethral strictures and aortic surgery. suprapubic rather than urethral catheters. *Eur J Vasc Surg.* 1990;4(5):535-538.
130. Horgan AF, Prasad B, Waldron DJ, O'Sullivan DC. Acute urinary retention. comparison of suprapubic and urethral catheterisation. *Br J Urol.* 1992;70(2):149-151.
131. Hofmeister FJ, Martens WE, Strelbel RL. Foley catheter or suprapubic tube? *Obstet Gynecol.* 1970;107(5):767-779.

132. Van Nagell, JR, Penny RM, Roddick JW. Suprapubic bladder drainage following radical hysterectomy. *Obstet Gynecol.* 1972;113(6):849-850.
133. Verbrugh HA, Mintjes-de Groot, Andriess R, Hamersma K, van Dijk A. Postoperative prophylaxis with norfloxacin in patients requiring bladder catheters. *Eur J Clin Microbiol Infect Dis.* 1988;7(4):490-494.
134. Noll F, Russe O, Kling E, Botel U, Schreiter F. Intermittent catheterisation versus percutaneous suprapubic cystostomy in the early management of traumatic spinal cord lesions. *Paraplegia.* 1988;26(1):4-9.
135. Weld KJ, Dmochowski RR, Foster HE, Jr. Effect of bladder management on urological complications in spinal cord injured patients. *J Urol.* 2000;163(3):768-772.
136. Lloyd LK, Kuhlemeier KV, Fine PR, Stover SL. Initial bladder management in spinal cord injury: Does it make a difference? *J Urol.* 1986;135(3):523-527.
137. Schumm K, Lam TB. Types of urethral catheters for management of short-term voiding problems in hospitalised adults.[update of cochrane database syst rev. 2004;(1):CD004013; PMID: 14974052]. *Cochrane Database Syst Rev.* 2008(2):004013.
138. Johnson JR, Kuskowski MA, Wilt TJ. Systematic review: Antimicrobial urinary catheters to prevent catheter-associated urinary tract infection in hospitalized patients. *Ann Intern Med.* 2006;144(2):116-126.
139. Niel-Weise BS, Arend SM, van den Broek, PJ. Is there evidence for recommending silver-coated urinary catheters in guidelines? *J Hosp Infect.* 2002;52(2):81-87.
140. van den Eijkel, E., Griffiths P. Catheter valves for indwelling urinary catheters: A systematic review. *Br J Community Nurs.* 2006;11(3):111-2, 114.
141. Crnich CJ, Drinka PJ. Does the composition of urinary catheters influence clinical outcomes and the results of research studies? *Infect Control Hosp Epidemiol.* 2007;28(1):102-103.
142. Reiche T, Lisby G, Jorgensen S, Christensen AB, Nordling J. A prospective, controlled, randomized study of the effect of a slow-release silver device on the frequency of urinary tract infection in newly catheterized patients. *BJU Int.* 2000;85(1):54-59.
143. Schaeffer AJ, Story KO, Johnson SM. Effect of silver oxide/trichloroisocyanuric acid antimicrobial urinary drainage system on catheter-associated bacteriuria. *J Urol.* 1988;139(1):69-73.
144. De Ridder, D. J., Everaert K, Fernandez LG, et al. Intermittent catheterisation with hydrophilic-coated catheters (SpeediCath) reduces the risk of clinical urinary tract infection in spinal cord injured patients: A prospective randomised parallel comparative trial. *Eur Urol.* 2005;48(6):991-995.
145. Cindolo L, Palmieri EA, Autorino R, Salzano L, Altieri V. Standard versus hydrophilic catheterization in the adjuvant treatment of patients with superficial bladder cancer. *Urol Int.* 2004;73(1):19-22.
146. Stensballe J, Looms D, Nielsen PN, Tvede M. Hydrophilic-coated catheters for intermittent catheterisation reduce urethral micro trauma: A prospective, randomised, participant-blinded, crossover study of three different types of catheters. *Eur Urol.* 2005;48(6):978-983.

147. Vapnek JM, Maynard FM, Kim J, Vapnek JM, Maynard FM, Kim J. A prospective randomized trial of the LoFric hydrophilic coated catheter versus conventional plastic catheter for clean intermittent catheterization. *J Urol.* 2003;169(3):994-998.
148. Monson T, Kunin CM. Evaluation of a polymer-coated indwelling catheter in prevention of infection. *J Urol.* 1974;111(2):220-222.
149. Shafik A. The electrified catheter. role in sterilizing urine and decreasing bacteriuria. *World J Urol.* 1993;11(3):183-185.
150. Leone M, Garnier F, Antonini F, Bimar MC, Albanese J, Martin C. Comparison of effectiveness of two urinary drainage systems in intensive care unit: A prospective, randomized clinical trial. *Intensive Care Med.* 2003;29(4):551-554.
151. Classen DC, Larsen RA, Burke JP, Stevens LE. Prevention of catheter-associated bacteriuria: Clinical trial of methods to block three known pathways of infection. *Am J Infect Control.* 1991;19(3):136-142.
152. al-Juburi AZ, Cicmanec J. New apparatus to reduce urinary drainage associated with urinary tract infections. *Urology.* 1989;33(2):97-101.
153. DeGroot-Kosolcharoen J, Guse R, Jones JM. Evaluation of a urinary catheter with a preconnected closed drainage bag. *Infect Control Hosp Epidemiol.* 1988;9(2):72-76.
154. Klarskov P, Bischoff N, Bremmelgaard A, Hald T. Catheter-associated bacteriuria. A controlled trial with bardex(TM) urinary drainage system. *Acta Obstet Gynecol Scand.* 1986;65(4):295-299.
155. Keys TF, Maker MD, Segura JW. Bacteriuria during closed urinary drainage: An evaluation of top-vented versus bag-vented systems. *J Urol.* 1979;122(1):49-51.
156. Platt R, Polk BF, Murdock B, Rosner B. Reduction of mortality associated with nosocomial urinary tract infection. *Lancet.* 1983;1(8330):893-897.
157. Monson TP, Macalalad FV, Hamman JW, Kunin CM. Evaluation of a vented drainage system in prevention of bacteriuria. *J Urol.* 1977;117(2):216-219.
158. Cleland V, Cox F, Berggren H, MacInnis MR. Prevention of bacteriuria in female patients with welling catheters. *Nurs Res.* 1971;20(4):309-318.
159. Seymour C. Audit of catheter-associated UTI using silver alloy-coated foley catheters. *Br J Nurs.* 2006;15(11):598-603.
160. Gentry H, Cope S. Using silver to reduce catheter-associated urinary tract infections. *Nurs Stand.* 2005;19(50):51-54.
161. Madeo M, Davies D, Johnson G, Owen E, Wadsworth P, Martin CR. The impact of using silver alloy urinary catheters in reducing the incidence of urinary tract infections in the critical care setting. *Br J Infect Control.* 2004;5(1):21-24.
162. Rupp ME, Fitzgerald T, Marion N, et al. Effect of silver-coated urinary catheters: Efficacy, cost-effectiveness, and antimicrobial resistance. *Am J Infect Control.* 2004;32(8):445-450.

163. Lai KK, Fontecchio SA, Lai KK, Fontecchio SA. Use of silver-hydrogel urinary catheters on the incidence of catheter-associated urinary tract infections in hospitalized patients. *Am J Infect Control*. 2002;30(4):221-225.
164. Akiyama H, Okamoto S. Prophylaxis of indwelling urethral catheter infection: Clinical experience with a modified foley catheter and drainage system. *J Urol*. 1979;121(1):40-42.
165. Bologna RA, Tu LM, Polansky M, Fraimow HD, Gordon DA, Whitmore KE. Hydrogel/silver ion-coated urinary catheter reduces nosocomial urinary tract infection rates in intensive care unit patients: A multicenter study. *Urology*. 1999;54(6):982-987.
166. Newton T, Still JM, Law E. A comparison of the effect of early insertion of standard latex and silver-impregnated latex foley catheters on urinary tract infections in burn patients. *Infect Control Hosp Epidemiol*. 2002;23(4):217-218.
167. Butler HK, Kunin CM. Evaluation of polymyxin catheter lubricant and impregnated catheters. *J Urol*. 1968;100(4):560-566.
168. Mooro H, Kamel M. Prevention of catheter fever by the use of furacin urethral inserts. *J Egypt Med Assoc*. 1966;49(8):550-553.
169. Pachler J, Frimodt-Moller C. A comparison of prelubricated hydrophilic and non-hydrophilic polyvinyl chloride catheters for urethral catheterization. *BJU Int*. 1999;83(7):767-769.
170. Chen TY, Ponsot Y, Carmel M, Bouffard N, Kennelly MJ, Tu LM. Multi-centre study of intraurethral valve-pump catheter in women with a hypocontractile or acontractile bladder. *Eur Urol*. 2005;48(4):628-633.
171. Danchaivijitr S, Chokeloidkaew S, Prutsachativuthi S, Trakulsomboon S. Should the open urinary drainage system be continued? *J Med Assoc Thai*. 1988;71 Suppl 3:14-18.
172. Wille JC, Blusse van Oud Alblas, A., Thewessen EA. Nosocomial catheter-associated bacteriuria: A clinical trial comparing two closed urinary drainage systems. *J Hosp Infect*. 1993;25(3):191-198.
173. Hardyck C, Petrinovich L. Reducing urinary tract infections in catheterized patients. *Ostomy Wound*. 1998;44(12):36-8, 40, 42-3.
174. Montagnino BA, Gonzales ET, Jr., Roth DR. Open catheter drainage after urethral surgery. *J Urol*. 1988;140(5):1250-1252.
175. Madeo M, Barr B, Owen E. A study to determine whether the use of a pre-connect urinary catheter system reduces the incidence of nosocomial urinary tract infections. *Br J Infect Control*. 2005;6(4):22-25.
176. Leone M, Garnier F, Dubuc M, Bimar MC, Martin C. Prevention of nosocomial urinary tract infection in ICU patients: Comparison of effectiveness of two urinary drainage systems. *Chest*. 2001;120(1):220-224.
177. Blenkarn JJ. Prevention of bacteriuria during urinary catheterization of patients in an intensive care unit: Evaluation of the 'ureofix 500' closed drainage system. *J Hosp Infect*. 1985;6(2):187-193.

178. Drach GW, Lacy SS, Cox CE, 2nd. Prevention of catheter-induced post-prostatectomy infection. effects of systemic cephaloridine and local irrigation with neomycin-polymyxin through closed-drainage catheter system. *J Urol*. 1971;105(6):840-842.
179. Plowman R, Graves N, Esquivel J, Roberts JA. An economic model to assess the cost and benefits of the routine use of silver alloy coated urinary catheters to reduce the risk of urinary tract infections in catheterized patients. *J Hosp Infect*. 2001;48(1):33-42.
180. Platt R, Polk BF, Murdock B, Rosner B. Prevention of catheter-associated urinary tract infection: A cost-benefit analysis. *Infect Control Hosp Epidemiol*. 1989;10(2):60-64.
181. Saint S, Veenstra DL, Sullivan SD, Chenoweth C, Fendrick AM. The potential clinical and economic benefits of silver alloy urinary catheters in preventing urinary tract infection. *Arch Intern Med*. 2000;160(17):2670-2675.
182. Niel-Weise BS, van den Broek, P. J. Antibiotic policies for short-term catheter bladder drainage in adults. *Cochrane Libr*. 2006;4.
183. Niel-Weise BS, van den Broek, P. J. Urinary catheter policies for long-term bladder drainage. *Cochrane Libr*. 2006;4.
184. Griffiths R, Fernandez R. Strategies for the removal of short-term indwelling urethral catheters in adults. *Cochrane Libr*. 2007(2).
185. Esposito S, Noviello S, Leone S, Marvaso A, Drago L, Marchetti F, The LSG. A pilot study on prevention of catheter-related urinary tract infections with fluoroquinolones. *J Chemother*. 2006;18(5):494-501.
186. Wazait HD, Patel HR, Van Der Meulen, J. H. P., et al. A pilot randomized double-blind placebo-controlled trial on the use of antibiotics on urinary catheter removal to reduce the rate of urinary tract infection: The pitfalls of ciprofloxacin. *BJU Int*. 2004;94(7):1048-1050.
187. Lukkarinen O, Hellstrom P, Leppilahti M, Kontituri M, Tammela T. Antibiotic prophylaxis in patients with urinary retention undergoing transurethral prostatectomy. *Ann Chir Gynaecol*. 1997;86(3):239-242.
188. Lukkarinen O, Hellstrom P, Leppilahti M, Kontturi M, Tammela T. Prospective randomized open study between ciprofloxacin and a combination of sulfadiazine and trimethoprim in antibiotic prophylaxis in connection with transurethral prostatectomy. *Scand J Urol Nephrol*. 1996;30(1):33-36.
189. Vollaard EJ, Clasener HA, Zambon JV, Joosten HJ, van Griethuysen, AJ. Prevention of catheter-associated gram-negative bacilluria with norfloxacin by selective decontamination of the bowel and high urinary concentration. *J Antimicrob Chemother*. 1989;23(6):915-922.
190. Grabe M, Forsgren A, Hellsten S. A short antibiotic course given in conjunction with and after catheter removal consecutive to transurethral prostatic resection. *Scand J Urol Nephrol*. 1984;18(3):193-199.
191. Little PJ, Pearson S, Peddie BA, Greenslade NF, Utley WL. Amoxicillin in the prevention of catheter-induced urinary infection. *J Infect Dis*. 1974;129(0):suppl:S241-2.
192. Clarke SA, Samuel M, Boddy SA. Are prophylactic antibiotics necessary with clean intermittent catheterization? A randomized controlled trial. *J Pediatr Surg*. 2005;40(3):568-571.

193. Waites KB, Canupp KC, Armstrong S, DeVivo MJ. Effect of cranberry extract on bacteriuria and pyuria in persons with neurogenic bladder secondary to spinal cord injury. *J Spinal Cord Med.* 2004;27(1):35-40.
194. Firestein M, Mendelson G, Gronich D, Granot E, Ben-Israel J, Raz R. Can antibiotic use during routine replacement of long-term urinary catheter prevent bacteriuria? *Infectious Diseases in Clinical Practice.* 2001;10(3):133-135.
195. Schlager TA, Anderson S, Trudell J, Hendley JO. Effect of cranberry juice on bacteriuria in children with neurogenic bladder receiving intermittent catheterization. *J Pediatr.* 1999;135(6):698-702.
196. Schiotz HA, Guttu K. Value of urinary prophylaxis with methenamine in gynecologic surgery. *Acta Obstet Gynecol Scand.* 2002;81(8):743-746.
197. Tyreman NO, Andersson PO, Kroon L, Orstam S. Urinary tract infection after vaginal surgery. effect of prophylactic treatment with methenamine hippurate. *Acta Obstet Gynecol Scand.* 1986;65(7):731-733.
198. Pearman JW, Bailey M, Harper WE, Pearman JW, Bailey M, Harper WE. Comparison of the efficacy of "trisdine" and kanamycin-colistin bladder instillations in reducing bacteriuria during intermittent catheterisation of patients with acute spinal cord trauma. *Br J Urol.* 1988;62(2):140-144.
199. Pearman JW, Bailey M, Riley LP. Bladder instillations of trisdine compared with catheter introducer for reduction of bacteriuria during intermittent catheterisation of patients with acute spinal cord trauma. *Br J Urol.* 1991;67(5):483-490.
200. Adesanya AA, Osegbe DN, Amaku EO. The use of intermittent chlorhexidine bladder irrigation in the prevention of post-prostatectomy infective complications. *Int Urol Nephrol.* 1993;25(4):359-367.
201. Ball AJ, Carr TW, Gillespie WA, Kelly M, Simpson RA, Smith PJ. Bladder irrigation with chlorhexidine for the prevention of urinary infection after transurethral operations: A prospective controlled study.[see comment]. *J Urol.* 1987;138(3):491-494.
202. van den Broek, P. J., Daha TJ, Mouton RP. Bladder irrigation with povidone-iodine in prevention of urinary-tract infections associated with intermittent urethral catheterisation. *Lancet.* 1985;1(8428):563-565.
203. Savage JE, Phillips B, Lifshitz S, et al. Bacteriuria in closed bladder drainage versus continuous irrigation in patients undergoing intracavitary radium for treatment of gynecologic cancer. *Gynecol Oncol.* 1982;13(1):26-30.
204. Kirk D, Dunn M, Bullock DW, Mitchell JP, Hobbs SJ. Hibitane bladder irrigation in the prevention of catheter-associated urinary infection. *Br J Urol.* 1979;51(6):528-531.
205. Chamberlain G, Holt E. Automatic bladder irrigation after vaginal surgery. *Br J Urol.* 1975;47(6):641-643.
206. Clark LW. Neomycin in the prevention of postcatheterization bacteriuria. *Med J Aust.* 1973;1(21):1034-1036.
207. Washington EA. Instillation of 3% hydrogen peroxide or distilled vinegar in urethral catheter drainage bag to decrease catheter-associated bacteriuria. *Biol Res Nurs.* 2001;3(2):78-87.

208. Sweet DE, Goodpasture HC, Holl K, Smart S, Alexander H, Hedari A. Evaluation of H₂O₂ prophylaxis of bacteriuria in patients with long-term indwelling foley catheters: A randomized controlled study. *Infect Control*. 1985;6(7):263-266.
209. Thompson RL, Haley CE, Searcy MA, et al. Catheter-associated bacteriuria. failure to reduce attack rates using periodic instillations of a disinfectant into urinary drainage systems. *JAMA*. 1984;251(6):747-751.
210. Gillespie WA, Simpson RA, Jones JE, Nashef L, Teasdale C, Speller DC. Does the addition of disinfectant to urine drainage bags prevent infection in catheterised patients? *Lancet*. 1983;1(8332):1037-1039.
211. Maizels M, Schaeffer AJ. Decreased incidence of bacteriuria associated with periodic instillations of hydrogen peroxide into the urethral catheter drainage bag. *J Urol*. 1980;123(6):841-845.
212. Webster J, Hood RH, Burrige CA, Doidge ML, Phillips CM, George N. Water or antiseptic for periurethral cleaning before urinary catheterization: A randomized controlled trial. *Am J Infect Control*. 2001;29(6):389-394.
213. Bardwell TA. Comparing meatal cleansing. *Aust Nurs J*. 1999;7(2):30-31.
214. Sanderson PJ, Weissler S. A comparison of the effect of chlorhexidine antiseptics, soap and antibiotics on bacteriuria, perineal colonization and environmental contamination in spinally injured patients. *J Hosp Infect*. 1990;15(3):235-243.
215. Burke JP, Garibaldi RA, Britt MR, Jacobson JA, Conti M, Alling DW. Prevention of catheter-associated urinary tract infections. efficacy of daily meatal care regimens. *Am J Med*. 1981;70(3):655-658.
216. Ross PW, Smylie HG. A comparison of two regimes of catheterisation in the prophylaxis of urinary tract infection in gynaecological patients. *Br J Urol*. 1966;38(2):194-198.
217. Keerasuntonpong A, Thearawiboon W, Panthawan A, et al. Incidence of urinary tract infections in patients with short-term indwelling urethral catheters: A comparison between a 3-day urinary drainage bag change and no change regimens. *Am J Infect Control*. 2003;31(1):9-12.
218. Dille CA, Kirchhoff KT, Sullivan JJ, Larson E. Increasing the wearing time of vinyl urinary drainage bags by decontamination with bleach. *Arch Phys Med Rehabil*. 1993;74(4):431-437.
219. Priefer BA, Duthie EH, Jr., Gambert SR. Frequency of urinary catheter change and clinical urinary tract infection. study in hospital-based, skilled nursing home. *Urology*. 1982;20(2):141-142.
220. Fera P, Glashan RQ, Bruschini H, Lelis MAS, Baretta MCC, Srougi M. Lubricated urethral catheters with lidocaine versus gentamycin for clean intermittent catheterization. *International Braz J Urol*. 2002;28(1):50-56.
221. Giannantoni A, Di Stasi SM., Scivoletto G, Virgili G, Dolci S, Porena M. Intermittent catheterization with a prelubricated catheter in spinal cord injured patients: A prospective randomized crossover study. *J Urol*. 2001;166(1):130-133.

222. Cohen A. A microbiological comparison of a povidone-iodine lubricating gel and a control as catheter lubricants. *J Hosp Infect.* 1985;6 Suppl A:155-161.
223. Kunin CM, Finkelberg Z. Evaluation of an intraurethral lubricating catheter in prevention of catheter-induced urinary tract infections. *J Urol.* 1971;106(6):928-930.
224. Darouiche RO, Goetz L, Kaldis T, Cerra-Stewart C, AlSharif A, Priebe M. Impact of StatLock securing device on symptomatic catheter-related urinary tract infection: A prospective, randomized, multicenter clinical trial. *Am J Infect Control.* 2006;34(9):555-560.
225. Darouiche RO, Thornby JI, Cerra-Stewart C, Donovan WH, Hull RA. Bacterial interference for prevention of urinary tract infection: A prospective, randomized, placebo-controlled, double-blind pilot trial. *Clinical infectious diseases : an official publication of the Infectious Diseases Society of America.* 2005;41(10):1531-1534.
226. Moore KN. Two methods for cleaning catheters used for intermittent catheterization: Sunlight(TM) liquid detergent and cetrimide 1:30 (savlon(TM)). *Canadian Journal of Rehabilitation.* 1990;4(2):87-92.
227. Alessandri F, Mistrangelo E, Lijoi D, Ferrero S, Ragni N. A prospective, randomized trial comparing immediate versus delayed catheter removal following hysterectomy. *Acta Obstet Gynecol Scand.* 2006;85(6):716-720.
228. Schiotz HA. Comparison of 1 and 3 days' transurethral foley catheterization after vaginal plastic surgery. *International Urogynecology Journal.* 1995;6(3):158-161.
229. Polliack T, Bluvshstein V, Philo O, et al. Clinical and economic consequences of volume- or time-dependent intermittent catheterization in patients with spinal cord lesions and neuropathic bladder (structured abstract). *Spinal Cord.* 2005;43(10):615-619.
230. Anton HA, Chambers K, Clifton J, Tasaka J. Clinical utility of a portable ultrasound device in intermittent catheterization. *Archives of Physical Medicine & Rehabilitation.* 1998;79(2):172-175.
231. Moyad RH, Persky L. Vesical irrigation and urosepsis. *Invest Urol.* 1968;6(1):21-25.
232. Shohet I, Alagam M, Shafir R, Tsur H, Cohen B. Postoperative catheterization and prophylactic antimicrobials in children with hypospadias. *Urology.* 1983;22(4):391-393.
233. Anonymous. Evaluation of aseptic techniques and chlorhexidine on the rate of catheter-associated urinary-tract infection. southampton infection control team. *Lancet.* 1982;1(8263):89-91.
234. Cafferkey MT, Conneely B, Falkiner FR, Gillespie WA, Murphy D. Post-operative urinary infection and septicaemia in urology. *J Hosp Infect.* 1980;1(4):315-320.
235. Salomon J, Denys P, Merle C, et al. Prevention of urinary tract infection in spinal cord-injured patients: Safety and efficacy of a weekly oral cyclic antibiotic (WOCA) programme with a 2 year follow-up - an observational prospective study. *J Antimicrob Chemother.* 2006;57(4):784-788.

236. Wibell L, Scheynius A, Norrman K. Methenamine-hippurate and bacteriuria in the geriatric patient with a catheter. *Acta Med Scand.* 1980;207(6):469-473.
237. Kostiala AA, Nyren P, Jokinen EJ, Runeberg L. Prospective study on the appearance of antibody-coated bacteria in patients with an indwelling urinary catheter. *Nephron.* 1982;30(3):279-285.
238. Nyren P, Runeberg L, Kostiala AI, Renkonen OV, Roine R. Prophylactic methenamine hippurate or nitrofurantoin in patients with an indwelling urinary catheter. *Ann Clin Res.* 1981;13(1):16-21.
239. Norrman K, Wibell L. Treatment with methenamine hippurate in the patient with a catheter. *J Int Med Res.* 1976;4(2):115-117.
240. Pearman JW. The value of kanamycin-colistin bladder instillations in reducing bacteriuria during intermittent catheterisation of patients with acute spinal cord injury. *Br J Urol.* 1979;51(5):367-374.
241. Cox F, Smith RF, Elliott JP, Quinn EL. Neomycin-polymyxin prophylaxis of urinary-tract infection associated with indwelling catheters. *Antimicrob Agents Chemother.* 1966;6:165-168.
242. Thornton GF, Lytton B, Andriole VT. Bacteriuria during indwelling catheter drainage. effect of constant bladder rinse. *JAMA.* 1966;195(3):179-183.
243. Wongsatanapong W. Prevention of urinary tract infection by instillation of chlorhexidine into urinary drainage bags. *J Med Assoc Thai.* 1988;71 Suppl 3:19-21.
244. Sujka SK, Petrelli NJ, Herrera L. Incidence of urinary tract infections in patients requiring long-term catheterization after abdominoperineal resection for rectal carcinoma: Does betadine(TM) in the foley drainage bag make a difference? *European Journal of Surgical Oncology.* 1987;13(4):341-343.
245. Samuels P, Fisher P. The use of hydrogen peroxide in catheter drainage units. *AUAA J.* 1983;3(3):5-9.
246. Koskeroglu N, Durmaz G, Bahar M, Kural M, Yelken B. The role of meatal disinfection in preventing catheter-related bacteriuria in an intensive care unit: A pilot study in turkey. *J Hosp Infect.* 2004;56(3):236-238.
247. Matsumoto T, Sakumoto M, Takahashi K, Kumazawa J. Prevention of catheter-associated urinary tract infection by meatal disinfection. *Dermatology.* 1997;195 Suppl 2:73-77.
248. Stelling JD, Hale AM. Protocol for changing condom catheters in males with spinal cord injury. *SCI Nurs.* 1996;13(2):28-34.
249. Reid RI, Webster O, Pead PJ, Maskell R. Comparison of urine bag-changing regimens in elderly catheterised patients. *Lancet.* 1982;2(8301):754-756.
250. Kambal C, Chance J, Cope S, Beck J. Catheter-associated UTIs in patients after major gynaecological surgery. *Prof Nurse.* 2004;19(9):515-518.
251. Schiotz HA. Antiseptic catheter gel and urinary tract infection after short-term postoperative catheterization in women. *Arch Gynecol Obstet.* 1996;258(2):97-100.
252. Harrison LH. Comparison of a microbicidal povidone-iodine gel and a placebo gel as catheter lubricants. *J Urol.* 1980;124(3):347-349.

253. Chavigny KH. The use of polymixin B as a urethral lubricant to reduce the post-instrumental incidence of bacteriuria in females. an exploratory study. *Int J Nurs Stud.* 1975;12(1):33-42.
254. Nooyen RL. Prophylaxis of catheter-induced urinary tract infection in obstetric patients. *J Am Osteopath Assoc.* 1966;66(3):324-326.
255. Sims L, Ballard N. A comparison of two methods of catheter cleansing and storage used with clean intermittent catheterization. *Rehabil Nurs Res.* 1993;2(2):87-92.
256. Wald H, Epstein A, Kramer A. Extended use of indwelling urinary catheters in postoperative hip fracture patients. *Med Care.* 2005;43(10):1009-1017.
257. Wyatt TD, Timoney R. The effect of introducing a policy for catheter care on the catheter infection rate in a small hospital. *J Hosp Infect.* 1987;9(3):230-234.
258. Hoy WE, Kissel SM, Freeman RB, Sterling WA, Jr. Altered patterns of posttransplant urinary tract infections associated with perioperative antibiotics and curtailed catheterization. *Am J Kidney Dis.* 1985;6(4):212-216.
259. Pickard WG, Grundy DJ. A comparison of two methods of sterile urethral catheterisation in spinal cord injured adults. *Paraplegia.* 1996;34(1):30-33.
260. Jain M, Miller L, Belt D, King D, Berwick DM. Decline in ICU adverse events, nosocomial infections and cost through a quality improvement initiative focusing on teamwork and culture change. *Qual Saf Health Care.* 2006;15(4):235-239.
261. Reilly L, Sullivan P, Ninni S, Fochesto D, Williams K, Fetherman B. Reducing foley catheter device days in an intensive care unit: Using the evidence to change practice. *AACN Adv Crit Care.* 2006;17(3):272-283.
262. Verdier R, Parer S, Jean-Pierre H, Dujols P, Picot MC. Impact of an infection control program in an intensive care unit in france. *Infect Control Hosp Epidemiol.* 2006;27(1):60-66.
263. Topal J, Conklin S, Camp K, Morris V, Balcezak T, Herbert P. Prevention of nosocomial catheter-associated urinary tract infections through computerized feedback to physicians and a nurse-directed protocol. *Am J Med Qual.* 2005;20(3):121-126.
264. Misset B, Timsit JF, Dumay MF, et al. A continuous quality-improvement program reduces nosocomial infection rates in the ICU. *Intensive Care Med.* 2004;30(3):395-400.
265. Rosenthal VD, Guzman S, Safdar N. Effect of education and performance feedback on rates of catheter-associated urinary tract infection in intensive care units in argentina. *Infect Control Hosp Epidemiol.* 2004;25(1):47-50.
266. Berg DE, Hershow RC, Ramirez CA, Weinstein RA. Control of nosocomial infections in an intensive care unit in guatemala city. *Clin Infect Dis.* 1995;21(3):588-593.
267. Cools HJ, van der Meer JW. Infection control in a skilled nursing facility: A 6-year survey. *J Hosp Infect.* 1988;12(2):117-124.

268. Saint S, Kaufman SR, Thompson M, Rogers MA, Chenoweth CE. A reminder reduces urinary catheterization in hospitalized patients. *Jt Comm J Qual Patient Saf.* 2005;31(8):455-462.
269. Huang WC, Wann SR, Lin SL, et al. Catheter-associated urinary tract infections in intensive care units can be reduced by prompting physicians to remove unnecessary catheters. *Infect Control Hosp Epidemiol.* 2004;25(11):974-978.
270. Cornia PB, Amory JK, Fraser S, Saint S, Lipsky BA. Computer-based order entry decreases duration of indwelling urinary catheterization in hospitalized patients. *Am J Med.* 2003;114(5):404-407.
271. Jacobson JA, Burke JP, Kasworm E. Effect of bacteriologic monitoring of urinary catheters on recognition and treatment of hospital-acquired urinary tract infections. *Infect Control.* 1981;2(3):227-232.
272. Fendler EJ, Ali Y, Hammond BS, Lyons MK, Kelley MB, Vowell NA. The impact of alcohol and sanitizer use on infection rates in an extended care facility. *Am J Infect Control.* 2002;30(4):226-233.
273. Fryklund B, Haeggman S, Burman LG. Transmission of urinary bacterial strains between patients with indwelling catheters - nursing in the same room and in separate rooms compared. *J Hosp Infect.* 1997;36(2):147-153.
274. Wyndaele JJ, De Taeye N. Early intermittent self-catheterisation after spinal cord injury. *Paraplegia.* 1990;28(2):76-80.
275. Goetz AM, Kedzuf S, Wagener M, Muder RR. Feedback to nursing staff as an intervention to reduce catheter-associated urinary tract infections. *Am J Infect Control.* 1999;27(5):402-404.
276. Dumigan DG, Kohan CA, Reed CR, Jekel JF, Fikrig MK. Utilizing national nosocomial infection surveillance system data to improve urinary tract infection rates in three intensive-care units. *Clin Perform Qual Health Care.* 1998;6(4):172-178.
277. Mayes J, Bliss J, Griffiths P. Preventing blockage of long-term indwelling catheters in adults: Are citric acid solutions effective? *Br J Community Nurs.* 2003;8(4):172-175.
278. Muncie HL, Jr., Hoopes JM, Damron DJ, Tenney JH, Warren JW. Once-daily irrigation of long-term urethral catheters with normal saline. lack of benefit. *Arch Intern Med.* 1989;149(2):441-443.
279. Kunin CM, Chin QF, Chambers S. Formation of encrustations on indwelling urinary catheters in the elderly: A comparison of different types of catheter materials in "blockers" and "nonblockers". *J Urol.* 1987;138(4):899-902.
280. Burns JR, Gauthier JF. Prevention of urinary catheter encrustations by acetohydroxamic acid. *J Urol.* 1984;132(3):455-456.
281. Ruwaldt MM. Irrigation of indwelling urinary catheters. *Urology.* 1983;21(2):127-129.

Infection Control Assessment Tool for Acute Care Hospitals

This tool is intended to assist in the assessment of infection control programs and practices in acute care hospitals. If feasible, direct observations of infection control practices are encouraged. To facilitate the assessment, health departments are encouraged to share this tool with hospitals in advance of their visit.

Overview

Section 1: Facility Demographics

Section 2: Infection Control Program and Infrastructure

Section 3: Direct Observation of Facility Practices (optional)

Section 4: Infection Control Guidelines and Other Resources

Infection Control Domains for Gap Assessment

- I. Infection Control Program and Infrastructure
- II. Infection Control Training, Competency, and Implementation of Policies and Practices
 - A. Hand Hygiene
 - B. Personal Protective Equipment (PPE)
 - C. Prevention of Catheter-associated Urinary Tract Infection (CAUTI)
 - D. Prevention of Central Line-associated Bloodstream Infection (CLABSI)
 - E. Prevention of Ventilator-associated Event (VAE)
 - F. Injection Safety
 - G. Prevention of Surgical Site Infection
 - H. Prevention of *Clostridium difficile* Infection (CDI)
 - I. Environmental Cleaning
 - J. Device Reprocessing
- III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs)



Section 1. Facility Demographics	
Facility Name (for health department use only)	
NHSN Facility Organization ID (for health department use only)	
State-assigned Unique ID	
Date of Assessment	
Type of Assessment	<input type="checkbox"/> On-site <input type="checkbox"/> Other (specify):
Rationale for Assessment (Select all that apply)	<input type="checkbox"/> Outbreak <input type="checkbox"/> Input from accrediting organization or state survey agency <input type="checkbox"/> NHSN data If YES, specify: <input type="checkbox"/> CAUTI <input type="checkbox"/> CLABSI <input type="checkbox"/> SSI <input type="checkbox"/> CDI <input type="checkbox"/> Other (specify:) <input type="checkbox"/> Collaborative (specify partner[s]):) <input type="checkbox"/> Other (specify):
Facility type	<input type="checkbox"/> Acute Care Hospital <input type="checkbox"/> Critical Access Hospital <input type="checkbox"/> Long-term Acute Care Hospital (LTACH) <input type="checkbox"/> Other (specify):
Number of Licensed Beds	
Number of Infection Preventionist Full-Time Equivalents	

Section 2: Infection Control Program and Infrastructure

I. Infection Control Program and Infrastructure		
Elements to be assessed	Assessment	Notes/Areas for Improvement
1. Hospital provides fiscal and human resource support for maintaining the infection prevention and control program.	<input type="radio"/> Yes <input type="radio"/> No	
2. The person(s) charged with directing the infection prevention and control program at the hospital is/are qualified and trained in infection control. Verify qualifications, which should include: (Check all that apply) <input type="checkbox"/> Successful completion of initial and recertification exams developed by the Certification Board for Infection Control & Epidemiology (CIC) AND/OR <input type="checkbox"/> Participation in infection control courses organized by recognized professional societies (e.g., APIC, SHEA)	<input type="radio"/> Yes <input type="radio"/> No	
3. Infection prevention and control program performs an annual facility infection risk assessment that evaluates and prioritizes potential risks for infections, contamination, and exposures and the program's preparedness to eliminate or mitigate such risks. <i>Note: Example of Facility Infection Risk Assessment Report and Plan is available in Section 4.</i>	<input type="radio"/> Yes <input type="radio"/> No	
4. Written infection control policies and procedures are available, current, and based on evidence-based guidelines (e.g., CDC/HICPAC), regulations, or standards. Verify the following: a. Respondent can describe the process for reviewing and updating policies (e.g., policies are dated and reviewed annually and when new guidelines are issued)	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No	
5. Infection prevention and control program provides infection prevention education to patients, family members, and other caregivers. Verify the following: a. Respondent can describe how this education is provided (e.g., information included in the admission or discharge packet, videos, signage, in-person training)	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Hand Hygiene		
<p>1. Hospital has a competency-based training program for hand hygiene.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all healthcare personnel, including all ancillary personnel not directly involved in patient care but potentially exposed to infectious agents (e.g., food tray handlers, housekeeping, volunteer personnel). b. Training is provided upon hire, prior to provision of care at this hospital. c. Training is provided at least annually. d. Personnel are required to demonstrate competency with hand hygiene following each training. e. Hospital maintains current documentation of hand hygiene competency for all personnel. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No 	
<p>2. Hospital regularly audits (monitors and documents) adherence to hand hygiene.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>3. Hospital provides feedback from audits to personnel regarding their hand hygiene performance.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	
<p>4. Supplies necessary for adherence to hand hygiene (e.g., soap, water, paper towels, alcohol-based hand rub) are readily accessible in patient care areas.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>5. Hand hygiene policies promote preferential use of alcohol-based hand rub over soap and water except when hands are visibly soiled (e.g., blood, body fluids) or after caring for a patient with known or suspected <i>C. difficile</i> or norovirus.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
B. Personal Protective Equipment (PPE)		
<p>1. Hospital has a competency-based training program for use of personal protective equipment (PPE).</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who use PPE. b. Training is provided upon hire, prior to provision of care at this hospital. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Training includes 1) appropriate indications for specific PPE components, 2) proper donning, doffing, adjustment, and wear of PPE, and 3) proper care, maintenance, useful life, and disposal of PPE. f. Personnel are required to demonstrate competency with selection and use of PPE (i.e., correct technique is observed by trainer) following each training. g. Hospital maintains current documentation of PPE competency for all personnel who use PPE. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No g. <input type="radio"/> Yes <input type="radio"/> No 	
<p>2. Hospital regularly audits (monitors and documents) adherence to proper PPE selection and use, including donning and doffing.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>3. Hospital provides feedback to personnel regarding their performance with selection and use of PPE.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	
<p>4. Supplies necessary for adherence to personal protective equipment recommendations specified under Standard and Transmission-based Precautions (e.g., gloves, gowns, mouth, eye, nose, and face protection) are available and located near point of use.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>5. The hospital's respiratory protection program provides annual respiratory fit testing for all personnel who are anticipated to require respiratory protection.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Hospital maintains supplies of respiratory protection devices (e.g., Powered air purifying respirator) to be used by personnel who cannot be fitted. b. Healthcare personnel are educated about factors that may compromise proper fit and function of respiratory protection devices (e.g., weight gain/loss, facial hair). 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
C. Prevention of Catheter-associated Urinary Tract Infection (CAUTI)		
1. Hospital has physician and/or nurse champions for CAUTI prevention activities.	<input type="radio"/> Yes <input type="radio"/> No	
2. Hospital has a competency-based training program for insertion of urinary catheters. Verify the following: <ol style="list-style-type: none"> a. Training is provided to all personnel who are given responsibility for insertion of urinary catheters. <i>Personnel</i> may include, but are not limited to, nurses, nursing assistants, medical assistants, technicians, and physicians. b. Training is provided upon hire, prior to being allowed to perform urinary catheter insertion. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with insertion (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with urinary catheter insertion for all personnel who insert urinary catheters. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No	
3. Hospital regularly audits (monitors and documents) adherence to recommended practices for insertion of urinary catheters. Verify the following: <ol style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No	
4. Hospital provides feedback from audits to personnel regarding their performance for insertion of urinary catheters. Verify the following: <ol style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
C. Prevention of Catheter-associated Urinary Tract Infection (CAUTI), continued		
<p>5. Hospital has a competency-based training program for <u>maintenance</u> of urinary catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who are given responsibility for urinary catheter maintenance (e.g., perineal care, emptying the drainage bag aseptically, maintaining the closed drainage system, maintaining unobstructed urine flow). Personnel may include, but are not limited to, nurses, nursing assistants, medical assistants, technicians, and transport personnel. b. Training is provided upon hire, prior to being allowed to perform urinary catheter maintenance. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with catheter maintenance (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with urinary catheter maintenance for all personnel who maintain urinary catheters. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p> <p>d. <input type="radio"/> Yes <input type="radio"/> No</p> <p>e. <input type="radio"/> Yes <input type="radio"/> No</p> <p>f. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>6. Hospital regularly audits (monitors and documents) adherence to recommended practices for <u>maintenance</u> of urinary catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>7. Hospital provides feedback from audits to personnel regarding their performance for <u>maintenance</u> of urinary catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>8. Patients with urinary catheters are assessed, at least daily, for continued need for the catheter.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods used to trigger the daily assessments (e.g., patient safety checklist, daily rounds, nurse directed protocol, reminders or stop orders). b. Hospital routinely audits adherence to daily assessment of urinary catheter need. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
C. Prevention of Catheter-associated Urinary Tract Infection (CAUTI), continued		
<p>9. Hospital monitors CAUTI data and uses it to direct prevention activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent is familiar with National Healthcare Safety Network (NHSN) CAUTI data. b. Respondent can describe how CAUTI data are used to direct prevention activities. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>10. Hospital provides feedback of CAUTI data to frontline personnel.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
D. Prevention of Central line-associated Bloodstream Infection (CLABSI)		
1. Hospital has physician and/or nurse champions for CLABSI prevention activities.	<input type="radio"/> Yes <input type="radio"/> No	
2. Hospital has a competency-based training program for insertion of central venous catheters. Verify the following: <ul style="list-style-type: none"> a. Training is provided to all personnel who are given responsibility for insertion of central venous catheters. Personnel may include, but are not limited to, physicians, physician assistants, and members of line insertion teams. b. Training is provided upon hire, prior to being allowed to perform central venous catheter insertion. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with insertion (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with central venous catheter insertion for all personnel who insert central venous catheters. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No	
3. Hospital regularly audits (monitors and documents) adherence to recommended practices for insertion of central venous catheters. Verify the following: <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No	
4. Hospital provides feedback from audits to personnel regarding their performance for insertion of central venous catheters. Verify the following: <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
D. Prevention of Central line-associated Bloodstream Infection (CLABSI), continued		
<p>5. Hospital has a competency-based training program for maintenance of central venous catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who maintain central venous catheters (e.g., scrub the hub, accessing the catheter, dressing changes). Personnel may include, but are not limited to, nurses, nursing assistants, physicians, and physician assistants. b. Training is provided upon hire, prior to being allowed to perform central venous catheter maintenance. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with maintenance (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with central venous catheter maintenance for all personnel who maintain central venous catheters. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p> <p>d. <input type="radio"/> Yes <input type="radio"/> No</p> <p>e. <input type="radio"/> Yes <input type="radio"/> No</p> <p>f. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>6. Hospital regularly audits (monitors and documents) adherence to recommended practices for maintenance of central venous catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>7. Hospital provides feedback from audits to personnel regarding their performance for maintenance of central venous catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>8. Patients with central venous catheters are assessed, at least daily, for continued need for the catheter.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods used to trigger the daily assessments (e.g., patient safety checklist, daily rounds, reminders). b. Hospital routinely audits adherence to daily assessment of central venous catheter need. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
D. Prevention of Central line-associated Bloodstream Infection (CLABSI), continued		
<p>9. Hospital monitors CLABSI data and uses it to direct prevention activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent is familiar with National Healthcare Safety network (NHSN) CLABSI data. b. Respondent can describe how CLABSI data are used to direct prevention activities. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>10. Hospital provides feedback of CLABSI data to frontline personnel.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
E. Prevention of Ventilator-associated Event (VAE)		
1. Hospital has physician and/or nurse champions for VAE prevention activities.	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Check if facility does not provide care to ventilated patients and move to item F. Injection Safety.	
2. Hospital has a competency-based training program addressing prevention of VAEs. Verify the following: <ul style="list-style-type: none"> a. Training is provided to all personnel who provide respiratory therapy for ventilated patients (e.g., suctioning, administration of aerosolized medications). Personnel may include, but are not limited to, respiratory therapists and nurses. b. Training is provided upon hire, prior to being allowed to provide respiratory therapy for ventilated patients. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with respiratory therapy practices (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with respiratory practices for all personnel who provide respiratory therapy for ventilated patients. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No	
3. Hospital regularly audits (monitors and documents) adherence to recommended practices for management of ventilated patients (e.g., suctioning, administration of aerosolized medications). Verify the following: <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No	
4. Hospital provides feedback from audits to personnel regarding their performance for management of ventilated patients. Verify the following: <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
E. Prevention of Ventilator-associated Event (VAE), continued		
<p>5. Patients requiring invasive ventilation are assessed, at least daily, for continued need for the ventilator.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods used to trigger the daily assessments (e.g., patient safety checklist, daily rounds, reminders) b. Hospital routinely audits adherence to daily assessment of ventilator need. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>6. Hospital has a program that includes daily spontaneous breathing trials and lightening of sedation in eligible patient.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>7. Hospital has an oral-hygiene program.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>8. Hospital monitors VAE data and uses it to direct prevention activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how VAE data are used to direct prevention activities. <p>If the hospital reports VAE data to NHSN, verify the following:</p> <ul style="list-style-type: none"> b. Respondent is familiar with NHSN VAE data. <p>If the hospital does not report VAE data to NHSN, verify the following:</p> <ul style="list-style-type: none"> c. Respondent can describe how VAE data are collected. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>Not Applicable <input type="radio"/></p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p> <p>Not Applicable <input type="radio"/></p>	
<p>9. Hospital provides feedback of VAE data to frontline personnel.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
F. Injection Safety (This element does not include assessment of pharmacy practices)		
<p>1. Hospital has a competency-based training program for preparation and administration of parenteral medications (e.g., SQ, IM, IV) outside of the pharmacy.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who prepare and/or administer injections and parenteral infusions. b. Training is provided upon hire, prior to being allowed to prepare and/or administer injections and parenteral infusions. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with preparation and/or administration of injections and parenteral infusions following each training. f. Hospital maintains current documentation of competency with preparation and/or administration procedures for all personnel who prepare and/or administer injections and parenteral infusions. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No 	
<p>2. Hospital regularly audits (monitors and documents) adherence to safe injection practices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>3. Hospital provides feedback from audits to personnel regarding their adherence to safe injection practices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	
<p>4. Hospital has a drug diversion prevention program that includes consultation with the IP program when drug tampering (involving alteration or substitution) is suspected or identified to assess patient safety risks.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how the hospital would assess risk to patients if tampering is suspected or identified. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
G. Prevention of Surgical Site Infection (SSI)		
<p>1. Hospital has a surgical care improvement program.</p> <p>Verify the following: The surgical care improvement program addresses appropriate prophylactic antibiotic use including:</p> <ul style="list-style-type: none"> a. Preoperative timing of prophylactic antibiotic administration (within 1 hour prior to incision or 2 hours for vancomycin or fluoroquinolones). b. Appropriate prophylactic antibiotic selection based on procedure type. c. Discontinuation of prophylactic antibiotics within 24 hours (48 hours for CABG or other cardiac surgery) after surgical end time. d. The surgical care improvement program addresses prompt removal of urinary catheter on post-op day 1 or 2, unless there is a documented appropriate reason for continued use. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Check if facility does not perform surgeries and move to item H. <i>Clostridium difficile</i> Infection.</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No 	
<p>2. Hospital regularly audits (monitors and documents) adherence to elements of surgical care improvement program.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>3. Hospital provides feedback from audits to personnel regarding their adherence to elements of the surgical care improvement program.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
G. Prevention of Surgical Site Infection (SSI) , continued		
<p>4. Hospital regularly audits (monitors and documents) adherence to recommended infection control practices for SSI prevention.</p> <p>Verify the following:</p> <p>Auditing includes:</p> <ul style="list-style-type: none"> a. Adherence to preoperative surgical scrub and hand hygiene b. Appropriate use of surgical attire and drapes c. Adherence to aseptic technique and sterile field d. Proper ventilation requirements in surgical suites e. Minimization of traffic in the operating room f. Adherence to cleaning and disinfection of environmental surfaces g. Respondent can describe process used for audits. h. Respondent can describe frequency of audits. i. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p> <p>d. <input type="radio"/> Yes <input type="radio"/> No</p> <p>e. <input type="radio"/> Yes <input type="radio"/> No</p> <p>f. <input type="radio"/> Yes <input type="radio"/> No</p> <p>g. <input type="radio"/> Yes <input type="radio"/> No</p> <p>h. <input type="radio"/> Yes <input type="radio"/> No</p> <p>i. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>5. Hospital provides feedback from audits to personnel regarding their adherence to surgical infection control practices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>6. Hospital monitors SSI data and uses it to direct prevention activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent is familiar with NHSN SSI data. b. Respondent can describe how SSI data are used to direct prevention activities. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>7. Hospital provides feedback of SSI data to surgeons and other surgical personnel.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
H. Prevention of <i>Clostridium difficile</i> Infection (CDI)		
1. Hospital has physician and/or nurse champions for CDI prevention activities.	<input type="radio"/> Yes <input type="radio"/> No	
2. Hospital regularly audits (monitors and documents) adherence to recommended infection control practices for CDI prevention. Verify the following: Auditing includes: <ol style="list-style-type: none"> a. Adherence to hand hygiene b. Appropriate use of PPE c. Compliance with Contact Precautions, including use of dedicated or disposable equipment d. Adherence to cleaning and disinfection procedures, including use of sporicidal disinfectants if part of hospital policy e. Respondent can describe process used for audits. f. Respondent can describe frequency of audits. g. Respondent can describe process for improvement when non-adherence is observed. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No g. <input type="radio"/> Yes <input type="radio"/> No	
3. Hospital provides feedback from audits to personnel regarding their adherence to recommended infection control practices for CDI prevention. Verify the following: <ol style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	
4. Hospital has specific antibiotic stewardship strategies in place to reduce CDI. <i>Note: Please see section III.8 for full assessment of antibiotic stewardship program.</i> Verify the following: <ol style="list-style-type: none"> a. Hospital has strategies to reduce unnecessary use of antibiotics that are high-risk for CDI (e.g., fluoroquinolones, 3rd/4th generation cephalosporins). b. Hospital reviews appropriateness of antibiotics prescribed for treatment of other conditions (e.g., urinary tract infection) for patients with new or recent CDI diagnosis. c. Hospital educates providers about the risk of CDI with antibiotics. d. Hospital educates patients and family members about the risk of CDI with antibiotics. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No	
5. Hospital monitors CDI data and uses it to direct prevention activities. Verify the following: <ol style="list-style-type: none"> a. Respondent is familiar with NHSN CDI data. b. Respondent can describe how CDI data are used to direct prevention activities. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	
6. Hospital provides feedback of CDI data to frontline personnel. Verify the following: <ol style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
I. Environmental Cleaning		
<p>1. Hospital has a competency-based training program for environmental cleaning.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who clean and disinfect patient care areas. Personnel may include, but are not limited to, environmental services staff, nurses, nursing assistants, and technicians. b. Training is provided upon hire, prior to being allowed to perform environmental cleaning. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with environmental cleaning (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with environmental cleaning procedures for all personnel who clean and disinfect patient care areas. g. If the hospital contracts environmental services, the contractor has a comparable training program. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No g. <input type="radio"/> Yes <input type="radio"/> No <p>Not Applicable <input type="radio"/></p>	
<p>2. Hospital has policies that clearly define responsibilities for cleaning and disinfection of non-critical equipment, mobile devices, and other electronics (e.g., ICU monitors, ventilator surfaces, bar code scanners, point-of-care devices, mobile work stations, code carts, airway boxes).</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>3. Hospital has protocols to ensure that healthcare personnel can readily identify equipment that has been properly cleaned and disinfected and is ready for patient use (e.g., tagging system, placement in dedicated clean area).</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>4. Hospital regularly audits (monitors and documents) adherence to cleaning and disinfection procedures, including use of products in accordance with manufacturers' instructions (e.g., dilution, storage, shelf-life, contact time).</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits (e.g., monitoring technology, direct observation). b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>5. Hospital provides feedback from audits to personnel regarding their adherence to cleaning and disinfection procedures.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>J. Device Reprocessing</p> <p>This section refers to all medical devices that may be reused in the hospital. Device categories include:</p> <ul style="list-style-type: none"> • Critical items (e.g., surgical instruments) are objects that enter sterile tissue or the vascular system and must be sterile prior to use. • Semi-critical items (e.g., endoscopes for upper endoscopy and colonoscopy, laryngoscope blades) are objects that contact mucous membranes or non-intact skin and require, at a minimum, high-level disinfection prior to reuse. • Non-critical items (e.g., blood pressure cuffs, point-of-care devices) are objects that may come in contact with intact skin but not mucous membranes and should undergo cleaning and low- or intermediate-level disinfection depending on the nature and degree of contamination (See Environmental Cleaning Section I. above). <p>Single-use devices (SUDs) are labeled by the manufacturer for a single use and do not have reprocessing instructions. They may not be reused unless they have been reprocessed for reuse by entities which have complied with FDA regulatory requirements and have received FDA clearance to reprocess specific SUDs.</p>		
<p>1. Hospital has a competency-based training program for reprocessing of critical devices.</p> <p>Verify the following:</p> <ol style="list-style-type: none"> Training is provided to all personnel who reprocess critical devices. Training is provided upon hire, prior to being allowed to reprocess critical devices. Training is provided at least annually. Training is provided when new devices or protocols are introduced. Personnel are required to demonstrate competency with device reprocessing (i.e., correct technique is observed by trainer) following each training. Hospital maintains current documentation of competency with reprocessing procedures for all personnel who reprocess critical devices. If the hospital contracts reprocessing of critical devices, the contractor has a comparable training program which includes the specific devices used by the hospital. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ol style="list-style-type: none"> <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <p>Not Applicable <input type="radio"/></p>	
<p>2. Hospital regularly audits (monitors and documents) adherence to reprocessing procedures for critical devices.</p> <p>Verify the following:</p> <ol style="list-style-type: none"> Respondent can describe process used for audits. Respondent can describe frequency of audits. Audits occur in all locations where critical devices are reprocessed (e.g., central sterile reprocessing, operating suites), including locations where initial cleaning steps are performed (e.g., point of use). Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ol style="list-style-type: none"> <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
J. Device Reprocessing, continued		
<p>3. Hospital provides feedback from audits to personnel regarding their adherence to reprocessing procedures for critical devices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	
<p>4. Hospital has a competency-based training program for reprocessing of semi-critical devices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who reprocess semi-critical devices. b. Training is provided upon hire, prior to being allowed to reprocess semi-critical devices. c. Training is provided at least annually. d. Training is provided when new devices or protocols are introduced. e. Personnel are required to demonstrate competency with device reprocessing (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with reprocessing procedures for all personnel who reprocess semi-critical devices. g. If the hospital contracts reprocessing of semi-critical devices, the contractor has a comparable training program which includes the specific devices used by the hospital. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No g. <input type="radio"/> Yes <input type="radio"/> No Not Applicable <input type="radio"/> 	
<p>5. Hospital regularly audits (monitors and documents) adherence to reprocessing procedures for semi-critical devices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Audits occur in all locations where semi-critical devices are reprocessed (e.g., central sterile reprocessing, endoscopy suites), including locations where initial cleaning steps are performed (e.g., point of use). d. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No 	
<p>6. Hospital provides feedback from audits to personnel regarding their adherence to reprocessing procedures for semi-critical devices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
J. Device Reprocessing, continued		
<p>7. If hospital reuses single-use devices, the devices are reprocessed by an FDA-approved entity.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not Applicable <input type="radio"/> (hospital does not reuse single-use devices)</p>	
<p>8. Hospital maintains documentation of reprocessing activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Hospital maintains logs for each sterilizer cycle that include the results from each load. b. Hospital has documentation that the chemicals used for high-level disinfection are routinely tested for appropriate concentration and replaced appropriately. c. Hospital maintains documentation of reprocessing activities. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>9. Hospital allows adequate time for reprocessing to ensure adherence to all steps recommended by the device manufacturer, including drying and proper storage.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Hospital has an adequate supply of instruments for the volume of procedures performed to allow sufficient time for all reprocessing steps. b. Scheduling of procedures allows sufficient time for all reprocessing steps. c. Hospital does not routinely use immediate-use steam sterilization (IUSS). 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>10. IP program is consulted whenever new devices or products will be purchased or introduced to ensure implementation of appropriate reprocessing policies and procedures.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>11. Hospital has policies and procedures outlining hospital response (i.e., risk assessment and recall of device) in the event of a reprocessing error or failure.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. The IP can describe how the risk assessment would be performed including how the hospital would identify which patients may have been exposed to an improperly reprocessed device. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No 	

III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>1. Hospital has system in place for early detection and management of potentially infectious persons at initial points of entry to the hospital, including rapid isolation as appropriate.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Travel and occupational history is included as part of admission and triage protocols. b. Hospital has system to identify (flag) patients with targeted MDROs upon readmission so appropriate precautions can be applied. <p>The hospital has a respiratory/hygiene cough etiquette program that includes:</p> <ul style="list-style-type: none"> c. Posting signs at entrances d. Providing tissues and no-touch receptacles for disposal of tissues e. Providing hand hygiene supplies in or near waiting areas f. Offering facemasks to coughing patients and other symptomatic individuals upon entry to the facility g. Providing space in patient waiting areas (e.g., ED waiting room) and encouraging individuals with symptoms of respiratory infections to sit as far away from others as possible 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p> <p>d. <input type="radio"/> Yes <input type="radio"/> No</p> <p>e. <input type="radio"/> Yes <input type="radio"/> No</p> <p>f. <input type="radio"/> Yes <input type="radio"/> No</p> <p>g. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>2. Hospital has systems in place for early detection and isolation of infectious patients identified during the hospital stay, including rapid isolation of patients as appropriate.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. There is a mechanism for prompt notification of the IP by the clinical microbiology laboratory when novel resistance patterns and/or targeted antimicrobial-resistant pathogens are detected. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>3. Hospital has system in place for INTER-facility communication of infectious status and isolation needs of patients prior to transfer to other facilities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods employed to ensure infectious status and isolation needs are communicated with receiving facilities. b. The hospital has system to notify receiving facilities of microbiological tests (e.g., cultures) that are pending at the time of transfer. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs), continued		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>4. Hospital has system in place for INTER-facility communication to identify infectious status and isolation needs of patients prior to accepting patients from other facilities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods employed to ensure infectious status and isolation needs are obtained from transferring facilities. b. The hospital has system to follow-up on microbiological results (e.g., cultures) that are pending at the time of transfer. c. If the hospital identifies an infection that may be related to care provided at another facility (e.g., hospital, nursing home, clinic), the facility is notified. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>5. Hospital has system in place for INTRA-facility communication to identify infectious status and isolation needs of patients prior to transfer to other units or shared spaces (e.g., radiology, physical therapy, emergency department) within the hospital.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods employed to ensure infectious status and isolation needs are communicated with receiving units. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>6. Hospital has a surveillance program to monitor incidence of epidemiologically-important organisms (e.g., CRE) and targeted healthcare-associated infections.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how the hospital determines which organisms and HAIs to track. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>7. Hospital uses surveillance data to implement corrective actions rapidly when transmission of epidemiologically-important organisms (e.g., CRE) or increased rates or persistently elevated rates of healthcare-associated infections are detected.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Data collection method allows for timely response to identified problems. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p>	

III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs), continued

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>8. Hospital has an antibiotic stewardship program that meets the 7 CDC core elements listed below (a – g).</p> <p><i>Note: The antibiotic stewardship program should be assessed in consultation with personnel knowledgeable about antibiotic stewardship activities (e.g., physician or pharmacist stewardship lead). Responses can be obtained from or cross-checked with the NHSN Annual Hospital Survey Antibiotic Stewardship Practice questions (Q 23 – 34) if available.</i></p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Hospital leadership commitment <ul style="list-style-type: none"> o Hospital has a written statement of support from leadership that supports efforts to improve antibiotic use (antibiotic stewardship) <u>AND/OR</u> o Hospital provides salary support for dedicated time for antibiotic stewardship activities. b. Program leadership (accountability) <ul style="list-style-type: none"> o There is a leader responsible for outcomes of stewardship activities at the hospital. c. Drug expertise <ul style="list-style-type: none"> o There is at least one pharmacist responsible for improving antibiotic use at the hospital. d. Act (at least one prescribing improvement action below) <ul style="list-style-type: none"> o Hospital has a policy that requires prescribers to document an indication for all antibiotics in the medical record or during order entry. o Hospital has hospital-specific treatment recommendations, based on national guidelines and local susceptibility, to assist with antibiotic selection for common clinical conditions. o There is a formal procedure for all clinicians to review the appropriateness of all antibiotics at or after 48 hours from the initial orders (e.g., antibiotic time out). o Hospital has specified antibiotic agents that need to be approved by a physician or pharmacist prior to dispensing at the hospital. o Physician or pharmacist reviews courses of therapy for specified antibiotic agents and communicates results with prescribers. e. Track <ul style="list-style-type: none"> o Hospital monitors antibiotic use (consumption). f. Report <ul style="list-style-type: none"> o Prescribers receive feedback by the stewardship program about how they can improve their antibiotic prescribing. g. Educate <ul style="list-style-type: none"> o Stewardship program provides education to clinicians and other relevant staff on improving antibiotic use. 	<p>○ Yes ○ No</p> <p>a. ○ Yes ○ No</p> <p>b. ○ Yes ○ No</p> <p>c. ○ Yes ○ No</p> <p>d. ○ Yes ○ No</p> <p>e. ○ Yes ○ No</p> <p>f. ○ Yes ○ No</p> <p>g. ○ Yes ○ No</p>	

III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs), continued		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>9. Hospital has occupational health program that, in addition to complying with state and federal requirements (e.g., OSHA), has policies regarding contact of personnel with patients when personnel have potentially transmissible conditions.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. The program has work-exclusion policies that encourage reporting of illnesses and do not penalize with loss of wages, benefits or job status. b. Personnel are educated regarding prompt reporting of illness to their supervisor and the occupational health programs. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>10. Hospital follows recommendations of the Advisory Committee on Immunization Practices (ACIP) for immunization of healthcare personnel, including offering Hepatitis B and influenza vaccination.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>11. Hospital is compliant with mandatory reporting requirements for notifiable diseases, healthcare-associated infections (as appropriate), and potential outbreaks.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Hospital can identify point(s) of contact at the local or state health department for HAI concerns. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>12. Hospital implements infection control measures relevant to construction, renovation, demolition, and repairs including performance of an infection control risk assessment (ICRA) before a project gets underway.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. IP program is consulted anytime construction, renovation, demolition, or repairs will be performed. b. ICRA elements are included in all contracts related to construction, renovation, demolition, and repairs. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

Section 3: Direct Observation of Facility Practices (optional)

Certain infection control lapses (e.g., reuse of syringes on more than one patient or to access a medication container that is used for subsequent patients; reuse of lancets) can result in bloodborne pathogen transmission and should be halted immediately. Identification of such lapses warrants appropriate notification and testing of potentially affected patients.

Examples of Auditing Tools for Direct Observations:

- **General Infection Control**

Centers for Medicare & Medicaid Services Hospital Infection Control

Worksheet: <http://www.cms.gov/Medicare/Provider-Enrollment-and-Certification/SurveyCertificationGenInfo/Downloads/Survey-and-Cert-Letter-15-12-Attachment-1.pdf>

Auditing checklists available for observations of:

- Hand hygiene
- Personal protective equipment use
- Indwelling urinary catheter insertion and maintenance
- Central venous catheter insertion and maintenance
- Injection safety
- Environmental services
- Equipment reprocessing (non-critical, semi-critical, critical reusable and single-use devices)
- Ventilator/respiratory therapy
- Spinal injection procedures
- Point of care devices
- Transmission-based precautions (Contact, Droplet, Airborne)
- Surgical procedures

- **Hand Hygiene Auditing Tools**

- Measuring Hand Hygiene Adherence: Overcoming the Challenges: http://www.jointcommission.org/assets/1/18/hh_monograph.pdf
- iScrub: <http://compepi.cs.uiowa.edu/index.php/Research/iScrub>

- **Personal Protective Equipment (PPE) Donning and Doffing**

- CDC Sequence for Donning and Removing Personal Protective Equipment <http://www.cdc.gov/hai/pdfs/ppe/PPE-Sequence.pdf>

- **Urinary Catheter Appropriate Use, Insertion, and Maintenance**

- American Nurses Association CAUTI Prevention Tool: <http://nursingworld.org/CAUTI-Tool>
- CDC TAP CAUTI Toolkit Implementation Guide: <http://www.cdc.gov/hai/prevent/tap/resources.html>

- **Central Venous Catheter Appropriate Use, Insertion, and Maintenance**

- CDC Checklist for Prevention of Central Line-Associated Blood Stream Infections: <http://www.cdc.gov/HAI/pdfs/bsi/checklist-for-CLABSI.pdf>
- AHRQ Tools for Reducing CLABSI: <http://www.ahrq.gov/professionals/education/curriculum-tools/clabsitools/index.html>

- **Safe Injection Practices**

- Injection Safety
Checklist: <http://www.oneandonlycampaign.org/sites/default/files/upload/pdf/Injection%20Safety%20Checklist-508.pdf>

- **Environmental Infection Control**

- CDC Environmental Checklist for Monitoring Terminal Cleaning: <http://www.cdc.gov/HAI/toolkits/Environmental-Cleaning-Checklist-10-6-2010.pdf>
- CDC Environmental Cleaning Evaluation Worksheet: <http://www.cdc.gov/HAI/toolkits/Evaluating-Environmental-Cleaning.html>
- Infection Control Risk Assessment (ICRA) Matrix of Precautions for Construction & Renovation: http://www.ashe.org/advocacy/organizations/CDC/pdfs/assessment_icra.pdf

Section 4: Infection Control Guidelines and Other Resources

- **General Infection Prevention**

- CDC/HICPAC Guidelines and recommendations: http://www.cdc.gov/HAI/prevent/prevent_pubs.html

- **Facility Infection Risk Assessment**

- Infection Prevention Annual Report and Plan: <http://apicchapter26.org/Data%20files/Minutes%202011/IC%20Risk%20Assessment%20guide.pdf>

- **Hand Hygiene**

- Guideline for Hand Hygiene in Healthcare Settings: <http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf>
- Hand Hygiene in Healthcare Settings: <http://www.cdc.gov/handhygiene>

- **Personal Protective Equipment**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation2007.pdf>
- Guidance for the Selection and Use of Personal Protective Equipment in Healthcare Settings: <http://www.cdc.gov/HAI/prevent/ppe.html>

- **Catheter-associated Urinary Tract Infection (CAUTI)**

- Guideline for Prevention of Catheter-associated Urinary Tract Infections, 2009: <http://www.cdc.gov/hicpac/pdf/CAUTI/CAUTIGuideline2009final.pdf>

- **Central line-associated Bloodstream Infection (CLABSI)**

- Guideline for Prevention of Intravascular Catheter-related Infections, 2011: <http://www.cdc.gov/hicpac/pdf/guidelines/bsi-guidelines-2011.pdf>

- **Ventilator-associated Event (VAE)**

- Guidelines for Preventing Healthcare-associated Pneumonia, 2003: http://www.cdc.gov/hicpac/pdf/guidelines/CDCpneumo_guidelines.pdf

- **Surgical Site Infection (SSI)**

- Guidelines for the Prevention of Surgical Site Infection, 1999: http://www.cdc.gov/hicpac/pdf/guidelines/SSI_1999.pdf

- **Safe Injection Practices**
 - 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
 - CDC Injection Safety Web Materials: <http://www.cdc.gov/injectionsafety>
 - CDC training video and related Safe Injection Practices Campaign materials: <http://oneandonlycampaign.org>

- ***Clostridium difficile* Infection (CDI) and Multidrug-Resistant Organisms (MDRO), including antimicrobial stewardship**
 - 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
 - Management of Multi-Drug Resistant Organisms in Healthcare Settings, 2006: <http://www.cdc.gov/hicpac/pdf/guidelines/MDROGuideline2006.pdf>
 - SHEA-IDSA Strategies to Prevention *Clostridium difficile* Infections in Acute Care Hospitals: 2014 Update: <http://www.jstor.org/stable/10.1086/676023>
 - SHEA-IDSA Guideline: <http://www.cdc.gov/HAI/pdfs/cdiff/Cohen-IDSA-SHEA-CDI-guidelines-2010.pdf>
 - CDC's Core Elements of Hospital Antibiotic Stewardship Program: <http://www.cdc.gov/getsmart/healthcare/implementation/core-elements.html>
 - CDC Implementation Resources for Antibiotic Stewardship: <http://www.cdc.gov/getsmart/healthcare/implementation.html>
 - EPA Listing of disinfectant products with sporicidal activity against *C. difficile*: http://www.epa.gov/oppad001/list_k_clostridium.pdf

- **Environmental Infection Control, including Infection Control Risk Assessment (ICRA)**
 - Guidelines for Environmental Infection Control in Healthcare Facilities: http://www.cdc.gov/hicpac/pdf/guidelines/eic_in_HCF_03.pdf
 - 2014 Facility Guidelines Institute (FGI) Guidelines for Hospitals and Outpatient Facilities: http://www.fgiguideines.org/guidelines2014_HOP.php

- **Equipment Reprocessing**
 - Guideline for Disinfection and Sterilization in Healthcare Facilities: http://www.cdc.gov/hicpac/pdf/guidelines/Disinfection_Nov_2008.pdf
 - FDA regulations on reprocessing of single-use devices: <http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm071434>

- **Point-of-Care Testing**

- Infection Prevention during Blood Glucose Monitoring and Insulin Administration: <http://www.cdc.gov/injectionsafety/blood-glucose-monitoring.html>
- Frequently Asked Questions (FAQs) regarding Assisted Blood Glucose Monitoring and Insulin Administration: http://www.cdc.gov/injectionsafety/providers/blood-glucose-monitoring_faqs.html

- **Respiratory Hygiene/Cough Etiquette**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
- Recommendations for Preventing the Spread of Influenza: <http://www.cdc.gov/flu/professionals/infectioncontrol/>

- **Healthcare Personnel Safety**

- Guideline for Infection Control in Healthcare Personnel: <http://www.cdc.gov/hicpac/pdf/InfectControl98.pdf>
- Immunization of Healthcare Personnel: <http://www.cdc.gov/vaccines/adults/rec-vac/hcw.html>
- Occupational Safety & Health Administration (OSHA) Bloodborne Pathogen and Needlestick Prevention Standard: <https://www.osha.gov/SLTC/bloodbornepathogens/index.html>
- Hospital Respiratory Protection Program Toolkit: <http://www.cdc.gov/niosh/docs/2015-117/pdfs/2015-117.pdf>

- **Resources to assist with evaluation and response to breaches in infection control**

- Patel PR, Srinivasan A, Perz JF. Developing a broader approach to management of infection control breaches in health care settings. Am J Infect Control 2008; 36(10):685-90. [http://www.ajicjournal.org/article/S0196-6553\(08\)00683-4/abstract](http://www.ajicjournal.org/article/S0196-6553(08)00683-4/abstract)
- Steps for Evaluating an Infection Control Breach: http://www.cdc.gov/hai/outbreaks/steps_for_eval_IC_breach.html
- Patient Notification Toolkit: <http://www.cdc.gov/injectionsafety/pntoolkit/index.html>

Management of Multidrug-Resistant Organisms In Healthcare Settings, 2006

Jane D. Siegel, MD; Emily Rhinehart, RN MPH CIC; Marguerite Jackson, PhD; Linda Chiarello, RN MS; the Healthcare Infection Control Practices Advisory Committee

Acknowledgement:

The authors and HICPAC gratefully acknowledge Dr. Larry Strausbaugh for his many contributions and valued guidance in the preparation of this guideline.



Healthcare Infection Control Practices Advisory Committee (HICPAC):

Chair

Patrick J. Brennan, MD
Professor of Medicine
Division of Infectious Diseases
University of Pennsylvania Medical School

Executive Secretary

Michael Bell, MD
Division of Healthcare Quality Promotion
National Center for Infectious Diseases
Centers for Disease Control and Prevention

Members

BRINSKO, Vicki L., RN, BA
Infection Control Coordinator
Vanderbilt University Medical Center

DELLINGER, E. Patchen., MD
Professor of Surgery
University of Washington School of
Medicine

ENGEL, Jeffrey, MD
Head General Communicable Disease Control
Branch
North Carolina State Epidemiologist

GORDON, Steven M., MD
Chairman, Department of Infections Diseases
Hospital Epidemiologist
Cleveland Clinic Foundation
Department of Infectious Disease

HARRELL, Lizzie J., PhD, D(ABMM)
Research Professor of Molecular Genetics,
Microbiology and Pathology
Associate Director, Clinical Microbiology
Duke University Medical Center
O'BOYLE, Carol, PhD, RN
Assistant Professor, School of Nursing
University of Minnesota

PEGUES, David Alexander, MD
Division of Infectious Diseases
David Geffen School of Medicine at UCLA

PERROTTA, Dennis M. PhD., CIC
Adjunct Associate Professor of Epidemiology
University of Texas School of Public Health
Texas A&M University School of Rural Public
Health

PITT, Harriett M., MS, CIC, RN
Director, Epidemiology
Long Beach Memorial Medical Center

RAMSEY, Keith M., MD
Professor of Medicine
Medical Director of Infection Control
The Brody School of Medicine at East Carolina
University

SINGH, Nalini, MD, MPH
Professor of Pediatrics
Epidemiology and International Health
The George Washington University Children's National
Medical Center

STEVENSON, Kurt Brown, MD, MPH
Division of Infectious Diseases
Department of Internal Medicine
The Ohio State University Medical Center

SMITH, Philip W., MD
Chief, Section of Infectious Diseases
Department of Internal Medicine
University of Nebraska Medical Center

HICPAC membership (past)

Robert A. Weinstein, MD (Chair)
Cook County Hospital
Chicago, IL

Jane D. Siegel, MD (Co-Chair)
University of Texas Southwestern Medical Center
Dallas, TX

Michele L. Pearson, MD
(Executive Secretary)
Centers for Disease Control and Prevention
Atlanta, GA

Raymond Y.W. Chinn, MD
Sharp Memorial Hospital
San Diego, CA

Alfred DeMaria, Jr, MD
Massachusetts Department of Public Health
Jamaica Plain, MA

James T. Lee, MD, PhD
University of Minnesota
Minneapolis, MN

William A. Rutala, PhD, MPH
University of North Carolina Health Care System
Chapel Hill, NC

William E. Scheckler, MD
University of Wisconsin
Madison, WI

Beth H. Stover, RN
Kosair Children's Hospital
Louisville, KY

Marjorie A. Underwood, RN, BSN CIC
Mt. Diablo Medical Center
Concord, CA

HICPAC Liaisons

William B. Baine, MD
Liaison to Agency for Healthcare Quality
Research

Joan Blanchard, RN, MSN, CNOR
Liaison to Association of periOperative
Registered Nurses

Patrick J. Brennan, MD
Liaison to Board of Scientific Counselors

Nancy Bjerke, RN, MPH, CIC
Liaison to Association of Professionals in
Infection Prevention and Control

Jeffrey P. Engel, MD
Liaison to Advisory Committee on Elimination of
Tuberculosis

David Henderson, MD
Liaison to National Institutes of Health

Lorine J. Jay MPH, RN, CPHQ
Liaison to Healthcare Resources Services
Administration

Stephen F. Jencks, MD, MPH
Liaison to Center for Medicare and Medicaid Services

Sheila A. Murphey, MD
Liaison to Food and Drug Administration

Mark Russi, MD, MPH
Liaison to American College of Occupational and
Environmental Medicine

Rachel L. Stricof, MPH
Liaison to Advisory Committee on Elimination of
Tuberculosis

Michael L. Tapper, MD
Liaison to Society for Healthcare Epidemiology of
America

Robert A. Wise, MD
Liaison to Joint Commission on the Accreditation of
Healthcare Organizations

Authors' Associations

Jane D. Siegel, MD
Professor of Pediatrics
Department of Pediatrics
University of Texas Southwestern Medical Center

Emily Rhinehart RN MPH CIC CPHQ
Vice President
AIG Consultants, Inc.

Marguerite Jackson, RN PhD CIC
Director, Administrative Unit, National Tuberculosis
Curriculum Consortium, Department of Medicine
University of California San Diego

Linda Chiarello, RN MS
Division of Healthcare Quality Promotion
National Center for Infectious Diseases, CDC

I. Introduction

Multidrug-resistant organisms (MDROs), including methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-resistant enterococci (VRE) and certain gram-negative bacilli (GNB) have important infection control implications that either have not been addressed or received only limited consideration in previous isolation guidelines. Increasing experience with these organisms is improving understanding of the routes of transmission and effective preventive measures. Although transmission of MDROs is most frequently documented in acute care facilities, all healthcare settings are affected by the emergence and transmission of antimicrobial-resistant microbes. The severity and extent of disease caused by these pathogens varies by the population(s) affected and by the institution(s) in which they are found. Institutions, in turn, vary widely in physical and functional characteristics, ranging from long-term care facilities (LTCF) to specialty units (e.g., intensive care units [ICU], burn units, neonatal ICUs [NICUs]) in tertiary care facilities. Because of this, the approaches to prevention and control of these pathogens need to be tailored to the specific needs of each population and individual institution. The prevention and control of MDROs is a national priority - one that requires that all healthcare facilities and agencies assume responsibility(1) (2). The following discussion and recommendations are provided to guide the implementation of strategies and practices to prevent the transmission of MRSA, VRE, and other MDROs. The administration of healthcare organizations and institutions should ensure that appropriate strategies are fully implemented, regularly evaluated for effectiveness, and adjusted such that there is a consistent decrease in the incidence of targeted MDROs. Successful prevention and control of MDROs requires administrative and scientific leadership and a financial and human resource commitment(3-5). Resources must be made available for infection prevention and control, including expert consultation, laboratory support, adherence monitoring, and data analysis. Infection prevention and control professionals have found that healthcare personnel (HCP) are more receptive and adherent to the recommended control measures when organizational leaders participate in efforts to reduce MDRO transmission(3).

II. Background

MDRO definition. For epidemiologic purposes, MDROs are defined as microorganisms, predominantly bacteria, that are resistant to one or more classes of antimicrobial agents (1). Although the names of certain MDROs describe resistance to only one agent (e.g., MRSA, VRE), these pathogens are frequently resistant to most available antimicrobial agents. These highly resistant organisms deserve special attention in healthcare facilities (2). In addition to MRSA and VRE, certain GNB, including those producing extended spectrum beta-lactamases (ESBLs) and others that are resistant to multiple classes of antimicrobial agents, are of particular concern.¹ In addition to *Escherichia coli* and *Klebsiella pneumoniae*, these include strains of *Acinetobacter baumannii* resistant to all antimicrobial agents, or all except imipenem,(6-12), and organisms such as *Stenotrophomonas maltophilia* (12-14), *Burkholderia cepacia* (15, 16), and *Ralstonia pickettii*(17) that are intrinsically resistant to the broadest-spectrum antimicrobial agents. In some residential settings (e.g., LTCFs), it is important to control multidrug-resistant *S. pneumoniae* (MDRSP) that are resistant to penicillin and other broad-spectrum agents such as macrolides and fluoroquinolones (18, 19). Strains of *S. aureus* that have intermediate susceptibility or are resistant to vancomycin (i.e., vancomycin-intermediate *S. aureus* [VISA], vancomycin-resistant *S. aureus* [VRSA]) (20-30) have affected specific populations, such as hemodialysis patients.

Clinical importance of MDROs. In most instances, MDRO infections have clinical manifestations that are similar to infections caused by susceptible pathogens. However, options for treating patients with these infections are often extremely limited. For example, until recently, only vancomycin provided effective therapy for potentially life-threatening MRSA infections and during the 1990's there were virtually no antimicrobial agents to treat infections caused by VRE. Although antimicrobials are now available for treatment of MRSA and VRE infections, resistance to each new agent has already emerged in clinical

¹ Multidrug-resistant strains of *M. tuberculosis* are not addressed in this document because of the markedly different patterns of transmission and spread of the pathogen and the very different control interventions that are needed for prevention of *M. tuberculosis* infection. Current recommendations for prevention and control of tuberculosis can be found at: <http://www.cdc.gov/mmwr/pdf/rr/rr5417.pdf>

isolates(31-37). Similarly, therapeutic options are limited for ESBL-producing isolates of gram-negative bacilli, strains of *A. baumannii* resistant to all antimicrobial agents except imipenem(8-11, 38) and intrinsically resistant *Stenotrophomonas* sp.(12-14, 39). These limitations may influence antibiotic usage patterns in ways that suppress normal flora and create a favorable environment for development of colonization when exposed to potential MDR pathogens (i.e., selective advantage)(40).

Increased lengths of stay, costs, and mortality also have been associated with MDROs (41-46). Two studies documented increased mortality, hospital lengths of stay, and hospital charges associated with multidrug-resistant gram-negative bacilli (MDR-GNBs), including an NICU outbreak of ESBL-producing *Klebsiella pneumoniae* (47) and the emergence of third-generation cephalosporin resistance in *Enterobacter* spp. in hospitalized adults (48). Vancomycin resistance has been reported to be an independent predictor of death from enterococcal bacteremia(44, 49-53). Furthermore, VRE was associated with increased mortality, length of hospital stay, admission to the ICU, surgical procedures, and costs when VRE patients were compared with a matched hospital population (54).

However, MRSA may behave differently from other MDROs. When patients with MRSA have been compared to patients with methicillin-susceptible *S. aureus* (MSSA), MRSA-colonized patients more frequently develop symptomatic infections(55, 56). Furthermore, higher case fatality rates have been observed for certain MRSA infections, including bacteremia(57-62), poststernotomy mediastinitis(63), and surgical site infections(64). These outcomes may be a result of delays in the administration of vancomycin, the relative decrease in the bactericidal activity of vancomycin(65), or persistent bacteremia associated with intrinsic characteristics of certain MRSA strains (66). Mortality may be increased further by *S. aureus* with reduced vancomycin susceptibility (VISA) (26, 67). Also some studies have reported an association between MRSA infections and increased length of stay, and healthcare costs(46, 61, 62), while others have not(64). Finally, some hospitals have observed an increase in the overall occurrence of staphylococcal infections following the introduction of MRSA into a hospital or special-care unit(68, 69).

III. Epidemiology of MDROs

Trends: Prevalence of MDROs varies temporally, geographically, and by healthcare setting(70, 71). For example, VRE emerged in the eastern United States in the early 1990s, but did not appear in the western United States until several years later, and MDRSP varies in prevalence by state(72). The type and level of care also influence the prevalence of MDROs. ICUs, especially those at tertiary care facilities, may have a higher prevalence of MDRO infections than do non-ICU settings (73, 74). Antimicrobial resistance rates are also strongly correlated with hospital size, tertiary-level care, and facility type (e.g., LTCF)(75, 76). The frequency of clinical infection caused by these pathogens is low in LTCFs(77, 78). Nonetheless, MDRO infections in LTCFs can cause serious disease and mortality, and colonized or infected LTCF residents may serve as reservoirs and vehicles for MDRO introduction into acute care facilities (78-88). Another example of population differences in prevalence of target MDROs is in the pediatric population. Point prevalence surveys conducted by the Pediatric Prevention Network (PPN) in eight U.S. PICUs and 7 U.S. NICUs in 2000 found $\leq 4\%$ of patients were colonized with MRSA or VRE compared with 10-24% were colonized with ceftazidime- or aminoglycoside-resistant gram-negative bacilli; $< 3\%$ were colonized with ESBL-producing gram negative bacilli. Despite some evidence that MDRO burden is greatest in adult hospital patients, MDRO require similar control efforts in pediatric populations as well(89).

During the last several decades, the prevalence of MDROs in U.S. hospitals and medical centers has increased steadily(90, 91). MRSA was first isolated in the United States in 1968. By the early 1990s, MRSA accounted for 20%-25% of *Staphylococcus aureus* isolates from hospitalized patients(92). In 1999, MRSA accounted for $>50\%$ of *S. aureus* isolates from patients in ICUs in the National Nosocomial Infection Surveillance (NNIS) system; in 2003, 59.5% of *S. aureus* isolates in NNIS ICUs were MRSA (93). A similar rise in prevalence has occurred with VRE (94). From 1990 to 1997, the prevalence of VRE in enterococcal isolates from hospitalized patients increased from $<1\%$ to approximately 15% (95). VRE accounted for almost 25% of enterococcus isolates in NNIS ICUs in 1999 (94), and 28.5% in 2003 (93).

GNB resistant to ESBLs, fluoroquinolones, carbapenems, and aminoglycosides also have increased in prevalence. For example, in 1997, the SENTRY Antimicrobial Surveillance Program found that among *K. pneumoniae* strains isolated in the United States, resistance rates to ceftazidime and other third-generation cephalosporins were 6.6%, 9.7%, 5.4%, and 3.6% for bloodstream, pneumonia, wound, and urinary tract infections, respectively (95) In 2003, 20.6% of all *K. pneumoniae* isolates from NNIS ICUs were resistant to these drugs ((93)). Similarly, between 1999 and 2003, *Pseudomonas aeruginosa* resistance to fluoroquinolone antibiotics increased from 23% to 29.5% in NNIS ICUs(74). Also, a 3-month survey of 15 Brooklyn hospitals in 1999 found that 53% of *A. baumannii* strains exhibited resistance to carbapenems and 24% of *P. aeruginosa* strains were resistant to imipenem (10). During 1994-2000, a national review of ICU patients in 43 states found that the overall susceptibility to ciprofloxacin decreased from 86% to 76% and was temporally associated with increased use of fluoroquinolones in the United States (96).

Lastly, an analysis of temporal trends of antimicrobial resistance in non-ICU patients in 23 U.S. hospitals during 1996-1997 and 1998-1999 (97) found significant increases in the prevalence of resistant isolates including MRSA, ciprofloxacin-resistant *P. aeruginosa*, and ciprofloxacin- or ofloxacin-resistant *E. coli*. Several factors may have contributed to these increases including: selective pressure exerted by exposure to antimicrobial agents, particularly fluoroquinolones, outside of the ICU and/or in the community(7, 96, 98); increasing rates of community-associated MRSA colonization and infection(99, 100); inadequate adherence to infection control practices; or a combination of these factors.

Important concepts in transmission. Once MDROs are introduced into a healthcare setting, transmission and persistence of the resistant strain is determined by the availability of vulnerable patients, selective pressure exerted by antimicrobial use, increased potential for transmission from larger numbers of colonized or infected patients (“colonization pressure”)(101, 102); and the impact of implementation and adherence to prevention efforts. Patients vulnerable to colonization and infection include those with severe disease, especially those with compromised host defenses from underlying medical conditions; recent surgery; or indwelling medical devices (e.g., urinary catheters or endotracheal

tubes(103, 104)). Hospitalized patients, especially ICU patients, tend to have more risk factors than non-hospitalized patients do, and have the highest infection rates. For example, the risk that an ICU patient will acquire VRE increases significantly once the proportion of ICU patients colonized with VRE exceeds 50%(101) or the number days of exposure to a VRE-patient exceeds 15 days(105). A similar effect of colonization pressure has been demonstrated for MRSA in a medical ICU(102). Increasing numbers of infections with MDROs also have been reported in non-ICU areas of hospitals(97).

There is ample epidemiologic evidence to suggest that MDROs are carried from one person to another via the hands of HCP(106-109). Hands are easily contaminated during the process of care-giving or from contact with environmental surfaces in close proximity to the patient(110-113). The latter is especially important when patients have diarrhea and the reservoir of the MDRO is the gastrointestinal tract(114-117). Without adherence to published recommendations for hand hygiene and glove use(111) HCP are more likely to transmit MDROs to patients. Thus, strategies to increase and monitor adherence are important components of MDRO control programs(106, 118).

Opportunities for transmission of MDROs beyond the acute care hospital results from patients receiving care at multiple healthcare facilities and moving between acute-care, ambulatory and/or chronic care, and LTC environments. System-wide surveillance at LDS Hospital in Salt Lake City, Utah, monitored patients identified as being infected or colonized with MRSA or VRE, and found that those patients subsequently received inpatient or outpatient care at as many as 62 different healthcare facilities in that system during a 5-year span(119).

Role of colonized HCP in MDRO transmission. Rarely, HCP may introduce an MDRO into a patient care unit(120-123). Occasionally, HCP can become persistently colonized with an MDRO, but these HCP have a limited role in transmission, unless other factors are present. Additional factors that can facilitate transmission, include chronic sinusitis(120), upper respiratory infection(123), and dermatitis(124).

Implications of community-associated MRSA (CA-MRSA). The emergence of new epidemic strains of MRSA in the community, among patients without established MRSA risk factors, may present new challenges to MRSA control in healthcare settings(125-128). Historically, genetic analyses of MRSA isolated from patients in hospitals worldwide revealed that a relatively small number of MRSA strains have unique qualities that facilitate their transmission from patient to patient within healthcare facilities over wide geographic areas, explaining the dramatic increases in HAIs caused by MRSA in the 1980s and early 1990s(129). To date, most MRSA strains isolated from patients with CA-MRSA infections have been microbiologically distinct from those endemic in healthcare settings, suggesting that some of these strains may have arisen *de novo* in the community via acquisition of methicillin resistance genes by established methicillin-susceptible *S. aureus* (MSSA) strains(130-132). Two pulsed-field types, termed USA300 and USA400 according to a typing scheme established at CDC, have accounted for the majority of CA-MRSA infections characterized in the United States, whereas pulsed-field types USA100 and USA200 are the predominant genotypes endemic in healthcare settings(133).

USA300 and USA400 genotypes almost always carry type IV of the staphylococcal chromosomal cassette (SCC) *mec*, the mobile genetic element that carries the *mecA* methicillin-resistance gene (133, 134). This genetic cassette is smaller than types I through III, the types typically found in healthcare associated MRSA strains, and is hypothesized to be more easily transferable between *S. aureus* strains.

CA-MRSA infection presents most commonly as relatively minor skin and soft tissue infections, but severe invasive disease, including necrotizing pneumonia, necrotizing fasciitis, severe osteomyelitis, and a sepsis syndrome with increased mortality have also been described in children and adults(134-136).

Transmission within hospitals of MRSA strains first described in the community (e.g. USA300 and USA400) are being reported with increasing frequency(137-140). Changing resistance patterns of MRSA in ICUs in the NNIS system from 1992 to 2003 provide additional evidence that the new epidemic MRSA strains are becoming established

healthcare-associated as well as community pathogens(90). Infections with these strains have most commonly presented as skin disease in community settings. However, intrinsic virulence characteristics of the organisms can result in clinical manifestations similar to or potentially more severe than traditional healthcare-associated MRSA infections among hospitalized patients. The prevalence of MRSA colonization and infection in the surrounding community may therefore affect the selection of strategies for MRSA control in healthcare settings.

IV. MDRO Prevention and Control

Prevention of Infections. Preventing infections will reduce the burden of MDROs in healthcare settings. Prevention of antimicrobial resistance depends on appropriate clinical practices that should be incorporated into all routine patient care. These include optimal management of vascular and urinary catheters, prevention of lower respiratory tract infection in intubated patients, accurate diagnosis of infectious etiologies, and judicious antimicrobial selection and utilization. Guidance for these preventive practices include the Campaign to Reduce Antimicrobial Resistance in Healthcare Settings (www.cdc.gov/drugresistance/healthcare/default.htm), a multifaceted, evidence-based approach with four parallel strategies: infection prevention; accurate and prompt diagnosis and treatment; prudent use of antimicrobials; and prevention of transmission. Campaign materials are available for acute care hospitals, surgical settings, dialysis units, LTCFs and pediatric acute care units.

To reduce rates of central-venous-line associated bloodstream infections(CVL-BSIs) and ventilator-associated pneumonia (VAP), a group of bundled evidence-based clinical practices have been implemented in many U.S. healthcare facilities(118, 141-144). One report demonstrated a sustained effect on the reduction in CVL-BSI rates with this approach(145). Although the specific effect on MDRO infection and colonization rates have not been reported, it is logical that decreasing these and other healthcare-associated infections will in turn reduce antimicrobial use and decrease opportunities for emergence and transmission of MDROs.

Prevention and Control of MDRO transmission

Overview of the MDRO control literature. Successful control of MDROs has been documented in the United States and abroad using a variety of combined interventions. These include improvements in hand hygiene, use of Contact Precautions until patients are culture-negative for a target MDRO, active surveillance cultures (ASC), education, enhanced environmental cleaning, and improvements in communication about patients with MDROs within and between healthcare facilities.

Representative studies include:

- Reduced rates of MRSA transmission in The Netherlands, Belgium, Denmark, and other Scandinavian countries after the implementation of aggressive and sustained infection control interventions (i.e., ASC; preemptive use of Contact Precautions upon admission until proven culture negative; and, in some instances, closure of units to new admissions). MRSA generally accounts for a very small proportion of *S. aureus* clinical isolates in these countries(146-150).
- Reduced rates of VRE transmission in healthcare facilities in the three-state Siouland region (Iowa, Nebraska, and South Dakota) following formation of a coalition and development of an effective region-wide infection control intervention that included ASC and isolation of infected patients. The overall prevalence rate of VRE in the 30 participating facilities decreased from 2.2% in 1997 to 0.5% in 1999(151).
- Eradication of endemic MRSA infections from two NICUs. The first NICU included implementation of ASC, Contact Precautions, use of triple dye on the umbilical cord, and systems changes to improve surveillance and adherence to recommended practices and to reduce overcrowding(152). The second NICU used ASC and Contact Precautions; surgical masks were included in the barriers used for Contact Precautions(153).
- Control of an outbreak and eventual eradication of VRE from a burn unit over a 13-month period with implementation of aggressive culturing, environmental cleaning, and barrier isolation(154).
- Control of an outbreak of VRE in a NICU over a 3-year period with implementation of ASC, other infection control measures such as use of a waterless hand disinfectant, and mandatory in-service education(155).

- Eradication of MDR-strains of *A. baumannii* from a burn unit over a 16-month period with implementation of strategies to improve adherence to hand hygiene, isolation, environmental cleaning, and temporary unit closure(38).
- In addition, more than 100 reports published during 1982-2005 support the efficacy of combinations of various control interventions to reduce the burden of MRSA, VRE, and MDR-GNBs (Tables 1 and 2). Case-rate reduction or pathogen eradication was reported in a majority of studies.
- VRE was eradicated in seven special-care units(154, 156-160), two hospitals(161, 162), and one LTCF(163).
- MRSA was eradicated from nine special-care units(89, 152, 153, 164-169), two hospitals(170), one LTCF(167), and one Finnish district(171). Furthermore, four MRSA reports described continuing success in sustaining low endemic MDRO rates for over 5 years(68, 166, 172, 173).
- An MDR-GNB was eradicated from 13 special-care units(8, 9, 38, 174-180) and two hospitals (11, 181).

These success stories testify to the importance of having dedicated and knowledgeable teams of healthcare professionals who are willing to persist for years, if necessary, to control MDROs. Eradication and control of MDROs, such as those reported, frequently required periodic reassessment and the addition of new and more stringent interventions over time (tiered strategy). For example, interventions were added in a stepwise fashion during a 3-year effort that eventually eradicated MRSA from an NICU(152). A series of interventions was adopted throughout the course of a year to eradicate VRE from a burn unit(154). Similarly, eradication of carbapenem-resistant strains of *A. baumannii* from a hospital required multiple and progressively more intense interventions over several years(11).

Nearly all studies reporting successful MDRO control employed a median of 7 to 8 different interventions concurrently or sequentially (Table 1). These figures may underestimate the actual number of control measures used, because authors of these reports may have considered their earliest efforts routine (e.g., added emphasis on handwashing), and did not include them as interventions, and some "single measures" are, in fact, a complex

combination of several interventions. The use of multiple concurrent control measures in these reports underscores the need for a comprehensive approach for controlling MDROs.

Several factors affect the ability to generalize the results of the various studies reviewed, including differences in definition, study design, endpoints and variables measured, and period of follow-up. Two-thirds of the reports cited in Tables 1 and 2 involved perceived outbreaks, and one-third described efforts to reduce endemic transmission. Few reports described preemptive efforts or prospective studies to control MDROs before they had reached high levels within a unit or facility.

With these and other factors, it has not been possible to determine the effectiveness of individual interventions, or a specific combination of interventions, that would be appropriate for all healthcare facilities to implement in order to control their target MDROs. Randomized controlled trials are necessary to acquire this level of evidence. An NIH-sponsored, randomized controlled trial on the prevention of MRSA and VRE transmission in adult ICUs is ongoing and may provide further insight into optimal control measures (<http://clinicaltrials.gov/ct/show/NCT00100386?order=1>). This trial compares the use of education (to improve adherence to hand hygiene) and Standard Precautions to the use of ASC and Contact Precautions.

Control Interventions. The various types of interventions used to control or eradicate MDROs may be grouped into seven categories. These include administrative support, judicious use of antimicrobials, surveillance (routine and enhanced), Standard and Contact Precautions, environmental measures, education and decolonization. These interventions provide the basis for the recommendations for control of MDROs in healthcare settings that follow this review and as summarized in Table 3. In the studies reviewed, these interventions were applied in various combinations and degrees of intensity, with differences in outcome.

- 1. Administrative support.** In several reports, administrative support and involvement were important for the successful control of the target MDRO(3, 152, 182-185), and authorities in infection control have strongly recommended such support(2, 106, 107,

186). There are several examples of MDRO control interventions that require administrative commitment of fiscal and human resources. One is the use of ASC(8, 38, 68, 107, 114, 151, 152, 167, 168, 183, 184, 187-192). Other interventions that require administrative support include: 1) implementing system changes to ensure prompt and effective communications e.g., computer alerts to identify patients previously known to be colonized/infected with MDROs(184, 189, 193, 194); 2), providing the necessary number and appropriate placement of hand washing sinks and alcohol-containing hand rub dispensers in the facility(106, 195); 3) maintaining staffing levels appropriate to the intensity of care required(152, 196-202); and 4) enforcing adherence to recommended infection control practices (e.g., hand hygiene, Standard and Contact Precautions) for MDRO control. Other measures that have been associated with a positive impact on prevention efforts, that require administrative support, are direct observation with feedback to HCP on adherence to recommended precautions and keeping HCP informed about changes in transmission rates(3, 152, 182, 203-205). A “How-to guide” for implementing change in ICUs, including analysis of structure, process, and outcomes when designing interventions, can assist in identification of needed administrative interventions(195). Lastly, participation in existing, or the creation of new, city-wide, state-wide, regional or national coalitions, to combat emerging or growing MDRO problems is an effective strategy that requires administrative support(146, 151, 167, 188, 206, 207).

2. Education. Facility-wide, unit-targeted, and informal, educational interventions were included in several successful studies(3, 189, 193, 208-211). The focus of the interventions was to encourage a behavior change through improved understanding of the problem MDRO that the facility was trying to control. Whether the desired change involved hand hygiene, antimicrobial prescribing patterns, or other outcomes, enhancing understanding and creating a culture that supported and promoted the desired behavior, were viewed as essential to the success of the intervention. Educational campaigns to enhance adherence to hand hygiene practices in conjunction with other control measures have been associated temporally with decreases in MDRO transmission in various healthcare settings(3, 106, 163).

3. *Judicious use of antimicrobial agents.* While a comprehensive review of antimicrobial stewardship is beyond the scope of this guideline, recommendations for control of MDROs must include attention to judicious antimicrobial use. A temporal association between formulary changes and decreased occurrence of a target MDRO was found in several studies, especially in those that focused on MDR-GNBs(98, 177, 209, 212-218). Occurrence of *C. difficile*-associated disease has also been associated with changes in antimicrobial use(219). Although some MRSA and VRE control efforts have attempted to limit antimicrobial use, the relative importance of this measure for controlling these MDROs remains unclear(193, 220). Limiting antimicrobial use alone may fail to control resistance due to a combination of factors; including 1) the relative effect of antimicrobials on providing initial selective pressure, compared to perpetuating resistance once it has emerged; 2) inadequate limits on usage; or 3) insufficient time to observe the impact of this intervention. With the intent of addressing #2 and #3 above in the study design, one study demonstrated a decrease in the prevalence of VRE associated with a formulary switch from ticarcillin-clavulanate to piperacillin-tazobactam(221).

The CDC Campaign to Prevent Antimicrobial Resistance that was launched in 2002 provides evidence-based principles for judicious use of antimicrobials and tools for implementation(222) www.cdc.gov/drugresistance/healthcare. This effort targets all healthcare settings and focuses on effective antimicrobial treatment of infections, use of narrow spectrum agents, treatment of infections and not contaminants, avoiding excessive duration of therapy, and restricting use of broad-spectrum or more potent antimicrobials to treatment of serious infections when the pathogen is not known or when other effective agents are unavailable. Achieving these objectives would likely diminish the selective pressure that favors proliferation of MDROs. Strategies for influencing antimicrobial prescribing patterns within healthcare facilities include education; formulary restriction; prior-approval programs, including pre-approved indications; automatic stop orders; academic interventions to counteract pharmaceutical influences on prescribing patterns; antimicrobial cycling(223-226);

computer-assisted management programs(227-229); and active efforts to remove redundant antimicrobial combinations(230). A systematic review of controlled studies identified several successful practices. These include social marketing (i.e. consumer education), practice guidelines, authorization systems, formulary restriction, mandatory consultation, and peer review and feedback. It further suggested that online systems that provide clinical information, structured order entry, and decision support are promising strategies(231). These changes are best accomplished through an organizational, multidisciplinary, antimicrobial management program(232).

- 4. MDRO surveillance.** Surveillance is a critically important component of any MDRO control program, allowing detection of newly emerging pathogens, monitoring epidemiologic trends, and measuring the effectiveness of interventions. Multiple MDRO surveillance strategies have been employed, ranging from surveillance of clinical microbiology laboratory results obtained as part of routine clinical care, to use of ASC to detect asymptomatic colonization.

Surveillance for MDROs isolated from routine clinical cultures.

Antibiograms. The simplest form of MDRO surveillance is monitoring of clinical microbiology isolates resulting from tests ordered as part of routine clinical care. This method is particularly useful to detect emergence of new MDROs not previously detected, either within an individual healthcare facility or community-wide. In addition, this information can be used to prepare facility- or unit-specific summary antimicrobial susceptibility reports that describe pathogen-specific prevalence of resistance among clinical isolates. Such reports may be useful to monitor for changes in known resistance patterns that might signal emergence or transmission of MDROs, and also to provide clinicians with information to guide antimicrobial prescribing practices(233-235).

MDRO Incidence Based on Clinical Culture Results. Some investigators have used clinical microbiology results to calculate measures of incidence of MDRO isolates in specific populations or patient care locations (e.g. new MDRO

isolates/1,000 patient days, new MDRO isolates per month)(205, 236, 237). Such measures may be useful for monitoring MDRO trends and assessing the impact of prevention programs, although they have limitations. Because they are based solely on positive culture results without accompanying clinical information, they do not distinguish colonization from infection, and may not fully demonstrate the burden of MDRO-associated disease. Furthermore, these measures do not precisely measure acquisition of MDRO colonization in a given population or location. Isolating an MDRO from a clinical culture obtained from a patient several days after admission to a given unit or facility does not establish that the patient acquired colonization in that unit. On the other hand, patients who acquire MDRO colonization may remain undetected by clinical cultures(107). Despite these limitations, incidence measures based on clinical culture results may be highly correlated with actual MDRO transmission rates derived from information using ASC, as demonstrated in a recent multicenter study(237). These results suggest that incidence measures based on clinical cultures alone might be useful surrogates for monitoring changes in MDRO transmission rates.

MDRO Infection Rates. Clinical cultures can also be used to identify targeted MDRO infections in certain patient populations or units(238, 239). This strategy requires investigation of clinical circumstances surrounding a positive culture to distinguish colonization from infection, but it can be particularly helpful in defining the clinical impact of MDROs within a facility.

Molecular typing of MDRO isolates. Many investigators have used molecular typing of selected isolates to confirm clonal transmission to enhance understanding of MDRO transmission and the effect of interventions within their facility(38, 68, 89, 92, 138, 152, 190, 193, 236, 240).

Surveillance for MDROs by Detecting Asymptomatic Colonization

Another form of MDRO surveillance is the use of active surveillance cultures (ASC) to identify patients who are colonized with a targeted MDRO(38, 107, 241). This

approach is based upon the observation that, for some MDROs, detection of colonization may be delayed or missed completely if culture results obtained in the course of routine clinical care are the primary means of identifying colonized patients(8, 38, 107, 114, 151, 153, 167, 168, 183, 184, 187, 189, 191-193, 242-244). Several authors report having used ASC when new pathogens emerge in order to define the epidemiology of the particular agent(22, 23, 107, 190). In addition, the authors of several reports have concluded that ASC, in combination with use of Contact Precautions for colonized patients, contributed directly to the decline or eradication of the target MDRO(38, 68, 107, 151, 153, 184, 217, 242). However, not all studies have reached the same conclusion. Poor control of MRSA despite use of ASC has been described(245). A recent study failed to identify cross-transmission of MRSA or MSSA in a MICU during a 10 week period when ASC were obtained, despite the fact that culture results were not reported to the staff(246). The investigators suggest that the degree of cohorting and adherence to Standard Precautions might have been the important determinants of transmission prevention, rather than the use of ASC and Contact Precautions for MRSA-colonized patients. The authors of a systematic review of the literature on the use of isolation measures to control healthcare-associated MRSA concluded that there is evidence that concerted efforts that include ASC and isolation can reduce MRSA even in endemic settings. However, the authors also noted that methodological weaknesses and inadequate reporting in published research make it difficult to rule out plausible alternative explanations for reductions in MRSA acquisition associated with these interventions, and therefore concluded that the precise contribution of active surveillance and isolation alone is difficult to assess(247).

Mathematical modeling studies have been used to estimate the impact of ASC use in control of MDROs. One such study evaluating interventions to decrease VRE transmission indicated that use of ASC (versus no cultures) could potentially decrease transmission 39% and that with pre-emptive isolation plus ASC, transmission could be decreased 65%(248). Another mathematical model examining the use of ASC and isolation for control of MRSA predicted that isolating colonized or

infected patients on the basis of clinical culture results is unlikely to be successful at controlling MRSA, whereas use of active surveillance and isolation can lead to successful control, even in settings where MRSA is highly endemic.(249) There is less literature on the use of ASC in controlling MDR-GNBs. Active surveillance cultures have been used as part of efforts to successful control of MDR-GNBs in outbreak settings. The experience with ASC as part of successful control efforts in endemic settings is mixed. One study reported successful reduction of extended-spectrum beta-lactamase –producing Enterobacteriaceae over a six year period using a multifaceted control program that included use of ASC(245). Other reports suggest that use of ASC is not necessary to control endemic MDR-GNBs.(250, 251).

More research is needed to determine the circumstances under which ASC are most beneficial(252), but their use should be considered in some settings, especially if other control measures have been ineffective. When use of ASC is incorporated into MDRO prevention programs, the following should be considered:

- The decision to use ASC as part of an infection prevention and control program requires additional support for successful implementation, including: 1) personnel to obtain the appropriate cultures, 2) microbiology laboratory personnel to process the cultures, 3) mechanism for communicating results to caregivers, 4) concurrent decisions about use of additional isolation measures triggered by a positive culture (e.g. Contact Precautions) and 5) mechanism for assuring adherence to the additional isolation measures.
- The populations targeted for ASC are not well defined and vary among published reports. Some investigators have chosen to target specific patient populations considered at high risk for MDRO colonization based on factors such as location (e.g. ICU with high MDRO rates), antibiotic exposure history, presence of underlying diseases, prolonged duration of stay, exposure to other MDRO-colonized patients, patients transferred from other facilities known to have a high prevalence of MDRO carriage, or having a history of recent hospital or nursing home stays(107, 151, 253). A more commonly employed strategy involves obtaining surveillance cultures from all patients admitted to units experiencing

high rates of colonization/infection with the MDROs of interest, unless they are already known to be MDRO carriers(153, 184, 242, 254). In an effort to better define target populations for active surveillance, investigators have attempted to create prediction rules to identify subpopulations of patients at high risk for colonization on hospital admission(255, 256). Decisions about which populations should be targeted for active surveillance should be made in the context of local determinations of the incidence and prevalence of MDRO colonization within the intervention facility as well as other facilities with whom patients are frequently exchanged(257).

- Optimal timing and interval of ASC are not well defined. In many reports, cultures were obtained at the time of admission to the hospital or intervention unit or at the time of transfer to or from designated units (e.g., ICU)(107). In addition, some hospitals have chosen to obtain cultures on a periodic basis [e.g., weekly(8, 153, 159) to detect silent transmission. Others have based follow-up cultures on the presence of certain risk factors for MDRO colonization, such as antibiotic exposure, exposure to other MDRO colonized patients, or prolonged duration of stay in a high risk unit(253).
- Methods for obtaining ASC must be carefully considered, and may vary depending upon the MDRO of interest.
 - MRSA: Studies suggest that cultures of the nares identify most patients with MRSA and perirectal and wound cultures can identify additional carriers(152, 258-261).
 - VRE: Stool, rectal, or perirectal swabs are generally considered a sensitive method for detection of VRE. While one study suggested that rectal swabs may identify only 60% of individuals harboring VRE, and may be affected by VRE stool density(262), this observation has not been reported elsewhere in the literature.
 - MDR-GNBs: Several methods for detection of MDR-GNBs have been employed, including use of peri-rectal or rectal swabs alone or in combination with oro-pharyngeal, endotracheal, inguinal, or wound cultures. The absence of standardized screening media for many gram-

negative bacilli can make the process of isolating a specific MDR-GNB a relatively labor-intensive process(38, 190, 241, 250).

- Rapid detection methods: Using conventional culture methods for active surveillance can result in a delay of 2-3 days before results are available. If the infection control precautions (e.g., Contact Precautions) are withheld until the results are available, the desired infection control measures could be delayed. If empiric precautions are used pending negative surveillance culture results, precautions may be unnecessarily implemented for many, if not most, patients. For this reason, investigators have sought methods for decreasing the time necessary to obtain a result from ASC. Commercially available media containing chromogenic enzyme substrates (CHROMagar MRSA(263, 264) has been shown to have high sensitivity and specificity for identification of MRSA and facilitate detection of MRSA colonies in screening cultures as early as 16 hours after inoculation. In addition, real-time PCR-based tests for rapid detection of MRSA directly from culture swabs (< 1-2 hours) are now commercially available(265-267), as well as PCR-based tests for detection of vanA and van B genes from rectal swabs(268). The impact of rapid testing on the effectiveness of active surveillance as a prevention strategy, however, has not been fully determined. Rapid identification of MRSA in one study was associated with a significant reduction in MRSA infections acquired in the medical ICU, but not the surgical ICU(265). A mathematical model characterizing MRSA transmission dynamics predicted that, in comparison to conventional culture methods, the use of rapid detection tests may decrease isolation needs in settings of low-endemicity and result in more rapid reduction in prevalence in highly-endemic settings(249).
- Some MDRO control reports described surveillance cultures of healthcare personnel during outbreaks, but colonized or infected healthcare personnel are rarely the source of ongoing transmission, and this strategy should be reserved for settings in which specific healthcare personnel have been epidemiologically implicated in the transmission of MDROs(38, 92, 152-154, 188).

5. Infection Control Precautions. Since 1996 CDC has recommended the use of Standard and Contact Precautions for MDROs “judged by an infection control program...to be of special clinical and epidemiologic significance.” This recommendation was based on general consensus and was not necessarily evidence-based. No studies have directly compared the efficacy of Standard Precautions alone versus Standard Precautions and Contact Precautions, with or without ASC, for control of MDROs. Some reports mention the use of one or both sets of precautions as part of successful MDRO control efforts; however, the precautions were not the primary focus of the study intervention(164, 190, 205, 269-271). The NIH-sponsored study mentioned earlier (Section: *Overview of the MDRO control literature*) may provide some answers, <http://clinicaltrials.gov/ct/show/NCT00100386?order=1>).

Standard Precautions have an essential role in preventing MDRO transmission, even in facilities that use Contact Precautions for patients with an identified MDRO. Colonization with MDROs is frequently undetected; even surveillance cultures may fail to identify colonized persons due to lack of sensitivity, laboratory deficiencies, or intermittent colonization due to antimicrobial therapy(262). Therefore, Standard Precautions must be used in order to prevent transmission from potentially colonized patients. Hand hygiene is an important component of Standard Precautions. The authors of the *Guideline for Hand Hygiene in Healthcare Settings*(106) cited nine studies that demonstrated a temporal relationship between improved adherence to recommended hand hygiene practices and control of MDROs. It is noteworthy that in one report the frequency of hand hygiene did not improve with use of Contact Precautions but did improve when gloves were used (per Standard Precautions) for contact with MDRO patients(272).

MDRO control efforts frequently involved changes in isolation practices, especially during outbreaks. In the majority of reports, Contact Precautions were implemented for all patients found to be colonized or infected with the target MDRO (See Table 2).

Some facilities also preemptively used Contact Precautions, in conjunction with ASC, for all new admissions or for all patients admitted to a specific unit, until a negative screening culture for the target MDRO was reported(30, 184, 273).

Contact Precautions are intended to prevent transmission of infectious agents, including epidemiologically important microorganisms, which are transmitted by direct or indirect contact with the patient or the patient's environment. A single-patient room is preferred for patients who require Contact Precautions. When a single-patient room is not available, consultation with infection control is necessary to assess the various risks associated with other patient placement options (e.g., cohorting, keeping the patient with an existing roommate). HCP caring for patients on Contact Precautions should wear a gown and gloves for all interactions that may involve contact with the patient or potentially contaminated areas in the patient's environment. Donning gown and gloves upon room entry and discarding before exiting the patient room is done to contain pathogens, especially those that have been implicated in transmission through environmental contamination (e.g., VRE, *C. difficile*, noroviruses and other intestinal tract agents; RSV)(109, 111, 274-277).

Cohorting and other MDRO control strategies. In several reports, cohorting of patients(152, 153, 167, 183, 184, 188, 189, 217, 242), cohorting of staff(184, 217, 242, 278), use of designated beds or units(183, 184), and even unit closure(38, 146, 159, 161, 279, 280) were necessary to control transmission. Some authors indicated that implementation of the latter two strategies were the turning points in their control efforts; however, these measures usually followed many other actions to prevent transmission. In one, two-center study, moving MRSA-positive patients into single rooms or cohorting these patients in designated bays failed to reduce transmission in ICUs. However, in this study adherence to recommendations for hand hygiene between patient contacts was only 21%(281). Other published studies, including one commissioned by the American Institute of Architects and the Facility Guidelines Institute (www.aia.org/aah_gd_hospcons), have documented a beneficial relationship between private rooms and reduction in risk of acquiring MDROs(282). Additional

studies are needed to define the specific contribution of using single-patient rooms and/or cohorting on preventing transmission of MDROs.

Duration of Contact Precautions. The necessary duration of Contact Precautions for patients treated for infection with an MDRO, but who may continue to be colonized with the organism at one or more body sites, remains an unresolved issue. Patients may remain colonized with MDROs for prolonged periods; shedding of these organisms may be intermittent, and surveillance cultures may fail to detect their presence(84, 250, 283). The 1995 HICPAC guideline for preventing the transmission of VRE suggested three negative stool/perianal cultures obtained at weekly intervals as a criterion for discontinuation of Contact Precautions(274). One study found these criteria generally reliable(284). However, this and other studies have noted a recurrence of VRE positive cultures in persons who subsequently receive antimicrobial therapy and persistent or intermittent carriage of VRE for more than 1 year has been reported(284-286). Similarly, colonization with MRSA can be prolonged(287, 288). Studies demonstrating initial clearance of MRSA following decolonization therapy have reported a high frequency of subsequent carriage(289, 290). There is a paucity of information in the literature on when to discontinue Contact Precautions for patients colonized with a MDR-GNB, possibly because infection and colonization with these MDROs are often associated with outbreaks. Despite the uncertainty about when to discontinue Contact Precautions, the studies offer some guidance. In the context of an outbreak, prudence would dictate that Contact Precautions be used indefinitely for all previously infected and known colonized patients. Likewise, if ASC are used to detect and isolate patients colonized with MRSA or VRE, and there is no decolonization of these patients, it is logical to assume that Contact Precautions would be used for the duration of stay in the setting where they were first implemented. In general, it seems reasonable to discontinue Contact Precautions when three or more surveillance cultures for the target MDRO are repeatedly negative over the course of a week or two in a patient who has not received antimicrobial therapy for several weeks, especially in the absence of a

draining wound, profuse respiratory secretions, or evidence implicating the specific patient in ongoing transmission of the MDRO within the facility.

Barriers used for contact with patients infected or colonized with MDROs.

Three studies evaluated the use of gloves with or without gowns for all patient contacts to prevent VRE acquisition in ICU settings(30, 105, 273). Two of the studies showed that use of both gloves and gowns reduced VRE transmission(30, 105) while the third showed no difference in transmission based on the barriers used(273). One study in a LTCF compared the use of gloves only, with gloves plus contact isolation, for patients with four MDROs, including VRE and MRSA, and found no difference(86). However, patients on contact isolation were more likely to acquire MDR-*K. pneumoniae* strains that were prevalent in the facility; reasons for this were not specifically known. In addition to differences in outcome, differing methodologies make comparisons difficult. Specifically, HCP adherence to the recommended protocol, the influence of added precautions on the number of HCP-patient interactions, and colonization pressure were not consistently assessed.

Impact of Contact Precautions on patient care and well-being. There are limited data regarding the impact of Contact Precautions on patients. Two studies found that HCP, including attending physicians, were half as likely to enter the rooms of(291), or examine(292), patients on Contact Precautions. Other investigators have reported similar observations on surgical wards(293). Two studies reported that patients in private rooms and on barrier precautions for an MDRO had increased anxiety and depression scores(294, 295). Another study found that patients placed on Contact Precautions for MRSA had significantly more preventable adverse events, expressed greater dissatisfaction with their treatment, and had less documented care than control patients who were not in isolation(296). Therefore, when patients are placed on Contact Precautions, efforts must be made by the healthcare team to counteract these potential adverse effects.

6. Environmental measures. The potential role of environmental reservoirs, such as surfaces and medical equipment, in the transmission of VRE and other MDROs has been the subject of several reports(109-111, 297, 298). While environmental cultures are not routinely recommended(299), environmental cultures were used in several studies to document contamination, and led to interventions that included the use of dedicated noncritical medical equipment(217, 300), assignment of dedicated cleaning personnel to the affected patient care unit(154), and increased cleaning and disinfection of frequently-touched surfaces (e.g., bedrails, charts, bedside commodes, doorknobs). A common reason given for finding environmental contamination with an MDRO was the lack of adherence to facility procedures for cleaning and disinfection. In an educational and observational intervention, which targeted a defined group of housekeeping personnel, there was a persistent decrease in the acquisition of VRE in a medical ICU(301). Therefore, monitoring for adherence to recommended environmental cleaning practices is an important determinant for success in controlling transmission of MDROs and other pathogens in the environment(274, 302).

In the MDRO reports reviewed, enhanced environmental cleaning was frequently undertaken when there was evidence of environmental contamination and ongoing transmission. Rarely, control of the target MDRO required vacating a patient care unit for complete environmental cleaning and assessment(175, 279).

7. Decolonization. Decolonization entails treatment of persons colonized with a specific MDRO, usually MRSA, to eradicate carriage of that organism. Although some investigators have attempted to decolonize patients harboring VRE(220), few have achieved success. However, decolonization of persons carrying MRSA in their nares has proved possible with several regimens that include topical mupirocin alone or in combination with orally administered antibiotics (e.g., rifampin in combination with trimethoprim- sulfamethoxazole or ciprofloxacin) plus the use of an antimicrobial soap for bathing(303). In one report, a 3-day regimen of baths with povidone-iodine and nasal therapy with mupirocin resulted in eradication of nasal MRSA

colonization(304). These and other methods of MRSA decolonization have been thoroughly reviewed.(303, 305-307).

Decolonization regimens are not sufficiently effective to warrant routine use. Therefore, most healthcare facilities have limited the use of decolonization to MRSA outbreaks, or other high prevalence situations, especially those affecting special-care units. Several factors limit the utility of this control measure on a widespread basis: 1) identification of candidates for decolonization requires surveillance cultures; 2) candidates receiving decolonization treatment must receive follow-up cultures to ensure eradication; and 3) recolonization with the same strain, initial colonization with a mupirocin-resistant strain, and emergence of resistance to mupirocin during treatment can occur(289, 303, 308-310). HCP implicated in transmission of MRSA are candidates for decolonization and should be treated and culture negative before returning to direct patient care. In contrast, HCP who are colonized with MRSA, but are asymptomatic, and have not been linked epidemiologically to transmission, do not require decolonization.

IV. Discussion

This review demonstrates the depth of published science on the prevention and control of MDROs. Using a combination of interventions, MDROs in endemic, outbreak, and non-endemic settings have been brought under control. However, despite the volume of literature, an appropriate set of evidence-based control measures that can be universally applied in all healthcare settings has not been definitively established. This is due in part to differences in study methodology and outcome measures, including an absence of randomized, controlled trials comparing one MDRO control measure or strategy with another. Additionally, the data are largely descriptive and quasi-experimental in design(311). Few reports described preemptive efforts or prospective studies to control MDROs before they had reached high levels within a unit or facility. Furthermore, small hospitals and LTCFs are infrequently represented in the literature.

A number of questions remain and are discussed below.

Impact on other MDROs from interventions targeted to one MDRO Only one report described control efforts directed at more than one MDRO, i.e., MDR-GNB and MRSA(312). Several reports have shown either decreases or increases in other pathogens with efforts to control one MDRO. For example, two reports on VRE control efforts demonstrated an increase in MRSA following the prioritization of VRE patients to private rooms and cohort beds(161). Similarly an outbreak of *Serratia marcescens* was temporally associated with a concurrent, but unrelated, outbreak of MRSA in an NICU(313). In contrast, Wright and colleagues reported a decrease in MRSA and VRE acquisition in an ICU during and after their successful effort to eradicate an MDR-strain of *A. baumannii* from the unit(210).

Colonization with multiple MDROs appears to be common(314, 315). One study found that nearly 50% of residents in a skilled-care unit in a LTCF were colonized with a target MDRO and that 26% were co-colonized with >1 MDRO; a detailed analysis showed that risk factors for colonization varied by pathogen(316). One review of the literature(317) reported that patient risk factors associated with colonization with MRSA, VRE, MDR-GNB, *C. difficile* and *Candida sp* were the same. This review concluded that control programs that focus on only one organism or one antimicrobial drug are unlikely to succeed because vulnerable patients will continue to serve as a magnet for other MDROs.

Costs. Several authors have provided evidence for the cost-effectiveness of approaches that use ASC(153, 191, 253, 318, 319). However, the supportive evidence often relied on assumptions, projections, and estimated attributable costs of MDRO infections. Similar limitations apply to a study suggesting that gown use yields a cost benefit in controlling transmission of VRE in ICUs(320). To date, no studies have directly compared the benefits and costs associated with different MDRO control strategies.

Feasibility. The subject of feasibility, as it applies to the extrapolation of results to other healthcare settings, has not been addressed. For example, smaller hospitals and LTCFs may lack the on-site laboratory services needed to obtain ASC in a timely manner. This factor could limit the applicability of an aggressive program based on obtaining ASC and preemptive placement of patients on Contact Precautions in these settings. However, with

the growing problem of antimicrobial resistance, and the recognized role of all healthcare settings for control of this problem, it is imperative that appropriate human and fiscal resources be invested to increase the feasibility of recommended control strategies in every setting.

Factors that influence selection of MDRO control measures. Although some common principles apply, the preceding literature review indicates that no single approach to the control of MDROs is appropriate for all healthcare facilities. Many factors influence the choice of interventions to be applied within an institution, including:

- **Type and significance of problem MDROs within the institution.** Many facilities have an MRSA problem while others have ESBL-producing *K. pneumoniae*. Some facilities have no VRE colonization or disease; others have high rates of VRE colonization without disease; and still others have ongoing VRE outbreaks. The magnitude of the problem also varies. Healthcare facilities may have very low numbers of cases, e.g., with a newly introduced strain, or may have prolonged, extensive outbreaks or colonization in the population. Between these extremes, facilities may have low or high levels of endemic colonization and variable levels of infection.
- **Population and healthcare-settings.** The presence of high-risk patients (e.g., transplant, hematopoietic stem-cell transplant) and special-care units (e.g. adult, pediatric, and neonatal ICUs; burn; hemodialysis) will influence surveillance needs and could limit the areas of a facility targeted for MDRO control interventions. Although it appears that MDRO transmission seldom occurs in ambulatory and outpatient settings, some patient populations (e.g., hemodialysis, cystic fibrosis) and patients receiving chemotherapeutic agents are at risk for colonization and infection with MDROs. Furthermore, the emergence of VRSA within the outpatient setting(22, 23, 25) demonstrates that even these settings need to make MDRO prevention a priority.

Differences of opinion on the optimal strategy to control MDROs. Published guidance on the control of MDROs reflects areas of ongoing debate on optimal control strategies. A key issue is the use of ASC in control efforts and preemptive use of Contact Precautions pending negative surveillance culture results(107, 321, 322). The various guidelines currently available exhibit a spectrum of approaches, which their authors deem to be evidence-based. One guideline for control of MRSA and VRE, the Society for Healthcare Epidemiology of America (SHEA) guideline from 2003(107), emphasizes routine use of ASC and Contact Precautions. That position paper does not address control of MDR-GNBs. The salient features of SHEA recommendations for MRSA and VRE control and the recommendations in this guideline for control of MDROs, including MRSA and VRE, have been compared(323); recommended interventions are similar. Other guidelines for VRE and MRSA, e.g., those proffered by the Michigan Society for Infection Control (www.msic-online.org/resource_sections/aro_guidelines), emphasize consistent practice of Standard Precautions and tailoring the use of ASC and Contact Precautions to local conditions, the specific MDROs that are prevalent and being transmitted, and the presence of risk factors for transmission. A variety of approaches have reduced MDRO rates(3, 164, 165, 209, 214, 240, 269, 324). Therefore, selection of interventions for controlling MDRO transmission should be based on assessments of the local problem, the prevalence of various MDRO and feasibility. Individual facilities should seek appropriate guidance and adopt effective measures that fit their circumstances and needs. Most studies have been in acute care settings; for non-acute care settings (e.g., LCTF, small rural hospitals), the optimal approach is not well defined.

Two-Tiered Approach for Control of MDROs. Reports describing successful control of MDRO transmission in healthcare facilities have included seven categories of interventions (Table 3). As a rule, these reports indicate that facilities confronted with an MDRO problem selected a combination of control measures, implemented them, and reassessed their impact. In some cases, new measures were added serially to further enhance control efforts. This evidence indicates that the control of MDROs is a dynamic process that requires a systematic approach tailored to the problem and healthcare setting. The nature of this evidence gave rise to the two-tiered approach to MDRO control

recommended in this guideline. This approach provides the flexibility needed to prevent and control MDRO transmission in every kind of facility addressed by this guideline. Detailed recommendations for MDRO control in all healthcare settings follow and are summarized in Table 3. Table 3, which applies to all healthcare settings, contains two tiers of activities. In the first tier are the baseline level of MDRO control activities designed to ensure recognition of MDROs as a problem, involvement of healthcare administrators, and provision of safeguards for managing unidentified carriers of MDROs.

With the emergence of an MDRO problem that cannot be controlled with the basic set of infection control measures, additional control measures should be selected from the second tier of interventions presented in Table 3. Decisions to intensify MDRO control activity arise from surveillance observations and assessments of the risk to patients in various settings. Circumstances that may trigger these decisions include:

- Identification of an MDRO from even one patient in a facility or special unit with a highly vulnerable patient population (e.g., an ICU, NICU, burn unit) that had previously not encountered that MDRO.
- Failure to decrease the prevalence or incidence of a specific MDRO (e.g., incidence of resistant clinical isolates) despite infection control efforts to stop its transmission. (Statistical process control charts or other validated methods that account for normal variation can be used to track rates of targeted MDROs)(205, 325, 326).

The combination of new or increased frequency of MDRO isolates and patients at risk necessitates escalation of efforts to achieve or re-establish control, i.e., to reduce rates of transmission to the lowest possible level. Intensification of MDRO control activities should begin with an assessment of the problem and evaluation of the effectiveness of measures in current use. Once the problem is defined, appropriate additional control measures should be selected from the second tier of Table 3. A knowledgeable infection prevention and control professional or healthcare epidemiologist should make this determination. This approach requires support from the governing body and medical staff of the facility. Once interventions are implemented, ongoing surveillance should be used to determine whether selected control measures are effective and if additional measures or consultation are

indicated. The result of this process should be to decrease MDRO rates to minimum levels. Healthcare facilities must not accept ongoing MDRO outbreaks or high endemic rates as the status quo. With selection of infection control measures appropriate to their situation, all facilities *can achieve* the desired goal and reduce the MDRO burden substantially.

V. Prevention of transmission of Multidrug Resistant Organisms (Table 3)

The CDC/HICPAC system for categorizing recommendations is as follows:

Category IA Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiologic studies.

Category IB Strongly recommended for implementation and supported by some experimental, clinical, or epidemiologic studies and a strong theoretical rationale.

Category IC Required for implementation, as mandated by federal and/or state regulation or standard.

Category II Suggested for implementation and supported by suggestive clinical or epidemiologic studies or a theoretical rationale.

No recommendation Unresolved issue. Practices for which insufficient evidence or no consensus regarding efficacy exists.

V.A. General recommendations for all healthcare settings independent of the prevalence of multidrug resistant organism (MDRO) infections or the population served.

V.A.1. Administrative measures

V.A.1.a. Make MDRO prevention and control an organizational patient safety priority.(3, 146, 151, 154, 182, 185, 194, 205, 208, 210, 242, 327, 328)

Category IB

V.A.1.b. Provide administrative support, and both fiscal and human resources, to prevent and control MDRO transmission within the healthcare organization (3, 9, 146, 152, 182-184, 208, 328, 329) *Category IB*

V.A.1.c. In healthcare facilities without expertise for analyzing epidemiologic data, recognizing MDRO problems, or devising effective control strategies (e.g., small or rural hospitals, rehabilitation centers, long-term care facilities [LTCFs], freestanding ambulatory centers), identify experts who can provide consultation as needed.(151, 188) *Category II*

V.A.1.d. Implement systems to communicate information about reportable MDROs [e.g., VRSA, VISA, MRSA, Penicillin resistant *S. pneumoniae*(PRSP)] to administrative personnel and as required by state and local health

- authorities (www.cdc.gov/epo/dphsi/nndsshis.htm). Refer to websites for updated requirements of local and state health departments. *Category II/IC*
- V.A.1.e. Implement a multidisciplinary process to monitor and improve healthcare personnel (HCP) adherence to recommended practices for Standard and Contact Precautions(3, 105, 182, 184, 189, 242, 273, 312, 330). *Category IB*
 - V.A.1.f. Implement systems to designate patients known to be colonized or infected with a targeted MDRO and to notify receiving healthcare facilities and personnel prior to transfer of such patients within or between facilities.(87, 151) *Category IB*
 - V.A.1.g. Support participation of the facility or healthcare system in local, regional, and national coalitions to combat emerging or growing MDRO problems.(41, 146, 151, 167, 188, 206, 207, 211, 331). *Category IB*
 - V.A.1.h. Provide updated feedback at least annually to healthcare providers and administrators on facility and patient-care-unit trends in MDRO infections. Include information on changes in prevalence or incidence of infection, results of assessments for system failures, and action plans to improve adherence to and effectiveness of recommended infection control practices to prevent MDRO transmission.(152, 154, 159, 184, 204, 205, 242, 312, 332) *Category IB*
 - V.A.2. Education and training of healthcare personnel
 - V.A.2.a. Provide education and training on risks and prevention of MDRO transmission during orientation and periodic educational updates for healthcare personnel; include information on organizational experience with MDROs and prevention strategies.(38, 152, 154, 173, 176, 189, 190, 203, 204, 217, 242, 330, 333, 334) *Category IB*
 - V.A.3. Judicious use of antimicrobial agents. The goal of the following recommendations is to ensure that systems are in place to promote optimal treatment of infections and appropriate antimicrobial use.
 - V.A.3.a. In hospitals and LTCFs, ensure that a multidisciplinary process is in place to review antimicrobial utilization, local susceptibility patterns

(antibiograms), and antimicrobial agents included in the formulary to foster appropriate antimicrobial use.(209, 212, 214, 215, 217, 242, 254, 334-339)

Category IB

V.A.3.b. Implement systems (e.g., computerized physician order entry, comment in microbiology susceptibility report, notification from a clinical pharmacist or unit director) to prompt clinicians to use the appropriate antimicrobial agent and regimen for the given clinical situation.(156, 157, 161, 166, 174, 175, 212, 214, 218, 254, 334, 335, 337, 340-346) *Category IB*

V.A.3.b.i. Provide clinicians with antimicrobial susceptibility reports and analysis of current trends, updated at least annually, to guide antimicrobial prescribing practices.(342, 347) *Category IB*

V.A.3.b.ii. In settings that administer antimicrobial agents but have limited electronic communication system infrastructures to implement physician prompts (e.g., LTCFs, home care and infusion companies), implement a process for appropriate review of prescribed antimicrobials. Prepare and distribute reports to prescribers that summarize findings and provide suggestions for improving antimicrobial use. (342, 348, 349) *Category II*

V.A.4. Surveillance

V.A.4.a. In microbiology laboratories, use standardized laboratory methods and follow published guidance for determining antimicrobial susceptibility of targeted (e.g., MRSA, VRE, MDR-ESBLs) and emerging (e.g., VRSA, MDR-*Acinetobacter baumannii*) MDROs.(8, 154, 177, 190, 193, 209, 254, 347, 350-353) *Category IB*

V.A.4.b. In all healthcare organizations, establish systems to ensure that clinical microbiology laboratories (in-house and out-sourced) promptly notify infection control staff or a medical director/ designee when a novel resistance pattern for that facility is detected.(9, 22, 154, 162, 169) *Category IB*

V.A.4.c. In hospitals and LTCFs, develop and implement laboratory protocols for storing isolates of selected MDROs for molecular typing when needed to

confirm transmission or delineate the epidemiology of the MDRO within the healthcare setting.(7, 8, 38, 140, 153, 154, 187, 190, 208, 217, 354, 355)

Category IB

- V.A.4.d. Prepare facility-specific antimicrobial susceptibility reports as recommended by the Clinical and Laboratory Standards Institute (CLSI) (www.phppo.cdc.gov/dls/master/default.aspx); monitor these reports for evidence of changing resistance patterns that may indicate the emergence or transmission of MDROs.(347, 351, 356, 357) *Category IB/IC*
 - V.A.4.d.i. In hospitals and LTCFs with special-care units (e.g., ventilator-dependent, ICU, or oncology units), develop and monitor unit-specific antimicrobial susceptibility reports.(358-361) *Category IB*
 - V.A.4.d.ii. Establish a frequency for preparing summary reports based on volume of clinical isolates, with updates at least annually.(347, 362) *Category II/IC*
 - V.A.4.d.iii. In healthcare organizations that outsource microbiology laboratory services (e.g., ambulatory care, home care, LTCFs, smaller acute care hospitals), specify by contract that the laboratory provide either facility-specific susceptibility data or local or regional aggregate susceptibility data in order to identify prevalent MDROs and trends in the geographic area served.(363) *Category II*
- V.A.4.e. Monitor trends in the incidence of target MDROs in the facility over time using appropriate statistical methods to determine whether MDRO rates are decreasing and whether additional interventions are needed.(152, 154, 183, 193, 205, 209, 217, 242, 300, 325, 326, 364, 365) *Category IA*
 - V.A.4.e.i. Specify isolate origin (i.e., location and clinical service) in MDRO monitoring protocols in hospitals and other large multi-unit facilities with high-risk patients.(8, 38, 152-154, 217, 358, 361) *Category IB*
 - V.A.4.e.ii. Establish a baseline (e.g., incidence) for targeted MDRO isolates by reviewing results of clinical cultures; if more timely or localized information is needed, perform baseline point prevalence studies of colonization in high-risk units. When possible, distinguish

colonization from infection in analysis of these data.(152, 153, 183, 184, 189, 190, 193, 205, 242, 365) *Category IB*

V.A.5. Infection control precautions to prevent transmission of MDROs

V.A.5.a. Follow Standard Precautions during all patient encounters in all settings in which healthcare is delivered.(119, 164, 255, 315, 316) *Category IB*

V.A.5.b. Use masks according to Standard Precautions when performing splash-generating procedures (e.g., wound irrigation, oral suctioning, intubation); when caring for patients with open tracheostomies and the potential for projectile secretions; and in circumstances where there is evidence of transmission from heavily colonized sources (e.g., burn wounds). Masks are not otherwise recommended for prevention of MDRO transmission from patients to healthcare personnel during routine care (e.g., upon room entry).(8, 22, 151, 152, 154, 189, 190, 193, 208, 240, 366) *Category IB*

V.A.5.c. Use of Contact Precautions

V.A.5.c.i. In *acute-care hospitals*, implement Contact Precautions routinely for all patients infected with target MDROs and for patients that have been previously identified as being colonized with target MDROs (e.g., patients transferred from other units or facilities who are known to be colonized). (11, 38, 68, 114, 151, 183, 188, 204, 217, 242, 304) *Category IB*

V.A.5.c.ii. In LTCFs, consider the individual patient's clinical situation and prevalence or incidence of MDRO in the facility when deciding whether to implement or modify Contact Precautions in addition to Standard Precautions for a patient infected or colonized with a target MDRO. *Category II*

V.A.5.c.ii.1. For relatively healthy residents (e.g., mainly independent) follow Standard Precautions, making sure that gloves and gowns are used for contact with uncontrolled secretions, pressure ulcers, draining wounds, stool incontinence, and ostomy tubes/bags. (78-80, 85, 151, 367, 368) *Category II*

- V.A.5.c.ii.2. For ill residents (e.g., those totally dependent upon healthcare personnel for healthcare and activities of daily living, ventilator-dependent) and for those residents whose infected secretions or drainage cannot be contained, use Contact Precautions in addition to Standard Precautions.(316, 369, 370) *Category II*
- V.A.5.c.iii. For MDRO colonized or infected patients without draining wounds, diarrhea, or uncontrolled secretions, establish ranges of permitted ambulation, socialization, and use of common areas based on their risk to other patients and on the ability of the colonized or infected patients to observe proper hand hygiene and other recommended precautions to contain secretions and excretions.(151, 163, 371) *Category II*
- V.A.5.d. In *ambulatory settings*, use Standard Precautions for patients known to be infected or colonized with target MDROs, making sure that gloves and gowns are used for contact with uncontrolled secretions, pressure ulcers, draining wounds, stool incontinence, and ostomy tubes and bags. *Category II*
- V.A.5.e. In *home care settings*
- Follow Standard Precautions making sure to use gowns and gloves for contact with uncontrolled secretions, pressure ulcers, draining wounds, stool incontinence, and ostomy tubes and bags. *Category II*
 - Limit the amount of reusable patient-care equipment that is brought into the home of patients infected or colonized with MDROs. When possible, leave patient-care equipment in the home until the patient is discharged from home care services. *Category II*
 - If noncritical patient-care equipment (e.g., stethoscopes) cannot remain in the home, clean and disinfect items before removing them from the home, using a low to intermediate level disinfectant, or place reusable items in a plastic bag for transport

to another site for subsequent cleaning and disinfection.

Category II

V.A.5.e.i. No recommendation is made for routine use of gloves, gowns, or both to prevent MDRO transmission in ambulatory or home care settings. *Unresolved issue*

V.A.5.e.ii. In *hemodialysis units*, follow the “Recommendations to Prevent Transmission of Infections in Chronic Hemodialysis Patients”(372)(www.cms.hhs.gov/home/regsguidance.asp).

Category IC

V.A.5.f. Discontinuation of Contact Precautions. No recommendation can be made regarding when to discontinue Contact Precautions. *Unresolved issue* (See Background for discussion of options)

V.A.5.g. Patient placement in hospitals and LTCFs

V.A.5.g.i. When single-patient rooms are available, assign priority for these rooms to patients with known or suspected MDRO colonization or infection. Give highest priority to those patients who have conditions that may facilitate transmission, e.g., uncontained secretions or excretions.(8, 38, 110, 151, 188, 208, 240, 304) *Category IB*

V.A.5.g.ii. When single-patient rooms are not available, cohort patients with the same MDRO in the same room or patient-care area.(8, 38, 92, 151-153, 162, 183, 184, 188, 217, 242, 304) *Category IB*

V.A.5.g.iii. When cohorting patients with the same MDRO is not possible, place MDRO patients in rooms with patients who are at low risk for acquisition of MDROs and associated adverse outcomes from infection and are likely to have short lengths of stay. *Category II*

V.A.6. Environmental measures

V.A.6.a. Clean and disinfect surfaces and equipment that may be contaminated with pathogens, including those that are in close proximity to the patient (e.g., bed rails, over bed tables) and frequently-touched surfaces in the patient care environment (e.g., door knobs, surfaces in and surrounding toilets in patients’ rooms) on a more frequent schedule compared to that for minimal

touch surfaces (e.g., horizontal surfaces in waiting rooms).(111, 297, 373)
Category IB

V.A.6.b. Dedicate noncritical medical items to use on individual patients known to be infected or colonized with MDROs.(38, 217, 324, 374, 375) *Category IB*

V.A.6.c. Prioritize room cleaning of patients on Contact Precautions. Focus on cleaning and disinfecting frequently touched surfaces (e.g., bedrails, bedside commodes, bathroom fixtures in the patient's room, doorknobs) and equipment in the immediate vicinity of the patient.(109, 110, 114-117, 297, 301, 373, 376, 377) *Category IB*

V.B. Intensified interventions to prevent MDRO transmission

The interventions presented below have been utilized in various combinations to reduce transmission of MDROs in healthcare facilities. Neither the effectiveness of individual components nor that of specific combinations of control measures has been assessed in controlled trials. Nevertheless, various combinations of control elements selected under the guidance of knowledgeable content experts have repeatedly reduced MDRO transmission rates in a variety of healthcare settings.

V.B.1. Indications and approach

V.B.1.a. Indications for intensified MDRO control efforts (VII.B.1.a.i and VII.B.1.a.ii) should result in selection and implementation of one or more of the interventions described in VII.B.2 to VII.B.8 below. Individualize the selection of control measures according to local considerations(8, 11, 38, 68, 114, 152-154, 183-185, 189, 190, 193, 194, 209, 217, 242, 312, 364, 365). *Category IB*

V.B.1.a.i. When incidence or prevalence of MDROs are not decreasing despite implementation of and correct adherence to the routine control measures described above, intensify MDRO control efforts by adopting one or more of the interventions described below.(92, 152, 183, 184, 193, 365) *Category IB*

V.B.1.a.ii. When the *first* case or outbreak of an epidemiologically important MDRO (e.g., VRE, MRSA, VISA, VRSA, MDR-GNB) is identified

within a healthcare facility or unit.(22, 23, 25, 68, 170, 172, 184, 240, 242, 378) *Category IB*

V.B.1.b. Continue to monitor the incidence of target MDRO infection and colonization after additional interventions are implemented. If rates do not decrease, implement more interventions as needed to reduce MDRO transmission.(11, 38, 68, 92, 152, 175, 184, 365) *Category IB*

V.B.2. Administrative measures

V.B.2.a. Identify persons with experience in infection control and the epidemiology of MDRO, either in house or through outside consultation, for assessment of the local MDRO problem and for the design, implementation, and evaluation of appropriate control measures (3, 68, 146, 151-154, 167, 184, 190, 193, 242, 328, 377). *Category IB*

V.B.2.b. Provide necessary leadership, funding, and day-to-day oversight to implement interventions selected. Involve the governing body and leadership of the healthcare facility or system that have organizational responsibility for this and other infection control efforts.(8, 38, 152, 154, 184, 189, 190, 208) *Category IB*

V.B.2.c. Evaluate healthcare system factors for their role in creating or perpetuating transmission of MDROs, including: staffing levels, education and training, availability of consumable and durable resources, communication processes, policies and procedures, and adherence to recommended infection control measures (e.g., hand hygiene and Standard or Contact Precautions). Develop, implement, and monitor action plans to correct system failures.(3, 8, 38, 152, 154, 172, 173, 175, 188, 196, 198, 199, 208, 217, 280, 324, 379, 380) *Category IB*

V.B.2.d. During the process, update healthcare providers and administrators on the progress and effectiveness of the intensified interventions. Include information on changes in prevalence, rates of infection and colonization; results of assessments and corrective actions for system failures; degrees of adherence to recommended practices; and action plans to improve

adherence to recommended infection control practices to prevent MDRO transmission.(152, 154, 159, 184, 204, 205, 312, 332, 381) *Category IB*

V.B.3. Educational interventions

Intensify the frequency of MDRO educational programs for healthcare personnel, especially those who work in areas in which MDRO rates are not decreasing. Provide individual or unit-specific feedback when available.(3, 38, 152, 154, 159, 170, 182, 183, 189, 190, 193, 194, 204, 205, 209, 215, 218, 312) *Category IB*

V.B.4. Judicious use of antimicrobial agents

Review the role of antimicrobial use in perpetuating the MDRO problem targeted for intensified intervention. Control and improve antimicrobial use as indicated. Antimicrobial agents that may be targeted include vancomycin, third-generation cephalosporins, and anti-anaerobic agents for VRE(217); third-generation cephalosporins for ESBLs(212, 214, 215); and quinolones and carbapenems(80, 156, 166, 174, 175, 209, 218, 242, 254, 329, 334, 335, 337, 341). *Category IB*

V.B.5. Surveillance

V.B.5.a. Calculate and analyze prevalence and incidence rates of targeted MDRO infection and colonization in populations at risk; when possible, distinguish colonization from infection(152, 153, 183, 184, 189, 190, 193, 205, 215, 242, 365). *Category IB*

V.B.5.a.i. Include only one isolate per patient, not multiple isolates from the same patient, when calculating rates(347, 382). *Category II*

V.B.5.a.ii. Increase the frequency of compiling and monitoring antimicrobial susceptibility summary reports for a targeted MDRO as indicated by an increase in incidence of infection or colonization with that MDRO. *Category II*

V.B.5.b. Develop and implement protocols to obtain active surveillance cultures (ASC) for targeted MDROs from patients in populations at risk (e.g., patients in intensive care, burn, bone marrow/stem cell transplant, and oncology units; patients transferred from facilities known to have high

MDRO prevalence rates; roommates of colonized or infected persons; and patients known to have been previously infected or colonized with an MDRO).(8, 38, 68, 114, 151-154, 167, 168, 183, 184, 187-190, 192, 193, 217, 242) *Category IB*

- V.B.5.b.i. Obtain ASC from areas of skin breakdown and draining wounds. In addition, include the following sites according to target MDROs:
 - V.B.5.b.i.1. For MRSA: Sampling the anterior nares is usually sufficient; throat, endotracheal tube aspirate, percutaneous gastrostomy sites, and perirectal or perineal cultures may be added to increase the yield. Swabs from several sites may be placed in the same selective broth tube prior to transport.(117, 383, 384) *Category IB*
 - V.B.5.b.i.2. For VRE: Stool, rectal, or perirectal samples should be collected.(154, 193, 217, 242)
Category IB
 - V.B.5.b.i.3. For MDR-GNB: Endotracheal tube aspirates or sputum should be cultured if a respiratory tract reservoir is suspected, (e.g., *Acinetobacter* spp., *Burkholderia* spp.).(385, 386) *Category IB*.
- V.B.5.b.ii. Obtain surveillance cultures for the target MDRO from patients at the time of admission to high-risk areas, e.g., ICUs, and at periodic intervals as needed to assess MDRO transmission.(8, 151, 154, 159, 184, 208, 215, 242, 387) *Category IB*
- V.B.5.c. Conduct culture surveys to assess the efficacy of the enhanced MDRO control interventions.
 - V.B.5.c.i. Conduct serial (e.g., weekly, until transmission has ceased and then decreasing frequency) unit-specific point prevalence culture surveys of the target MDRO to determine if transmission has decreased or ceased.(107, 167, 175, 184, 188, 218, 339) *Category IB*
 - V.B.5.c.ii. Repeat point-prevalence culture surveys at routine intervals or at time of patient discharge or transfer until transmission has ceased.(8, 152-154, 168, 178, 190, 215, 218, 242, 388) *Category IB*

- V.B.5.c.iii. If indicated by assessment of the MDRO problem, collect cultures to assess the colonization status of roommates and other patients with substantial exposure to patients with known MDRO infection or colonization.(25, 68, 167, 193) *Category IB*
- V.B.5.d. Obtain cultures of healthcare personnel for target MDRO when there is epidemiologic evidence implicating the healthcare staff member as a source of ongoing transmission.(153, 365) *Category IB*
- V.B.6. Enhanced infection control precautions
 - V.B.6.a. Use of Contact Precautions
 - V.B.6.a.i. Implement Contact Precautions routinely for all patients colonized or infected with a target MDRO.(8, 11, 38, 68, 114, 151, 154, 183, 188, 189, 217, 242, 304) *Category IA*
 - V.B.6.a.ii. Because environmental surfaces and medical equipment, especially those in close proximity to the patient, may be contaminated, don gowns and gloves *before or upon entry* to the patient's room or cubicle.(38, 68, 154, 187, 189, 242) *Category IB*
 - V.B.6.a.iii. In LTCFs, modify Contact Precautions to allow MDRO-colonized/infected patients whose site of colonization or infection can be appropriately contained and who can observe good hand hygiene practices to enter common areas and participate in group activities.(78, 86, 151, 367) *Category IB*
 - V.B.6.b. When ASC are obtained as part of an intensified MDRO control program, implement Contact Precautions until the surveillance culture is reported negative for the target MDRO.(8, 30, 153, 389, 390) *Category IB*
 - V.B.6.c. No recommendation is made regarding universal use of gloves, gowns, or both in high-risk units in acute-care hospitals.(153, 273, 312, 320, 391)
Unresolved issue
- V.B.7. Implement policies for patient admission and placement as needed to prevent transmission of a problem MDRO.(183, 184, 189, 193, 242, 339, 392)
Category IB

- V.B.7.a.i. Place MDRO patients in single-patient rooms.(6, 151, 158, 160, 166, 170, 187, 208, 240, 282, 393-395) *Category IB*
 - V.B.7.a.ii. Cohort patients with the same MDRO in designated areas (e.g., rooms, bays, patient care areas).(8, 151, 152, 159, 161, 176, 181, 183, 184, 188, 208, 217, 242, 280, 339, 344) *Category IB*
 - V.B.7.a.iii. When transmission continues despite adherence to Standard and Contact Precautions and cohorting patients, assign dedicated nursing and ancillary service staff to the care of MDRO patients only. Some facilities may consider this option when intensified measures are first implemented.(184, 217, 242, 278) *Category IB*
 - V.B.7.a.iv. Stop new admissions to the unit of facility if transmission continues despite the implementation of the enhanced control measures described above. (Refer to state or local regulations that may apply upon closure of hospital units or services.).(9, 38, 146, 159, 161, 168, 175, 205, 279, 280, 332, 339, 396) *Category IB*
- V.B.8. Enhanced environmental measures
- V.B.8.a. Implement patient-dedicated or single-use disposable noncritical equipment (e.g., blood pressure cuff, stethoscope) and instruments and devices.(38, 104, 151, 156, 159, 163, 181, 217, 324, 329, 367, 389, 390, 394) *Category IB*
 - V.B.8.b. Intensify and reinforce training of environmental staff who work in areas targeted for intensified MDRO control and monitor adherence to environmental cleaning policies. Some facilities may choose to assign dedicated staff to targeted patient care areas to enhance consistency of proper environmental cleaning and disinfection services.(38, 154, 159, 165, 172, 173, 175, 178-181, 193, 205, 208, 217, 279, 301, 327, 339, 397) *Category IB*
 - V.B.8.c. Monitor (i.e., supervise and inspect) cleaning performance to ensure consistent cleaning and disinfection of surfaces in close proximity to the patient and those likely to be touched by the patient and HCP (e.g.,

- bedrails, carts, bedside commodes, doorknobs, faucet handles).(8, 38, 109, 111, 154, 169, 180, 208, 217, 301, 333, 398) *Category IB*
- V.B.8.d. Obtain environmental cultures (e.g., surfaces, shared medical equipment) when there is epidemiologic evidence that an environmental source is associated with ongoing transmission of the targeted MDRO.(399-402) *Category IB*
- V.B.8.e. Vacate units for environmental assessment and intensive cleaning when previous efforts to eliminate environmental reservoirs have failed.(175, 205, 279, 339, 403) *Category II*
- V.B.9. Decolonization
- V.B.9.a. Consult with physicians with expertise in infectious diseases and/or healthcare epidemiology on a case-by-case basis regarding the appropriate use of decolonization therapy for patients or staff during limited periods of time, as a component of an intensified MRSA control program).(152, 168, 170, 172, 183, 194, 304) *Category II*
- V.B.9.b. When decolonization for MRSA is used, perform susceptibility testing for the decolonizing agent against the target organism in the individual being treated or the MDRO strain that is epidemiologically implicated in transmission. Monitor susceptibility to detect emergence of resistance to the decolonizing agent. Consult with a microbiologist for appropriate testing for mupirocin resistance, since standards have not been established.(289, 290, 304, 308) *Category IB*
- V.B.9.b.i. Because mupirocin-resistant strains may emerge and because it is unusual to eradicate MRSA when multiple body sites are colonized, do not use topical mupirocin *routinely* for MRSA decolonization of patients as a component of MRSA control programs in any healthcare setting.(289, 404) *Category IB*
- V.B.9.b.ii. Limit decolonization of HCP found to be colonized with MRSA to persons who have been epidemiologically linked as a likely source of ongoing transmission to patients. Consider reassignment of HCP

if decolonization is not successful and ongoing transmission to patients persists.(120, 122, 168) *Category IB*

- V.B.9.c. No recommendation can be made for decolonizing patients with VRE or MDR-GNB. Regimens and efficacy of decolonization protocols for VRE and MDR-GNB have not been established.(284, 286, 288, 307, 387, 405)
Unresolved issue

Glossary - Multidrug-Resistant Organisms

Ambulatory care settings. Facilities that provide health care to patients who do not remain overnight (e.g., hospital-based outpatient clinics, nonhospital-based clinics and physician offices, urgent care centers, surgicenters, free-standing dialysis centers, public health clinics, imaging centers, ambulatory behavioral health and substance abuse clinics, physical therapy and rehabilitation centers, and dental practices).

Cohorting. In the context of this guideline, this term applies to the practice of grouping patients infected or colonized with the same infectious agent together to confine their care to one area and prevent contact with susceptible patients (cohorting patients). During outbreaks, healthcare personnel may be assigned to a cohort of patients to further limit opportunities for transmission (cohorting staff).

Contact Precautions. Contact Precautions are a set of practices used to prevent transmission of infectious agents that are spread by direct or indirect contact with the patient or the patient's environment. Contact Precautions also apply where the presence of excessive wound drainage, fecal incontinence, or other discharges from the body suggest an increased transmission risk. A single patient room is preferred for patients who require Contact Precautions. When a single patient room is not available, consultation with infection control is helpful to assess the various risks associated with other patient placement options (e.g., cohorting, keeping the patient with an existing roommate). In multi-patient rooms, ≥ 3 feet spatial separation of between beds is advised to reduce the opportunities for inadvertent sharing of items between the infected/colonized patient and other patients. Healthcare personnel caring for patients on Contact Precautions wear a gown and gloves for all interactions that may involve contact with the patient or potentially contaminated areas in the patient's environment. Donning of gown and gloves upon room entry, removal before exiting the patient room and performance of hand hygiene immediately upon exiting are done to contain pathogens.

Epidemiologically important pathogens. Infectious agents that have one or more of the following characteristics: 1) A propensity for transmission within healthcare facilities based on published reports and the occurrence of temporal or geographic clusters of ≥ 2 patients, (e.g., VRE, MRSA and MSSA, *Clostridium difficile*, norovirus, RSV, influenza, rotavirus, *Enterobacter* spp; *Serratia* spp., group A streptococcus). However, for group A streptococcus, most experts consider a single case of healthcare-associated disease a trigger for investigation and enhanced control measures because of the devastating outcomes associated with HAI group A streptococcus infections. For susceptible bacteria that are known to be associated with asymptomatic colonization, isolation from normally sterile body fluids in patients with significant clinical disease would be the trigger to consider the organism as epidemiologically important. 2) Antimicrobial resistance implications:

- Resistance to first-line therapies (e.g., MRSA, VRE, VISA, VRSA, ESBL-producing organisms).
- Unusual or usual agents with unusual patterns of resistance within a facility, (e.g., the first isolate of *Burkholderia cepacia* complex or *Ralstonia* spp. in non-CF patients or a quinolone-resistant strain of *Pseudomonas* in a facility).
- Difficult to treat because of innate or acquired resistance to multiple classes of antimicrobial agents (e.g., *Stenotrophomonas maltophilia*, *Acinetobacter* spp.).

3) Associated with serious clinical disease, increased morbidity and mortality (e.g., MRSA and MSSA, group A streptococcus); or 4) A newly discovered or reemerging pathogen. The strategies described for MDROs may be applied for control of epidemiologically important organisms other than MDROs.

Hand hygiene. A general term that applies to any one of the following: 1) handwashing with plain (nonantimicrobial) soap and water); 2) antiseptic hand wash (soap containing antiseptic agents and water); 3) antiseptic hand rub (waterless antiseptic product, most often alcohol-based, rubbed on all surfaces of hands); or 4) surgical hand antisepsis

(antiseptic hand wash or antiseptic hand rub performed preoperatively by surgical personnel to eliminate transient hand flora and reduce resident hand flora).

Healthcare-associated infection (HAI). An infection that develops in a patient who is cared for in any setting where healthcare is delivered (e.g., acute care hospital, chronic care facility, ambulatory clinic, dialysis center, surgicenter, home) and is related to receiving health care (i.e., was not incubating or present at the time healthcare was provided). In ambulatory and home settings, HAI would apply to any infection that is associated with a medical or surgical intervention performed in those settings.

Healthcare epidemiologist A person whose primary training is medical (M.D., D.O.) and/or masters or doctorate-level epidemiology who has received advanced training in healthcare epidemiology. Typically these professionals direct or provide consultation to an infection prevention and control program in a hospital, long term care facility (LTCF), or healthcare delivery system (also see infection prevention and control professional).

Healthcare personnel (HCP). All paid and unpaid persons who work in a healthcare setting, also known as healthcare workers (e.g. any person who has professional or technical training in a healthcare-related field and provides patient care in a healthcare setting or any person who provides services that support the delivery of healthcare such as dietary, housekeeping, engineering, maintenance personnel).

Home care. A wide-range of medical, nursing, rehabilitation, hospice, and social services delivered to patients in their place of residence (e.g., private residence, senior living center, assisted living facility). Home health-care services include care provided by home health aides and skilled nurses, respiratory therapists, dietitians, physicians, chaplains, and volunteers; provision of durable medical equipment; home infusion therapy; and physical, speech, and occupational therapy.

Infection prevention and control professional (ICP). A person whose primary training is in either nursing, medical technology, microbiology, or epidemiology and who has acquired

specialized training in infection control. Responsibilities may include collection, analysis, and feedback of infection data and trends to healthcare providers; consultation on infection risk assessment, prevention and control strategies; performance of education and training activities; implementation of evidence-based infection control practices or those mandated by regulatory and licensing agencies; application of epidemiologic principles to improve patient outcomes; participation in planning renovation and construction projects (e.g., to ensure appropriate containment of construction dust); evaluation of new products or procedures on patient outcomes; oversight of employee health services related to infection prevention; implementation of preparedness plans; communication within the healthcare setting, with local and state health departments, and with the community at large concerning infection control issues; and participation in research.

Infection prevention and control program. A multidisciplinary program that includes a group of activities to ensure that recommended practices for the prevention of healthcare-associated infections are implemented and followed by healthcare personnel, making the healthcare setting safe from infection for patients and healthcare personnel. The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) requires the following five components of an infection prevention and control program for accreditation: 1) *surveillance*: monitoring patients and healthcare personnel for acquisition of infection and/or colonization; 2) *investigation*: identification and analysis of infection problems or undesirable trends; 3) *prevention*: implementation of measures to prevent transmission of infectious agents and to reduce risks for device- and procedure-related infections; 4) *control*: evaluation and management of outbreaks; and 5) *reporting*: provision of information to external agencies as required by state and federal law and regulation (www.jcaho.org). The infection prevention and control program staff has the ultimate authority to determine infection control policies for a healthcare organization with the approval of the organization's governing body.

Long-term care facilities (LTCFs). An array of residential and outpatient facilities designed to meet the bio-psychosocial needs of persons with sustained self-care deficits. These include skilled nursing facilities, chronic disease hospitals, nursing homes, foster and group homes, institutions for the developmentally disabled, residential care facilities, assisted

living facilities, retirement homes, adult day health care facilities, rehabilitation centers, and long-term psychiatric hospitals.

Mask. A term that applies collectively to items used to cover the nose and mouth and includes both procedure masks and surgical masks (www.fda.gov/cdrh/ode/guidance/094.html#4).

Multidrug-resistant organisms (MDROs). In general, bacteria (excluding *M. tuberculosis*) that are resistant to one or more classes of antimicrobial agents and usually are resistant to all but one or two commercially available antimicrobial agents (e.g., MRSA, VRE, extended spectrum beta-lactamase [ESBL]-producing or intrinsically resistant gram-negative bacilli).

Nosocomial infection. Derived from two Greek words “nosos” (disease) and “komeion” (to take care of). Refers to any infection that develops during or as a result of an admission to an acute care facility (hospital) and was not incubating at the time of admission.

Standard Precautions. A group of infection prevention practices that apply to all patients, regardless of suspected or confirmed diagnosis or presumed infection status. Standard Precautions are a combination and expansion of Universal Precautions and Body Substance Isolation. Standard Precautions are based on the principle that all blood, body fluids, secretions, excretions except sweat, nonintact skin, and mucous membranes may contain transmissible infectious agents. Standard Precautions includes hand hygiene, and depending on the anticipated exposure, use of gloves, gown, mask, eye protection, or face shield. Also, equipment or items in the patient environment likely to have been contaminated with infectious fluids must be handled in a manner to prevent transmission of infectious agents, (e.g. wear gloves for handling, contain heavily soiled equipment, properly clean and disinfect or sterilize reusable equipment before use on another patient).

1. IOM (1998), eds. Harrison, P. F. & Lederberg, J. (National Academy Press, Washington, DC), pp. 8-74.
2. Shlaes, D. M., Gerding, D. N., John, J. F., Jr., Craig, W. A., Bornstein, D. L., Duncan, R. A., Eckman, M. R., Farrer, W. E., Greene, W. H., Lorian, V., *et al.* (1997) *Infect Control Hosp Epidemiol* **18**, 275-291.
3. Larson, E. L., Early, E., Cloonan, P., Sugrue, S., & Parides, M. (2000) *Behav Med* **26**, 14-22.
4. Goldmann, D. A., Weinstein, R. A., Wenzel, R. P., Tablan, O. C., Duma, R. J., Gaynes, R. P., Schlosser, J., & Martone, W. J. (1996) *JAMA* **275**, 234-240.
5. Murthy, R. (2001) *Chest* **119**, 405S-411S.
6. Mahgoub, S., Ahmed, J., & Glatt, A. E. (2002) *Infect Control Hosp Epidemiol* **23**, 477-479.
7. Fournier, P. E. & Richet, H. (2006) *Clin Infect Dis* **42**, 692-699.
8. Fierobe, L., Lucet, J. C., Decre, D., Muller-Serieys, C., Deleuze, A., Joly-Guillou, M. L., Mantz, J., & Desmonts, J. M. (2001) *Infect Control Hosp Epidemiol* **22**, 35-40.
9. Ling, M. L., Ang, A., Wee, M., & Wang, G. C. (2001) *Infect Control Hosp Epidemiol* **22**, 48-49.
10. Landman, D., Quale, J. M., Mayorga, D., Adedeji, A., Vangala, K., Ravishankar, J., Flores, C., & Brooks, S. (2002) *Arch Intern Med* **162**, 1515-1520.
11. Urban, C., Segal-Maurer, S., & Rahal, J. J. (2003) *Clin Infect Dis* **36**, 1268-1274.
12. Gales, A. C., Jones, R. N., Forward, K. R., Linares, J., Sader, H. S., & Verhoef, J. (2001) *Clin Infect Dis* **32 Suppl 2**, S104-113.
13. del Toro, M. D., Rodriguez-Bano, J., Herrero, M., Rivero, A., Garcia-Ordenez, M. A., Corzo, J., & Perez-Cano, R. (2002) *Medicine (Baltimore)* **81**, 228-239.
14. Hanes, S. D., Demirkan, K., Tolley, E., Boucher, B. A., Croce, M. A., Wood, G. C., & Fabian, T. C. (2002) *Clin Infect Dis* **35**, 228-235.
15. Saiman, L. & Siegel, J. (2003) *Infect Control Hosp Epidemiol* **24**, S6-52.
16. Loukil, C., Saizou, C., Doit, C., Bidet, P., Mariani-Kurkdjian, P., Aujard, Y., Beaufils, F., & Bingen, E. (2003) *Infect Control Hosp Epidemiol* **24**, 707-710.
17. Ryan, M. P., Pembroke, J. T., & Adley, C. C. (2006) *J Hosp Infect* **62**, 278-284.
18. Fry, A. M., Udeagu, C. C., Soriano-Gabarro, M., Fridkin, S., Musinski, D., LaClaire, L., Elliott, J., Cook, D. J., Kornblum, J., Layton, M., *et al.* (2005) *Infect Control Hosp Epidemiol* **26**, 239-247.
19. Carter, R. J., Sorenson, G., Heffernan, R., Kiehlbauch, J. A., Kornblum, J. S., Leggiadro, R. J., Nixon, L. J., Wertheim, W. A., Whitney, C. G., & Layton, M. (2005) *Infect Control Hosp Epidemiol* **26**, 248-255.
20. Whitener, C. J., Park, S. Y., Browne, F. A., Parent, L. J., Julian, K., Bozdogan, B., Appelbaum, P. C., Chaitram, J., Weigel, L. M., Jernigan, J., *et al.* (2004) *Clin Infect Dis* **38**, 1049-1055.
21. CDC (1997) *MMWR Morb Mortal Wkly Rep* **46 (33)**, 765-766.
22. CDC (2002) *MMWR Morb Mortal Wkly Rep* **51 (26)**, 565-567.
23. CDC (2002) *MMWR - Morbidity & Mortality Weekly Report* **51(40)**, 902.
24. CDC (2004) *MMWR Morb Mortal Wkly Rep* **53**, 322-323.
25. Chang, S., Sievert, D. M., Hageman, J. C., Boulton, M. L., Tenover, F. C., Downes, F. P., Shah, S., Rudrik, J. T., Pupp, G. R., Brown, W. J., *et al.* (2003) *N Engl J Med* **348**, 1342-1347.

26. Fridkin, S. K., Hageman, J., McDougal, L. K., Mohammed, J., Jarvis, W. R., Perl, T. M., & Tenover, F. C. (2003) *Clin Infect Dis* **36**, 429-439.
27. Hageman, J. C., Fridkin, S. K., Mohammed, J. M., Steward, C. D., Gaynes, R. P., & Tenover, F. C. (2003) *Infect Control Hosp Epidemiol* **24**, 356-361.
28. Rotun, S. S., McMath, V., Schoonmaker, D. J., Maupin, P. S., Tenover, F. C., Hill, B. C., & Ackman, D. M. (1999) *Emerg Infect Dis* **5**, 147-149.
29. Smith, T. L., Pearson, M. L., Wilcox, K. R., Cruz, C., Lancaster, M. V., Robinson-Dunn, B., Tenover, F. C., Zervos, M. J., Band, J. D., White, E., *et al.* (1999) *N Engl J Med* **340**, 493-501.
30. Srinivasan, A., Dick, J. D., & Perl, T. M. (2002) *Clin Microbiol Rev* **15**, 430-438.
31. Gonzales, R. D., Schreckenberger, P. C., Graham, M. B., Kelkar, S., DenBesten, K., & Quinn, J. P. (2001) *Lancet* **357**, 1179.
32. Soltani, M., Beighton, D., Philpott-Howard, J., & Woodford, N. (2001) *Antimicrob Agents Chemother* **45**, 645-646.
33. Pai, M. P., Rodvold, K. A., Schreckenberger, P. C., Gonzales, R. D., Petrolatti, J. M., & Quinn, J. P. (2002) *Clin Infect Dis* **35**, 1269-1272.
34. Pillai, S. K., Sakoulas, G., Wennersten, C., Eliopoulos, G. M., Moellering, R. C., Jr., Ferraro, M. J., & Gold, H. S. (2002) *J Infect Dis* **186**, 1603-1607.
35. Hershberger, E., Donabedian, S., Konstantinou, K., & Zervos, M. J. (2004) *Clin Infect Dis* **38**, 92-98.
36. Mangili, A., Bica, I., Snyderman, D. R., & Hamer, D. H. (2005) *Clin Infect Dis* **40**, 1058-1060.
37. Sabol, K., Patterson, J. E., Lewis, J. S., 2nd, Owens, A., Cadena, J., & Jorgensen, J. H. (2005) *Antimicrob Agents Chemother* **49**, 1664-1665.
38. Simor, A. E., Lee, M., Vearncombe, M., Jones-Paul, L., Barry, C., Gomez, M., Fish, J. S., Cartotto, R. C., Palmer, R., & Louie, M. (2002) *Infect Control Hosp Epidemiol* **23**, 261-267.
39. Clarke, N. M., Patterson, J., & Lynch, I. J. (2003) *Curr Opin Crit Care* **9**, 413-423.
40. Martone, W. J. (1998) *Infect Control Hosp Epidemiol* **19**, 539-545.
41. The Brooklyn Antibiotic Task Force (2002) *Infect Control Hosp Epidemiol* **23**, 106-108.
42. Wilson, S. J., Knipe, C. J., Zieger, M. J., Gabehart, K. M., Goodman, J. E., Volk, H. M., & Sood, R. (2004) *Am J Infect Control* **32**, 342-344.
43. Qavi, A., Segal-Maurer, S., Mariano, N., Urban, C., Rosenberg, C., Burns, J., Chiang, T., Maurer, J., & Rahal, J. J. (2005) *Infect Control Hosp Epidemiol* **26**, 63-68.
44. Song, X., Srinivasan, A., Plaut, D., & Perl, T. M. (2003) *Infect Control Hosp Epidemiol* **24**, 251-256.
45. Aloush, V., Navon-Venezia, S., Seigman-Igra, Y., Cabili, S., & Carmeli, Y. (2006) *Antimicrob Agents Chemother* **50**, 43-48.
46. Cosgrove, S. E. (2006) *Clin Infect Dis* **42 Suppl 2**, S82-89.
47. Stone, P. W., Gupta, A., Loughrey, M., Della-Latta, P., Cimiotti, J., Larson, E., Rubenstein, D., & Saiman, L. (2003) *Infect Control Hosp Epidemiol* **24**, 601-606.
48. Cosgrove, S. E., Kaye, K. S., Eliopoulos, G. M., & Carmeli, Y. (2002) *Arch Intern Med* **162**, 185-190.
49. Linden, P. K., Pasculle, A. W., Manez, R., Kramer, D. J., Fung, J. J., Pinna, A. D., & Kusne, S. (1996) *Clin Infect Dis* **22**, 663-670.
50. Vergis, E. N., Hayden, M. K., Chow, J. W., Snyderman, D. R., Zervos, M. J., Linden, P. K., Wagener, M. M., Schmitt, B., & Muder, R. R. (2001) *Ann Intern Med* **135**, 484-492.
51. Salgado, C. D. & Farr, B. M. (2003) *Infect Control Hosp Epidemiol* **24**, 690-698.

52. DiazGranados, C. A. & Jernigan, J. A. (2005) *J Infect Dis* **191**, 588-595.
53. DiazGranados, C. A., Zimmer, S. M., Klein, M., & Jernigan, J. A. (2005) *Clin Infect Dis* **41**, 327-333.
54. Carmeli, Y., Eliopoulos, G., Mozaffari, E., & Samore, M. (2002) *Arch Intern Med* **162**, 2223-2228.
55. Davis, K. A., Stewart, J. J., Crouch, H. K., Florez, C. E., & Hospenthal, D. R. (2004) *Clin Infect Dis* **39**, 776-782.
56. Muder, R. R., Brennen, C., Wagener, M. M., Vickers, R. M., Rihs, J. D., Hancock, G. A., Yee, Y. C., Miller, J. M., & Yu, V. L. (1991) *Ann Intern Med* **114**, 107-112.
57. Cosgrove, S. E., Sakoulas, G., Perencevich, E. N., Schwaber, M. J., Karchmer, A. W., & Carmeli, Y. (2003) *Clin Infect Dis* **36**, 53-59.
58. Melzer, M., Eykyn, S. J., Gransden, W. R., & Chinn, S. (2003) *Clin Infect Dis* **37**, 1453-1460.
59. Selvey, L. A., Whitby, M., & Johnson, B. (2000) *Infect Control Hosp Epidemiol* **21**, 645-648.(s).
60. Romero-Vivas, J., Rubio, M., Fernandez, C., & Picazo, J. J. (1995) *Clin Infect Dis* **21**, 1417-1423.
61. Blot, S. I., Vandewoude, K. H., Hoste, E. A., & Colardyn, F. A. (2002) *Arch Intern Med* **162**, 2229-2235.
62. Reed, S. D., Friedman, J. Y., Engemann, J. J., Griffiths, R. I., Anstrom, K. J., Kaye, K. S., & al., e. (2005) *Infect Control Hosp Epidemiol* **26**, 175-183.
63. Mekontso-Dessap, A., Kirsch, M., Brun-Buisson, C., & Loisanche, D. (2001) *Clin Infect Dis* **32**, 877-883.
64. Engemann, J. J., Carmeli, Y., Cosgrove, S. E., Fowler, V. G., Bronstein, M. Z., Trivette, S. L., Briggs, J. P., Sexton, D. J., & Kaye, K. S. (2003) *Clin Infect Dis* **36**, 592-598.
65. Jones, R. N. (2006) *Clin Infect Dis* **42 Suppl 1**, S13-24.
66. Fowler, V. G., Jr., Sakoulas, G., McIntyre, L. M., Meka, V. G., Arbeit, R. D., Cabell, C. H., Stryjewski, M. E., Eliopoulos, G. M., Reller, L. B., Corey, G. R., *et al.* (2004) *J Infect Dis* **190**, 1140-1149.
67. Woods, C. W., Cheng, A. C., Fowler, V. G., Jr., Moorefield, M., Frederick, J., Sakoulas, G., Meka, V. G., Tenover, F. C., Zwadyk, P., & Wilson, K. H. (2004) *Clin Infect Dis* **38**, 1188-1191.
68. Jernigan, J. A., Clemence, M. A., Stott, G. A., Titus, M. G., Alexander, C. H., Palumbo, C. M., & Farr, B. M. (1995) *Infect Control Hosp Epidemiol* **16**, 686-696.
69. Stamm, A. M., Long, M. N., & Belcher, B. (1993) *Am J Infect Control* **21**, 70-74.
70. Harbarth, S., Albrich, W., Goldmann, D. A., & Huebner, J. (2001) *Lancet Infect Dis* **1**, 251-261.
71. Zinn, C. S., Westh, H., & Rosdahl, V. T. (2004) *Microb Drug Resist* **10**, 160-168.
72. Whitney, C. G., Farley, M. M., Hadler, J., Harrison, L. H., Lexau, C., Reingold, A., Lefkowitz, L., Cieslak, P. R., Cetron, M., Zell, E. R., *et al.* (2000) *N Engl J Med* **343**, 1917-1924.
73. Kollef, M. H. & Fraser, V. J. (2001) *Ann Intern Med* **134**, 298-314.
74. Fridkin, S. K. (2001) *Crit Care Med* **29**, N64-68.
75. Diekema, D. J., BootsMiller, B. J., Vaughn, T. E., Woolson, R. F., Yankey, J. W., Ernst, E. J., Flach, S. D., Ward, M. M., Franciscus, C. L., Pfaller, M. A., *et al.* (2004) *Clin Infect Dis* **38**, 78-85.

76. Polgreen, P. M., Beekmann, S. E., Chen, Y. Y., Doern, G. V., Pfaller, M. A., Brueggemann, A. B., Herwaldt, L. A., & Diekema, D. J. (2006) *Infect Control Hosp Epidemiol* **27**, 252-256.
77. Bradley, S. F., Terpenning, M. S., Ramsey, M. A., Zarins, L. T., Jorgensen, K. A., Sottile, W. S., Schaberg, D. R., & Kauffman, C. A. (1991) *Ann Intern Med* **115**, 417-422.
78. Brennen, C., Wagener, M. M., & Muder, R. R. (1998) *J Am Geriatr Soc* **46**, 157-160.
79. Strausbaugh, L. J., Crossley, K. B., Nurse, B. A., & Thrupp, L. D. (1996) *Infect Control Hosp Epidemiol* **17**, 129-140.
80. Bradley, S. F. (1999) *Infect Control Hosp Epidemiol* **20**, 362-366.
81. Bradley, S. F. (1999) *Am J Med* **106**, 2S-10S; discussion 48S-52S.
82. Wiener, J., Quinn, J. P., Bradford, P. A., Goering, R. V., Nathan, C., Bush, K., & Weinstein, R. A. (1999) *Jama* **281**, 517-523.
83. McNeil, S. A., Mody, L., & Bradley, S. F. (2002) *Geriatrics* **57**, 16-18, 21-14, 27.
84. Pacio, G. A., Visintainer, P., Maguire, G., Wormser, G. P., Raffalli, J., & Montecalvo, M. A. (2003) *Infect Control Hosp Epidemiol* **24**, 246-250.
85. Rahimi, A. R. (1998) *J Am Geriatr Soc* **46**, 1555-1557.
86. Trick, W. E., Weinstein, R. A., DeMarais, P. L., Tomaska, W., Nathan, C., McAllister, S. K., Hageman, J. C., Rice, T. W., Westbrook, G., & Jarvis, W. R. (2004) *J Am Geriatr Soc* **52**, 2003-2009.
87. Ben-Ami, R., Schwaber, M. J., Navon-Venezia, S., Schwartz, D., Giladi, M., Chmelnitsky, I., Leavitt, A., & Carmeli, Y. (2006) *Clin Infect Dis* **42**, 925-934.
88. Elizaga, M. L., Weinstein, R. A., & Hayden, M. K. (2002) *Clin Infect Dis* **34**, 441-446.
89. Saiman, L., Cronquist, A., Wu, F., Zhou, J., Rubenstein, D., Eisner, W., Kreiswirth, B. N., & Della-Latta, P. (2003) *Infect Control Hosp Epidemiol* **24**, 317-321.
90. Klevens, R. M., Edwards, J. R., Tenover, F. C., McDonald, L. C., Horan, T., & Gaynes, R. (2006) *Clin Infect Dis* **42**, 389-391.
91. Gaynes, R. & Edwards, J. R. (2005) *Clin Infect Dis* **41**, 848-854.
92. Boyce, J. M., Jackson, M. M., Pugliese, G., Batt, M. D., Fleming, D., Garner, J. S., Hartstein, A. I., Kauffman, C. A., Simmons, M., Weinstein, R., *et al.* (1994) *Infect Control Hosp Epidemiol* **15**, 105-115.
93. NNIS (2003) *Am J Infect Control* **31**, 481-498.
94. Fridkin, S. K., Edwards, J. R., Courval, J. M., Hill, H., Tenover, F. C., Lawton, R., Gaynes, R. P., & McGowan, J. E., Jr. (2001) *Ann Intern Med* **135**, 175-183.
95. Jones, R. N. (2001) *Chest* **119**, 397S-404S.
96. Neuhauser, M. M., Weinstein, R. A., Rydman, R., Danziger, L. H., Karam, G., & Quinn, J. P. (2003) *JAMA* **289**, 885-888.
97. Fridkin, S. K., Hill, H. A., Volkova, N. V., Edwards, J. R., Lawton, R. M., Gaynes, R. P., & McGowan, J. E., Jr. (2002) *Emerg Infect Dis* **8**, 697-701.
98. Madaras-Kelly, K. J., Remington, R. E., Lewis, P. G., & Stevens, D. L. (2006) *Infect Control Hosp Epidemiol* **27**, 155-169.
99. Fridkin, S. K., Hageman, J. C., Morrison, M., Sanza, L. T., Como-Sabetti, K., Jernigan, J. A., Harriman, K., Harrison, L. H., Lynfield, R., & Farley, M. M. (2005) *N Engl J Med* **352**, 1436-1444.
100. Kuehnert, M. J., Kruszon-Moran, D., Hill, H. A., McQuillan, G., McAllister, S. K., Fosheim, G., McDougal, L. K., Chaitram, J., Jensen, B., Fridkin, S. K., *et al.* (2006) *J Infect Dis* **193**, 172-179.

101. Bonten, M. J., Slaughter, S., Ambergen, A. W., Hayden, M. K., van Voorhis, J., Nathan, C., & Weinstein, R. A. (1998) *Arch Intern Med* **158**, 1127-1132.
102. Merrer, J., Santoli, F., Appere de Vecchi, C., Tran, B., De Jonghe, B., & Outin, H. (2000) *Infect Control Hosp Epidemiol* **21**, 718-723.
103. Lautenbach, E., Patel, J. B., Bilker, W. B., Edelstein, P. H., & Fishman, N. O. (2001) *Clin Infect Dis* **32**, 1162-1171.
104. Goetz, A. M., Rihs, J. D., Wagener, M. M., & Muder, R. R. (1998) *Am J Infect Control* **26**, 558-562.
105. Puzniak, L. A., Leet, T., Mayfield, J., Kollef, M., & Mundy, L. M. (2002) *Clin Infect Dis* **35**, 18-25.
106. CDC (2002) *MMWR* **51(16)**, 1-44.
107. Muto, C. A., Jernigan, J. A., Ostrowsky, B. E., Richet, H. M., Jarvis, W. R., Boyce, J. M., & Farr, B. M. (2003) *Infect Control Hosp Epidemiol* **24**, 362-386.
108. Almuneef, M. A., Baltimore, R. S., Farrel, P. A., Reagan-Cirincione, P., & Dembry, L. M. (2001) *Clin Infect Dis* **32**, 220-227.
109. Duckro, A. N., Blom, D. W., Lyle, E. A., Weinstein, R. A., & Hayden, M. K. (2005) *Arch Intern Med* **165**, 302-307.
110. Boyce, J. M., Potter-Bynoe, G., Chenevert, C., & King, T. (1997) *Infect Control Hosp Epidemiol* **18**, 622-627.(mj).
111. Bhalla, A., Pultz, N. J., Gries, D. M., Ray, A. J., Eckstein, E. C., Aron, D. C., & Donskey, C. J. (2004) *Infect Control Hosp Epidemiol* **25**, 164-167.
112. Larson, E. L., Cimiotti, J. P., Haas, J., Nesin, M., Allen, A., Della-Latta, P., & Saiman, L. (2005) *Pediatr Crit Care Med* **6**, 457-461.
113. Lee, Y. L., Cesario, T., Lee, R., Nothvogel, S., Nassar, J., Farsad, N., & Thrupp, L. (1994) *Am J Infect Control* **22**, 346-351.
114. Boyce, J. M., Opal, S. M., Chow, J. W., Zervos, M. J., Potter-Bynoe, G., Sherman, C. B., Romulo, R. L., Fortna, S., & Medeiros, A. A. (1994) *J Clin Microbiol* **32**, 1148-1153.
115. Gerding, D. N., Johnson, S., Peterson, L. R., Mulligan, M. E., & Silva, J., Jr. (1995) *Infect Control Hosp Epidemiol* **16**, 459-477.
116. Donskey, C. J. (2004) *Clin Infect Dis* **39**, 219-226.
117. Boyce, J. M., Havill, N. L., & Maria, B. (2005) *J Clin Microbiol* **43**, 5992-5995.
118. www.ihl.org/IHI/Programs/Campaign.
119. Evans, R., Lloyd, J. F., Abouzelof, R. H., Taylor, C. W., Anderson, V. R., & Samore, M. H. (2004) *Medinfo* **2004**, 212-216.
120. Boyce, J. M., Opal, S. M., Potter-Bynoe, G., & Medeiros, A. A. (1993) *Clin Infect Dis* **17**, 496-504.
121. Zawacki, A., O'Rourke, E., Potter-Bynoe, G., Macone, A., Harbarth, S., & Goldmann, D. (2004) *Infect Control Hosp Epidemiol* **25**, 1083-1089.
122. Faibis, F., Laporte, C., Fiacre, A., Delisse, C., Lina, G., Demachy, M.-C., & Botterel, F. (2005) *Infect Control Hosp Epidemiol* **26**, 213-215.
123. Sheretz, R. J., Reagan, D. R., Hampton, K. D., Robertson, K. L., Streed, S. A., Hoen, H. M., Thomas, R., & Gwaltney, J. M., Jr. (1996) *Ann Intern Med* **124**, 539-547.
124. Wang, J. T., Chang, S. C., Ko, W. J., Chang, Y. Y., Chen, M. L., Pan, H. J., & Luh, K. T. (2001) *J Hosp Infect* **47**, 104-109.
125. Herold, B. C., Immergluck, L. C., Maranan, M. C., Lauderdale, D. S., Gaskin, R. E., Boyle-Vavra, S., Leitch, C. D., & Daum, R. S. (1998) *JAMA* **279**, 593-598.

126. CDC (1999) *MMWR - Morbidity & Mortality Weekly Report* **48**, 707-710.
127. Fergie, J. E. & Purcell, K. (2001) *Pediatr Infect Dis J* **20**, 860-863.
128. Sattler, C. A., Mason, E. O., Jr., & Kaplan, S. L. (2002) *Pediatr Infect Dis J* **21**, 910-917.
129. Enright, M. C., Robinson, D. A., Randle, G., Feil, E. J., Grundmann, H., & Spratt, B. G. (2002) *Proc Natl Acad Sci U S A* **99**, 7687-7692.
130. Pan, E. S., Diep, B. A., Carleton, H. A., Charlebois, E. D., Sensabaugh, G. F., Haller, B. L., & Perdreau-Remington, F. (2003) *Clin Infect Dis* **37**, 1384-1388.
131. Daum, R. S., Ito, T., Hiramatsu, K., Hussain, F., Mongkolrattanothai, K., Jamklang, M., & Boyle-Vavra, S. (2002) *J Infect Dis* **186**, 1344-1347.
132. Said-Salim, B., Mathema, B., & Kreiswirth, B. N. (2003) *Infect Control Hosp Epidemiol* **24**, 451-455.
133. McDougal, L. K., Steward, C. D., Killgore, G. E., Chaitram, J. M., McAllister, S. K., & Tenover, F. C. (2003) *J Clin Microbiol* **41**, 5113-5120.
134. Zetola, N., Francis, J. S., Nuermberger, E. L., & Bishai, W. R. (2005) *Lancet Infect Dis* **5**, 275-286.
135. Adem, P. V., Montgomery, C. P., Husain, A. N., Koogler, T. K., Arangelovich, V., Humilier, M., Boyle-Vavra, S., & Daum, R. S. (2005) *N Engl J Med* **353**, 1245-1251.
136. Bocchini, C. E., Hulten, K. G., Mason, E. O., Jr., Gonzalez, B. E., Hammerman, W. A., & Kaplan, S. L. (2006) *Pediatrics* **117**, 433-440.
137. Healy, C. M., Hulten, K. G., Palazzi, D. L., Campbell, J. R., & Baker, C. J. (2004) *Clin Infect Dis* **39**, 1460-1466.
138. Saiman, L., O'keefe, M., Graham, P. L., Wu, F., Said-Salim, B., Kreiswirth, B., LaSala, A., Schlievert, P. M., & Della Latta, P. (2003) *Clin Infect Dis* **37**, 1313-1319.
139. Eckhardt, C., Halvosa, J. S., Ray, S. M., & Blumberg, H. M. (2003) *Infect Control Hosp Epidemiol* **24**, 460-461.
140. Seybold, U., Kourbatova, E. V., Johnson, J. G., Halvosa, S. J., Wang, Y. F., King, M. D., Ray, S. M., & Blumberg, H. M. (2006) *Clin Infect Dis* **42**, 647-656.
141. Berenholtz, S. M., Pronovost, P. J., Lipsett, P. A., Hobson, D., Earsing, K., Farley, J. E., Milanovich, S., Garrett-Mayer, E., Winters, B. D., Rubin, H. R., *et al.* (2004) *Crit Care Med* **32**, 2014-2020.
142. Coopersmith, C. M., Rebmann, T. L., Zack, J. E., Ward, M. R., Corcoran, R. M., Schallom, M. E., Sona, C. S., Buchman, T. G., Boyle, W. A., Polish, L. B., *et al.* (2002) *Crit Care Med* **30**, 59-64.
143. Babcock, H. M., Zack, J. E., Garrison, T., Trovillion, E., Jones, M., Fraser, V. J., & Kollef, M. H. (2004) *Chest* **125**, 2224-2231.
144. Warren, D. K., Zack, J. E., Cox, M. J., Cohen, M. M., & Fraser, V. J. (2003) *Crit Care Med* **31**, 1959-1963.
145. Eggimann, P., Hugonnet, S., Sax, H., Harbarth, S., Chevrolet, J. C., & Pittet, D. (2005) *Ann Intern Med* **142**, 875-876.
146. Verhoef, J., Beaujean, D., Blok, H., Baars, A., Meyler, A., van der Werken, C., & Weersink, A. (1999) *Eur J Clin Microbiol Infect Dis* **18**, 461-466.
147. Salmenlinna, S., Lyytikainen, O., Kotilainen, P., Scotford, R., Siren, E., & Vuopio-Varkila, J. (2000) *Eur J Clin Microbiol Infect Dis* **19**, 101-107.
148. Struelens, M. J., Ronveaux, O., Jans, B., & Mertens, R. (1996) *Infect Control Hosp Epidemiol* **17**, 503-508.

149. Voss, A., Milatovic, D., Wallrauch-Schwarz, C., Rosdahl, V. T., & Braveny, I. (1994) *Eur J Clin Microbiol Infect Dis* **13**, 50-55.
150. Rosdahl, V. T. & Knudsen, A. M. (1991) *Infect Control Hosp Epidemiol* **12**, 83-88.
151. Ostrowsky, B. E., Trick, W. E., Sohn, A. H., Quirk, S. B., Holt, S., Carson, L. A., Hill, B. C., Arduino, M. J., Kuehnert, M. J., & Jarvis, W. R. (2001) *N Engl J Med* **344**, 1427-1433.
152. Haley, R. W., Cushion, N. B., Tenover, F. C., Bannerman, T. L., Dryer, D., Ross, J., Sanchez, P. J., & Siegel, J. D. (1995) *J Infect Dis* **171**, 614-624.
153. Jernigan, J. A., Titus, M. G., Groschel, D. H., Getchell-White, S., & Farr, B. M. (1996) *Am J Epidemiol* **143**, 496-504.
154. Falk, P. S., Winnike, J., Woodmansee, C., Desai, M., & Mayhall, C. G. (2000) *Infect Control Hosp Epidemiol* **21**, 575-582.
155. Sherer, C. R., Sprague, B. M., Campos, J. M., Nambiar, S., Temple, R., Short, B., & Singh, N. (2005) *Emerg Infect Dis* **11**, 1470-1472.
156. Nourse, C., Byrne, C., Murphy, H., Kaufmann, M. E., Clarke, A., & Butler, K. (2000) *Epidemiol Infect* **124**, 53-59.
157. Rubin, L. G., Tucci, V., Cercenado, E., Eliopoulos, G., & Isenberg, H. D. (1992) *Infect Control Hosp Epidemiol* **13**, 700-705.
158. Karanfil, L. V., Murphy, M., Josephson, A., Gaynes, R., Mandel, L., Hill, B. C., & Swenson, J. M. (1992) *Infect Control Hosp Epidemiol* **13**, 195-200.
159. Hanna, H., Umphrey, J., Tarrand, J., Mendoza, M., & Raad, I. (2001) *Infect Control Hosp Epidemiol* **22**, 217-219.
160. Dembry, L. M., Uzokwe, K., & Zervos, M. J. (1996) *Infect Control Hosp Epidemiol* **17**, 286-292.
161. Bartley, P. B., Schooneveldt, J. M., Looke, D. F., Morton, A., Johnson, D. W., & Nimmo, G. R. (2001) *J Hosp Infect* **48**, 43-54.
162. Christiansen, K. J., Tibbett, P. A., Beresford, W., Pearman, J. W., Lee, R. C., Coombs, G. W., Kay, I. D., O'Brien, F. G., Palladino, S., Douglas, C. R., *et al.* (2004) *Infect Control Hosp Epidemiol* **25**, 384-390.
163. Armstrong-Evans, M., Litt, M., McArthur, M. A., Willey, B., Cann, D., Liska, S., Nusinowitz, S., Gould, R., Blacklock, A., Low, D. E., *et al.* (1999) *Infect Control Hosp Epidemiol* **20**, 312-317.
164. Webster, J., Faoagali, J. L., & Cartwright, D. (1994) *J Paediatr Child Health* **30**, 59-64.
165. Zafar, A. B., Butler, R. C., Reese, D. J., Gaydos, L. A., & Mennonna, P. A. (1995) *Am J Infect Control* **23**, 200-208.
166. Carrier, M., Marchand, R., Auger, P., Hebert, Y., Pellerin, M., Perrault, L. P., Cartier, R., Bouchard, D., Poirier, N., & Page, P. (2002) *J Thorac Cardiovasc Surg* **123**, 40-44.
167. Kotilainen, P., Routamaa, M., Peltonen, R., Evesti, P., Eerola, E., Salmenlinna, S., Vuopio-Varkila, J., & Rossi, T. (2001) *Arch Intern Med* **161**, 859-863.
168. Back, N. A., Linnemann, C. C., Jr., Staneck, J. L., & Kotagal, U. R. (1996) *Infect Control Hosp Epidemiol* **17**, 227-231.
169. Embil, J. M., McLeod, J. A., Al-Barrak, A. M., Thompson, G. M., Aoki, F. Y., Witwicki, E. J., Stranc, M. F., Kabani, A. M., Nicoll, D. R., & Nicolle, L. E. (2001) *Burns* **27**, 681-688.
170. Rao, N., Jacobs, S., & Joyce, L. (1988) *Infect Control Hosp Epidemiol* **9**, 255-260.
171. Kotilainen, P., Routamaa, M., Peltonen, R., Oksi, J., Rintala, E., Meurman, O., Lehtonen, O. P., Eerola, E., Salmenlinna, S., Vuopio-Varkila, J., *et al.* (2003) *Emerg Infect Dis* **9**, 169-175.
172. Cohen, S. H., Morita, M. M., & Bradford, M. (1991) *Am J Med* **91**, 233S-237S.

173. Adeyemi-Doro, F. A., Scheel, O., Lyon, D. J., & Cheng, A. F. (1997) *Infect Control Hosp Epidemiol* **18**, 765-767.
174. van der Zwet, W. C., Parlevliet, G. A., Savelkoul, P. H., Stoof, J., Kaiser, A. M., Koeleman, J. G., & Vandenbroucke-Grauls, C. M. (1999) *J Hosp Infect* **42**, 295-302.
175. Macrae, M. B., Shannon, K. P., Rayner, D. M., Kaiser, A. M., Hoffman, P. N., & French, G. L. (2001) *J Hosp Infect* **49**, 183-192.
176. Villari, P., Crispino, M., Salvadori, A., & Scarcella, A. (2001) *Infect Control Hosp Epidemiol* **22**, 630-634.
177. Paterson, D. L., Singh, N., Rihs, J. D., Squier, C., Rihs, B. L., & Muder, R. R. (2001) *Clin Infect Dis* **33**, 126-128.
178. Bukholm, G., Tannaes, T., Kjelsberg, A. B., & Smith-Erichsen, N. (2002) *Infect Control Hosp Epidemiol* **23**, 441-446.
179. Roberts, S. A., Findlay, R., & Lang, S. D. (2001) *J Hosp Infect* **48**, 228-232.
180. Hollander, R., Ebke, M., Barck, H., & von Pritzbuier, E. (2001) *J Hosp Infect* **48**, 207-213.
181. Podnos, Y. D., Cinat, M. E., Wilson, S. E., Cooke, J., Gornick, W., & Thrupp, L. D. (2001) *Surgical Infections* **2**, 297-301.
182. Pittet, D., Hugonnet, S., Harbarth, S., Mourouga, P., Sauvan, V., Touveneau, S., & Perneger, T. V. (2000) *Lancet* **356**, 1307-1312.
183. Murray-Leisure, K. A., Geib, S., Graceley, D., Rubin-Slutsky, A. B., Saxena, N., Muller, H. A., & Hamory, B. H. (1990) *Infect Control Hosp Epidemiol* **11**, 343-350.
184. Jochimsen, E. M., Fish, L., Manning, K., Young, S., Singer, D. A., Baker, R., & Jarvis, W. R. (1999) *Infect Control Hosp Epidemiol* **20**, 106-109.
185. Calfee, D. P. & Farr, B. M. (2002) *Infect Control Hosp Epidemiol* **23**, 407-410.
186. Scheckler, W. E., Brimhall, D., Buck, A. S., Farr, B. M., Friedman, C., Garibaldi, R. A., Gross, P. A., Harris, J. A., Hierholzer, W. J., Jr., Martone, W. J., *et al.* (1998) *Infect Control Hosp Epidemiol* **19**, 114-124.
187. Boyce, J. M., Mermel, L. A., Zervos, M. J., Rice, L. B., Potter-Bynoe, G., Giorgio, C., & Medeiros, A. A. (1995) *Infect Control Hosp Epidemiol* **16**, 634-637.
188. Nicolle, L. E., Dyck, B., Thompson, G., Roman, S., Kabani, A., Plourde, P., Fast, M., & Embil, J. (1999) *Infect Control Hosp Epidemiol* **20**, 202-205.
189. Lucet, J. C., Decre, D., Fichelle, A., Joly-Guillou, M. L., Pernet, M., Deblangy, C., Kosmann, M. J., & Regnier, B. (1999) *Clin Infect Dis* **29**, 1411-1418.
190. D'Agata, E. M., Thayer, V., & Schaffner, W. (2000) *Infect Control Hosp Epidemiol* **21**, 588-591.
191. Papia, G., Louie, M., Tralla, A., Johnson, C., Collins, V., & Simor, A. E. (1999) *Infect Control Hosp Epidemiol* **20**, 473-477.
192. Siddiqui, A. H., Harris, A. D., Hebden, J., Wilson, P. D., Morris, J. G., Jr., & Roghmann, M. C. (2002) *Am J Infect Control* **30**, 40-43.
193. Byers, K. E., Anglim, A. M., Anneski, C. J., Germanson, T. P., Gold, H. S., Durbin, L. J., Simonton, B. M., & Farr, B. M. (2001) *Infect Control Hosp Epidemiol* **22**, 140-147.
194. Harbarth, S., Martin, Y., Rohner, P., Henry, N., Auckenthaler, R., & Pittet, D. (2000) *J Hosp Infect* **46**, 43-49.
195. Curtis, J. R., Cook, D. J., Wall, R. J., Angus, D. C., Bion, J., Kacmarek, R., Kane-Gill, S. L., Kirchhoff, K. T., Levy, M., Mitchell, P. H., *et al.* (2006) *Crit Care Med* **34**, 211-218.
196. Arnow, P., Allyn, P. A., Nichols, E. M., Hill, D. L., Pezzlo, M., & Bartlett, R. H. (1982) *J Trauma* **22**, 954-959.

197. Fridkin, S. K., Pear, S. M., Williamson, T. H., Galgiani, J. N., & Jarvis, W. R. (1996) *Infect Control Hosp Epidemiol* **17**, 150-158.
198. Harbarth, S., Sudre, P., Dharan, S., Cadenas, M., & Pittet, D. (1999) *Infect Control Hosp Epidemiol* **20**, 598-603.
199. Vicca, A. F. (1999) *J Hosp Infect* **43**, 109-113.
200. Robert, J., Fridkin, S. K., Blumberg, H. M., Anderson, B., White, N., Ray, S. M., Chan, J., & Jarvis, W. R. (2000) *Infect Control Hosp Epidemiol* **21**, 12-17.(mj).
201. Jackson, M., Chiarello, L. A., Gaynes, R. P., & Gerberding, J. L. (2002) *Am J Infect Control* **30**, 199-206.
202. Grundmann, H., Hori, S., Winter, B., Tami, A., & Austin, D. J. (2002) *J Infect Dis* **185**, 481-488.
203. Dubbert, P. M., Dolce, J., Richter, W., Miller, M., & Chapman, S. W. (1990) *Infect Control Hosp Epidemiol* **11**, 191-193.
204. Nettleman, M. D., Trilla, A., Fredrickson, M., & Pfaller, M. (1991) *Am J Med* **91**, 228S-232S.
205. Curran, E. T., Benneyan, J. C., & Hood, J. (2002) *Infect Control Hosp Epidemiol* **23**, 13-18.
206. Gerber, S. I., Jones, R. C., Scott, M. V., Price, J. S., Dworkin, M. S., Filippell, M. B., Rearick, T., Pur, S. L., McAuley, J. B., Lavin, M. A., *et al.* (2006) *Infect Control Hosp Epidemiol* **27**, 139-145.
207. Chicago Antimicrobial Resistance Project.
208. Rampling, A., Wiseman, S., Davis, L., Hyett, A. P., Walbridge, A. N., Payne, G. C., & Cornaby, A. J. (2001) *J Hosp Infect* **49**, 109-116.
209. Rice, L. B., Eckstein, E. C., DeVente, J., & Shlaes, D. M. (1996) *Clin Infect Dis* **23**, 118-124.
210. Wright, M. O., Hebden, J. N., Harris, A. D., Shanholtz, C. B., Standiford, H. C., Furuno, J. P., & Perencevich, E. N. (2004) *Infect Control Hosp Epidemiol* **25**, 167-168.
211. Smith, D. L., Dushoff, J., Perencevich, E. N., Harris, A. D., & Levin, S. A. (2004) *Proc Natl Acad Sci U S A* **101**, 3709-3714.
212. Rahal, J. J., Urban, C., Horn, D., Freeman, K., Segal-Maurer, S., Maurer, J., Mariano, N., Marks, S., Burns, J. M., Dominick, D., *et al.* (1998) *JAMA* **280**, 1233-1237.
213. Rahal, J. J., Urban, C., & Segal-Maurer, S. (2002) *Clin Infect Dis* **34**, 499-503.
214. Meyer, K. S., Urban, C., Eagan, J. A., Berger, B. J., & Rahal, J. J. (1993) *Ann Intern Med* **119**, 353-358.
215. Pena, C., Pujol, M., Ardanuy, C., Ricart, A., Pallares, R., Linares, J., Ariza, J., & Gudiol, F. (1998) *Antimicrob Agents Chemother* **42**, 53-58.
216. Quale, J. M., Landman, D., Bradford, P. A., Visalli, M., Ravishankar, J., Flores, C., Mayorga, D., Vangala, K., & Adedeji, A. (2002) *Clin Infect Dis* **35**, 834-841.
217. Rupp, M. E., Marion, N., Fey, P. D., Bolam, D. L., Iwen, P. C., Overfelt, C. M., & Chapman, L. (2001) *Infect Control Hosp Epidemiol* **22**, 301-303.
218. Calil, R., Marba, S. T., von Nowakonski, A., & Tresoldi, A. T. (2001) *Am J Infect Control* **29**, 133-138.
219. McDonald, L. C. (2005) *Infect Control Hosp Epidemiol* **26**, 672-675.
220. Harbarth, S., Cosgrove, S., & Carmeli, Y. (2002) *Antimicrob Agents Chemother* **46**, 1619-1628.
221. Winston, L. G., Charlebois, E. D., Pang, S., Bangsberg, D. R., Perdreau-Remington, F., & Chambers, H. F. (2004) *Am J Infect Control* **32**, 462-469.
222. Brinsley, K., Srinivasan, A., Sinkowitz-Cochran, R., Lawton, R., McIntyre, R., Kravitz, G., Burke, B., Shadowen, R., & Cardo, D. (2005) *Am J Infect Control* **33**, 53-54.

223. Bruno-Murtha, L. A., Bruschi, J., Bor, D., Li, W., & Zucker, D. (2005) *Infect Control Hosp Epidemiol* **26**, 81-87.
224. Fridkin, S. K. (2003) *Clin Infect Dis* **36**, 1438-1444.
225. John, J. F., Jr. (2000) *Infect Control Hosp Epidemiol* **21**, 9-11.
226. McGowan, J. E., Jr. (2000) *Infect Control Hosp Epidemiol* **21**, S36-43.
227. Evans, R. S., Pestotnik, S. L., Classen, D. C., Clemmer, T. P., Weaver, L. K., Orme, J. F., Jr., Lloyd, J. F., & Burke, J. P. (1998) *N Engl J Med* **338**, 232-238.
228. Huskins, W. C. (2001) *Semin Pediatr Infect Dis* **12**, 138-146.
229. Mullett, C. J., Evans, R. S., Christenson, J. C., & Dean, J. M. (2001) *Pediatrics* **108**, E75.
230. Glowacki, R. C., Schwartz, D. N., Itokazu, G. S., Wisniewski, M. F., Kieszkowski, P., & Weinstein, R. A. (2003) *Clin Infect Dis* **37**, 59-64.
231. Parrino, T. A. (2005) *Pharmacotherapy* **25**, 289-298.
232. Paterson, D. L. (2006) *Clin Infect Dis* **42 Suppl 2**, S90-95.
233. Binkley, S., Fishman, N. O., LaRosa, L. A., Marr, A. M., Nachamkin, I., Wordell, D., Bilker, W. B., & Lautenbach, E. (2006) *Infect Control Hosp Epidemiol* **27**, 682-687.
234. McGowan, J. E., Jr. & Tenover, F. C. (2004) *Nat Rev Microbiol* **2**, 251-258.
235. Fridkin, S. K., Edwards, J. R., Tenover, F. C., Gaynes, R. P., & McGowan, J. E., Jr. (2001) *Clin Infect Dis* **33**, 324-330.
236. Foca, M., Jakob, K., Whittier, S., Della Latta, P., Factor, S., Rubenstein, D., & Saiman, L. (2000) *N Engl J Med* **343**, 695-700.
237. Huang (In press) *J Infect Dis*.
238. Gaynes, R. P. & Emori, T. G. (2001) in *Saunders Infection Control Reference Service*, eds. Abrutyn, E., Goldmann, D. A., & Scheckler, W. E. (W.B. Saunders Company, Philadelphia, PA), pp. 40-44.
239. Pottinger, J. M., Herwaldt, L. A., & Perl, T. M. (1997) *Infect Control Hosp Epidemiol* **18**, 513-527.
240. Hartstein, A. I., LeMonte, A. M., & Iwamoto, P. K. (1997) *Infect Control Hosp Epidemiol* **18**, 42-48.
241. Piagnerelli, M., Kennes, B., Brogniez, Y., Deplano, A., & Govaerts, D. (2000) *Infect Control Hosp Epidemiol* **21**, 651-653.
242. Montecalvo, M. A., Jarvis, W. R., Uman, J., Shay, D. K., Petrullo, C., Rodney, K., Gedris, C., Horowitz, H. W., & Wormser, G. P. (1999) *Ann Intern Med* **131**, 269-272.
243. Talon, D. R. & Bertrand, X. (2001) *Infect Control Hosp Epidemiol* **22**, 505-509.
244. Lucet, J. C., Grenet, K., Armand-Lefevre, L., Harnal, M., Bouvet, E., Regnier, B., & al., e. (2005) *Infect Control Hosp Epidemiol* **26**.
245. Troche, G., Joly, L. M., Guibert, M., & Zazzo, J. F. (2005) *Infect Control Hosp Epidemiol* **26**, 161-165.
246. Nijssen, S., Bonten, M. J., & Weinstein, R. A. (2005) *Clin Infect Dis* **40**, 405-409.
247. Cooper, B. S., Stone, S. P., Kibbler, C. C., Cookson, B. D., Roberts, J. A., Medley, G. F., Duckworth, G., Lai, R., & Ebrahim, S. (2004) *Bmj* **329**, 533.
248. Perencevich, E. N., Fisman, D. N., Lipsitch, M., Harris, A. D., Morris, J. G., Jr., & Smith, D. L. (2004) *Clin Infect Dis* **38**, 1108-1115.
249. Bootsma, M. C., Diekmann, O., & Bonten, M. J. (2006) *Proc Natl Acad Sci U S A* **103**, 5620-5625.
250. Gardam, M. A., Burrows, L. L., Kus, J. V., Brunton, J., Low, D. E., Conly, J. M., & Humar, A. (2002) *J Infect Dis* **186**, 1754-1760.

251. Thouverez, M., Talon, D., & Bertrand, X. (2004) *Infect Control Hosp Epidemiol* **25**, 838-841.
252. Armeanu, E. & Bonten, M. J. (2005) *Clin Infect Dis* **41**, 210-216.
253. Muto, C. A., Giannetta, E. T., Durbin, L. J., Simonton, B. M., & Farr, B. M. (2002) *Infect Control Hosp Epidemiol* **23**, 429-435.
254. Morris, J. G., Jr., Shay, D. K., Hebden, J. N., McCarter, R. J., Jr., Perdue, B. E., Jarvis, W., Johnson, J. A., Dowling, T. C., Polish, L. B., & Schwalbe, R. S. (1995) *Ann Intern Med* **123**, 250-259.
255. Furuno, J. P., McGregor, J. C., Harris, A. D., Johnson, J. A., Johnson, J. K., Langenberg, P., Venezia, R. A., Finkelstein, J., Smith, D. L., Strauss, S. M., et al. (2006) *Arch Intern Med* **166**, 580-585.
256. Harbarth, S., Sax, H., Fankhauser-Rodriguez, C., Schrenzel, J., Agostinho, A., & Pittet, D. (2006) *Am J Med* **119**, 275 e215-223.
257. Lee, T. A., Hacek, D. M., Stroupe, K. T., Collins, S. M., & Peterson, L. R. (2005) *Infect Control Hosp Epidemiol* **26**, 39-46.
258. Manian, F. A., Senkel, D., Zack, J., & Meyer, L. (2002) *Infect Control Hosp Epidemiol* **23**, 516-519.
259. Troillet, N., Carmeli, Y., Samore, M. H., Dakos, J., Eichelberger, K., DeGirolami, P. C., & Karchmer, A. W. (1998) *Infect Control Hosp Epidemiol* **19**, 181-185.
260. Sanford, M. D., Widmer, A. F., Bale, M. J., Jones, R. N., & Wenzel, R. P. (1994) *Clin Infect Dis* **19**, 1123-1128.
261. Lucet, J. C., Chevret, S., Durand-Zaleski, I., Chastang, C., & Regnier, B. (2003) *Arch Intern Med* **163**, 181-188.
262. D'Agata, E. M., et al. (2002) *Clin Infect Dis* **34**, 167-172.
263. Flayhart, D., Hindler, J. F., Bruckner, D. A., Hall, G., Shrestha, R. K., Vogel, S. A., Richter, S. S., Howard, W., Walther, R., & Carroll, K. C. (2005) *J Clin Microbiol* **43**, 5536-5540.
264. Perry, J. D., Davies, A., Butterworth, L. A., Hopley, A. L., Nicholson, A., & Gould, F. K. (2004) *J Clin Microbiol* **42**, 4519-4523.
265. Harbarth, S., Masuet-Aumatell, C., Schrenzel, J., Francois, P., Akakpo, C., Renzi, G., Pugin, J., Ricou, B., & Pittet, D. (2006) *Crit Care* **10**, R25.
266. Huletsky, A., Lebel, P., Picard, F. J., Bernier, M., Gagnon, M., Boucher, N., & Bergeron, M. G. (2005) *Clin Infect Dis* **40**, 976-981.
267. Warren, D. K., Liao, R. S., Merz, L. R., Eveland, M., & Dunne, W. M., Jr. (2004) *J Clin Microbiol* **42**, 5578-5581.
268. Palladino, S., Kay, I. D., Flexman, J. P., Boehm, I., Costa, A. M., Lambert, E. J., & Christiansen, K. J. (2003) *J Clin Microbiol* **41**, 2483-2486.
269. Fazal, B. A., Telzak, E. E., Blum, S., Turett, G. S., Petersen-Fitzpatrick, F. E., & Lorian, V. (1996) *Infect Control Hosp Epidemiol* **17**, 372-374.
270. Toltzis, P., Hoyen, C., & et al. (1999) *Pediatrics* **103** (4 Pt1), 719-723.
271. Weinstein, R. A. & Kabins, S. A. (1981) *Am J Med* **70**, 449-454.
272. Kim, P. W., Roghmann, M. C., Perencevich, E. N., & Harris, A. D. (2003) *Am J Infect Control* **31**, 97-103.
273. Slaughter, S., Hayden, M. K., Nathan, C., Hu, T. C., Rice, T., Van Voorhis, J., Matushek, M., Franklin, C., & Weinstein, R. A. (1996) *Ann Intern Med* **125**, 448-456.
274. CDC (1995) *MMWR Recomm Rep* **44** (RR-12), 1-13.
275. Evans, M. R., Meldrum, R., Lane, W., Gardner, D., Ribeiro, C. D., Gallimore, C. I., & Westmoreland, D. (2002) *Epidemiol Infect* **129**, 355-360.

276. Hall, C. B., Douglas, R. G., Jr., Schnabel, K. C., & Geiman, J. M. (1981) *Infect Immun* **33**, 779-783.
277. Wu, H. M., Fornek, M., Kellogg, J. S., Chapin, A. R., Gibson, K., Schwab, E., Spencer, C., & Henning, K. (2005) *Infect Control Hosp Epidemiol* **26**, 802-810.
278. Austin, D. J., Bonten, M. J., Weinstein, R. A., Slaughter, S., & Anderson, R. M. (1999) *Proc Natl Acad Sci U S A* **96**, 6908-6913.
279. Law, M. R., Gill, O. N., & Turner, A. (1988) *Epidemiol Infect* **101**, 301-309.
280. Ruchel, R., Mergeryan, H., Boger, O., Langefeld, C., & Witte, W. (1999) *Infect Control Hosp Epidemiol* **20**, 353-355.
281. Cepeda, J. A., Whitehouse, T., Cooper, B., Hails, J., Jones, K., Kwaku, F., Taylor, L., Hayman, S., Cookson, B., Shaw, S., *et al.* (2005) *Lancet* **365**, 295-304.
282. Mulin, B., Rouget, C., Clement, C., Bailly, P., Julliot, M. C., Viel, J. F., Thouverez, M., Vieille, I., Barale, F., & Talon, D. (1997) *Infect Control Hosp Epidemiol* **18**, 499-503.
283. Nouwen, J. L., Ott, A., Kluytmans-Vandenbergh, M. F., Boelens, H. A., Hofman, A., van Belkum, A., & Verbrugh, H. A. (2004) *Clin Infect Dis* **39**, 806-811.
284. Byers, K. E., Anglim, A. M., Anneski, C. J., & Farr, B. M. (2002) *Infect Control Hosp Epidemiol* **23**, 207-211.
285. Baden, L. R., Thiemke, W., Skolnik, A., Chambers, R., Strymish, J., Gold, H. S., Moellering, R. C., Jr., & Eliopoulos, G. M. (2001) *Clin Infect Dis* **33**, 1654-1660.
286. Donskey, C. J., Huyen, C. K., Das, S. M., Helfand, M. S., & Hecker, M. T. (2002) *Infect Control Hosp Epidemiol* **23**, 436-440.
287. Ridenour, G. A., Wong, E. S., Call, M. A., & Climo, M. W. (2006) *Infect Control Hosp Epidemiol* **27**, 271-278.
288. Scanvic, A., Denic, L., Gaillon, S., Giry, P., Andremont, A., & Lucet, J. C. (2001) *Clin Infect Dis* **32**, 1393-1398.
289. Kauffman, C. A., Terpenning, M. S., He, X., Zarins, L. T., Ramsey, M. A., Jorgensen, K. A., Sottile, W. S., & Bradley, S. F. (1993) *Am J Med* **94**, 371-378.
290. Strausbaugh, L. J., Jacobson, C., Sewell, D. L., Potter, S., & Ward, T. T. (1992) *Infect Control Hosp Epidemiol* **13**, 151-159.
291. Kirkland, K. B. & Weinstein, J. M. (1999) *Lancet* **354**, 1177-1178.
292. Saint, S., Higgins, L. A., Nallamotheu, B. K., & Chenoweth, C. (2003) *Am J Infect Control* **31**, 354-356.
293. Evans, H. L., Shaffer, M. M., Hughes, M. G., Smith, R. L., Chong, T. W., Raymond, D. P., Pelletier, S. J., Pruett, T. L., & Sawyer, R. G. (2003) *Surgery* **134**, 180-188.
294. Catalano, G., Houston, S. H., Catalano, M. C., Butera, A. S., Jennings, S. M., Hakala, S. M., Burrows, S. L., Hickey, M. G., Duss, C. V., Skelton, D. N., *et al.* (2003) *South Med J* **96**, 141-145.
295. Tarzi, S., Kennedy, P., Stone, S., & Evans, M. (2001) *J Hosp Infect* **49**, 250-254.
296. Stelfox, H. T., Bates, D. W., & Redelmeier, D. A. (2003) *JAMA* **290**, 1899-1905.
297. Hota, B. (2004) *Clin Infect Dis* **39**, 1182-1189.
298. Martinez, J. A., Ruthazer, R., Hansjosten, K., Barefoot, L., & Snyderman, D. R. (2003) *Arch Intern Med* **163**, 1905-1912.
299. CDC (2003) *MMWR* **52(RR10);1-42**.
300. Simor, A. E. (2001) *Infect Control Hosp Epidemiol* **22**, 459-463.
301. Hayden, M. K., Bonten, M. J., Blom, D. W., Lyle, E. A., van de Vijver, D. A., & Weinstein, R. A. (2006) *Clin Infect Dis* **42**, 1552-1560.

302. Lai, K. K., Kelley, A. L., Melvin, Z. S., Belliveau, P. P., & Fontecchio, S. A. (1998) *Infect Control Hosp Epidemiol* **19**, 647-652.
303. Boyce, J. M. (2001) *J Hosp Infect* **48 Suppl A**, S9-14.
304. Montesinos, I., Salido, E., Delgado, T., Lecuona, M., & Sierra, A. (2003) *Infect Control Hosp Epidemiol* **24**, 667-672.
305. Chen, S. F. (2005) *Pediatr Infect Dis J* **24**, 79-80.
306. Kaplan, S. L. (2005) *Pediatr Infect Dis J* **24**, 457-458.
307. Loeb, M., Main, C., Walker-Dilks, C., & Eady, A. (2003) *Cochrane Database Syst Rev*, CD003340.
308. Deshpande, L. M., Fix, A. M., Pfaller, M. A., & Jones, R. N. (2002) *Diagn Microbiol Infect Dis* **42**, 283-290.
309. Mody, L., Kauffman, C. A., McNeil, S. A., Galecki, A. T., & Bradley, S. F. (2003) *Clin Infect Dis* **37**, 1467-1474.
310. Walker, E. S., Vasquez, J. E., Dula, R., Bullock, H., & Sarubbi, F. A. (2003) *Infect Control Hosp Epidemiol* **24**, 342-346.
311. Harris, A. D., Bradham, D. D., Baumgarten, M., Zuckerman, I. H., Fink, J. C., & Perencevich, E. N. (2004) *Clin Infect Dis* **38**, 1586-1591.
312. Eveillard, M., Eb, F., Tramier, B., Schmit, J. L., Lescure, F. X., Biendo, M., Canarelli, B., Daoudi, F., Laurans, G., Rousseau, F., *et al.* (2001) *J Hosp Infect* **47**, 116-124.
313. Campbell, J. R., Zaccaria, E., Mason, E. O., Jr., & Baker, C. J. (1998) *Infect Control Hosp Epidemiol* **19**, 924-928.
314. Harris, A. D., Nemoy, L., Johnson, J. A., Martin-Carnahan, A., Smith, D. L., Standiford, H., & Perencevich, E. N. (2004) *Infect Control Hosp Epidemiol* **25**, 105-108.
315. Warren, D. K., Nitin, A., Hill, C., Fraser, V. J., & Kollef, M. H. (2004) *Infect Control Hosp Epidemiol* **25**, 99-104.
316. Trick, W. E., Weinstein, R. A., DeMarais, P. L., Kuehnert, M. J., Tomaska, W., Nathan, C., Rice, T. W., McAllister, S. K., Carson, L. A., & Jarvis, W. R. (2001) *J Am Geriatr Soc* **49**, 270-276.
317. Safdar, N. & Maki, D. G. (2002) *Ann Intern Med* **136**, 834-844.
318. Montecalvo, M. A., Jarvis, W. R., Uman, J., Shay, D. K., Petrullo, C., Horowitz, H. W., & Wormser, G. P. (2001) *Infect Control Hosp Epidemiol* **22**, 437-442.
319. Rubinovitch, B. & Pittet, D. (2001) *J Hosp Infect* **47**, 9-18.
320. Puzniak, L. A., Gillespie, K. N., Leet, T., Kollef, M., & Mundy, L. M. (2004) *Infect Control Hosp Epidemiol* **25**, 418-424.
321. Cookson, B. (1997) *Bmj* **314**, 664-665.
322. Farr, B. M. & Jarvis, W. R. (2002) *Infect Control Hosp Epidemiol* **23**, 65-68.
323. Strausbaugh, L. J., Siegel, J. D., & Weinstein, R. A. (2006) *Clin Infect Dis* **42**, 828-835.
324. Brooks, S., Khan, A., Stoica, D., Griffith, J., Friedeman, L., Mukherji, R., Hameed, R., & Schupf, N. (1998) *Infect Control Hosp Epidemiol* **19**, 333-336.
325. Benneyan, J. C., Lloyd, R. C., & Plsek, P. E. (2003) *Qual Saf Health Care* **12**, 458-464.
326. Gustafson, T. L. (2000) *Am J Infect Control* **28**, 406-414.
327. Aubry-Damon, H., Legrand, P., Brun-Buisson, C., Astier, A., Soussy, C. J., & Leclercq, R. (1997) *Clin Infect Dis* **25**, 647-653.
328. Cooper, B. S., Medley, G. F., Stone, S. P., Kibbler, C. C., Cookson, B. D., Roberts, J. A., Duckworth, G., Lai, R., & Ebrahim, S. (2004) *Proc Natl Acad Sci U S A* **101**, 10223-10228.

329. Brown, A. R., Amyes, S. G., Paton, R., Plant, W. D., Stevenson, G. M., Winney, R. J., & Miles, R. S. (1998) *J Hosp Infect* **40**, 115-124.
330. Cromer, A. L., Hutsell, S. O., Latham, S. C., Bryant, K. G., Wacker, B. B., Smith, S. A., Bendyk, H. A., Valainis, G. T., & Carney, M. C. (2004) *Am J Infect Control* **32**, 451-455.
331. Pittsburgh Regional Project.
332. Assadian, O., Berger, A., Aspöck, C., Mustafa, S., Kohlhauser, C., & Hirschl, A. M. (2002) *Infect Control Hosp Epidemiol* **23**, 457-461.
333. Byers, K. E., Durbin, L. J., Simonton, B. M., Anglim, A. M., Adal, K. A., & Farr, B. M. (1998) *Infect Control Hosp Epidemiol* **19**, 261-264.
334. Patterson, J. E., Hardin, T. C., Kelly, C. A., Garcia, R. C., & Jorgensen, J. H. (2000) *Infect Control Hosp Epidemiol* **21**, 455-458.
335. Bantar, C., Sartori, B., Vesco, E., Heft, C., Saul, M., Salamone, F., & Oliva, M. E. (2003) *Clin Infect Dis* **37**, 180-186.
336. Bisson, G., Fishman, N. O., Patel, J. B., Edelstein, P. H., & Lautenbach, E. (2002) *Infect Control Hosp Epidemiol* **23**, 254-260.
337. Carling, P., Fung, T., Killion, A., Terrin, N., & Barza, M. (2003) *Infect Control Hosp Epidemiol* **24**, 699-706.
338. Quale, J., Landman, D., Saurina, G., Atwood, E., DiTore, V., & Patel, K. (1996) *Clin Infect Dis* **23**, 1020-1025.
339. Sample, M. L., Gravel, D., Oxley, C., Toye, B., Garber, G., & Ramotar, K. (2002) *Infect Control Hosp Epidemiol* **23**, 468-470.
340. Burke, J. P. & Pestotnik, S. L. (1999) *J Chemother* **11**, 530-535.
341. Cooper, E., Paull, A., & O'Reilly, M. (2002) *Infect Control Hosp Epidemiol* **23**, 151-153.
342. Lagerlov, P., Loeb, M., Andrew, M., & Hjortdahl, P. (2000) *Qual Health Care* **9**, 159-165.
343. Lemmen, S. W., Zollmann, D., Gastmeier, P., & Lutticken, R. (2001) *Am J Infect Control* **29**, 89-93.
344. Liu, S. C., Leu, H. S., Yen, M. Y., Lee, P. I., & Chou, M. C. (2002) *Am J Infect Control* **30**, 381-385.
345. Monnet, D. L. (1998) *Infect Control Hosp Epidemiol* **19**, 552-559.
346. Pestotnik, S. L., Classen, D. C., Evans, R. S., & Burke, J. P. (1996) *Ann Intern Med* **124**, 884-890.
347. NCCLS (2002).
348. Kupronis, B. A., Richards, C. L., & Whitney, C. G. (2003) *J Am Geriatr Soc* **51**, 1520-1525.
349. Viray, M., Linkin, D., Maslow, J. N., Stieritz, D. D., Carson, L. S., Bilker, W. B., & Lautenbach, E. (2005) *Infect Control Hosp Epidemiol* **26**, 56-62.
350. Chaitram, J. M., Jevitt, L. A., Lary, S., & Tenover, F. C. (2003) *J Clin Microbiol* **41**, 2372-2377.
351. Ernst, E. J., Diekema, D. J., BootsMiller, B. J., Vaughn, T., Yankey, J. W., Flach, S. D., Ward, M. M., Franciscus, C. L., Acosta, E., Pfaller, M. A., *et al.* (2004) *Diagn Microbiol Infect Dis* **49**, 141-145.
352. Ginocchio, C. C. (2002) *Am J Health Syst Pharm* **59**, S7-11.
353. Stevenson, K. B., Samore, M., Barbera, J., Moore, J. W., Hannah, E., Houck, P., Tenover, F. C., & Gerberding, J. L. (2003) *Diagn Microbiol Infect Dis* **47**, 303-311.
354. Gupta, A., Della-Latta, P., Todd, B., San Gabriel, P., Haas, J., Wu, F., Rubenstein, D., & Saiman, L. (2004) *Infect Control Hosp Epidemiol* **25**, 210-215.

355. Rodriguez-Bano, J., Navarro, M. D., Romero, L., Muniain, M. A., Perea, E. J., Perez-Cano, R., Hernandez, J. R., & Pascual, A. (2006) *Clin Infect Dis* **42**, 37-45.
356. Bhavnani, S. M., Hammel, J. P., Forrest, A., Jones, R. N., & Ambrose, P. G. (2003) *Clin Infect Dis* **37**, 344-350.
357. Halstead, D. C., Gomez, N., & McCarter, Y. S. (2004) *J Clin Microbiol* **42**, 1-6.
358. Fridkin, S. K., Steward, C. D., Edwards, J. R., Pryor, E. R., McGowan, J. E., Jr., Archibald, L. K., Gaynes, R. P., & Tenover, F. C. (1999) *Clin Infect Dis* **29**, 245-252.
359. Lang, A., De Fina, G., Meyer, R., Aschbacher, R., Rizza, F., Mayr, O., & Casini, M. (2001) *Eur J Clin Microbiol Infect Dis* **20**, 657-660.
360. White, R. L., Friedrich, L. V., Mihm, L. B., & Bosso, J. A. (2000) *Clin Infect Dis* **31**, 16-23.
361. Zoutman, D. E. & Ford, B. D. (2005) *Am J Infect Control* **33**, 1-5.
362. cms.
363. Peterson, L. R., Hamilton, J. D., Baron, E. J., Tompkins, L. S., Miller, J. M., Wilfert, C. M., Tenover, F. C., & Thomson Jr, R. B., Jr. (2001) *Clin Infect Dis* **32**, 605-611.
364. Calfee, D. P., Giannetta, E. T., Durbin, L. J., Germanson, T. P., & Farr, B. M. (2003) *Clin Infect Dis* **37**, 326-332.
365. Thompson, R. L., Cabezudo, I., & Wenzel, R. P. (1982) *Ann Intern Med* **97**, 309-317.
366. Lacey, S., Flaxman, D., Scales, J., & Wilson, A. (2001) *J Hosp Infect* **48**, 308-311.
367. Greenaway, C. A. & Miller, M. A. (1999) *Infect Control Hosp Epidemiol* **20**, 341-343.
368. Spindel, S. J., Strausbaugh, L. J., & Jacobson, C. (1995) *Infect Control Hosp Epidemiol* **16**, 217-223.
369. Bula, C. J., Ghilardi, G., Wietlisbach, V., Petignat, C., & Francioli, P. (2004) *J Am Geriatr Soc* **52**, 700-706.
370. High, K. P., Bradley, S., Loeb, M., Palmer, R., Quagliarello, V., & Yoshikawa, T. (2005) *Clin Infect Dis* **40**, 114-122.
371. Silverblatt, F. J., Tibert, C., Mikolich, D., Blazek-D'Arezzo, J., Alves, J., Tack, M., & Agatiello, P. (2000) *J Am Geriatr Soc* **48**, 1211-1215.
372. CDC (2001) *MMWR* **50(RR05)**, 1-43.
373. Samore, M. H., Venkataraman, L., DeGirolami, P. C., Arbeit, R. D., & Karchmer, A. W. (1996) *Am J Med* **100**, 32-40.
374. Brooks, S. E., Veal, R. O., Kramer, M., Dore, L., Schupf, N., & Adachi, M. (1992) *Infect Control Hosp Epidemiol* **13**, 98-103.
375. Jernigan, J. A., Siegman-Igra, Y., Guerrant, R. C., & Farr, B. M. (1998) *Infect Control Hosp Epidemiol* **19**, 494-499.
376. Chang, V. T. & Nelson, K. (2000) *Clin Infect Dis* **31**, 717-722.
377. Nicolle, L. E. (2000) *Clin Infect Dis* **31**, 752-756.
378. Bonten, M. J., Slaughter, S., Hayden, M. K., Nathan, C., van Voorhis, J., & Weinstein, R. A. (1998) *Crit Care Med* **26**, 2001-2004.
379. Loeb, M. B., Craven, S., McGeer, A. J., Simor, A. E., Bradley, S. F., Low, D. E., Armstrong-Evans, M., Moss, L. A., & Walter, S. D. (2003) *Am J Epidemiol* **157**, 40-47.
380. McDonald, L. C., Banerjee, S. N., & Jarvis, W. R. (1998) *Infect Control Hosp Epidemiol* **19**, 772-777.
381. Montecalvo, M. A., de Lencastre, H., Carraher, M., Gedris, C., Chung, M., VanHorn, K., & Wormser, G. P. (1995) *Infect Control Hosp Epidemiol* **16**, 680-685.
382. Shannon, K. P. & French, G. L. (2002) *J Antimicrob Chemother* **50**, 965-969.

383. Singh, K., Gavin, P. J., Vescio, T., Thomson Jr, R. B., Jr., Deddish, R. B., Fisher, A., Noskin, G. A., & Peterson, L. R. (2003) *J Clin Microbiol* **41**, 2755-2757.
384. Grmek-Kosnik, I., Ihan, A., Dermota, U., Rems, M., Kosnik, M., & Jorn Kolmos, H. (2005) *J Hosp Infect* **61**, 155-161.
385. Villegas, M. V. & Hartstein, A. I. (2003) *Infect Control Hosp Epidemiol* **24**, 284-295.
386. Ramsey, A. H., Skonieczny, P., Coolidge, D. T., Kurzynski, T. A., Proctor, M. E., & Davis, J. P. (2001) *Infect Control Hosp Epidemiol* **22**, 423-426.
387. Harbarth, S., Liassine, N., Dharan, S., Herrault, P., Auckenthaler, R., & Pittet, D. (2000) *Clin Infect Dis* **31**, 1380-1385.
388. Lucet, J. C., Chevret, S., Decre, D., Vanjak, D., Macrez, A., Bedos, J. P., Wolff, M., & Regnier, B. (1996) *Clin Infect Dis* **22**, 430-436.
389. Malik, R. K., Montecalvo, M. A., Reale, M. R., Li, K., Maw, M., Munoz, J. L., Gedris, C., van Horn, K., Carnevale, K. A., Levi, M. H., *et al.* (1999) *Pediatr Infect Dis J* **18**, 352-356.
390. Stosor, V., Kruszynski, J., Suriano, T., Noskin, G. A., & Peterson, L. R. (1999) *Infect Control Hosp Epidemiol* **20**, 653-659.
391. Srinivasan, A., Song, X., Ross, T., Merz, W., Brower, R., & Perl, T. M. (2002) *Infect Control Hosp Epidemiol* **23**, 424-428.
392. Rumbak, M. J. & Cancio, M. R. (1995) *Crit Care Med* **23**, 1200-1203.
393. Quale, J., Landman, D., Atwood, E., Kreiswirth, B., Willey, B. M., Ditore, V., Zaman, M., Patel, K., Saurina, G., Huang, W., *et al.* (1996) *Am J Infect Control* **24**, 372-379.
394. Livornese, L. L., Jr., Dias, S., Samel, C., Romanowski, B., Taylor, S., May, P., Pitsakis, P., Woods, G., Kaye, D., Levison, M. E., *et al.* (1992) *Ann Intern Med* **117**, 112-116.
395. Gastmeier, P., Schwab, F., Geffers, C., & Ruden, H. (2004) *Infect Control Hosp Epidemiol* **25**, 109-113.
396. Ridwan, B., Mascini, E., van Der Reijden, N., Verhoef, J., & Bonten, M. (2002) *Bmj* **324**, 666-668.
397. Hitomi, S., Kubota, M., Mori, N., Baba, S., Yano, H., Okuzumi, K., & Kimura, S. (2000) *J Hosp Infect* **46**, 123-129.
398. Weber, D. J. & Rutala, W. A. (1997) *Infect Control Hosp Epidemiol* **18**, 306-309.
399. Schelenz, S. & French, G. (2000) *J Hosp Infect* **46**, 23-30.
400. Kirschke, D. L., Jones, T. F., Craig, A. S., Chu, P. S., Mayernick, G. G., Patel, J. A., & Schaffner, W. (2003) *N Engl J Med* **348**, 214-220.
401. Srinivasan, A., Wolfenden, L. L., Song, X., Mackie, K., Hartsell, T. L., Jones, H. D., Diette, G. B., Orens, J. B., Yung, R. C., Ross, T. L., *et al.* (2003) *N Engl J Med* **348**, 221-227.
402. Mangram, A. & Jarvis, W. R. (1996) *Infect Control Hosp Epidemiol* **17**, 718-720.
403. Vriens, M. R., Fluit, A. C., Troelstra, A., Verhoef, J., & van der Werken, C. (2002) *Infect Control Hosp Epidemiol* **23**, 491-494.
404. Cederna, J. E., Terpenning, M. S., Ensberg, M., Bradley, S. F., & Kauffman, C. A. (1990) *Infect Control Hosp Epidemiol* **11**, 13-16.
405. Hachem, R. & Raad, I. (2002) *Infect Control Hosp Epidemiol* **23**, 43-44.
406. Lui, S. L., Luk, W. K., Cheung, C. Y., Chan, T. M., Lai, K. N., & Peiris, J. S. (2001) *Transplantation* **71**, 59-64.
407. Zafar, A. B., Sylvester, L. K., & Beidas, S. O. (2002) *Am J Infect Control* **30**, 425-429.
408. Darouiche, R., Wright, C., Hamill, R., Koza, M., Lewis, D., & Markowski, J. (1991) *Antimicrob Agents Chemother* **35**, 1612-1615.

409. Goetz, M. B., Mulligan, M. E., Kwok, R., O'Brien, H., Caballes, C., & Garcia, J. P. (1992) *Am J Med* **92**, 607-614.
410. Pan, A., Carnevale, G., Catenazzi, P., Colombini, P., Crema, L., Dolcetti, L., Ferrari, L., Mondello, P., Signorini, L., Tinelli, C., *et al.* (2005) *Infect Control Hosp Epidemiol* **26**, 127-133.
411. Silvestri, L., Milanese, M., Oblach, L., Fontana, F., Gregori, D., Guerra, R., & van Saene, H. K. (2002) *Am J Infect Control* **30**, 391-399.
412. Weber, J. M., Sheridan, R. L., Schulz, J. T., Tompkins, R. G., & Ryan, C. M. (2002) *Infect Control Hosp Epidemiol* **23**, 549-551.

Table 1. Categorization of Reports about Control of MDROs in Healthcare Settings, 1982-2005

MDRO	MDR-GNB	MRSA	VRE
No. of Studies Reviewed/category	30	35	39
Types of Healthcare Facilities from which Study or Report Arose			
No. (%) from academic facilities ^α	30 (100)	28 (80)	33 (85)
No. (%) from other hospitals	0	4 (11)	3 (8)
No. (%) from LTCFs	0	1 (3)	2 (5)
No. (%) from multiple facilities in a region	0	2 (6)	1 (2)
Unit of Study for MDRO Control Efforts			
Special unit ^β	20	13	19
Hospital	10	19	17
LTCF	0	1	2
Region	0	2	1
Nature of Study or Report on MDRO Control^χ			
Outbreak	22	19	28
Non-outbreak	8	16	11
Total Period of Observation after Interventions Introduced			
Less than 1 year	17	14	25
1-2 years	6	6	6
2-5 years	5	11	8
Greater than 5 years	2	4	
Numbers of Control Measures Employed in Outbreaks/Studies			
Range	2-12	0-11	1-12
Median	7	7	8
Mode	8	7	9

^α Variably described as university hospitals, medical school affiliated hospitals, VA teaching hospitals, and, to a much lesser extent, community teaching hospitals

^β Includes intensive care units, burn units, dialysis units, hematology/oncology units, neonatal units, neonatal intensive care units, and, in a few instances, individual wards of a hospital

^χ Based on authors' description – if they called their experience an outbreak or not; authors vary in use of term so there is probable overlap between two categories

Table 2. Control Measures for MDROs Employed in Studies Performed in Healthcare Settings, 1982-2005

Focus of MDRO (No. of Studies)	MDR-GNB (n=30)	MRSA (n=35)	VRE (n=39)
No. (%) of Studies Using Control Measure			
Education of staff, patients or visitors	19 (63)	11 (31)	20 (53)
Emphasis on handwashing	16 (53)	21 (60)	9 (23)
Use of antiseptics for handwashing	8 (30)	12 (36)	16 (41)
Contact Precautions or glove use ^α	20 (67)	27 (77)	34 (87)
Private Rooms	4 (15)	10 (28)	10 (27)
Segregation of cases	4 (15)	3 (9)	5 (14)
Cohorting of Patients	11 (37)	12 (34)	14 (36)
Cohorting of Staff	2 (7)	6 (17)	9 (23)
Change in Antimicrobial Use	12 (41)	1 (3)	17 (44)
Surveillance cultures of patients	19 (63)	34 (97)	36 (92)
Surveillance cultures of staff	9 (31)	8 (23)	7 (19)
Environmental cultures	15 (50)	14 (42)	15 (38)
Extra cleaning & disinfection	11 (37)	7 (21)	20 (51)
Dedicated Equipment	5 (17)	0	12 (32)
Decolonization	3 (10)	25 (71)	4 (11)
Ward closure to new admission or to all patients	6 (21)	4 (12)	5 (14)
Other miscellaneous measures	6 (22) ^β	9 (27) ^χ	17 (44) ^δ

^α Contact Precautions mentioned specifically, use of gloves with gowns or aprons mentioned, barrier precautions, strict isolation, all included under this heading

^β includes signage, record flagging, unannounced inspections, selective decontamination, and peer compliance monitoring (1 to 4 studies employing any of these measures)

^χ includes requirements for masks, signage, record tracking, alerts, early discharge, and preventive isolation of new admissions pending results of screening cultures (1 to 4 studies employing any of these measures)

^δ includes computer flags, signage, requirement for mask, one-to-one nursing, changing type of thermometer used, and change in rounding sequence (1 to 7 studies employing any of these measures)

References for Tables 1 and 2

MDR-GNBs: (6, 8, 9, 11, 16, 38, 174, 175, 180, 209, 210, 213-215, 218, 334, 388, 406, 407)

MRSA: (68, 89, 152, 153, 165-173, 183, 188, 194, 204, 205, 208, 240, 269, 279, 280, 289, 304, 312, 327, 365, 392, 397, 408-412)

Table 3.

Tier 1. General Recommendations for Routine Prevention and Control of MDROs in Healthcare Settings						
Administrative Measures/Adherence Monitoring	MDRO Education	Judicious Antimicrobial Use	Surveillance	Infection Control Precautions to Prevent Transmission	Environmental Measures	Decolonization
<p>Make MDRO prevention/control an organizational priority. Provide administrative support and both fiscal and human resources to prevent and control MDRO transmission. <i>(IB)</i></p> <p>Identify experts who can provide consultation and expertise for analyzing epidemiologic data, recognizing MDRO problems, or devising effective control strategies, as needed. <i>(II)</i></p> <p>Implement systems to communicate information about reportable MDROs to administrative personnel and state/local health departments. <i>(II)</i></p> <p>Implement a multi-disciplinary process to monitor and improve HCP adherence to recommended practices for Standard and Contact Precautions. <i>(IB)</i></p> <p>Implement systems to designate patients known to be colonized or infected with a targeted MDRO and to notify receiving healthcare facilities or personnel prior to transfer of such patients within or between facilities. <i>(IB)</i></p> <p>Support participation in local, regional and/or national coalitions to combat emerging or growing MDRO problems. <i>(IB)</i></p> <p>Provide updated feedback at least annually to healthcare providers and administrators on facility and patient-care unit MDRO infections. Include information on changes in prevalence and incidence, problem assessment and performance improvement plans. <i>(IB)</i></p>	<p>Provide education and training on risks and prevention of MDRO transmission during orientation and periodic educational updates for HCP; include information on organizational experience with MDROs and prevention strategies. <i>(IB)</i></p>	<p>In hospitals and LTCFs, ensure that a multi-disciplinary process is in place to review local susceptibility patterns (antibiograms), and antimicrobial agents included in the formulary, to foster appropriate antimicrobial use. <i>(IB)</i></p> <p>Implement systems (e.g., CPOE, susceptibility report comment, pharmacy or unit director notification) to prompt clinicians to use the appropriate agent and regimen for the given clinical situation. <i>(IB)</i></p> <p>Provide clinicians with antimicrobial susceptibility reports and analysis of current trends, updated at least annually, to guide antimicrobial prescribing practices. <i>(IB)</i></p> <p>In settings with limited electronic communication system infrastructures to implement physician prompts, etc., at a minimum implement a process to review antibiotic use. Prepare and distribute reports to providers. <i>(II)</i></p>	<p>Use standardized laboratory methods and follow published guidelines for determining antimicrobial susceptibilities of targeted and emerging MDROs.</p> <p>Establish systems to ensure that clinical micro labs (in-house and outsourced) promptly notify infection control or a medical director/designee when a novel resistance pattern for that facility is detected. <i>(IB)</i></p> <p>In hospitals and LTCFs:</p> <p>...develop and implement laboratory protocols for storing isolates of selected MDROs for molecular typing when needed to confirm transmission or delineate epidemiology of MDRO in facility. <i>(IB)</i></p> <p>...establish laboratory-based systems to detect and communicate evidence of MDROs in clinical isolates <i>(IB)</i></p> <p>...prepare facility-specific antimicrobial susceptibility reports as recommended by CLSI; monitor reports for evidence of changing resistance that may indicate emergence or transmission of MDROs <i>(IA/IC)</i></p> <p>...develop and monitor special-care unit-specific antimicrobial susceptibility reports (e.g., ventilator-dependent units, ICUs, oncology units). <i>(IB)</i></p> <p>...monitor trends in incidence of target MDROs in the facility over time to determine if MDRO rates are decreasing or if additional interventions are needed. <i>(IA)</i></p>	<p>Follow Standard Precautions in all healthcare settings. <i>(IB)</i></p> <p>Use of Contact Precautions (CP):</p> <p>--- In <u>acute care settings</u>: Implement CP for all patients known to be colonized/infected with target MDROs. <i>(IB)</i></p> <p>--- In <u>LTCFs</u>: Consider the individual patient's clinical situation and facility resources in deciding whether to implement CP <i>(II)</i></p> <p>--- In <u>ambulatory and home care settings</u>, follow Standard Precautions <i>(II)</i></p> <p>---In <u>hemodialysis units</u>: Follow dialysis specific guidelines <i>(IC)</i></p> <p>No recommendation can be made regarding when to discontinue CP. <i>(Unresolved issue)</i></p> <p>Masks are not recommended for routine use to prevent transmission of MDROs from patients to HCWs. Use masks according to Standard Precautions when performing splash-generating procedures, caring for patients with open tracheostomies with potential for projectile secretions, and when there is evidence for transmission from heavily colonized sources (e.g., burn wounds).</p> <p>Patient placement in hospitals and LTCFs:</p> <p>When single-patient rooms are available, assign priority for these rooms to patients with known or suspected MDRO colonization or infection. Give highest priority to those patients who have conditions that may facilitate transmission, e.g., uncontained secretions or excretions. When single-patient rooms are not available, cohort patients with the same MDRO in the same room or patient-care area. <i>(IB)</i></p> <p>When cohorting patients with the same MDRO is not possible, place MDRO patients in rooms with patients who are at low risk for acquisition of MDROs and associated adverse outcomes from infection and are likely to have short lengths of stay. <i>(II)</i></p>	<p>Follow recommended cleaning, disinfection and sterilization guidelines for maintaining patient care areas and equipment.</p> <p>Dedicate non-critical medical items to use on individual patients known to be infected or colonized with an MDRO. Prioritize room cleaning of patients on Contact Precautions. Focus on cleaning and disinfecting frequently touched surfaces (e.g., bed rails, bedside commodes, bathroom fixtures in patient room, doorknobs) and equipment in immediate vicinity of patient.</p>	<p>Not recommended routinely</p>

Tier 2. Recommendations for Intensified MDRO control efforts

Institute one or more of the interventions described below when 1) incidence or prevalence of MDROs are not decreasing despite the use of routine control measures; or 2) the *first* case or outbreak of an epidemiologically important MDRO (e.g., VRE, MRSA, VISA, VRSA, MDR-GNB) is identified within a healthcare facility or unit *(IB)* Continue to monitor the incidence of target MDRO infection and colonization; if rates do not decrease, implement additional interventions as needed to reduce MDRO transmission.

Administrative Measures/Adherence Monitoring	MDRO Education	Judicious Antimicrobial Use	Surveillance	Infection Control Precautions to Prevent Transmission	Environmental Measures	Decolonization
<p>Obtain expert consultation from persons with experience in infection control and the epidemiology of MDROs, either in-house or through outside consultation, for assessment of the local MDRO problem and guidance in the design, implementation and evaluation of appropriate control measures. <i>(IB)</i></p> <p>Provide necessary leadership, funding and day-to-day oversight to implement interventions selected. <i>(IB)</i></p> <p>Evaluate healthcare system factors for role in creating or perpetuating MDRO transmission, including staffing levels, education and training, availability of consumable and durable resources; communication processes, and adherence to infection control measures. <i>(IB)</i></p> <p>Update healthcare providers and administrators on the progress and effectiveness of the intensified interventions. <i>(IB)</i></p>	<p>Intensify the frequency of educational programs for healthcare personnel, especially for those who work in areas where MDRO rates are not decreasing. Provide individual or unit-specific feedback when available. <i>(IB)</i></p>	<p>Review the role of antimicrobial use in perpetuating the MDRO problem targeted for intensified intervention. Control and improve antimicrobial use as indicated. Antimicrobial agents that may be targeted include vancomycin, third-^d generation cephalosporins, anti-anaerobic agents for VRE; third generation cephalosporins for ESBLs; and quinolones and carbapenems. <i>(IB)</i></p>	<p>Calculate and analyze incidence rates of target MDROs (single isolates/patient; location-, service-specific) <i>(IB)</i></p> <p>Increase frequency of compiling, monitoring antimicrobial susceptibility summary reports <i>(II)</i></p> <p>Implement laboratory protocols for storing isolates of selected MDROs for molecular typing; perform typing if needed <i>(IB)</i></p> <p>Develop and implement protocols to obtain active surveillance cultures from patients in populations at risk. <i>(IB)</i> (See recommendations for appropriate body sites and culturing methods.)</p> <p>Conduct culture surveys to assess efficacy of intensified MDRO control interventions.</p> <p>Conduct serial (e.g., weekly) unit-specific point prevalence culture surveys of the target MDRO to determine if transmission has decreased or ceased. <i>(IB)</i></p> <p>Repeat point-prevalence culture-surveys at routine intervals and at time of patient discharge or transfer until transmission has ceased. <i>(IB)</i></p> <p>If indicated by assessment of the MDRO problem, collect cultures to assess the colonization status of roommates and other patients with substantial exposure to patients with known MDRO infection or colonization. <i>(IB)</i></p> <p>Obtain cultures from HCP for target MDROs when there is epidemiologic evidence implicating the staff member as a source of ongoing transmission. <i>(IB)</i></p>	<p>Use of Contact Precautions: Implement Contact Precautions (CP) routinely for all patients colonized or infected with a target MDRO. <i>(IA)</i> Don gowns and gloves before or upon entry to the patient’s room or cubicle. <i>(IB)</i> In LTCFs, modify CP to allow MDRO-colonized/infected patients whose site of colonization or infection can be appropriately contained and who can observe good hand hygiene practices to enter common areas and participate in group activities When active surveillance cultures are obtained as part of an intensified MDRO control program, implement CP until the surveillance culture is reported negative for the target MDRO <i>(IB)</i></p> <p>No recommendation is made for universal use of gloves and/or gowns. <i>(Unresolved issue)</i></p> <p>Implement policies for patient admission and placement as needed to prevent transmission of the problem MDRO. <i>(IB)</i></p> <p>When single-patient rooms are available, assign priority for these rooms to patients with known or suspected MDRO colonization or infection. Give highest priority to those patients who have conditions that may facilitate transmission, e.g., uncontained secretions or excretions. When single-patient rooms are not available, cohort patients with the same MDRO in the same room or patient-care area. <i>(IB)</i></p> <p>When cohorting patients with the same MDRO is not possible, place MDRO patients in rooms with patients who are at low risk for acquisition of MDROs and associated adverse outcomes from infection and are likely to have short lengths of stay. <i>(II)</i></p> <p>Stop new admissions to the unit or facility if transmission continues despite the implementation of the intensified control measures. <i>(IB)</i></p>	<p>Implement patient.-dedicated use of non-critical equipment <i>(IB)</i></p> <p>Intensify and reinforce training of environmental staff who work in areas targeted for intensified MDRO control. Some facilities may choose to assign dedicated staff to targeted patient care areas to enhance consistency of proper environmental cleaning and disinfection services <i>(IB)</i></p> <p>Monitor cleaning performance to ensure consistent cleaning and disinfection of surfaces in close proximity to the patient and those likely to be touched by the patient and HCWs (e.g., bedrails, carts, bedside commodes, doorknobs, faucet handles) <i>(IB)</i>.</p> <p>Obtain environmental cultures (e.g., surfaces, shared equipment) only when epidemiologically implicated in transmission <i>(IB)</i></p> <p>Vacate units for environmental assessment and intensive cleaning when previous efforts to control environmental transmission have failed <i>(II)</i></p>	<p>Consult with experts on a case-by-case basis regarding the appropriate use of decolonization therapy for patients or staff during limited period of time as a component of an intensified MRSA control program <i>(II)</i></p> <p>When decolonization for MRSA is used, perform susceptibility testing for the decolonizing agent against the target organism or the MDRO strain epidemiologically implicated in transmission. Monitor susceptibility to detect emergence of resistance to the decolonizing agent. Consult with microbiologists for appropriate testing for mupirocin resistance, since standards have not been established.</p> <p>Do not use topical mupirocin routinely for MRSA decolonization of patients as a component of MRSA control programs in any healthcare setting. <i>(IB)</i></p> <p>Limit decolonization to HCP found to be colonized with MRSA who have been epidemiologically implicated in ongoing transmission of MRSA to patients. <i>(IB)</i></p> <p>No recommendation can be made for decolonization of patients who carry VRE or MDR-GNB.</p>

Infection Prevention and Control Assessment Tool for Outpatient Settings

This tool is intended to assist in the assessment of infection control programs and practices in outpatient settings. In order to complete the assessment, direct observation of infection control practices will be necessary. To facilitate the assessment, health departments are encouraged to share this tool with facilities in advance of their visit.

Overview

Section 1: Facility Demographics

Section 2: Infection Control Program and Infrastructure

Section 3: Direct Observation of Facility Practices

Section 4: Infection Control Guidelines and Other Resources

Infection Control Domains for Gap Assessment

- I. Infection Control Program and Infrastructure
- II. Infection Control Training and Competency
- III. Healthcare Personnel Safety
- IV. Surveillance and Disease Reporting
- V.a/b. Hand Hygiene
- VI.a/b. Personal Protective Equipment (PPE)
- VII.a/b. Injection Safety
- VIII.a/b. Respiratory Hygiene/Cough Etiquette
- IX.a/b. Point-of-Care Testing (if applicable)
- X.a/b. Environmental Cleaning
- XI.a/b. Device Reprocessing (if applicable)
- XII. Sterilization of Reusable Devices (if applicable)
- XIII. High-level Disinfection of Reusable Devices (if applicable)



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

Section 1: Facility Demographics			
Facility Name (for health department use only)			
NHSN Facility Organization ID (for health department use only)			
State-assigned Unique ID			
Date of Assessment			
Type of Assessment	<input type="checkbox"/> On-site <input type="checkbox"/> Other (specify):		
Rationale for Assessment (Select all that apply)	<input type="checkbox"/> Outbreak <input type="checkbox"/> Input from accrediting organization or state survey agency <input type="checkbox"/> Other (specify):		
Is the facility licensed by the state?	<input type="checkbox"/> Yes <input type="checkbox"/> No		
Is the facility certified by the Centers for Medicare & Medicaid Services (CMS)?	<input type="checkbox"/> Yes <input type="checkbox"/> No		
Is the facility accredited?	<input type="checkbox"/> Yes <input type="checkbox"/> No If yes, list the accreditation organization: <ul style="list-style-type: none"> <input type="checkbox"/> Accreditation Association for Ambulatory Health Care (AAAHC) <input type="checkbox"/> American Association for Accreditation of Ambulatory Surgery Facilities (AAAASF) <input type="checkbox"/> American Osteopathic Association (AOA) <input type="checkbox"/> The Joint Commission (TJC) <input type="checkbox"/> Other (specify): 		
Is the facility affiliated with a hospital?	<input type="checkbox"/> Yes (specify – for health department use only): <input type="checkbox"/> No		
Which procedures are performed by the facility? Select all that apply.	<input type="checkbox"/> Chemotherapy	<input type="checkbox"/> Endoscopy	<input type="checkbox"/> Ear/Nose/Throat
	<input type="checkbox"/> Imaging (MRI/CT)	<input type="checkbox"/> Immunizations	<input type="checkbox"/> OB/Gyn
	<input type="checkbox"/> Ophthalmologic	<input type="checkbox"/> Orthopedic	<input type="checkbox"/> Pain remediation
	<input type="checkbox"/> Plastic/reconstructive	<input type="checkbox"/> Podiatry	<input type="checkbox"/> Other (specify):
What is the primary procedure-type performed by the facility? Select only one.	<input type="checkbox"/> Chemotherapy	<input type="checkbox"/> Endoscopy	<input type="checkbox"/> Ear/Nose/Throat
	<input type="checkbox"/> Imaging (MRI/CT)	<input type="checkbox"/> Immunizations	<input type="checkbox"/> OB/Gyn
	<input type="checkbox"/> Ophthalmologic	<input type="checkbox"/> Orthopedic	<input type="checkbox"/> Pain remediation
	<input type="checkbox"/> Plastic/reconstructive	<input type="checkbox"/> Podiatry	<input type="checkbox"/> Other (specify):
How many physicians work at the facility?			
What is the average number of patients seen per week?			

Section 2: Infection Control Program and Infrastructure

I. Infection Control Program and Infrastructure		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Written infection prevention policies and procedures are available, current, and based on evidence-based guidelines (e.g., CDC/HICPAC), regulations, or standards.</p> <p><i>Note: Policies and procedures should be appropriate for the services provided by the facility and should extend beyond OSHA bloodborne pathogen training</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>B. Infection prevention policies and procedures are re-assessed at least annually or according to state or federal requirements, and updated if appropriate.</p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>C. At least one individual trained in infection prevention is employed by or regularly available (e.g., by contract) to manage the facility's infection control program.</p> <p><i>Note: Examples of training may include: Successful completion of initial and/or recertification exams developed by the Certification Board for Infection Control & Epidemiology; participation in infection control courses organized by the state or recognized professional societies (e.g., APIC, SHEA).</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>D. Facility has system for early detection and management of potentially infectious persons at initial points of patient encounter.</p> <p><i>Note: System may include taking a travel and occupational history, as appropriate, and elements described under respiratory hygiene/cough etiquette.</i></p>	<input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training and Competency		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Facility has a competency-based training program that provides job-specific training on infection prevention policies and procedures to healthcare personnel.</p> <p><i>Note: This includes those employed by outside agencies and available by contract or on a volunteer basis to the facility.</i></p> <p><i>See sections below for more specific assessment of training related to: hand hygiene, personal protective equipment (PPE), injection safety, environmental cleaning, point-of-care testing, and device reprocessing</i></p>	<input type="radio"/> Yes <input type="radio"/> No	

III. Healthcare Personnel Safety		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Facility has an exposure control plan that is tailored to the specific requirements of the facility (e.g., addresses potential hazards posed by specific services provided by the facility).</p> <p><i>Note: A model template, which includes a guide for creating an exposure control plan that meets the requirements of the OSHA Bloodborne Pathogens Standard is available at: https://www.osha.gov/Publications/osha3186.pdf</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>B. HCP for whom contact with blood or other potentially infectious material is anticipated are trained on the OSHA bloodborne pathogen standard upon hire and at least annually.</p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>C. Following an exposure event, post-exposure evaluation and follow-up, including prophylaxis as appropriate, are available at no cost to employee and are supervised by a licensed healthcare professional.</p> <p><i>Note: An exposure incident refers to a specific eye, mouth, other mucous membrane, non-intact skin, or parenteral contact with blood or other potentially infectious materials that results from the performance of an individual's duties.</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>D. Facility tracks HCP exposure events and evaluates event data and develops/implements corrective action plans to reduce incidence of such events.</p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>E. Facility follows recommendations of the Advisory Committee on Immunization Practices (ACIP) for immunization of HCP, including offering Hepatitis B and influenza vaccination.</p> <p><i>Note: Immunization of Health-Care Personnel: Recommendations of the ACIP available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/rr6007a1.htm</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>F. All HCP receive baseline tuberculosis (TB) screening prior to placement, and those with potential for ongoing exposure to TB receive periodic screening (if negative) at least annually.</p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>G. If respirators are used, the facility has a respiratory protection program that details required worksite-specific procedures and elements for required respirator use, including provision of medical clearance, training, and fit testing as appropriate.</p>	<input type="radio"/> Yes <input type="radio"/> No Not Applicable <input type="radio"/>	
<p>H. Facility has well-defined policies concerning contact of personnel with patients when personnel have potentially transmissible conditions. These policies include:</p> <ul style="list-style-type: none"> i. Work-exclusion policies that encourage reporting of illnesses and do not penalize with loss of wages, benefits, or job status. ii. Education of personnel on prompt reporting of illness to supervisor. 	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	

IV. Surveillance and Disease Reporting		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. An updated list of diseases reportable to the public health authority is readily available to all personnel.	<input type="radio"/> Yes <input type="radio"/> No	
B. Facility can demonstrate knowledge of and compliance with mandatory reporting requirements for notifiable diseases, healthcare associated infections (as appropriate), and for potential outbreaks.	<input type="radio"/> Yes <input type="radio"/> No	
C. Patients who have undergone procedures at the facility are educated regarding signs and symptoms of infection that may be associated with the procedure and instructed to notify the facility if such signs or symptoms occur.	<input type="radio"/> Yes <input type="radio"/> No	

V.a. Hand Hygiene		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. All HCP are educated regarding appropriate indications for hand hygiene: i. Upon hire, prior to provision of care ii. Annually	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	
B. HCP are required to demonstrate competency with hand hygiene following each training	<input type="radio"/> Yes <input type="radio"/> No	
C. Facility regularly audits (monitors and documents) adherence to hand hygiene.	<input type="radio"/> Yes <input type="radio"/> No	
D. Facility provides feedback from audits to personnel regarding their hand hygiene performance.	<input type="radio"/> Yes <input type="radio"/> No	
E. Hand hygiene policies promote preferential use of alcohol-based hand rub over soap and water in all clinical situations except when hands are visibly soiled (e.g., blood, body fluids) or after caring for a patient with known or suspected <i>C. difficile</i> or norovirus.	<input type="radio"/> Yes <input type="radio"/> No	

VI.a. Personal Protective Equipment (PPE)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. HCP who use PPE receive training on proper selection and use of PPE: i. Upon hire, prior to provision of care ii. Annually iii. When new equipment or protocols are introduced	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	
B. HCP are required to demonstrate competency with selection and use of PPE following each training.	<input type="radio"/> Yes <input type="radio"/> No	
C. Facility regularly audits (monitors and documents) adherence to proper PPE selection and use.	<input type="radio"/> Yes <input type="radio"/> No	
D. Facility provides feedback from audits to personnel regarding their performance with selection and use of PPE.	<input type="radio"/> Yes <input type="radio"/> No	

VII.a. Injection Safety (This element does not include assessment of pharmacy/compounding practices)

Elements to be assessed	Assessment	Notes/Areas for Improvement
A. HCP who prepare and/or administer parenteral medications receive training on safe injection practices: <ul style="list-style-type: none"> i. Upon hire, prior to being allowed to prepare and/or administer parenteral medications ii. Annually iii. When new equipment or protocols are introduced 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Yes <input type="radio"/> No</p>	
B. HCP are required to demonstrate competency with safe injection practices following each training.	<input type="radio"/> Yes <input type="radio"/> No	
C. Facility regularly audits (monitors and documents) adherence to safe injection practices.	<input type="radio"/> Yes <input type="radio"/> No	
D. Facility provides feedback from audits to personnel regarding their adherence to safe injection practices.	<input type="radio"/> Yes <input type="radio"/> No	
E. Facility has policies and procedures to track HCP access to controlled substances to prevent narcotics theft/diversion. <i>Note: Policies and procedures should address: how data are reviewed, how facility would respond to unusual access patterns, how facility would assess risk to patients if tampering (alteration or substitution) is suspected or identified, and who the facility would contact if diversion is suspected or identified.</i>	<input type="radio"/> Yes <input type="radio"/> No	

VIII.a. Respiratory Hygiene/Cough Etiquette

Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Facility has policies and procedures to contain respiratory secretions in persons who have signs and symptoms of a respiratory infection, beginning at point of entry to the facility and continuing through the duration of the visit. Policies include: <ul style="list-style-type: none"> i. Offering facemasks to coughing patients and other symptomatic persons upon entry to the facility, at a minimum, during periods of increased respiratory infection activity in the community. ii. Providing space in waiting rooms and encouraging persons with symptoms of respiratory infections to sit as far away from others as possible. <i>Note: If available, facilities may wish to place patients with symptoms of a respiratory infection in a separate area while waiting for care.</i>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Yes <input type="radio"/> No</p>	
B. Facility educates HCP on the importance of infection prevention measures to contain respiratory secretions to prevent the spread of respiratory pathogens.	<input type="radio"/> Yes <input type="radio"/> No	

IX.a. Point-of-Care Testing (e.g., blood glucose meters, INR monitor)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. HCP who perform point-of-care testing receive training on recommended practices: i. Upon hire, prior to being allowed to perform point-of-care testing ii. Annually iii. When new equipment or protocols are introduced	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
B. HCP are required to demonstrate competency with recommended practices for point-of-care testing following each training.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
C. Facility regularly audits (monitors and documents) adherence to recommended practices during point-of-care testing.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
D. Facility provides feedback from audits to personnel regarding their adherence to recommended practices.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

X.a. Environmental Cleaning		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Facility has written policies and procedures for routine cleaning and disinfection of environmental surfaces, including identification of responsible personnel.	<input type="radio"/> Yes <input type="radio"/> No	
B. Personnel who clean and disinfect patient care areas (e.g., environmental services, technicians, nurses) receive training on cleaning procedures i. Upon hire, prior to being allowed to perform environmental cleaning ii. Annually iii. When new equipment or protocols are introduced <i>Note: If environmental cleaning is performed by contract personnel, facility should verify this is provided by contracting company.</i>	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	
C. HCP are required to demonstrate competency with environmental cleaning procedures following each training.	<input type="radio"/> Yes <input type="radio"/> No	
D. Facility regularly audits (monitors and documents) adherence to cleaning and disinfection procedures, including using products in accordance with manufacturer's instructions (e.g., dilution, storage, shelf-life, contact time).	<input type="radio"/> Yes <input type="radio"/> No	
E. Facility provides feedback from audits to personnel regarding their adherence to cleaning and disinfection procedures.	<input type="radio"/> Yes <input type="radio"/> No	
F. Facility has a policy/procedure for decontamination of spills of blood or other body fluids.	<input type="radio"/> Yes <input type="radio"/> No	

X.a. Environmental Cleaning, continued

Operating Room

Elements to be assessed	Assessment	Notes/Areas for Improvement
G. Operating rooms are terminally cleaned after last procedure of the day.	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Not applicable	
H. Facility regularly audits (monitors and documents) adherence to recommended infection control practices for surgical infection prevention including: <ul style="list-style-type: none"> i. Adherence to preoperative surgical scrub and hand hygiene ii. Appropriate use of surgical attire and drapes iii. Adherence to aseptic technique and sterile field iv. Proper ventilation requirements in surgical suites v. Minimization of traffic in the operating room vi. Adherence to cleaning and disinfection of environmental surfaces 	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Not applicable	
I. Facility provides feedback from audits to personnel regarding their adherence to surgical infection prevention practices.	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Not applicable	

XI.a. Device Reprocessing

The following basic information allows for a general assessment of policies and procedures related to reprocessing of reusable medical devices. Outpatient facilities that are performing on-site sterilization or high-level disinfection of reusable medical devices should refer to the more detailed checklists in separate sections of this document devoted to those issues.

Categories of Medical Devices:

- **Critical items** (e.g., surgical instruments) are objects that enter sterile tissue or the vascular system and must be sterile prior to use (see Sterilization Section).
- **Semi-critical items** (e.g., endoscopes for upper endoscopy and colonoscopy, vaginal probes) are objects that contact mucous membranes or non-intact skin and require, at a minimum, high-level disinfection prior to reuse (see High-level Disinfection Section).
- **Non-critical items** (e.g., blood pressure cuffs) are objects that may come in contact with intact skin but not mucous membranes and should undergo cleaning and low- or intermediate-level disinfection depending on the nature and degree of contamination.

Single-use devices (SUDs) are labeled by the manufacturer for a single use and do not have reprocessing instructions. They may *not* be reprocessed for reuse except by entities which have complied with FDA regulatory requirements and have received FDA clearance to reprocess specific SUDs.

Note: Cleaning must always be performed prior to sterilization and disinfection

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Facility has policies and procedures to ensure that reusable medical devices are cleaned and reprocessed appropriately prior to use on another patient.</p> <p><i>Note: This includes clear delineation of responsibility among HCP for cleaning and disinfection of equipment including, non-critical equipment, mobile devices, and other electronics (e.g., point-of-care devices) that might not be reprocessed in a centralized reprocessing area.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>B. The individual(s) in charge of infection prevention at the facility is consulted whenever new devices or products will be purchased or introduced to ensure implementation of appropriate reprocessing policies and procedures.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>C. HCP responsible for reprocessing reusable medical devices receive hands-on training on proper selection and use of PPE and recommended steps for reprocessing assigned devices:</p> <ul style="list-style-type: none"> i. Upon hire, prior to being allowed to reprocess devices ii. Annually iii. When new devices are introduced or policies/procedures change. <p><i>Note: If device reprocessing is performed by contract personnel, facility should verify this is provided by contracting company.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>D. HCP are required to demonstrate competency with reprocessing procedures (i.e., correct technique is observed by trainer) following each training.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	

XI.a. Device Reprocessing, continued

Elements to be assessed	Assessment	Notes/Areas for Improvement
E. Facility regularly audits (monitors and documents) adherence to reprocessing procedures.	<input type="radio"/> Yes <input type="radio"/> No	
F. Facility provides feedback from audits to personnel regarding their adherence to reprocessing procedures.	<input type="radio"/> Yes <input type="radio"/> No	
G. Facility has protocols to ensure that HCP can readily identify devices that have been properly reprocessed and are ready for patient use (e.g., tagging system, storage in designated area).	<input type="radio"/> Yes <input type="radio"/> No	
H. Facility has policies and procedures outlining facility response (i.e., risk assessment and recall of device) in the event of a reprocessing error or failure.	<input type="radio"/> Yes <input type="radio"/> No	
I. Routine maintenance for reprocessing equipment (e.g., automated endoscope reprocessors, steam autoclave) is performed by qualified personnel in accordance with manufacturer instructions; confirm maintenance records are available.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

Section 3: Direct Observation of Facility Practices

Certain infection control lapses (e.g., reuse of syringes on more than one patient or to access a medication container that is used for subsequent patients; reuse of lancets) have resulted in bloodborne pathogen transmission and should be halted immediately. Identification of such lapses warrants appropriate notification and testing of potentially affected patients.

If an element is unable to be observed during an assessment (e.g., no patients received point-of-care testing during the visit), assess the element by interviewing appropriate personnel about facility practices. Notation should also be made in the notes section that the element was not able to be directly observed.

V.b. Hand hygiene		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Supplies necessary for adherence to hand hygiene (e.g., soap, water, paper towels, alcohol-based hand rub) are readily accessible to HCP in patient care areas.	<input type="radio"/> Yes <input type="radio"/> No	
Hand hygiene is performed correctly:		
B. Before contact with the patient	<input type="radio"/> Yes <input type="radio"/> No	
C. Before performing an aseptic task (e.g., insertion of IV or preparing an injection)	<input type="radio"/> Yes <input type="radio"/> No	
D. After contact with the patient	<input type="radio"/> Yes <input type="radio"/> No	
E. After contact with objects in the immediate vicinity of the patient	<input type="radio"/> Yes <input type="radio"/> No	
F. After contact with blood, body fluids or contaminated surfaces	<input type="radio"/> Yes <input type="radio"/> No	
G. After removing gloves	<input type="radio"/> Yes <input type="radio"/> No	
H. When moving from a contaminated-body site to a clean-body site during patient care	<input type="radio"/> Yes <input type="radio"/> No	

VI.b. Personal Protective Equipment (PPE)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Sufficient and appropriate PPE is available and readily accessible to HCP.	<input type="radio"/> Yes <input type="radio"/> No	
PPE is used correctly:		
B. PPE, other than respirator, is removed and discarded prior to leaving the patient's room or care area. If a respirator is used, it is removed and discarded (or reprocessed if reusable) <u>after</u> leaving the patient room or care area and closing the door.	<input type="radio"/> Yes <input type="radio"/> No	
C. Hand hygiene is performed immediately after removal of PPE.	<input type="radio"/> Yes <input type="radio"/> No	

VI.b. Personal Protective Equipment (PPE), continued		
Elements to be assessed	Assessment	Notes/Areas for Improvement
D. Gloves		
i. HCP wear gloves for potential contact with blood, body fluids, mucous membranes, non-intact skin, or contaminated equipment.	<input type="radio"/> Yes <input type="radio"/> No	
ii. HCP <u>do not</u> wear the same pair of gloves for the care of more than one patient.	<input type="radio"/> Yes <input type="radio"/> No	
iii. HCP <u>do not</u> wash gloves for the purpose of reuse.	<input type="radio"/> Yes <input type="radio"/> No	
E. Gowns		
i. HCP wear gowns to protect skin and clothing during procedures or activities where contact with blood or body fluids is anticipated.	<input type="radio"/> Yes <input type="radio"/> No Not Applicable <input type="radio"/>	
ii. HCP <u>do not</u> wear the same gown for the care of more than one patient.	<input type="radio"/> Yes <input type="radio"/> No Not Applicable <input type="radio"/>	
F. Facial protection		
i. HCP wear mouth, nose, and eye protection during procedures that are likely to generate splashes or sprays of blood or other body fluids.	<input type="radio"/> Yes <input type="radio"/> No Not Applicable <input type="radio"/>	

VII.b. Injection safety (This element does not include assessment of pharmacy/compounding practices)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Injections are prepared using aseptic technique in a clean area free from contamination or contact with blood, body fluids or contaminated equipment.	<input type="radio"/> Yes <input type="radio"/> No	
B. Needles and syringes are used for only one patient (this includes manufactured prefilled syringes and cartridge devices such as insulin pens).	<input type="radio"/> Yes <input type="radio"/> No	
C. The rubber septum on a medication vial is disinfected with alcohol prior to piercing.	<input type="radio"/> Yes <input type="radio"/> No	
D. Medication containers are entered with a new needle and a new syringe, even when obtaining additional doses for the same patient.	<input type="radio"/> Yes <input type="radio"/> No	
E. Single dose (single-use) medication vials, ampules, and bags or bottles of intravenous solution are used for only one patient.	<input type="radio"/> Yes <input type="radio"/> No	
F. Medication administration tubing and connectors are used for only one patient.	<input type="radio"/> Yes <input type="radio"/> No	
G. Multi-dose vials are dated by HCP when they are first opened and discarded within 28 days unless the manufacturer specifies a different (shorter or longer) date for that opened vial. <i>Note: This is different from the expiration date printed on the vial.</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/> (Facility does not use multi-dose vials or discards them after single patient use)	

VII.b. Injection safety (This element does not include assessment of pharmacy/compounding practices), continued

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>H. Multi-dose vials to be used for more than one patient are kept in a centralized medication area and <u>do not</u> enter the immediate patient treatment area (e.g., operating room, patient room/cubicle).</p> <p><i>Note: If multi-dose vials enter the immediate patient treatment area they should be dedicated for single-patient use and discarded immediately after use.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/> (Facility does not use multi-dose vials or discards them after single patient use)</p>	
I. All sharps are disposed of in a puncture-resistant sharps container.	<input type="radio"/> Yes <input type="radio"/> No	
J. Filled sharps containers are disposed of in accordance with state regulated medical waste rules.	<input type="radio"/> Yes <input type="radio"/> No	
K. All controlled substances (e.g., Schedule II, III, IV, V drugs) are kept locked within a secure area.	<input type="radio"/> Yes <input type="radio"/> No	
L. HCP wear a facemask (e.g., surgical mask) when placing a catheter or injecting material into the epidural or subdural space (e.g., during myelogram, epidural or spinal anesthesia).	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/> (Facility does not perform spinal injection procedures)</p>	

VIII.b. Respiratory Hygiene/Cough Etiquette

Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Facility:		
<p>i. Posts signs at entrances with instructions to patients with symptoms of respiratory infection to:</p> <p>a. Inform HCP of symptoms of a respiratory infection when they first register for care, and</p> <p>b. Practice Respiratory Hygiene/Cough Etiquette (cover their mouths/noses when coughing or sneezing, use and dispose of tissues, and perform hand hygiene after hands have been covered with respiratory secretions).</p>	<input type="radio"/> Yes <input type="radio"/> No	
ii. Provides tissues and no-touch receptacles for disposal of tissues.	<input type="radio"/> Yes <input type="radio"/> No	
iii. Provides resources for performing hand hygiene in or near waiting areas.	<input type="radio"/> Yes <input type="radio"/> No	

IX.b. Point-of-Care Testing (e.g., blood glucose meters, INR monitor)

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. New single-use, auto-disabling lancing device is used for each patient.</p> <p><i>Note: Lancet holder devices are not suitable for multi-patient use.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/></p>	
<p>B. If used for more than one patient, the point-of-care testing meter is cleaned and disinfected after every use according to manufacturer’s instructions.</p> <p><i>Note: If the manufacturer does not provide instructions for cleaning and disinfection, then the testing meter should not be used for >1 patient.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/></p>	

X.b. Environmental Cleaning

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Supplies necessary for appropriate cleaning and disinfection procedures (e.g., EPA-registered disinfectants) are available.</p> <p><i>Note: If environmental services are performed by contract personnel, facility should verify that appropriate EPA-registered products are provided by contracting company</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>B. High-touch surfaces in rooms where surgical or other invasive procedures (e.g., endoscopy, spinal injections) are performed are cleaned and then disinfected with an EPA-registered disinfectant after each procedure.</p>	<p><input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/></p>	
<p>C. Cleaners and disinfectants are used in accordance with manufacturer’s instructions (e.g., dilution, storage, shelf-life, contact time).</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>D. HCP engaged in environmental cleaning wear appropriate PPE to prevent exposure to infectious agents or chemicals (PPE can include gloves, gowns, masks, and eye protection).</p> <p><i>Note: The exact type of correct PPE depends on infectious or chemical agent and anticipated type of exposure.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	

XI.b. Device Reprocessing		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Policies, procedures, and manufacturer reprocessing instructions for reusable medical devices used in the facility are available in the reprocessing area(s).	<input type="radio"/> Yes <input type="radio"/> No	
B. Reusable medical devices are cleaned, reprocessed (disinfection or sterilization) and maintained according to the manufacturer instructions. <i>Note: If the manufacturer does not provide such instructions, the device may not be suitable for multi-patient use.</i>	<input type="radio"/> Yes <input type="radio"/> No	
C. Single-use devices are discarded after use and not used for more than one patient. <i>Note: If the facility elects to reuse single-use devices, these devices must be reprocessed prior to reuse by a third-party reprocessor that it is registered with the FDA as a third-party reprocessor and cleared by the FDA to reprocess the specific device in question. The facility should have documentation from the third party reprocessor confirming this is the case.</i>	<input type="radio"/> Yes <input type="radio"/> No	
D. Reprocessing area: i. Adequate space is allotted for reprocessing activities. ii. A workflow pattern is followed such that devices clearly flow from high contamination areas to clean/sterile areas (i.e., there is clear separation between soiled and clean workspaces).	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	
E. Adequate time for reprocessing is allowed to ensure adherence to all steps recommended by the device manufacturer, including drying and proper storage. <i>Note: Facilities should have an adequate supply of instruments for the volume of procedures performed and should schedule procedures to allow sufficient time for all reprocessing steps.</i>	<input type="radio"/> Yes <input type="radio"/> No	
F. HCP engaged in device reprocessing wear appropriate PPE to prevent exposure to infectious agents or chemicals (PPE can include gloves, gowns, masks, and eye protection). <i>Note: The exact type of correct PPE depends on infectious or chemical agent and anticipated type of exposure.</i>	<input type="radio"/> Yes <input type="radio"/> No	
G. Medical devices are stored in a manner to protect from damage and contamination.	<input type="radio"/> Yes <input type="radio"/> No	

XII. Sterilization of Reusable Devices

Note: If all device sterilization is performed off-site, skip to items M-O below.

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Devices are thoroughly cleaned according to manufacturer instructions and visually inspected for residual soil prior to sterilization.</p> <p><i>Note: Cleaning may be manual (i.e., using friction) and/or mechanical (e.g., with ultrasonic cleaners, washer-disinfector, washer-sterilizers).</i></p> <p><i>Ensure appropriately sized cleaning brushes are selected for cleaning device channels and lumens.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>B. Cleaning is performed as soon as practical after use (e.g., at the point of use) to prevent soiled materials from becoming dried onto devices.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>C. Enzymatic cleaner or detergent is used for cleaning and discarded according to manufacturer's instructions (typically after each use)</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>D. Cleaning brushes are disposable or, if reusable, cleaned and high-level disinfected or sterilized (per manufacturer's instructions) after use.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>E. After cleaning, instruments are appropriately wrapped/packaged for sterilization (e.g., package system selected is compatible with the sterilization process being performed, items are placed correctly into the basket, shelf or cart of the sterilizer so as not to impede the penetration of the sterilant, hinged instruments are open, instruments are disassembled if indicated by the manufacturer).</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>F. A chemical indicator (process indicator) is placed correctly in the instrument packs in every load.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>G. A biological indicator, intended specifically for the type and cycle parameters of the sterilizer, is used at least weekly for each sterilizer and with every load containing implantable items.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>H. For dynamic air removal-type sterilizers (e.g., prevacuum steam sterilizer), an air removal test (Bowie-Dick test) is performed in an empty dynamic-air removal sterilizer each day the sterilizer is used to verify efficacy of air removal.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>I. Sterile packs are labeled with a load number that indicates the sterilizer used, the cycle or load number, the date of sterilization, and, if applicable, the expiration date.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>J. Sterilization logs are current and include results from each load.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>K. Immediate-use steam sterilization, if performed, is only done in circumstances in which routine sterilization procedures cannot be performed.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	

XII. Sterilization of Reusable Devices, continued		
Note: If all device sterilization is performed off-site, skip to items M-O below.		
Elements to be assessed	Assessment	Notes/Areas for Improvement
L. Instruments that undergo immediate-use steam sterilization are used immediately and not stored.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
M. After sterilization, medical devices are stored so that sterility is not compromised.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
N. Sterile packages are inspected for integrity and compromised packages are reprocessed prior to use.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
O. The facility has a process to perform initial cleaning of devices (to prevent soiled materials from becoming dried onto devices) prior to transport to the off-site reprocessing facility.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

XIII. High-Level Disinfection of Reusable Devices		
Note: If all high-level disinfection is performed off-site, skip to items L-N below.		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Flexible endoscopes are inspected for damage and leak tested as part of each reprocessing cycle. Any device that fails the leak test is removed from clinical use and repaired.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
B. Devices are thoroughly cleaned according to manufacturer instructions and visually inspected for residual soil prior to high-level disinfection. <i>Note: Cleaning may be manual (i.e., using friction) and/or mechanical (e.g., with ultrasonic cleaners, washer-disinfector, washer-sterilizers).</i> <i>Ensure appropriately sized cleaning brushes are selected for cleaning device channels and lumens.</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
C. Cleaning is performed as soon as practical after use (e.g., at the point of use) to prevent soiled materials from becoming dried onto instruments.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
D. Enzymatic cleaner or detergent is used and discarded according to manufacturer instructions (typically after each use).	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
E. Cleaning brushes are disposable or, if reusable, cleaned and high-level disinfected or sterilized (per manufacturer instructions) after use.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
F. For chemicals used in high-level disinfection, manufacturer instructions are followed for: <ul style="list-style-type: none"> i. Preparation ii. Testing for appropriate concentration iii. Replacement (i.e., upon expiration or loss of efficacy) 	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

XIII. High-Level Disinfection of Reusable Devices, continued

Note: If all high-level disinfection is performed off-site, skip to items L-N below.

Elements to be assessed	Assessment	Notes/Areas for Improvement
G. If automated reprocessing equipment is used, proper connectors are used to assure that channels and lumens are appropriately disinfected.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
H. Devices are disinfected for the appropriate length of time as specified by manufacturer instructions.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
I. Devices are disinfected at the appropriate temperature as specified by manufacturer instructions.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
J. After high-level disinfection, devices are rinsed with sterile water, filtered water, or tap water followed by a rinse with 70% - 90% ethyl or isopropyl alcohol. <i>Note: There is no recommendation to use sterile or filtered water rather than tap water for rinsing semi-critical equipment that contact the mucous membranes of the rectum or vagina</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
K. Devices are dried thoroughly prior to reuse. <i>Note: For lumened instruments (e.g., endoscopes) this includes flushing all channels with alcohol and forcing air through channels.</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
L. After high-level disinfection, devices are stored in a manner to protect from damage or contamination. <i>Note: Endoscopes should be hung in a vertical position.</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
M. Facility maintains a log for each endoscopy procedure which includes: patient's name and medical record number (if available), procedure, date, endoscopist, system used to reprocess the endoscope (if more than one system could be used in the reprocessing area), and serial number or other identifier of the endoscope used.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
N. The facility has a process to perform initial cleaning of devices (to prevent soiled materials from becoming dried onto devices) prior to transport to the off-site reprocessing facility.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

Section 4: Infection Control Guidelines and Other Resources

- **General Infection Prevention**

- CDC/HICPAC Guidelines and recommendations: http://www.cdc.gov/HAI/prevent/prevent_pubs.html

- **Healthcare Personnel Safety**

- Guideline for Infection Control in Healthcare Personnel: <http://www.cdc.gov/hicpac/pdf/InfectControl98.pdf>
- Immunization of HealthCare Personnel: <http://www.cdc.gov/vaccines/spec-grps/hcw.htm>
- Occupational Safety & Health Administration (OSHA) Bloodborne Pathogens and Needlestick Prevention Standard: <http://www.osha.gov/SLTC/bloodbornepathogens/index.html>
- OSHA Respiratory Protection Standard: [https://www.osha.gov/pls/oshaweb/owadisp.show_document?p_id=12716&p_table=STANDARD S](https://www.osha.gov/pls/oshaweb/owadisp.show_document?p_id=12716&p_table=STANDARD_S)
- OSHA Respirator Fit Testing: https://www.osha.gov/video/respiratory_protection/fittesting_transcript.html

- **Hand Hygiene**

- Guideline for Hand Hygiene in Healthcare Settings: <http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf>
- Hand Hygiene in Healthcare Settings: <http://www.cdc.gov/handhygiene/>

Examples of tools that can be used to conduct a formal audit of hand hygiene practices:

- http://www.jointcommission.org/assets/1/18/hh_monograph.pdf
- <http://compepi.cs.uiowa.edu/index.php/Research/IScrub>

- **Personal Protective Equipment**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
- Guidance for the Selection and Use of Personal Protective Equipment in Healthcare Settings: <http://www.cdc.gov/HAI/prevent/ppe.html>

- **Injection Safety**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
- CDC Injection Safety Web Materials: <http://www.cdc.gov/injectionsafety/>

- CDC training video and related Safe Injection Practices Campaign materials: <http://www.oneandonlycampaign.org/>

- **Respiratory Hygiene/Cough Etiquette**
 - 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
 - Recommendations for preventing the spread of influenza: <http://www.cdc.gov/flu/professionals/infectioncontrol/>

- **Environmental Cleaning**
 - Guidelines for Environmental Infection Control in Healthcare Facilities: http://www.cdc.gov/hicpac/pdf/guidelines/eic_in_HCF_03.pdf
 - Options for Evaluating Environmental Infection Control: <http://www.cdc.gov/HAI/toolkits/Evaluating-Environmental-Cleaning.html>

- **Equipment Reprocessing**
 - Guideline for Disinfection and Sterilization in Healthcare Facilities: http://www.cdc.gov/hicpac/pdf/guidelines/Disinfection_Nov_2008.pdf
 - FDA regulations on reprocessing of single-use devices: <http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm071434>

- **Point-of-Care Testing**
 - Infection Prevention during Blood Glucose Monitoring and Insulin Administration: <http://www.cdc.gov/injectionsafety/blood-glucose-monitoring.html>
 - Frequently Asked Questions (FAQs) regarding Assisted Blood Glucose Monitoring and Insulin Administration: http://www.cdc.gov/injectionsafety/providers/blood-glucose-monitoring_faqs.html

- **Resources to assist with evaluation and response to breaches in infection control**
 - Patel PR, Srinivasan A, Perz JF. Developing a broader approach to management of infection control breaches in health care settings. Am J Infect Control. 2008 Dec;36(10):685-90
 - Steps for Evaluating an Infection Control Breach: http://www.cdc.gov/hai/outbreaks/steps_for_eval_IC_breach.html
 - Patient Notification Toolkit: <http://www.cdc.gov/injectionsafety/pntoolkit/index.html>



Guideline for Disinfection and Sterilization in Healthcare Facilities, 2008

William A. Rutala, Ph.D., M.P.H.^{1,2}, David J. Weber, M.D., M.P.H.^{1,2}, and the Healthcare
Infection Control Practices Advisory Committee (HICPAC)³

¹Hospital Epidemiology
University of North Carolina Health Care System
Chapel Hill, NC 27514

²Division of Infectious Diseases
University of North Carolina School of Medicine
Chapel Hill, NC 27599-7030

³HICPAC Members

Robert A. Weinstein, MD (Chair)
Cook County Hospital
Chicago, IL

Jane D. Siegel, MD (Co-Chair)
University of Texas Southwestern Medical Center
Dallas, TX

Michele L. Pearson, MD
(Executive Secretary)
Centers for Disease Control and Prevention
Atlanta, GA

Raymond Y.W. Chinn, MD
Sharp Memorial Hospital
San Diego, CA

Alfred DeMaria, Jr, MD
Massachusetts Department of Public Health
Jamaica Plain, MA

James T. Lee, MD, PhD
University of Minnesota
Minneapolis, MN

William A. Rutala, PhD, MPH
University of North Carolina Health Care System
Chapel Hill, NC

William E. Scheckler, MD
University of Wisconsin
Madison, WI

Beth H. Stover, RN
Kosair Children's Hospital
Louisville, KY

Marjorie A. Underwood, RN, BSN CIC
Mt. Diablo Medical Center
Concord, CA

This guideline discusses use of products by healthcare personnel in healthcare settings such as hospitals, ambulatory care and home care; the recommendations are not intended for consumer use of the products discussed.

Disinfection and Sterilization in Healthcare Facilities

Executive Summary

Introduction

Methods

Definition of Terms

Approach to Disinfection and Sterilization

 Critical Items

 Semicritical Items

 Noncritical Items

 Changes in Disinfection and Sterilization Since 1981

Disinfection of Healthcare Equipment

 Concerns with Implementing the Spaulding Scheme

 Reprocessing of Endoscopes

 Laparoscopes and Arthroscopes

 Tonometers, Cervical Diaphragm Fitting Rings, Cryosurgical Instruments, Endocavitary Probes

 Dental Instruments

 Disinfection of HBV, HCV, HIV or Tuberculosis-Contaminated Devices

 Disinfection in the Hemodialysis Unit

 Inactivation of *Clostridium difficile*

 OSHA Bloodborne Pathogen Standard

 Emerging Pathogens (*Cryptosporidium*, *Helicobacter pylori*, *E. coli* O157:H7, Rotavirus, Human

 Papilloma Virus, Norovirus, Severe Acute Respiratory Syndrome Coronavirus)

 Inactivation of Bioterrorist Agents

 Toxicological, Environmental, and Occupational Concerns

 Disinfection in Ambulatory Care, Home Care, and the Home

 Susceptibility of Antibiotic-Resistant Bacteria to Disinfectants

 Surface Disinfection: Should We Do It?

 Contact Time for Surface Disinfectants

 Air Disinfection

 Microbial Contamination of Disinfectants

Factors Affecting the Efficacy of Disinfection and Sterilization

 Number and Location of Microorganisms

 Innate Resistance of Microorganisms

 Concentration and Potency of Disinfectants

 Physical and Chemical Factors

 Organic and Inorganic Matter

 Duration of Exposure

 Biofilms

Cleaning

Disinfection

 Chemical Disinfectants

 Alcohol

 Overview

 Mode of Action

 Microbicidal Activity

 Uses

 Chlorine and Chlorine Compounds

 Overview

 Mode of Action

 Microbicidal Activity

- Uses
- Formaldehyde
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Glutaraldehyde
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Hydrogen Peroxide
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Iodophors
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Ortho-phthalaldehyde
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Peracetic Acid
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Peracetic Acid and Hydrogen Peroxide
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Phenolics
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Quaternary Ammonium Compounds
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Miscellaneous Inactivating Agents
 - Other Germicides
 - Ultraviolet Radiation
 - Pasteurization
 - Flushing- and Washer-Disinfectors
- Regulatory Framework for Disinfectants and Sterilants
- Neutralization of Germicides

Sterilization

Steam Sterilization

Overview

Mode of Action

Microbicidal Activity

Uses

Flash Sterilization

Overview

Uses

Low-Temperature Sterilization Technologies

Ethylene Oxide “Gas” Sterilization

Overview

Mode of Action

Microbicidal Activity

Uses

Hydrogen Peroxide Gas Plasma

Overview

Mode of Action

Microbicidal Activity

Uses

Peracetic Acid Sterilization

Overview

Mode of Action

Microbicidal Activity

Uses

Microbicidal Activity of Low-Temperature Sterilization Technology

Bioburden of Surgical Devices

Effect of Cleaning on Sterilization Efficacy

Other Sterilization Methods

Ionizing Radiation

Dry-Heat Sterilizers

Liquid Chemicals

Performic Acid

Filtration

Microwave

Glass Bead “Sterilizer”

Vaporized Hydrogen Peroxide

Ozone

Formaldehyde Steam

Gaseous Chlorine Dioxide

Vaporized Peracetic Acid

Infrared radiation

Sterilizing Practices

Overview

Sterilization Cycle Validation

Physical Facilities

Cleaning

Packaging

Loading

Storage

Monitoring (Mechanical, Chemical, Biological Indicators)

Reuse of Single-Use Medical Devices

Conclusion

Web-Based Disinfection and Sterilization Resources

Recommendations (Category IA, IB, IC, II)

Performance Indicators

Acknowledgements

Glossary

Tables and Figure

References

EXECUTIVE SUMMARY

The Guideline for Disinfection and Sterilization in Healthcare Facilities, 2008, presents evidence-based recommendations on the preferred methods for cleaning, disinfection and sterilization of patient-care medical devices and for cleaning and disinfecting the healthcare environment. This document supercedes the relevant sections contained in the 1985 Centers for Disease Control (CDC) Guideline for Handwashing and Environmental Control.¹ Because maximum effectiveness from disinfection and sterilization results from first cleaning and removing organic and inorganic materials, this document also reviews cleaning methods. The chemical disinfectants discussed for patient-care equipment include alcohols, glutaraldehyde, formaldehyde, hydrogen peroxide, iodophors, *ortho*-phthalaldehyde, peracetic acid, phenolics, quaternary ammonium compounds, and chlorine. The choice of disinfectant, concentration, and exposure time is based on the risk for infection associated with use of the equipment and other factors discussed in this guideline. The sterilization methods discussed include steam sterilization, ethylene oxide (ETO), hydrogen peroxide gas plasma, and liquid peracetic acid. When properly used, these cleaning, disinfection, and sterilization processes can reduce the risk for infection associated with use of invasive and noninvasive medical and surgical devices. However, for these processes to be effective, health-care workers should adhere strictly to the cleaning, disinfection, and sterilization recommendations in this document and to instructions on product labels.

In addition to updated recommendations, new topics addressed in this guideline include 1) inactivation of antibiotic-resistant bacteria, bioterrorist agents, emerging pathogens, and bloodborne pathogens; 2) toxicologic, environmental, and occupational concerns associated with disinfection and sterilization practices; 3) disinfection of patient-care equipment used in ambulatory settings and home care; 4) new sterilization processes, such as hydrogen peroxide gas plasma and liquid peracetic acid; and 5) disinfection of complex medical instruments (e.g., endoscopes).

INTRODUCTION

In the United States, approximately 46.5 million surgical procedures and even more invasive medical procedures—including approximately 5 million gastrointestinal endoscopies—are performed each year.² Each procedure involves contact by a medical device or surgical instrument with a patient's sterile tissue or mucous membranes. A major risk of all such procedures is the introduction of pathogens that can lead to infection. Failure to properly disinfect or sterilize equipment carries not only risk associated with breach of host barriers but also risk for person-to-person transmission (e.g., hepatitis B virus) and transmission of environmental pathogens (e.g., *Pseudomonas aeruginosa*).

Disinfection and sterilization are essential for ensuring that medical and surgical instruments do not transmit infectious pathogens to patients. Because sterilization of all patient-care items is not necessary, health-care policies must identify, primarily on the basis of the items' intended use, whether cleaning, disinfection, or sterilization is indicated.

Multiple studies in many countries have documented lack of compliance with established guidelines for disinfection and sterilization.³⁻⁶ Failure to comply with scientifically-based guidelines has led to numerous outbreaks.⁶⁻¹² This guideline presents a pragmatic approach to the judicious selection and proper use of disinfection and sterilization processes; the approach is based on well-designed studies assessing the efficacy (through laboratory investigations) and effectiveness (through clinical studies) of disinfection and sterilization procedures.

METHODS

This guideline resulted from a review of all MEDLINE articles in English listed under the MeSH headings of *disinfection* or *sterilization* (focusing on health-care equipment and supplies) from January 1980 through August 2006. References listed in these articles also were reviewed. Selected articles published before 1980 were reviewed and, if still relevant, included in the guideline. The three major peer-reviewed journals in infection control—*American Journal of Infection Control*, *Infection Control and Hospital Epidemiology*, and *Journal of Hospital Infection*—were searched for relevant articles published from January 1990 through August 2006. Abstracts presented at the annual meetings of the Society for Healthcare Epidemiology of America and Association for professionals in Infection Control and Epidemiology, Inc. during 1997–2006 also were reviewed; however, abstracts were not used to support the recommendations.

DEFINITION OF TERMS

Sterilization describes a process that destroys or eliminates all forms of microbial life and is carried out in health-care facilities by physical or chemical methods. Steam under pressure, dry heat, EtO gas, hydrogen peroxide gas plasma, and liquid chemicals are the principal sterilizing agents used in health-care facilities. Sterilization is intended to convey an absolute meaning; unfortunately, however, some health professionals and the technical and commercial literature refer to “disinfection” as “sterilization” and items as “partially sterile.” When chemicals are used to destroy all forms of microbiologic life, they can be called chemical sterilants. These same germicides used for shorter exposure periods also can be part of the disinfection process (i.e., high-level disinfection).

Disinfection describes a process that eliminates many or all pathogenic microorganisms, except bacterial spores, on inanimate objects (Tables 1 and 2). In health-care settings, objects usually are disinfected by liquid chemicals or wet pasteurization. Each of the various factors that affect the efficacy of

disinfection can nullify or limit the efficacy of the process.

Factors that affect the efficacy of both disinfection and sterilization include prior cleaning of the object; organic and inorganic load present; type and level of microbial contamination; concentration of and exposure time to the germicide; physical nature of the object (e.g., crevices, hinges, and lumens); presence of biofilms; temperature and pH of the disinfection process; and in some cases, relative humidity of the sterilization process (e.g., ethylene oxide).

Unlike sterilization, disinfection is not sporicidal. A few disinfectants will kill spores with prolonged exposure times (3–12 hours); these are called *chemical sterilants*. At similar concentrations but with shorter exposure periods (e.g., 20 minutes for 2% glutaraldehyde), these same disinfectants will kill all microorganisms except large numbers of bacterial spores; they are called *high-level disinfectants*. *Low-level disinfectants* can kill most vegetative bacteria, some fungi, and some viruses in a practical period of time (≤ 10 minutes). *Intermediate-level disinfectants* might be cidal for mycobacteria, vegetative bacteria, most viruses, and most fungi but do not necessarily kill bacterial spores. Germicides differ markedly, primarily in their antimicrobial spectrum and rapidity of action.

Cleaning is the removal of visible soil (e.g., organic and inorganic material) from objects and surfaces and normally is accomplished manually or mechanically using water with detergents or enzymatic products. Thorough cleaning is essential before high-level disinfection and sterilization because inorganic and organic materials that remain on the surfaces of instruments interfere with the effectiveness of these processes. *Decontamination* removes pathogenic microorganisms from objects so they are safe to handle, use, or discard.

Terms with the suffix *cide* or *cidal* for killing action also are commonly used. For example, a germicide is an agent that can kill microorganisms, particularly pathogenic organisms (“germs”). The term *germicide* includes both antiseptics and disinfectants. *Antiseptics* are germicides applied to living tissue and skin; *disinfectants* are antimicrobials applied only to inanimate objects. In general, antiseptics are used only on the skin and not for surface disinfection, and disinfectants are not used for skin antiseptics because they can injure skin and other tissues. Virucide, fungicide, bactericide, sporicide, and tuberculocide can kill the type of microorganism identified by the prefix. For example, a bactericide is an agent that kills bacteria.¹³⁻¹⁸

A RATIONAL APPROACH TO DISINFECTION AND STERILIZATION

More than 30 years ago, Earle H. Spaulding devised a rational approach to disinfection and sterilization of patient-care items and equipment.¹⁴ This classification scheme is so clear and logical that it has been retained, refined, and successfully used by infection control professionals and others when planning methods for disinfection or sterilization.^{1, 13, 15, 17, 19, 20} Spaulding believed the nature of disinfection could be understood readily if instruments and items for patient care were categorized as critical, semicritical, and noncritical according to the degree of risk for infection involved in use of the items. The CDC *Guideline for Handwashing and Hospital Environmental Control*²¹, *Guidelines for the Prevention of Transmission of Human Immunodeficiency Virus (HIV) and Hepatitis B Virus (HBV) to Health-Care and Public-Safety Workers*²², and *Guideline for Environmental Infection Control in Health-Care Facilities*²³ employ this terminology.

Critical Items

Critical items confer a high risk for infection if they are contaminated with any microorganism. Thus, objects that enter sterile tissue or the vascular system must be sterile because any microbial contamination could transmit disease. This category includes surgical instruments, cardiac and urinary catheters, implants, and ultrasound probes used in sterile body cavities. Most of the items in this category should be purchased as sterile or be sterilized with steam if possible. Heat-sensitive objects can be treated with EtO, hydrogen peroxide gas plasma; or if other methods are unsuitable, by liquid chemical sterilants. Germicides categorized as chemical sterilants include $\geq 2.4\%$ glutaraldehyde-based formulations, 0.95% glutaraldehyde with 1.64% phenol/phenate, 7.5% stabilized hydrogen peroxide, 7.35% hydrogen peroxide with 0.23% peracetic acid, 0.2% peracetic acid, and 0.08% peracetic acid with 1.0% hydrogen peroxide. Liquid chemical sterilants reliably produce sterility only if cleaning precedes treatment and if proper guidelines are followed regarding concentration, contact time, temperature, and pH.

Semicritical Items

Semicritical items contact mucous membranes or nonintact skin. This category includes respiratory therapy and anesthesia equipment, some endoscopes, laryngoscope blades²⁴, esophageal manometry probes, cystoscopes²⁵, anorectal manometry catheters, and diaphragm fitting rings. These medical devices should be free from all microorganisms; however, small numbers of bacterial spores are permissible. Intact mucous membranes, such as those of the lungs and the gastrointestinal tract, generally are resistant to infection by common bacterial spores but susceptible to other organisms, such as bacteria, mycobacteria, and viruses. Semicritical items minimally require high-level disinfection using chemical disinfectants. Glutaraldehyde, hydrogen peroxide, *ortho*-phthalaldehyde, and peracetic acid with hydrogen peroxide are cleared by the Food and Drug Administration (FDA) and are dependable high-level disinfectants provided the factors influencing germicidal procedures are met (Table 1). When a disinfectant is selected for use with certain patient-care items, the chemical compatibility after extended use with the items to be disinfected also must be considered.

High-level disinfection traditionally is defined as complete elimination of all microorganisms in or on an instrument, except for small numbers of bacterial spores. The FDA definition of high-level disinfection is a sterilant used for a shorter contact time to achieve a 6-log₁₀ kill of an appropriate *Mycobacterium* species. Cleaning followed by high-level disinfection should eliminate enough pathogens to prevent transmission of infection.^{26, 27}

Laparoscopes and arthroscopes entering sterile tissue ideally should be sterilized between patients. However, in the United States, this equipment sometimes undergoes only high-level disinfection between patients.²⁸⁻³⁰ As with flexible endoscopes, these devices can be difficult to clean and high-level disinfect or sterilize because of intricate device design (e.g., long narrow lumens, hinges). Meticulous

cleaning must precede any high-level disinfection or sterilization process. Although sterilization is preferred, no reports have been published of outbreaks resulting from high-level disinfection of these scopes when they are properly cleaned and high-level disinfected. Newer models of these instruments can withstand steam sterilization that for critical items would be preferable to high-level disinfection.

Rinsing endoscopes and flushing channels with sterile water, filtered water, or tap water will prevent adverse effects associated with disinfectant retained in the endoscope (e.g., disinfectant-induced colitis). Items can be rinsed and flushed using sterile water after high-level disinfection to prevent contamination with organisms in tap water, such as nontuberculous mycobacteria,^{10, 31, 32} *Legionella*,³³⁻³⁵ or gram-negative bacilli such as *Pseudomonas*.^{1, 17, 36-38} Alternatively, a tapwater or filtered water (0.2µ filter) rinse should be followed by an alcohol rinse and forced air drying.^{28, 38-40} Forced-air drying markedly reduces bacterial contamination of stored endoscopes, most likely by removing the wet environment favorable for bacterial growth.³⁹ After rinsing, items should be dried and stored (e.g., packaged) in a manner that protects them from recontamination.

Some items that may come in contact with nonintact skin for a brief period of time (i.e., hydrotherapy tanks, bed side rails) are usually considered noncritical surfaces and are disinfected with intermediate-level disinfectants (i.e., phenolic, iodophor, alcohol, chlorine)²³. Since hydrotherapy tanks have been associated with spread of infection, some facilities have chosen to disinfect them with recommended levels of chlorine^{23, 41}.

In the past, high-level disinfection was recommended for mouthpieces and spirometry tubing (e.g., glutaraldehyde) but cleaning the interior surfaces of the spirometers was considered unnecessary.⁴² This was based on a study that showed that mouthpieces and spirometry tubing become contaminated with microorganisms but there was no bacterial contamination of the surfaces inside the spirometers. Filters have been used to prevent contamination of this equipment distal to the filter; such filters and the proximal mouthpiece are changed between patients.

Noncritical Items

Noncritical items are those that come in contact with intact skin but not mucous membranes. Intact skin acts as an effective barrier to most microorganisms; therefore, the sterility of items coming in contact with intact skin is "not critical." In this guideline, noncritical items are divided into noncritical patient care items and noncritical environmental surfaces^{43, 44}. Examples of noncritical patient-care items are bedpans, blood pressure cuffs, crutches and computers⁴⁵. In contrast to critical and some semicritical items, most noncritical reusable items may be decontaminated where they are used and do not need to be transported to a central processing area. Virtually no risk has been documented for transmission of infectious agents to patients through noncritical items³⁷ when they are used as noncritical items and do not contact non-intact skin and/or mucous membranes. Table 1 lists several low-level disinfectants that may be used for noncritical items. Most Environmental Protection Agency (EPA)-registered disinfectants have a 10-minute label claim. However, multiple investigators have demonstrated the effectiveness of these disinfectants against vegetative bacteria (e.g., *Listeria*, *Escherichia coli*, *Salmonella*, vancomycin-resistant Enterococci, methicillin-resistant *Staphylococcus aureus*), yeasts (e.g., *Candida*), mycobacteria (e.g., *Mycobacterium tuberculosis*), and viruses (e.g. poliovirus) at exposure times of 30–60 seconds⁴⁶⁻⁶⁴. Federal law requires all applicable label instructions on EPA-registered products to be followed (e.g., use-dilution, shelf life, storage, material compatibility, safe use, and disposal). If the user selects exposure conditions (e.g., exposure time) that differ from those on the EPA-registered products label, the user assumes liability for any injuries resulting from off-label use and is potentially subject to enforcement action under Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA)⁶⁵.

Noncritical environmental surfaces include bed rails, some food utensils, bedside tables, patient furniture and floors. Noncritical environmental surfaces frequently touched by hand (e.g., bedside tables,

bed rails) potentially could contribute to secondary transmission by contaminating hands of health-care workers or by contacting medical equipment that subsequently contacts patients^{13, 46-48, 51, 66, 67}. Mops and reusable cleaning cloths are regularly used to achieve low-level disinfection on environmental surfaces. However, they often are not adequately cleaned and disinfected, and if the water-disinfectant mixture is not changed regularly (e.g., after every three to four rooms, at no longer than 60-minute intervals), the mopping procedure actually can spread heavy microbial contamination throughout the health-care facility⁶⁸. In one study, standard laundering provided acceptable decontamination of heavily contaminated mopheads but chemical disinfection with a phenolic was less effective.⁶⁸ Frequent laundering of mops (e.g., daily), therefore, is recommended. Single-use disposable towels impregnated with a disinfectant also can be used for low-level disinfection when spot-cleaning of noncritical surfaces is needed⁴⁵.

Changes in Disinfection and Sterilization Since 1981

The Table in the CDC *Guideline for Environmental Control* prepared in 1981 as a guide to the appropriate selection and use of disinfectants has undergone several important changes (Table 1).¹⁵ First, formaldehyde-alcohol has been deleted as a recommended chemical sterilant or high-level disinfectant because it is irritating and toxic and not commonly used. Second, several new chemical sterilants have been added, including hydrogen peroxide, peracetic acid^{58, 69, 70}, and peracetic acid and hydrogen peroxide in combination. Third, 3% phenolics and iodophors have been deleted as high-level disinfectants because of their unproven efficacy against bacterial spores, *M. tuberculosis*, and/or some fungi.^{55, 71} Fourth, isopropyl alcohol and ethyl alcohol have been excluded as high-level disinfectants¹⁵ because of their inability to inactivate bacterial spores and because of the inability of isopropyl alcohol to inactivate hydrophilic viruses (i.e., poliovirus, coxsackie virus).⁷² Fifth, a 1:16 dilution of 2.0% glutaraldehyde-7.05% phenol-1.20% sodium phenate (which contained 0.125% glutaraldehyde, 0.440% phenol, and 0.075% sodium phenate when diluted) has been deleted as a high-level disinfectant because this product was removed from the marketplace in December 1991 because of a lack of bactericidal activity in the presence of organic matter; a lack of fungicidal, tuberculocidal and sporicidal activity; and reduced virucidal activity.^{49, 55, 56, 71, 73-79} Sixth, the exposure time required to achieve high-level disinfection has been changed from 10-30 minutes to 12 minutes or more depending on the FDA-cleared label claim and the scientific literature.^{27, 55, 69, 76, 80-84} A glutaraldehyde and an ortho-phthalaldehyde have an FDA-cleared label claim of 5 minutes when used at 35°C and 25°C, respectively, in an automated endoscope reprocessor with FDA-cleared capability to maintain the solution at the appropriate temperature.⁸⁵

In addition, many new subjects have been added to the guideline. These include inactivation of emerging pathogens, bioterrorist agents, and bloodborne pathogens; toxicologic, environmental, and occupational concerns associated with disinfection and sterilization practices; disinfection of patient-care equipment used in ambulatory and home care; inactivation of antibiotic-resistant bacteria; new sterilization processes, such as hydrogen peroxide gas plasma and liquid peracetic acid; and disinfection of complex medical instruments (e.g., endoscopes).

DISINFECTION OF HEALTHCARE EQUIPMENT

Concerns about Implementing the Spaulding Scheme

One problem with implementing the aforementioned scheme is oversimplification. For example, the scheme does not consider problems with reprocessing of complicated medical equipment that often is heat-sensitive or problems of inactivating certain types of infectious agents (e.g., prions, such as Creutzfeldt-Jakob disease [CJD] agent). Thus, in some situations, choosing a method of disinfection remains difficult, even after consideration of the categories of risk to patients. This is true particularly for a few medical devices (e.g., arthroscopes, laparoscopes) in the critical category because of controversy about whether they should be sterilized or high-level disinfected.^{28, 86} Heat-stable scopes (e.g., many rigid scopes) should be steam sterilized. Some of these items cannot be steam sterilized because they are heat-sensitive; additionally, sterilization using ethylene oxide (EtO) can be too time-consuming for routine use between patients (new technologies, such as hydrogen peroxide gas plasma and peracetic acid reprocessor, provide faster cycle times). However, evidence that sterilization of these items improves patient care by reducing the infection risk is lacking^{29, 87-91}. Many newer models of these instruments can withstand steam sterilization, which for critical items is the preferred method.

Another problem with implementing the Spaulding scheme is processing of an instrument in the semicritical category (e.g., endoscope) that would be used in conjunction with a critical instrument that contacts sterile body tissues. For example, is an endoscope used for upper gastrointestinal tract investigation still a semicritical item when used with sterile biopsy forceps or in a patient who is bleeding heavily from esophageal varices? Provided that high-level disinfection is achieved, and all microorganisms except bacterial spores have been removed from the endoscope, the device should not represent an infection risk and should remain in the semicritical category⁹²⁻⁹⁴. Infection with spore-forming bacteria has not been reported from appropriately high-level disinfected endoscopes.

An additional problem with implementation of the Spaulding system is that the optimal contact time for high-level disinfection has not been defined or varies among professional organizations, resulting in different strategies for disinfecting different types of semicritical items (e.g., endoscopes, applanation tonometers, endocavitary transducers, cryosurgical instruments, and diaphragm fitting rings). Until simpler and effective alternatives are identified for device disinfection in clinical settings, following this guideline, other CDC guidelines^{1, 22, 95, 96} and FDA-cleared instructions for the liquid chemical sterilants/high-level disinfectants would be prudent.

Reprocessing of Endoscopes

Physicians use endoscopes to diagnose and treat numerous medical disorders. Even though endoscopes represent a valuable diagnostic and therapeutic tool in modern medicine and the incidence of infection associated with their use reportedly is very low (about 1 in 1.8 million procedures)⁹⁷, more healthcare-associated outbreaks have been linked to contaminated endoscopes than to any other medical device^{6-8, 12, 98}. To prevent the spread of health-care-associated infections, all heat-sensitive endoscopes (e.g., gastrointestinal endoscopes, bronchoscopes, nasopharygoscopes) must be properly cleaned and, at a minimum, subjected to high-level disinfection after each use. High-level disinfection can be expected to destroy all microorganisms, although when high numbers of bacterial spores are present, a few spores might survive.

Because of the types of body cavities they enter, flexible endoscopes acquire high levels of microbial contamination (bioburden) during each use⁹⁹. For example, the bioburden found on flexible gastrointestinal endoscopes after use has ranged from 10^5 colony forming units (CFU)/mL to 10^{10} CFU/mL, with the highest levels found in the suction channels⁹⁹⁻¹⁰². The average load on bronchoscopes before cleaning was 6.4×10^4 CFU/mL. Cleaning reduces the level of microbial contamination by 4–6 \log_{10} ^{83, 103}. Using human immunovirus (HIV)-contaminated endoscopes, several investigators have shown that cleaning completely eliminates the microbial contamination on the scopes^{104, 105}. Similarly, other investigators found that EtO sterilization or soaking in 2% glutaraldehyde for 20 minutes was effective only when the device first was properly cleaned¹⁰⁶.

FDA maintains a list of cleared liquid chemical sterilants and high-level disinfectants that can be used to reprocess heat-sensitive medical devices, such as flexible endoscopes (<http://www.fda.gov/cdrh/ode/germlab.html>). At this time, the FDA-cleared and marketed formulations include: $\geq 2.4\%$ glutaraldehyde, 0.55% *ortho*-phthalaldehyde (OPA), 0.95% glutaraldehyde with 1.64% phenol/phenate, 7.35% hydrogen peroxide with 0.23% peracetic acid, 1.0% hydrogen peroxide with 0.08% peracetic acid, and 7.5% hydrogen peroxide⁸⁵. These products have excellent antimicrobial activity; however, some oxidizing chemicals (e.g., 7.5% hydrogen peroxide, and 1.0% hydrogen peroxide with 0.08% peracetic acid [latter product is no longer marketed]) reportedly have caused cosmetic and functional damage to endoscopes⁶⁹. Users should check with device manufacturers for information about germicide compatibility with their device. If the germicide is FDA-cleared, then it is safe when used according to label directions; however, professionals should review the scientific literature for newly available data regarding human safety or materials compatibility. EtO sterilization of flexible endoscopes is infrequent because it requires a lengthy processing and aeration time (e.g., 12 hours) and is a potential hazard to staff and patients. The two products most commonly used for reprocessing endoscopes in the United States are glutaraldehyde and an automated, liquid chemical sterilization process that uses peracetic acid¹⁰⁷. The American Society for Gastrointestinal Endoscopy (ASGE) recommends glutaraldehyde solutions that do not contain surfactants because the soapy residues of surfactants are difficult to remove during rinsing¹⁰⁸. *ortho*-phthalaldehyde has begun to replace glutaraldehyde in many health-care facilities because it has several potential advantages over glutaraldehyde: is not known to irritate the eyes and nasal passages, does not require activation or exposure monitoring, and has a 12-minute high-level disinfection claim in the United States⁶⁹. Disinfectants that are not FDA-cleared and should not be used for reprocessing endoscopes include iodophors, chlorine solutions, alcohols, quaternary ammonium compounds, and phenolics. These solutions might still be in use outside the United States, but their use should be strongly discouraged because of lack of proven efficacy against all microorganisms or materials incompatibility.

FDA clearance of the contact conditions listed on germicide labeling is based on the manufacturer's test results (<http://www.fda.gov/cdrh/ode/germlab.html>). Manufacturers test the product under worst-case conditions for germicide formulation (i.e., minimum recommended concentration of the active ingredient), and include organic soil. Typically manufacturers use 5% serum as the organic soil and hard water as examples of organic and inorganic challenges. The soil represents the organic loading to which the device is exposed during actual use and that would remain on the device in the absence of cleaning. This method ensures that the contact conditions completely eliminate the test mycobacteria (e.g., 10^5 to 10^6 *Mycobacteria tuberculosis* in organic soil and dried on a scope) if inoculated in the most difficult areas for the disinfectant to penetrate and contact in the absence of cleaning and thus provides a margin of safety¹⁰⁹. For 2.4% glutaraldehyde that requires a 45-minute immersion at 25°C to achieve high-level disinfection (i.e., 100% kill of *M. tuberculosis*). FDA itself does not conduct testing but relies solely on the disinfectant manufacturer's data. Data suggest that *M. tuberculosis* levels can be reduced by at least 8 log₁₀ with cleaning (4 log₁₀)^{83, 101, 102, 110}, followed by chemical disinfection for 20 minutes at 20°C (4 to 6 log₁₀)^{83, 93, 111, 112}. On the basis of these data, APIC¹¹³, the Society of Gastroenterology Nurses and Associates (SGNA)^{38, 114, 115}, the ASGE¹⁰⁸, American College of Chest Physicians¹², and a multi-society guideline¹¹⁶ recommend alternative contact conditions with 2% glutaraldehyde to achieve high-level disinfection (e.g., that equipment be immersed in 2% glutaraldehyde at 20°C for at least 20 minutes for high-level disinfection). Federal regulations are to follow the FDA-cleared label claim for high-level disinfectants. The FDA-cleared labels for high-level disinfection with $>2\%$ glutaraldehyde at 25°C range from 20-90 minutes, depending upon the product based on three tier testing which includes AOAC sporicidal tests, simulated use testing with mycobacterial and in-use testing. The studies supporting the efficacy of $>2\%$ glutaraldehyde for 20 minutes at 20°C assume adequate cleaning prior to disinfection, whereas the FDA-cleared label claim incorporates an added margin of safety to accommodate possible lapses in cleaning practices. Facilities that have chosen to apply the 20 minute duration at 20°C have done so based on the IA recommendation in the July 2003 SHEA position paper, "Multi-society Guideline for Reprocessing Flexible Gastrointestinal Endoscopes"^{19, 57, 83, 94, 108, 111, 116-121}.

Flexible endoscopes are particularly difficult to disinfect¹²² and easy to damage because of their intricate design and delicate materials.¹²³ Meticulous cleaning must precede any sterilization or high-level disinfection of these instruments. Failure to perform good cleaning can result in sterilization or disinfection failure, and outbreaks of infection can occur. Several studies have demonstrated the importance of cleaning in experimental studies with the duck hepatitis B virus (HBV)^{106, 124}, HIV¹²⁵ and *Helicobacter pylori*.¹²⁶

An examination of health-care-associated infections related only to endoscopes through July 1992 found 281 infections transmitted by gastrointestinal endoscopy and 96 transmitted by bronchoscopy. The clinical spectrum ranged from asymptomatic colonization to death. *Salmonella* species and *Pseudomonas aeruginosa* repeatedly were identified as causative agents of infections transmitted by gastrointestinal endoscopy, and *M. tuberculosis*, atypical mycobacteria, and *P. aeruginosa* were the most common causes of infections transmitted by bronchoscopy¹². Major reasons for transmission were inadequate cleaning, improper selection of a disinfecting agent, and failure to follow recommended cleaning and disinfection procedures^{6, 8, 37, 98}, and flaws in endoscope design^{127, 128} or automated endoscope reprocessors.^{7, 98} Failure to follow established guidelines has continued to result in infections associated with gastrointestinal endoscopes⁸ and bronchoscopes^{7, 12}. Potential device-associated problems should be reported to the FDA Center for Devices and Radiologic Health. One multistate investigation found that 23.9% of the bacterial cultures from the internal channels of 71 gastrointestinal endoscopes grew $\geq 100,000$ colonies of bacteria after completion of all disinfection and sterilization procedures (nine of 25 facilities were using a product that has been removed from the marketplace [six facilities using 1:16 glutaraldehyde phenate], is not FDA-cleared as a high-level disinfectant [an iodophor] or no disinfecting agent) and before use on the next patient¹²⁹. The incidence of postendoscopic procedure infections from an improperly processed endoscope has not been rigorously assessed.

Automated endoscope reprocessors (AER) offer several advantages over manual reprocessing: they automate and standardize several important reprocessing steps¹³⁰⁻¹³², reduce the likelihood that an essential reprocessing step will be skipped, and reduce personnel exposure to high-level disinfectants or chemical sterilants. Failure of AERs has been linked to outbreaks of infections¹³³ or colonization^{7, 134}, and the AER water filtration system might not be able to reliably provide “sterile” or bacteria-free rinse water^{135, 136}. Establishment of correct connectors between the AER and the device is critical to ensure complete flow of disinfectants and rinse water^{7, 137}. In addition, some endoscopes such as the duodenoscopes (e.g., endoscopic retrograde cholangiopancreatography [ERCP]) contain features (e.g., elevator-wire channel) that require a flushing pressure that is not achieved by most AERs and must be reprocessed manually using a 2- to 5-mL syringe, until new duodenoscopes equipped with a wider elevator-channel that AERs can reliably reprocess become available¹³². Outbreaks involving removable endoscope parts^{138, 139} such as suction valves and endoscopic accessories designed to be inserted through flexible endoscopes such as biopsy forceps emphasize the importance of cleaning to remove all foreign matter before high-level disinfection or sterilization.¹⁴⁰ Some types of valves are now available as single-use, disposable products (e.g., bronchoscope valves) or steam sterilizable products (e.g., gastrointestinal endoscope valves).

AERs need further development and redesign^{7, 141}, as do endoscopes^{123, 142}, so that they do not represent a potential source of infectious agents. Endoscopes employing disposable components (e.g., protective barrier devices or sheaths) might provide an alternative to conventional liquid chemical high-level disinfection/sterilization^{143, 144}. Another new technology is a swallowable camera-in-a-capsule that travels through the digestive tract and transmits color pictures of the small intestine to a receiver worn outside the body. This capsule currently does not replace colonoscopies.

Published recommendations for cleaning and disinfecting endoscopic equipment should be strictly followed^{12, 38, 108, 113-116, 145-148}. Unfortunately, audits have shown that personnel do not consistently adhere to guidelines on reprocessing¹⁴⁹⁻¹⁵¹ and outbreaks of infection continue to occur.¹⁵²⁻¹⁵⁴ To ensure

reprocessing personnel are properly trained, each person who reprocesses endoscopic instruments should receive initial and annual competency testing^{38, 155}.

In general, endoscope disinfection or sterilization with a liquid chemical sterilant involves five steps after leak testing:

1. Clean: mechanically clean internal and external surfaces, including brushing internal channels and flushing each internal channel with water and a detergent or enzymatic cleaners (leak testing is recommended for endoscopes before immersion).
2. Disinfect: immerse endoscope in high-level disinfectant (or chemical sterilant) and perfuse (eliminates air pockets and ensures contact of the germicide with the internal channels) disinfectant into all accessible channels, such as the suction/biopsy channel and air/water channel and expose for a time recommended for specific products.
3. Rinse: rinse the endoscope and all channels with sterile water, filtered water (commonly used with AERs) or tap water (i.e., high-quality potable water that meets federal clean water standards at the point of use).
4. Dry: rinse the insertion tube and inner channels with alcohol, and dry with forced air after disinfection and before storage.

Store: store the endoscope in a way that prevents recontamination and promotes drying (e.g., hung vertically). Drying the endoscope (steps 3 and 4) is essential to greatly reduce the chance of recontamination of the endoscope by microorganisms that can be present in the rinse water^{116, 156}. One study demonstrated that reprocessed endoscopes (i.e., air/water channel, suction/biopsy channel) generally were negative (100% after 24 hours; 90% after 7 days [1 CFU of coagulase-negative *Staphylococcus* in one channel]) for bacterial growth when stored by hanging vertically in a ventilated cabinet¹⁵⁷. Other investigators found all endoscopes were bacteria-free immediately after high-level disinfection, and only four of 135 scopes were positive during the subsequent 5-day assessment (skin bacteria cultured from endoscope surfaces). All flush-through samples remained sterile¹⁵⁸. Because tapwater can contain low levels of microorganisms¹⁵⁹, some researchers have suggested that only sterile water (which can be prohibitively expensive)¹⁶⁰ or AER filtered water be used. The suggestion to use only sterile water or filtered water is not consistent with published guidelines that allow tapwater with an alcohol rinse and forced air-drying^{38, 108, 113} or the scientific literature.^{39, 93} In addition, no evidence of disease transmission has been found when a tap water rinse is followed by an alcohol rinse and forced-air drying. AERs produce filtered water by passage through a bacterial filter (e.g., 0.2 μ). Filtered rinse water was identified as a source of bacterial contamination in a study that cultured the accessory and suction channels of endoscopes and the internal chambers of AERs during 1996–2001 and reported 8.7% of samples collected during 1996–1998 had bacterial growth, with 54% being *Pseudomonas* species. After a system of hot water flushing of the piping (60°C for 60 minutes daily) was introduced, the frequency of positive cultures fell to approximately 2% with only rare isolation of >10 CFU/mL¹⁶¹. In addition to the endoscope reprocessing steps, a protocol should be developed that ensures the user knows whether an endoscope has been appropriately cleaned and disinfected (e.g., using a room or cabinet for processed endoscopes only) or has not been reprocessed. When users leave endoscopes on movable carts, confusion can result about whether the endoscope has been processed. Although one guideline recommended endoscopes (e.g., duodenoscopes) be reprocessed immediately before use¹⁴⁷, other guidelines do not require this activity^{38, 108, 115} and except for the Association of periOperative Registered Nurses (AORN), professional organizations do not recommend that reprocessing be repeated as long as the original processing is done correctly. As part of a quality assurance program, healthcare facility personnel can consider random bacterial surveillance cultures of processed endoscopes to ensure high-level disinfection or sterilization^{7, 162-164}. Reprocessed endoscopes should be free of microbial pathogens except for small numbers of relatively avirulent microbes that represent exogenous environmental contamination (e.g., coagulase-negative *Staphylococcus*, *Bacillus* species, diphtheroids). Although recommendations exist for the final rinse water used during endoscope reprocessing to be microbiologically cultured at least monthly¹⁶⁵, a microbiologic standard has not been

set, and the value of routine endoscope cultures has not been shown¹⁶⁶. In addition, neither the routine culture of reprocessed endoscopes nor the final rinse water has been validated by correlating viable counts on an endoscope to infection after an endoscopic procedure. If reprocessed endoscopes were cultured, sampling the endoscope would assess water quality and other important steps (e.g., disinfectant effectiveness, exposure time, cleaning) in the reprocessing procedure. A number of methods for sampling endoscopes and water have been described^{23, 157, 161, 163, 167, 168}. Novel approaches (e.g., detection of adenosine triphosphate [ATP]) to evaluate the effectiveness of endoscope cleaning^{169, 170} or endoscope reprocessing¹⁷¹ also have been evaluated, but no method has been established as a standard for assessing the outcome of endoscope reprocessing.

The carrying case used to transport clean and reprocessed endoscopes outside the health-care environment should not be used to store an endoscope or to transport the instrument within the health-care facility. A contaminated endoscope should never be placed in the carrying case because the case can also become contaminated. When the endoscope is removed from the case, properly reprocessed, and put back in the case, the case could recontaminate the endoscope. A contaminated carrying case should be discarded (Olympus America, June 2002, written communication).

Infection-control professionals should ensure that institutional policies are consistent with national guidelines and conduct infection-control rounds periodically (e.g., at least annually) in areas where endoscopes are reprocessed to ensure policy compliance. Breaches in policy should be documented and corrective action instituted. In incidents in which endoscopes were not exposed to a high-level disinfection process, patients exposed to potentially contaminated endoscopes have been assessed for possible acquisition of HIV, HBV, and hepatitis C virus (HCV). A 14-step method for managing a failure incident associated with high-level disinfection or sterilization has been described [Rutala WA, 2006 #12512]. The possible transmission of bloodborne and other infectious agents highlights the importance of rigorous infection control^{172, 173}.

Laparoscopes and Arthroscopes

Although high-level disinfection appears to be the minimum standard for processing laparoscopes and arthroscopes between patients^{28, 86, 174, 175}, this practice continues to be debated^{89, 90, 176}. However, neither side in the high-level disinfection versus sterilization debate has sufficient data on which to base its conclusions. Proponents of high-level disinfection refer to membership surveys²⁹ or institutional experiences⁸⁷ involving more than 117,000 and 10,000 laparoscopic procedures, respectively, that cite a low risk for infection (<0.3%) when high-level disinfection is used for gynecologic laparoscopic equipment. Only one infection in the membership survey was linked to spores. In addition, growth of common skin microorganisms (e.g., *Staphylococcus epidermidis*, diphtheroids) has been documented from the umbilical area even after skin preparation with povidone-iodine and ethyl alcohol. Similar organisms were recovered in some instances from the pelvic serosal surfaces or from the laparoscopic telescopes, suggesting that the microorganisms probably were carried from the skin into the peritoneal cavity^{177, 178}. Proponents of sterilization focus on the possibility of transmitting infection by spore-forming organisms. Researchers have proposed several reasons why sterility was not necessary for all laparoscopic equipment: only a limited number of organisms (usually ≤ 10) are introduced into the peritoneal cavity during laparoscopy; minimal damage is done to inner abdominal structures with little devitalized tissue; the peritoneal cavity tolerates small numbers of spore-forming bacteria; equipment is simple to clean and disinfect; surgical sterility is relative; the natural bioburden on rigid lumened devices is low¹⁷⁹; and no evidence exists that high-level disinfection instead of sterilization increases the risk for infection^{87, 89, 90}. With the advent of laparoscopic cholecystectomy, concern about high-level disinfection is justifiable because the degree of tissue damage and bacterial contamination is greater than with laparoscopic procedures in gynecology. Failure to completely disassemble, clean, and high-level disinfect laparoscope parts has led to infections in patients¹⁸⁰. Data from one study suggested that disassembly, cleaning, and proper reassembly of laparoscopic equipment used in gynecologic procedures before steam sterilization presents no risk for infection¹⁸¹.

As with laparoscopes and other equipment that enter sterile body sites, arthroscopes ideally should be sterilized before used. Older studies demonstrated that these instruments were commonly (57%) only high-level disinfected in the United States^{28, 86}. A later survey (with a response rate of only 5%) reported that high-level disinfection was used by 31% and a sterilization process in the remainder of the health-care facilities³⁰. High-level disinfection rather than sterilization presumably has been used because the incidence of infection is low and the few infections identified probably are unrelated to the use of high-level disinfection rather than sterilization. A retrospective study of 12,505 arthroscopic procedures found an infection rate of 0.04% (five infections) when arthroscopes were soaked in 2% glutaraldehyde for 15–20 minutes. Four infections were caused by *S. aureus*; the fifth was an anaerobic streptococcal infection⁸⁸. Because these organisms are very susceptible to high-level disinfectants, such as 2% glutaraldehyde, the infections most likely originated from the patient's skin. Two cases of *Clostridium perfringens* arthritis have been reported when the arthroscope was disinfected with glutaraldehyde for an exposure time that is not effective against spores^{182, 183}.

Although only limited data are available, the evidence does not demonstrate that high-level disinfection of arthroscopes and laparoscopes poses an infection risk to the patient. For example, a prospective study that compared the reprocessing of arthroscopes and laparoscopes (per 1,000 procedures) with EtO sterilization to high-level disinfection with glutaraldehyde found no statistically significant difference in infection risk between the two methods (i.e., EtO, 7.5/1,000 procedures; glutaraldehyde, 2.5/1,000 procedures)⁸⁹. Although the debate for high-level disinfection versus sterilization of laparoscopes and arthroscopes will go unsettled until well-designed, randomized clinical trials are published, this guideline should be followed^{1, 17}. That is, laparoscopes, arthroscopes, and other scopes that enter normally sterile tissue should be sterilized before each use; if this is not feasible, they should receive at least high-level disinfection.

Tonometers, Cervical Diaphragm Fitting Rings, Cryosurgical Instruments, and Endocavitary Probes

Disinfection strategies vary widely for other semicritical items (e.g., applanation tonometers, rectal/vaginal probes, cryosurgical instruments, and diaphragm fitting rings). FDA requests that device manufacturers include at least one validated cleaning and disinfection/sterilization protocol in the labeling for their devices. As with all medications and devices, users should be familiar with the label instructions. One study revealed that no uniform technique was in use for disinfection of applanation tonometers, with disinfectant contact times varying from <15 sec to 20 minutes²⁸. In view of the potential for transmission of viruses (e.g., herpes simplex virus [HSV], adenovirus 8, or HIV)¹⁸⁴ by tonometer tips, CDC recommended that the tonometer tips be wiped clean and disinfected for 5–10 minutes with either 3% hydrogen peroxide, 5000 ppm chlorine, 70% ethyl alcohol, or 70% isopropyl alcohol⁹⁵. However, more recent data suggest that 3% hydrogen peroxide and 70% isopropyl alcohol are not effective against adenovirus capable of causing epidemic keratoconjunctivitis and similar viruses and should not be used for disinfecting applanation tonometers^{49, 185, 186}. Structural damage to Schiottz tonometers has been observed with a 1:10 sodium hypochlorite (5,000 ppm chlorine) and 3% hydrogen peroxide¹⁸⁷. After disinfection, the tonometer should be thoroughly rinsed in tapwater and air dried before use. Although these disinfectants and exposure times should kill pathogens that can infect the eyes, no studies directly support this^{188, 189}. The guidelines of the American Academy of Ophthalmology for preventing infections in ophthalmology focus on only one potential pathogen: HIV.¹⁹⁰ Because a short and simple decontamination procedure is desirable in the clinical setting, swabbing the tonometer tip with a 70% isopropyl alcohol wipe sometimes is practiced.¹⁸⁹ Preliminary reports suggest that wiping the tonometer tip with an alcohol swab and then allowing the alcohol to evaporate might be effective in eliminating HSV, HIV, and adenovirus^{189, 191, 192}. However, because these studies involved only a few replicates and were conducted in a controlled laboratory setting, further studies are needed before this technique can be recommended. In addition, two reports have found that disinfection of pneumotonometer tips between uses with a 70% isopropyl alcohol wipe contributed to outbreaks of epidemic keratoconjunctivitis caused

by adenovirus type 8^{193, 194}.

Limited studies have evaluated disinfection techniques for other items that contact mucous membranes, such as diaphragm fitting rings, cryosurgical probes, transesophageal echocardiography probes¹⁹⁵, flexible cystoscopes¹⁹⁶ or vaginal/rectal probes used in sonographic scanning. Lettau, Bond, and McDougal of CDC supported the recommendation of a diaphragm fitting ring manufacturer that involved using a soap-and-water wash followed by a 15-minute immersion in 70% alcohol⁹⁶. This disinfection method should be adequate to inactivate HIV, HBV, and HSV even though alcohols are not classified as high-level disinfectants because their activity against picornaviruses is somewhat limited⁷². No data are available regarding inactivation of human papillomavirus (HPV) by alcohol or other disinfectants because in vitro replication of complete virions has not been achieved. Thus, even though alcohol for 15 minutes should kill pathogens of relevance in gynecology, no clinical studies directly support this practice.

Vaginal probes are used in sonographic scanning. A vaginal probe and all endocavitary probes without a probe cover are semicritical devices because they have direct contact with mucous membranes (e.g., vagina, rectum, pharynx). While use of the probe cover could be considered as changing the category, this guideline proposes use of a new condom/probe cover for the probe for each patient, and because condoms/probe covers can fail^{195, 197-199}, the probe also should be high-level disinfected. The relevance of this recommendation is reinforced with the findings that sterile transvaginal ultrasound probe covers have a very high rate of perforations even before use (0%, 25%, and 65% perforations from three suppliers).¹⁹⁹ One study found, after oocyte retrieval use, a very high rate of perforations in used endovaginal probe covers from two suppliers (75% and 81%)¹⁹⁹, other studies demonstrated a lower rate of perforations after use of condoms (2.0% and 0.9%)^{197 200}. Condoms have been found superior to commercially available probe covers for covering the ultrasound probe (1.7% for condoms versus 8.3% leakage for probe covers)²⁰¹. These studies underscore the need for routine probe disinfection between examinations. Although most ultrasound manufacturers recommend use of 2% glutaraldehyde for high-level disinfection of contaminated transvaginal transducers, the this agent has been questioned²⁰² because it might shorten the life of the transducer and might have toxic effects on the gametes and embryos²⁰³. An alternative procedure for disinfecting the vaginal transducer involves the mechanical removal of the gel from the transducer, cleaning the transducer in soap and water, wiping the transducer with 70% alcohol or soaking it for 2 minutes in 500 ppm chlorine, and rinsing with tap water and air drying²⁰⁴. The effectiveness of this and other methods²⁰⁰ has not been validated in either rigorous laboratory experiments or in clinical use. High-level disinfection with a product (e.g., hydrogen peroxide) that is not toxic to staff, patients, probes, and retrieved cells should be used until the effectiveness of alternative procedures against microbes of importance at the cavitory site is demonstrated by well-designed experimental scientific studies. Other probes such as rectal, cryosurgical, and transesophageal probes or devices also should be high-level disinfected between patients.

Ultrasound probes used during surgical procedures also can contact sterile body sites. These probes can be covered with a sterile sheath to reduce the level of contamination on the probe and reduce the risk for infection. However, because the sheath does not completely protect the probe, the probes should be sterilized between each patient use as with other critical items. If this is not possible, at a minimum the probe should be high-level disinfected and covered with a sterile probe cover.

Some cryosurgical probes are not fully immersible. During reprocessing, the tip of the probe should be immersed in a high-level disinfectant for the appropriate time; any other portion of the probe that could have mucous membrane contact can be disinfected by immersion or by wrapping with a cloth soaked in a high-level disinfectant to allow the recommended contact time. After disinfection, the probe should be rinsed with tap water and dried before use. Health-care facilities that use nonimmersible probes should replace them as soon as possible with fully immersible probes.

As with other high-level disinfection procedures, proper cleaning of probes is necessary to ensure the success of the subsequent disinfection²⁰⁵. One study demonstrated that vegetative bacteria

inoculated on vaginal ultrasound probes decreased when the probes were cleaned with a towel²⁰⁶. No information is available about either the level of contamination of such probes by potential viral pathogens such as HBV and HPV or their removal by cleaning (such as with a towel). Because these pathogens might be present in vaginal and rectal secretions and contaminate probes during use, high-level disinfection of the probes after such use is recommended.

Dental Instruments

Scientific articles and increased publicity about the potential for transmitting infectious agents in dentistry have focused attention on dental instruments as possible agents for pathogen transmission²⁰⁷.²⁰⁸ The American Dental Association recommends that surgical and other instruments that normally penetrate soft tissue or bone (e.g., extraction forceps, scalpel blades, bone chisels, periodontal scalers, and surgical burs) be classified as critical devices that should be sterilized after each use or discarded. Instruments not intended to penetrate oral soft tissues or bone (e.g., amalgam condensers, and air/water syringes) but that could contact oral tissues are classified as semicritical, but sterilization after each use is recommended if the instruments are heat-tolerant^{43, 209}. If a semicritical item is heat-sensitive, it should, at a minimum, be processed with high-level disinfection^{43, 210}. Handpieces can be contaminated internally with patient material and should be heat sterilized after each patient. Handpieces that cannot be heat sterilized should not be used.²¹¹ Methods of sterilization that can be used for critical or semicritical dental instruments and materials that are heat-stable include steam under pressure (autoclave), chemical (formaldehyde) vapor, and dry heat (e.g., 320°F for 2 hours). Dental professionals most commonly use the steam sterilizer²¹². All three sterilization procedures can damage some dental instruments, including steam-sterilized hand pieces²¹³. Heat-tolerant alternatives are available for most clinical dental applications and are preferred⁴³.

CDC has divided noncritical surfaces in dental offices into clinical contact and housekeeping surfaces⁴³. Clinical contact surfaces are surfaces that might be touched frequently with gloved hands during patient care or that might become contaminated with blood or other potentially infectious material and subsequently contact instruments, hands, gloves, or devices (e.g., light handles, switches, dental X-ray equipment, chair-side computers). Barrier protective coverings (e.g., clear plastic wraps) can be used for these surfaces, particularly those that are difficult to clean (e.g., light handles, chair switches). The coverings should be changed when visibly soiled or damaged and routinely (e.g., between patients). Protected surfaces should be disinfected at the end of each day or if contamination is evident. If not barrier-protected, these surfaces should be disinfected between patients with an intermediate-disinfectant (i.e., EPA-registered hospital disinfectant with tuberculocidal claim) or low-level disinfectant (i.e., EPA-registered hospital disinfectant with an HBV and HIV label claim)^{43, 214, 215}.

Most housekeeping surfaces need to be cleaned only with a detergent and water or an EPA-registered hospital disinfectant, depending of the nature of the surface and the type and degree of contamination. When housekeeping surfaces are visibly contaminated by blood or body substances, however, prompt removal and surface disinfection is a sound infection control practice and required by the Occupational Safety and Health Administration (OSHA)^{43, 214}.

Several studies have demonstrated variability among dental practices while trying to meet these recommendations^{216, 217}. For example, 68% of respondents believed they were sterilizing their instruments but did not use appropriate chemical sterilants or exposure times and 49% of respondents did not challenge autoclaves with biological indicators²¹⁶. Other investigators using biologic indicators have found a high proportion (15%–65%) of positive spore tests after assessing the efficacy of sterilizers used in dental offices. In one study of Minnesota dental offices, operator error, rather than mechanical malfunction²¹⁸, caused 87% of sterilization failures. Common factors in the improper use of sterilizers include chamber overload, low temperature setting, inadequate exposure time, failure to preheat the sterilizer, and interruption of the cycle.

Mail-return sterilization monitoring services use spore strips to test sterilizers in dental clinics, but

delay caused by mailing to the test laboratory could potentially cause false-negative results. Studies revealed, however, that the post-sterilization time and temperature after a 7-day delay had no influence on the test results²¹⁹. Delays (7 days at 27°C and 37°C, 3-day mail delay) did not cause any predictable pattern of inaccurate spore tests²²⁰.

Disinfection of HBV-, HCV-, HIV- or TB-Contaminated Devices

The CDC recommendation for high-level disinfection of HBV-, HCV-, HIV- or TB-contaminated devices is appropriate because experiments have demonstrated the effectiveness of high-level disinfectants to inactivate these and other pathogens that might contaminate semicritical devices^{61, 62, 73, 81, 105, 121, 125, 221-238}. Nonetheless, some healthcare facilities have modified their disinfection procedures when endoscopes are used with a patient known or suspected to be infected with HBV, HIV, or *M. tuberculosis*^{28, 239}. This is inconsistent with the concept of Standard Precautions that presumes all patients are potentially infected with bloodborne pathogens²²⁸. Several studies have highlighted the inability to distinguish HBV- or HIV-infected patients from noninfected patients on clinical grounds²⁴⁰⁻²⁴². In addition, mycobacterial infection is unlikely to be clinically apparent in many patients. In most instances, hospitals that altered their disinfection procedure used EtO sterilization on the endoscopic instruments because they believed this practice reduced the risk for infection^{28, 239}. EtO is not routinely used for endoscope sterilization because of the lengthy processing time. Endoscopes and other semicritical devices should be managed the same way regardless of whether the patient is known to be infected with HBV, HCV, HIV or *M. tuberculosis*.

An evaluation of a manual disinfection procedure to eliminate HCV from experimentally contaminated endoscopes provided some evidence that cleaning and 2% glutaraldehyde for 20 minutes should prevent transmission²³⁶. A study that used experimentally contaminated hysteroscopes detected HCV by polymerase chain reaction (PCR) in one (3%) of 34 samples after cleaning with a detergent, but no samples were positive after treatment with a 2% glutaraldehyde solution for 20 minutes¹²⁰. Another study demonstrated complete elimination of HCV (as detected by PCR) from endoscopes used on chronically infected patients after cleaning and disinfection for 3–5 minutes in glutaraldehyde¹¹⁸. Similarly, PCR was used to demonstrate complete elimination of HCV after standard disinfection of experimentally contaminated endoscopes²³⁶ and endoscopes used on HCV-antibody-positive patients had no detectable HCV RNA after high-level disinfection²⁴³. The inhibitory activity of a phenolic and a chlorine compound on HCV showed that the phenolic inhibited the binding and replication of HCV, but the chlorine was ineffective, probably because of its low concentration and its neutralization in the presence of organic matter²⁴⁴.

Disinfection in the Hemodialysis Unit

Hemodialysis systems include hemodialysis machines, water supply, water-treatment systems, and distribution systems. During hemodialysis, patients have acquired bloodborne viruses and pathogenic bacteria²⁴⁵⁻²⁴⁷. Cleaning and disinfection are important components of infection control in a hemodialysis center. EPA and FDA regulate disinfectants used to reprocess hemodialyzers, hemodialysis machines, and water-treatment systems.

Noncritical surfaces (e.g., dialysis bed or chair, countertops, external surfaces of dialysis machines, and equipment [scissors, hemostats, clamps, blood pressure cuffs, stethoscopes]) should be disinfected with an EPA-registered disinfectant unless the item is visibly contaminated with blood; in that case a tuberculocidal agent (or a disinfectant with specific label claims for HBV and HIV) or a 1:100 dilution of a hypochlorite solution (500–600 ppm free chlorine) should be used^{246, 248}. This procedure accomplishes two goals: it removes soil on a regular basis and maintains an environment that is consistent with good patient care. Hemodialyzers are disinfected with peracetic acid, formaldehyde, glutaraldehyde, heat pasteurization with citric acid, and chlorine-containing compounds²⁴⁹. Hemodialysis systems usually are disinfected by chlorine-based disinfectants (e.g., sodium hypochlorite), aqueous

formaldehyde, heat pasteurization, ozone, or peracetic acid^{250, 251}. All products must be used according to the manufacturers' recommendations. Some dialysis systems use hot-water disinfection to control microbial contamination.

At its high point, 82% of U.S. chronic hemodialysis centers were reprocessing (i.e., reusing) dialyzers for the same patient using high-level disinfection²⁴⁹. However, one of the large dialysis organizations has decided to phase out reuse and, by 2002 the percentage of dialysis facilities reprocessing hemodialyzers had decreased to 63%²⁵². The two commonly used disinfectants to reprocess dialyzers were peracetic acid and formaldehyde; 72% used peracetic acid and 20% used formaldehyde to disinfect hemodialyzers. Another 4% of the facilities used either glutaraldehyde or heat pasteurization in combination with citric acid²⁵². Infection-control recommendations, including disinfection and sterilization and the use of dedicated machines for hepatitis B surface antigen (HBsAg)-positive patients, in the hemodialysis setting were detailed in two reviews^{245, 246}. The Association for the Advancement of Medical Instrumentation (AAMI) has published recommendations for the reuse of hemodialyzers²⁵³.

Inactivation of *Clostridium difficile*

The source of health-care-associated acquisition of *Clostridium difficile* in nonepidemic settings has not been determined. The environment and carriage on the hands of health-care personnel have been considered possible sources of infection^{66, 254}. Carpeted rooms occupied by a patient with *C. difficile* were more heavily contaminated with *C. difficile* than were noncarpeted rooms²⁵⁵. Because *C. difficile* spore-production can increase when exposed to nonchlorine-based cleaning agents and the spores are more resistant than vegetative cells to commonly used surface disinfectants²⁵⁶, some investigators have recommended use of dilute solutions of hypochlorite (1,600 ppm available chlorine) for routine environmental disinfection of rooms of patients with *C. difficile*-associated diarrhea or colitis²⁵⁷, to reduce the incidence of *C. difficile* diarrhea²⁵⁸, or in units with high *C. difficile* rates.²⁵⁹ Stool samples of patients with symptomatic *C. difficile* colitis contain spores of the organism, as demonstrated by ethanol treatment of the stool to reduce the overgrowth of fecal flora when isolating *C. difficile* in the laboratory^{260, 261}. *C. difficile*-associated diarrhea rates were shown to have decreased markedly in a bone-marrow transplant unit (from 8.6 to 3.3 cases per 1,000 patient-days) during a period of bleach disinfection (1:10 dilution) of environmental surfaces compared with cleaning with a quaternary ammonium compound. Because no EPA-registered products exist that are specific for inactivating *C. difficile* spores, use of diluted hypochlorite should be considered in units with high *C. difficile* rates. Acidified bleach and regular bleach (5000 ppm chlorine) can inactivate 10^6 *C. difficile* spores in ≤ 10 minutes²⁶². However, studies have shown that asymptomatic patients constitute an important reservoir within the health-care facility and that person-to-person transmission is the principal means of transmission between patients. Thus, combined use of hand washing, barrier precautions, and meticulous environmental cleaning with an EPA-registered disinfectant (e.g., germicidal detergent) should effectively prevent spread of the organism²⁶³.

Contaminated medical devices, such as colonoscopes and thermometers, can be vehicles for transmission of *C. difficile* spores²⁶⁴. For this reason, investigators have studied commonly used disinfectants and exposure times to assess whether current practices can place patients at risk. Data demonstrate that 2% glutaraldehyde^{79, 265-267} and peracetic acid^{267, 268} reliably kill *C. difficile* spores using exposure times of 5–20 minutes. *ortho*-Phthalaldehyde and $\geq 0.2\%$ peracetic acid (WA Rutala, personal communication, April 2006) also can inactivate $\geq 10^4$ *C. difficile* spores in 10–12 minutes at 20°C²⁶⁸. Sodium dichloroisocyanurate at a concentration of 1000 ppm available chlorine achieved lower \log_{10} reduction factors against *C. difficile* spores at 10 min, ranging from 0.7 to 1.5, than 0.26% peracetic acid with \log_{10} reduction factors ranging from 2.7 to 6.0²⁶⁸.

OSHA Bloodborne Pathogen Standard

In December 1991, OSHA promulgated a standard entitled "Occupational Exposure to

Bloodborne Pathogens” to eliminate or minimize occupational exposure to bloodborne pathogens²¹⁴. One component of this requirement is that all equipment and environmental and working surfaces be cleaned and decontaminated with an appropriate disinfectant after contact with blood or other potentially infectious materials. Even though the OSHA standard does not specify the type of disinfectant or procedure, the OSHA original compliance document²⁶⁹ suggested that a germicide must be tuberculocidal to kill the HBV. To follow the OSHA compliance document a tuberculocidal disinfectant (e.g., phenolic, and chlorine) would be needed to clean a blood spill. However, in February 1997, OSHA amended its policy and stated that EPA-registered disinfectants labeled as effective against HIV and HBV would be considered as appropriate disinfectants “. . . provided such surfaces have not become contaminated with agent(s) or volumes of or concentrations of agent(s) for which higher level disinfection is recommended.” When bloodborne pathogens other than HBV or HIV are of concern, OSHA continues to require use of EPA-registered tuberculocidal disinfectants or hypochlorite solution (diluted 1:10 or 1:100 with water)^{215, 228}. Studies demonstrate that, in the presence of large blood spills, a 1:10 final dilution of EPA-registered hypochlorite solution initially should be used to inactivate bloodborne viruses^{63, 235} to minimize risk for infection to health-care personnel from percutaneous injury during cleanup.

Emerging Pathogens (*Cryptosporidium*, *Helicobacter pylori*, *Escherichia coli* O157:H7, Rotavirus, Human Papilloma Virus, Norovirus, Severe Acute Respiratory Syndrome [SARS] Coronavirus)

Emerging pathogens are of growing concern to the general public and infection-control professionals. Relevant pathogens include *Cryptosporidium parvum*, *Helicobacter pylori*, *E. coli* O157:H7, HIV, HCV, rotavirus, norovirus, severe acute respiratory syndrome (SARS) coronavirus, multidrug-resistant *M. tuberculosis*, and nontuberculous mycobacteria (e.g., *M. chelonae*). The susceptibility of each of these pathogens to chemical disinfectants and sterilants has been studied. With the exceptions discussed below, all of these emerging pathogens are susceptible to currently available chemical disinfectants and sterilants²⁷⁰.

Cryptosporidium is resistant to chlorine at concentrations used in potable water. *C. parvum* is not completely inactivated by most disinfectants used in healthcare including ethyl alcohol²⁷¹, glutaraldehyde^{271, 272}, 5.25% hypochlorite²⁷¹, peracetic acid²⁷¹, ortho-phthalaldehyde²⁷¹, phenol^{271, 272}, povidone-iodine^{271, 272}, and quaternary ammonium compounds²⁷¹. The only chemical disinfectants and sterilants able to inactivate greater than 3 log₁₀ of *C. parvum* were 6% and 7.5% hydrogen peroxide²⁷¹. Sterilization methods will fully inactivate *C. parvum*, including steam²⁷¹, EtO^{271, 273}, and hydrogen peroxide gas plasma²⁷¹. Although most disinfectants are ineffective against *C. parvum*, current cleaning and disinfection practices appear satisfactory to prevent healthcare-associated transmission. For example, endoscopes are unlikely to be an important vehicle for transmitting *C. parvum* because the results of bacterial studies indicate mechanical cleaning will remove approximately 10⁴ organisms, and drying results in rapid loss of *C. parvum* viability (e.g., 30 minutes, 2.9 log₁₀ decrease; and 60 minutes, 3.8 log₁₀ decrease)²⁷¹.

Chlorine at ~1 ppm has been found capable of eliminating approximately 4 log₁₀ of *E. coli* O157:H7 within 1 minute in a suspension test⁶⁴. Electrolyzed oxidizing water at 23°C was effective in 10 minutes in producing a 5-log₁₀ decrease in *E. coli* O157:H7 inoculated onto kitchen cutting boards²⁷⁴. The following disinfectants eliminated >5 log₁₀ of *E. coli* O157:H7 within 30 seconds: a quaternary ammonium compound, a phenolic, a hypochlorite (1:10 dilution of 5.25% bleach), and ethanol⁵³. Disinfectants including chlorine compounds can reduce *E. coli* O157:H7 experimentally inoculated onto alfalfa seeds or sprouts^{275, 276} or beef carcass surfaces²⁷⁷.

Data are limited on the susceptibility of *H. pylori* to disinfectants. Using a suspension test, one study assessed the effectiveness of a variety of disinfectants against nine strains of *H. pylori*⁶⁰. Ethanol (80%) and glutaraldehyde (0.5%) killed all strains within 15 seconds; chlorhexidine gluconate (0.05%, 1.0%), benzalkonium chloride (0.025%, 0.1%), alkyldiaminoethylglycine hydrochloride (0.1%), povidone-iodine (0.1%), and sodium hypochlorite (150 ppm) killed all strains within 30 seconds. Both ethanol

(80%) and glutaraldehyde (0.5%) retained similar bactericidal activity in the presence of organic matter; the other disinfectants showed reduced bactericidal activity. In particular, the bactericidal activity of povidone-iodine (0.1%) and sodium hypochlorite (150 ppm) markedly decreased in the presence of dried yeast solution with killing times increased to 5 - 10 minutes and 5 - 30 minutes, respectively.

Immersing biopsy forceps in formalin before obtaining a specimen does not affect the ability to culture *H. pylori* from the biopsy specimen²⁷⁸. The following methods are ineffective for eliminating *H. pylori* from endoscopes: cleaning with soap and water^{119, 279}, immersion in 70% ethanol for 3 minutes²⁸⁰, instillation of 70% ethanol¹²⁶, instillation of 30 ml of 83% methanol²⁷⁹, and instillation of 0.2% Hyamine solution²⁸¹. The differing results with regard to the efficacy of ethyl alcohol against *Helicobacter* are unexplained. Cleaning followed by use of 2% alkaline glutaraldehyde (or automated peracetic acid) has been demonstrated by culture to be effective in eliminating *H. pylori*^{119, 279, 282}. Epidemiologic investigations of patients who had undergone endoscopy with endoscopes mechanically washed and disinfected with 2.0%–2.3% glutaraldehyde have revealed no evidence of person-to-person transmission of *H. pylori*^{126, 283}. Disinfection of experimentally contaminated endoscopes using 2% glutaraldehyde (10-minute, 20-minute, 45-minute exposure times) or the peracetic acid system (with and without active peracetic acid) has been demonstrated to be effective in eliminating *H. pylori*¹¹⁹. *H. pylori* DNA has been detected by PCR in fluid flushed from endoscope channels after cleaning and disinfection with 2% glutaraldehyde²⁸⁴. The clinical significance of this finding is unclear. *In vitro* experiments have demonstrated a $>3.5\text{-log}_{10}$ reduction in *H. pylori* after exposure to 0.5 mg/L of free chlorine for 80 seconds²⁸⁵.

An outbreak of healthcare-associated rotavirus gastroenteritis on a pediatric unit has been reported²⁸⁶. Person to person through the hands of health-care workers was proposed as the mechanism of transmission. Prolonged survival of rotavirus on environmental surfaces (90 minutes to >10 days at room temperature) and hands (>4 hours) has been demonstrated. Rotavirus suspended in feces can survive longer^{287, 288}. Vectors have included hands, fomites, air, water, and food^{288, 289}. Products with demonstrated efficacy ($>3\text{ log}_{10}$ reduction in virus) against rotavirus within 1 minute include: 95% ethanol, 70% isopropanol, some phenolics, 2% glutaraldehyde, 0.35% peracetic acid, and some quaternary ammonium compounds^{59, 290-293}. In a human challenge study, a disinfectant spray (0.1% ortho-phenylphenol and 79% ethanol), sodium hypochlorite (800 ppm free chlorine), and a phenol-based product (14.7% phenol diluted 1:256 in tapwater) when sprayed onto contaminated stainless steel disks, were effective in interrupting transfer of a human rotavirus from stainless steel disk to fingerpads of volunteers after an exposure time of 3- 10 minutes. A quaternary ammonium product (7.05% quaternary ammonium compound diluted 1:128 in tapwater) and tapwater allowed transfer of virus⁵².

No data exist on the inactivation of HPV by alcohol or other disinfectants because *in vitro* replication of complete virions has not been achieved. Similarly, little is known about inactivation of noroviruses (members of the family *Caliciviridae* and important causes of gastroenteritis in humans) because they cannot be grown in tissue culture. Improper disinfection of environmental surfaces contaminated by feces or vomitus of infected patients is believed to play a role in the spread of noroviruses in some settings²⁹⁴⁻²⁹⁶. Prolonged survival of a norovirus surrogate (i.e., feline calicivirus virus [FCV], a closely related cultivable virus) has been demonstrated (e.g., at room temperature, FCV in a dried state survived for 21–18 days)²⁹⁷. Inactivation studies with FCV have shown the effectiveness of chlorine, glutaraldehyde, and iodine-based products whereas the quaternary ammonium compound, detergent, and ethanol failed to inactivate the virus completely.²⁹⁷ An evaluation of the effectiveness of several disinfectants against the feline calicivirus found that bleach diluted to 1000 ppm of available chlorine reduced infectivity of FCV by 4.5 logs in 1 minute. Other effective (\log_{10} reduction factor of >4 in virus) disinfectants included accelerated hydrogen peroxide, 5,000 ppm (3 min); chlorine dioxide, 1,000 ppm chlorine (1 min); a mixture of four quaternary ammonium compounds, 2,470 ppm (10 min); 79% ethanol with 0.1% quaternary ammonium compound (3 min); and 75% ethanol (10 min)²⁹⁸. A quaternary ammonium compound exhibited activity against feline calicivirus suspensions dried on hard surface carriers in 10 minutes²⁹⁹. Seventy percent ethanol and 70% 1-propanol reduced FCV by a 3–4- \log_{10}

reduction in 30 seconds³⁰⁰.

CDC announced that a previously unrecognized human virus from the coronavirus family is the leading hypothesis for the cause of a described syndrome of SARS³⁰¹. Two coronaviruses that are known to infect humans cause one third of common colds and can cause gastroenteritis. The virucidal efficacy of chemical germicides against coronavirus has been investigated. A study of disinfectants against coronavirus 229E found several that were effective after a 1-minute contact time; these included sodium hypochlorite (at a free chlorine concentration of 1,000 ppm and 5,000 ppm), 70% ethyl alcohol, and povidone-iodine (1% iodine)¹⁸⁶. In another study, 70% ethanol, 50% isopropanol, 0.05% benzalkonium chloride, 50 ppm iodine in iodophor, 0.23% sodium chlorite, 1% cresol soap and 0.7% formaldehyde inactivated >3 logs of two animal coronaviruses (mouse hepatitis virus, canine coronavirus) after a 10-minute exposure time³⁰². The activity of povidone-iodine has been demonstrated against human coronaviruses 229E and OC43³⁰³. A study also showed complete inactivation of the SARS coronavirus by 70% ethanol and povidone-iodine with an exposure times of 1 minute and 2.5% glutaraldehyde with an exposure time of 5 minute³⁰⁴. Because the SARS coronavirus is stable in feces and urine at room temperature for at least 1–2 days (WHO, 2003; http://www.who.int/csr/sars/survival_2003_05_04/en/index.html), surfaces might be a possible source of contamination and lead to infection with the SARS coronavirus and should be disinfected. Until more precise information is available, environments in which SARS patients are housed should be considered heavily contaminated, and rooms and equipment should be thoroughly disinfected daily and after the patient is discharged. EPA-registered disinfectants or 1:100 dilution of household bleach and water should be used for surface disinfection and disinfection on noncritical patient-care equipment. High-level disinfection and sterilization of semicritical and critical medical devices, respectively, does not need to be altered for patients with known or suspected SARS.

Free-living amoeba can be pathogenic and can harbor agents of pneumonia such as *Legionella pneumophila*. Limited studies have shown that 2% glutaraldehyde and peracetic acid do not completely inactivate *Acanthamoeba polyphaga* in a 20-minute exposure time for high-level disinfection. If amoeba are found to contaminate instruments and facilitate infection, longer immersion times or other disinfectants may need to be considered³⁰⁵.

Inactivation of Bioterrorist Agents

Publications have highlighted concerns about the potential for biological terrorism^{306, 307}. CDC has categorized several agents as “high priority” because they can be easily disseminated or transmitted from person to person, cause high mortality, and are likely to cause public panic and social disruption³⁰⁸. These agents include *Bacillus anthracis* (the cause of anthrax), *Yersinia pestis* (plague), variola major (smallpox), *Clostridium botulinum* toxin (botulism), *Francisella tularensis* (tularemia), filoviruses (Ebola hemorrhagic fever, Marburg hemorrhagic fever); and arenaviruses (Lassa [Lassa fever], Junin [Argentine hemorrhagic fever]), and related viruses³⁰⁸.

A few comments can be made regarding the role of sterilization and disinfection of potential agents of bioterrorism³⁰⁹. First, the susceptibility of these agents to germicides *in vitro* is similar to that of other related pathogens. For example, variola is similar to vaccinia^{72, 310, 311} and *B. anthracis* is similar to *B. atropaueus* (formerly *B. subtilis*)^{312, 313}. *B. subtilis* spores, for instance, proved as resistant as, if not more resistant than, *B. anthracis* spores (>6 log₁₀ reduction of *B. anthracis* spores in 5 minutes with acidified bleach [5,250 ppm chlorine])³¹³. Thus, one can extrapolate from the larger database available on the susceptibility of genetically similar organisms³¹⁴. Second, many of the potential bioterrorist agents are stable enough in the environment that contaminated environmental surfaces or fomites could lead to transmission of agents such as *B. anthracis*, *F. tularensis*, variola major, *C. botulinum* toxin, and *C. burnetti*³¹⁵. Third, data suggest that current disinfection and sterilization practices are appropriate for managing patient-care equipment and environmental surfaces when potentially contaminated patients are evaluated and/or admitted in a health-care facility after exposure to a bioterrorist agent. For example,

sodium hypochlorite can be used for surface disinfection (see <http://www.epa.gov/pesticides/factsheets/chemicals/bleachfactsheet.htm>). In instances where the health-care facility is the site of a bioterrorist attack, environmental decontamination might require special decontamination procedures (e.g., chlorine dioxide gas for *B. anthracis* spores). Because no antimicrobial products are registered for decontamination of biologic agents after a bioterrorist attack, EPA has granted a crises exemption for each product (see <http://www.epa.gov/pesticides/factsheets/chemicals/bleachfactsheet.htm>). Of only theoretical concern is the possibility that a bioterrorist agent could be engineered to be less susceptible to disinfection and sterilization processes³⁰⁹.

Toxicological, Environmental and Occupational Concerns

Health hazards associated with the use of germicides in healthcare vary from mucous membrane irritation to death, with the latter involving accidental injection by mentally disturbed patients³¹⁶. Although their degrees of toxicity vary³¹⁷⁻³²⁰, all disinfectants should be used with the proper safety precautions³²¹ and only for the intended purpose.

Key factors associated with assessing the health risk of a chemical exposure include the duration, intensity (i.e., how much chemical is involved), and route (e.g., skin, mucous membranes, and inhalation) of exposure. Toxicity can be acute or chronic. Acute toxicity usually results from an accidental spill of a chemical substance. Exposure is sudden and often produces an emergency situation. Chronic toxicity results from repeated exposure to low levels of the chemical over a prolonged period. Employers are responsible for informing workers about the chemical hazards in the workplace and implementing control measures. The OSHA Hazard Communication Standard (29 CFR 1910.1200, 1915.99, 1917.28, 1918.90, 1926.59, and 1928.21) requires manufacturers and importers of hazardous chemicals to develop Material Safety Data Sheets (MSDS) for each chemical or mixture of chemicals. Employers must have these data sheets readily available to employees who work with the products to which they could be exposed.

Exposure limits have been published for many chemicals used in health care to help provide a safe environment and, as relevant, are discussed in each section of this guideline. Only the exposure limits published by OSHA carry the legal force of regulations. OSHA publishes a limit as a time-weighted average (TWA), that is, the average concentration for a normal 8-hour work day and a 40-hour work week to which nearly all workers can be repeatedly exposed to a chemical without adverse health effects. For example, the permissible exposure limit (PEL) for EtO is 1.0 ppm, 8 hour TWA. The CDC National Institute for Occupational Safety and Health (NIOSH) develops recommended exposure limits (RELs). RELs are occupational exposure limits recommended by NIOSH as being protective of worker health and safety over a working lifetime. This limit is frequently expressed as a 40-hour TWA exposure for up to 10 hours per day during a 40-hour work week. These exposure limits are designed for inhalation exposures. Irritant and allergic effects can occur below the exposure limits, and skin contact can result in dermal effects or systemic absorption without inhalation. The American Conference on Governmental Industrial Hygienists (ACGIH) also provides guidelines on exposure limits³²². Information about workplace exposures and methods to reduce them (e.g., work practices, engineering controls, PPE) is available on the OSHA (<http://www.osha.gov>) and NIOSH (<http://www.cdc.gov/niosh>) websites.

Some states have excluded or limited concentrations of certain chemical germicides (e.g., glutaraldehyde, formaldehyde, and some phenols) from disposal through the sewer system. These rules are intended to minimize environmental harm. If health-care facilities exceed the maximum allowable concentration of a chemical (e.g., ≥ 5.0 mg/L), they have three options. First, they can switch to alternative products; for example, they can change from glutaraldehyde to another disinfectant for high-level disinfection or from phenolics to quaternary ammonium compounds for low-level disinfection. Second, the health-care facility can collect the disinfectant and dispose of it as a hazardous chemical. Third, the

facility can use a commercially available small-scale treatment method (e.g., neutralize glutaraldehyde with glycine).

Safe disposal of regulated chemicals is important throughout the medical community. For disposal of large volumes of spent solutions, users might decide to neutralize the microbicidal activity before disposal (e.g., glutaraldehyde). Solutions can be neutralized by reaction with chemicals such as sodium bisulfite^{323, 324} or glycine³²⁵.

European authors have suggested that instruments and ventilation therapy equipment should be disinfected by heat rather than by chemicals. The concerns for chemical disinfection include toxic side effects for the patient caused by chemical residues on the instrument or object, occupational exposure to toxic chemicals, and recontamination by rinsing the disinfectant with microbially contaminated tap water³²⁶.

Disinfection in Ambulatory Care, Home Care, and the Home

With the advent of managed healthcare, increasing numbers of patients are now being cared for in ambulatory-care and home settings. Many patients in these settings might have communicable diseases, immunocompromising conditions, or invasive devices. Therefore, adequate disinfection in these settings is necessary to provide a safe patient environment. Because the ambulatory-care setting (i.e., outpatient facility) provides the same risk for infection as the hospital, the Spaulding classification scheme described in this guideline should be followed (Table 1)¹⁷.

The home environment should be much safer than hospitals or ambulatory care. Epidemics should not be a problem, and cross-infection should be rare. The healthcare provider is responsible for providing the responsible family member information about infection-control procedures to follow in the home, including hand hygiene, proper cleaning and disinfection of equipment, and safe storage of cleaned and disinfected devices. Among the products recommended for home disinfection of reusable objects are bleach, alcohol, and hydrogen peroxide. APIC recommends that reusable objects (e.g., tracheostomy tubes) that touch mucous membranes be disinfected by immersion in 70% isopropyl alcohol for 5 minutes or in 3% hydrogen peroxide for 30 minutes. Additionally, a 1:50 dilution of 5.25%–6.15% sodium hypochlorite (household bleach) for 5 minutes should be effective³²⁷⁻³²⁹. Noncritical items (e.g., blood pressure cuffs, crutches) can be cleaned with a detergent. Blood spills should be handled according to OSHA regulations as previously described (see section on OSHA Bloodborne Pathogen Standard). In general, sterilization of critical items is not practical in homes but theoretically could be accomplished by chemical sterilants or boiling. Single-use disposable items can be used or reusable items sterilized in a hospital^{330, 331}.

Some environmental groups advocate “environmentally safe” products as alternatives to commercial germicides in the home-care setting. These alternatives (e.g., ammonia, baking soda, vinegar, Borax, liquid detergent) are not registered with EPA and should not be used for disinfecting because they are ineffective against *S. aureus*. Borax, baking soda, and detergents also are ineffective against *Salmonella* Typhi and *E. coli*; however, undiluted vinegar and ammonia are effective against *S. Typhi* and *E. coli*^{53, 332, 333}. Common commercial disinfectants designed for home use also are effective against selected antibiotic-resistant bacteria⁵³.

Public concerns have been raised that the use of antimicrobials in the home can promote development of antibiotic-resistant bacteria^{334, 335}. This issue is unresolved and needs to be considered further through scientific and clinical investigations. The public health benefits of using disinfectants in the home are unknown. However, some facts are known: many sites in the home kitchen and bathroom are microbially contaminated³³⁶, use of hypochlorites markedly reduces bacteria³³⁷, and good standards of hygiene (e.g., food hygiene, hand hygiene) can help reduce infections in the home^{338, 339}. In addition, laboratory studies indicate that many commercially prepared household disinfectants are effective against common pathogens⁵³ and can interrupt surface-to-human transmission of pathogens⁴⁸. The “targeted

hygiene concept”—which means identifying situations and areas (e.g., food-preparation surfaces and bathroom) where risk exists for transmission of pathogens—may be a reasonable way to identify when disinfection might be appropriate³⁴⁰.

Susceptibility of Antibiotic-Resistant Bacteria to Disinfectants

As with antibiotics, reduced susceptibility (or acquired “resistance”) of bacteria to disinfectants can arise by either chromosomal gene mutation or acquisition of genetic material in the form of plasmids or transposons^{338, 341-343, 344, 345, 346}. When changes occur in bacterial susceptibility that renders an antibiotic ineffective against an infection previously treatable by that antibiotic, the bacteria are referred to as “resistant.” In contrast, reduced susceptibility to disinfectants does not correlate with failure of the disinfectant because concentrations used in disinfection still greatly exceed the cidal level. Thus, the word “resistance” when applied to these changes is incorrect, and the preferred term is “reduced susceptibility” or “increased tolerance”^{344, 347}. No data are available that show that antibiotic-resistant bacteria are less sensitive to the liquid chemical germicides than antibiotic-sensitive bacteria at currently used germicide contact conditions and concentrations.

MRSA and vancomycin-resistant *Enterococcus* (VRE) are important health-care-associated agents. Some antiseptics and disinfectants have been known for years to be, because of MICs, somewhat less inhibitory to *S. aureus* strains that contain a plasmid-carrying gene encoding resistance to the antibiotic gentamicin³⁴⁴. For example, gentamicin resistance has been shown to also encode reduced susceptibility to propamidine, quaternary ammonium compounds, and ethidium bromide³⁴⁸, and MRSA strains have been found to be less susceptible than methicillin-sensitive *S. aureus* (MSSA) strains to chlorhexidine, propamidine, and the quaternary ammonium compound cetrimide³⁴⁹. In other studies, MRSA and MSSA strains have been equally sensitive to phenols and chlorhexidine, but MRSA strains were slightly more tolerant to quaternary ammonium compounds³⁵⁰. Two gene families (*qacCD* [now referred to as *smr*] and *qacAB*) are involved in providing protection against agents that are components of disinfectant formulations such as quaternary ammonium compounds. Staphylococci have been proposed to evade destruction because the protein specified by the *qacA* determinant is a cytoplasmic-membrane-associated protein involved in an efflux system that actively reduces intracellular accumulation of toxicants, such as quaternary ammonium compounds, to intracellular targets³⁵¹.

Other studies demonstrated that plasmid-mediated formaldehyde tolerance is transferable from *Serratia marcescens* to *E. coli*³⁵² and plasmid-mediated quaternary ammonium tolerance is transferable from *S. aureus* to *E. coli*³⁵³. Tolerance to mercury and silver also is plasmid borne^{341, 343-346}.

Because the concentrations of disinfectants used in practice are much higher than the MICs observed, even for the more tolerant strains, the clinical relevance of these observations is questionable. Several studies have found antibiotic-resistant hospital strains of common healthcare-associated pathogens (i.e., *Enterococcus*, *P. aeruginosa*, *Klebsiella pneumoniae*, *E. coli*, *S. aureus*, and *S. epidermidis*) to be equally susceptible to disinfectants as antibiotic-sensitive strains^{53, 354-356}. The susceptibility of glycopeptide-intermediate *S. aureus* was similar to vancomycin-susceptible, MRSA³⁵⁷. On the basis of these data, routine disinfection and housekeeping protocols do not need to be altered because of antibiotic resistance provided the disinfection method is effective^{358, 359}. A study that evaluated the efficacy of selected cleaning methods (e.g., QUAT-sprayed cloth, and QUAT-immersed cloth) for eliminating VRE found that currently used disinfection processes most likely are highly effective in eliminating VRE. However, surface disinfection must involve contact with all contaminated surfaces³⁵⁸. A new method using an invisible fluorescent marker to objectively evaluate the thoroughness of cleaning activities in patient rooms might lead to improvement in cleaning of all objects and surfaces but needs further evaluation³⁶⁰.

Lastly, does the use of antiseptics or disinfectants facilitate the development of disinfectant-tolerant organisms? Evidence and reviews indicate enhanced tolerance to disinfectants can be

developed in response to disinfectant exposure^{334, 335, 346, 347, 361}. However, the level of tolerance is not important in clinical terms because it is low and unlikely to compromise the effectiveness of disinfectants of which much higher concentrations are used^{347, 362}.

The issue of whether low-level tolerance to germicides selects for antibiotic-resistant strains is unsettled but might depend on the mechanism by which tolerance is attained. For example, changes in the permeability barrier or efflux mechanisms might affect susceptibility to both antibiotics and germicides, but specific changes to a target site might not. Some researchers have suggested that use of disinfectants or antiseptics (e.g., triclosan) could facilitate development of antibiotic-resistant microorganisms^{334, 335, 363}. Although evidence in laboratory studies indicates low-level resistance to triclosan, the concentrations of triclosan in these studies were low (generally <1 µg/mL) and dissimilar from the higher levels used in antimicrobial products (2,000–20,000 µg/mL)^{364, 365}. Thus, researchers can create laboratory-derived mutants that demonstrate reduced susceptibility to antiseptics or disinfectants. In some experiments, such bacteria have demonstrated reduced susceptibility to certain antibiotics³³⁵. There is no evidence that using antiseptics or disinfectants selects for antibiotic-resistant organisms in nature or that such mutants survive in nature³⁶⁶. In addition, the action of antibiotics and the action of disinfectants differ fundamentally. Antibiotics are selectively toxic and generally have a single target site in bacteria, thereby inhibiting a specific biosynthetic process. Germicides generally are considered nonspecific antimicrobials because of a multiplicity of toxic-effect mechanisms or target sites and are broader spectrum in the types of microorganisms against which they are effective^{344, 347}.

The rotational use of disinfectants in some environments (e.g., pharmacy production units) has been recommended and practiced in an attempt to prevent development of resistant microbes^{367, 368}. There have been only rare case reports that appropriately used disinfectants have resulted in a clinical problem arising from the selection or development of nonsusceptible microorganisms³⁶⁹.

Surface Disinfection

Is Surface Disinfection Necessary?

The effective use of disinfectants is part of a multibarrier strategy to prevent health-care–associated infections. Surfaces are considered noncritical items because they contact intact skin. Use of noncritical items or contact with noncritical surfaces carries little risk of causing an infection in patients or staff. Thus, the routine use of germicidal chemicals to disinfect hospital floors and other noncritical items is controversial³⁷⁰⁻³⁷⁵. A 1991 study expanded the Spaulding scheme by dividing the noncritical environmental surfaces into housekeeping surfaces and medical equipment surfaces³⁷⁶. The classes of disinfectants used on housekeeping and medical equipment surfaces can be similar. However, the frequency of decontaminating can vary (see Recommendations). Medical equipment surfaces (e.g., blood pressure cuffs, stethoscopes, hemodialysis machines, and X-ray machines) can become contaminated with infectious agents and contribute to the spread of health-care–associated infections^{248, 375}. For this reason, noncritical medical equipment surfaces should be disinfected with an EPA-registered low- or intermediate-level disinfectant. Use of a disinfectant will provide antimicrobial activity that is likely to be achieved with minimal additional cost or work.

Environmental surfaces (e.g., bedside table) also could potentially contribute to cross-transmission by contamination of health-care personnel from hand contact with contaminated surfaces, medical equipment, or patients^{50, 375, 377}. A paper reviews the epidemiologic and microbiologic data (Table 3) regarding the use of disinfectants on noncritical surfaces³⁷⁸.

Of the seven reasons to use a disinfectant on noncritical surfaces, five are particularly noteworthy and support the use of a germicidal detergent. First, hospital floors become contaminated with microorganisms from settling airborne bacteria: by contact with shoes, wheels, and other objects; and occasionally by spills. The removal of microbes is a component in controlling health-care–associated infections. In an investigation of the cleaning of hospital floors, the use of soap and water (80% reduction) was less effective in reducing the numbers of bacteria than was a phenolic disinfectant (94%–99.9%

reduction)³⁷⁹. However, a few hours after floor disinfection, the bacterial count was nearly back to the pretreatment level. Second, detergents become contaminated and result in seeding the patient's environment with bacteria. Investigators have shown that mop water becomes increasingly dirty during cleaning and becomes contaminated if soap and water is used rather than a disinfectant. For example, in one study, bacterial contamination in soap and water without a disinfectant increased from 10 CFU/mL to 34,000 CFU/mL after cleaning a ward, whereas contamination in a disinfectant solution did not change (20 CFU/mL)³⁸⁰. Contamination of surfaces close to the patient that are frequently touched by the patient or staff (e.g., bed rails) could result in patient exposures³⁸¹. In a study, using of detergents on floors and patient room furniture, increased bacterial contamination of the patients' environmental surfaces was found after cleaning (average increase = 103.6 CFU/24cm²)³⁸². In addition, a *P. aeruginosa* outbreak was reported in a hematology-oncology unit associated with contamination of the surface cleaning equipment when nongermicidal cleaning solutions instead of disinfectants were used to decontaminate the patients' environment³⁸³ and another study demonstrated the role of environmental cleaning in controlling an outbreak of *Acinetobacter baumannii*³⁸⁴. Studies also have shown that, in situations where the cleaning procedure failed to eliminate contamination from the surface and the cloth is used to wipe another surface, the contamination is transferred to that surface and the hands of the person holding the cloth^{381, 385}. Third, the CDC Isolation Guideline recommends that noncritical equipment contaminated with blood, body fluids, secretions, or excretions be cleaned and disinfected after use. The same guideline recommends that, in addition to cleaning, disinfection of the bedside equipment and environmental surfaces (e.g., bedrails, bedside tables, carts, commodes, door-knobs, and faucet handles) is indicated for certain pathogens, e.g., enterococci, which can survive in the inanimate environment for prolonged periods³⁸⁶. Fourth, OSHA requires that surfaces contaminated with blood and other potentially infectious materials (e.g., amniotic, pleural fluid) be disinfected. Fifth, using a single product throughout the facility can simplify both training and appropriate practice.

Reasons also exist for using a detergent alone on floors because noncritical surfaces contribute minimally to endemic health-care-associated infections³⁸⁷, and no differences have been found in health-care-associated infections rates when floors are cleaned with detergent rather than disinfectant^{382, 388, 389}. However, these studies have been small and of short duration and suffer from low statistical power because the outcome—healthcare-associated infections—is of low frequency. The low rate of infections makes the efficacy of an intervention statistically difficult to demonstrate. Because housekeeping surfaces are associated with the lowest risk for disease transmission, some researchers have suggested that either detergents or a disinfectant/detergent could be used³⁷⁶. No data exist that show reduced health-care-associated infection rates with use of surface disinfection of floors, but some data demonstrate reduced microbial load associated with the use of disinfectants. Given this information; other information showing that environmental surfaces (e.g., bedside table, bed rails) close to the patient and in outpatient settings³⁹⁰ can be contaminated with epidemiologically important microbes (such as VRE and MRSA)^{47, 390-394}, and data showing these organisms survive on various hospital surfaces^{395, 396}, some researchers have suggested that such surfaces should be disinfected on a regular schedule³⁷⁸. Spot decontamination on fabrics that remain in hospitals or clinic rooms while patients move in and out (e.g., privacy curtains) also should be considered. One study demonstrated the effectiveness of spraying the fabric with 3% hydrogen peroxide³⁹⁷. Future studies should evaluate the level of contamination on noncritical environmental surfaces as a function of high and low hand contact and whether some surfaces (e.g., bed rails) near the patient with high contact frequencies require more frequent disinfection. Regardless of whether a detergent or disinfectant is used on surfaces in a health-care facility, surfaces should be cleaned routinely and when dirty or soiled to provide an aesthetically pleasing environment and to prevent potentially contaminated objects from serving as a source for health-care-associated infections³⁹⁸. The value of designing surfaces (e.g. hexyl-polyvinylpyridine) that kill bacteria on contact³⁹⁹ or have sustained antimicrobial activity⁴⁰⁰ should be further evaluated.

Several investigators have recognized heavy microbial contamination of wet mops and cleaning cloths and the potential for spread of such contamination^{68, 401}. They have shown that wiping hard surfaces with contaminated cloths can contaminate hands, equipment, and other surfaces^{68, 402}. Data

have been published that can be used to formulate effective policies for decontamination and maintenance of reusable cleaning cloths. For example, heat was the most reliable treatment of cleaning cloths as a detergent washing followed by drying at 80°C for 2 hours produced elimination of contamination. However, the dry heating process might be a fire hazard if the mop head contains petroleum-based products or lint builds up within the equipment or vent hose (American Health Care Association, personal communication, March 2003). Alternatively, immersing the cloth in hypochlorite (4,000 ppm) for 2 minutes produced no detectable surviving organisms in 10 of 13 cloths⁴⁰³. If reusable cleaning cloths or mops are used, they should be decontaminated regularly to prevent surface contamination during cleaning with subsequent transfer of organisms from these surfaces to patients or equipment by the hands of health-care workers. Some hospitals have begun using a new mopping technique involving microfiber materials to clean floors. Microfibers are densely constructed, polyester and polyamide (nylon) fibers, that are approximately 1/16 the thickness of a human hair. The positively charged microfibers attract dust (which has a negative charge) and are more absorbent than a conventional, cotton-loop mop. Microfiber materials also can be wet with disinfectants, such as quaternary ammonium compounds. In one study, the microfiber system tested demonstrated superior microbial removal compared with conventional string mops when used with a detergent cleaner (94% vs 68%). The use of a disinfectant did not improve the microbial elimination demonstrated by the microfiber system (95% vs 94%). However, use of disinfectant significantly improved microbial removal when a conventional string mop was used (95% vs 68%) (WA Rutala, unpublished data, August 2006). The microfiber system also prevents the possibility of transferring microbes from room to room because a new microfiber pad is used in each room.

Contact Times for Surface Disinfectants

An important issue concerning use of disinfectants for noncritical surfaces in health-care settings is that the contact time specified on the label of the product is often too long to be practically followed. The labels of most products registered by EPA for use against HBV, HIV, or *M. tuberculosis* specify a contact time of 10 minutes. Such a long contact time is not practical for disinfection of environmental surfaces in a health-care setting because most health-care facilities apply a disinfectant and allow it to dry (~1 minute). Multiple scientific papers have demonstrated significant microbial reduction with contact times of 30 to 60 seconds^{46-56, 58-64}. In addition, EPA will approve a shortened contact time for any product for which the manufacturers will submit confirmatory efficacy data.

Currently, some EPA-registered disinfectants have contact times of one to three minutes. By law, users must follow all applicable label instructions for EPA-registered products. Ideally, product users should consider and use products that have the shortened contact time. However, disinfectant manufacturers also need to obtain EPA approval for shortened contact times so these products will be used correctly and effectively in the health-care environment.

Air Disinfection

Disinfectant spray-fog techniques for antimicrobial control in hospital rooms has been used. This technique of spraying of disinfectants is an unsatisfactory method of decontaminating air and surfaces and is not recommended for general infection control in routine patient-care areas³⁸⁶. Disinfectant fogging is rarely, if ever, used in U.S. healthcare facilities for air and surface disinfection in patient-care areas. Methods (e.g., filtration, ultraviolet germicidal irradiation, chlorine dioxide) to reduce air contamination in the healthcare setting are discussed in another guideline²³.

Microbial Contamination of Disinfectants

Contaminated disinfectants and antiseptics have been occasional vehicles of health-care infections and pseudoepidemics for more than 50 years. Published reports describing contaminated disinfectants and antiseptic solutions leading to health-care-associated infections have been summarized

⁴⁰⁴. Since this summary additional reports have been published ⁴⁰⁵⁻⁴⁰⁸. An examination of reports of disinfectants contaminated with microorganisms revealed noteworthy observations. Perhaps most importantly, high-level disinfectants/liquid chemical sterilants have not been associated with outbreaks due to intrinsic or extrinsic contamination. Members of the genus *Pseudomonas* (e.g., *P. aeruginosa*) are the most frequent isolates from contaminated disinfectants—recovered from 80% of contaminated products. Their ability to remain viable or grow in use-dilutions of disinfectants is unparalleled. This survival advantage for *Pseudomonas* results presumably from their nutritional versatility, their unique outer membrane that constitutes an effective barrier to the passage of germicides, and/or efflux systems ⁴⁰⁹. Although the concentrated solutions of the disinfectants have not been demonstrated to be contaminated at the point of manufacture, an undiluted phenolic can be contaminated by a *Pseudomonas* sp. during use ⁴¹⁰. In most of the reports that describe illness associated with contaminated disinfectants, the product was used to disinfect patient-care equipment, such as cystoscopes, cardiac catheters, and thermometers. Germicides used as disinfectants that were reported to have been contaminated include chlorhexidine, quaternary ammonium compounds, phenolics, and pine oil.

The following control measures should be instituted to reduce the frequency of bacterial growth in disinfectants and the threat of serious healthcare-associated infections from the use of such contaminated products ⁴⁰⁴. First, some disinfectants should not be diluted; those that are diluted must be prepared correctly to achieve the manufacturers' recommended use-dilution. Second, infection-control professionals must learn from the literature what inappropriate activities result in extrinsic contamination (i.e., at the point of use) of germicides and train users to prevent recurrence. Common sources of extrinsic contamination of germicides in the reviewed literature are the water to make working dilutions, contaminated containers, and general contamination of the hospital areas where the germicides are prepared and/or used. Third, stock solutions of germicides must be stored as indicated on the product label. EPA verifies manufacturers' efficacy claims against microorganisms. These measures should provide assurance that products meeting the EPA registration requirements can achieve a certain level of antimicrobial activity when used as directed.

FACTORS AFFECTING THE EFFICACY OF DISINFECTION AND STERILIZATION

The activity of germicides against microorganisms depends on a number of factors, some of which are intrinsic qualities of the organism, others of which are the chemical and external physical environment. Awareness of these factors should lead to better use of disinfection and sterilization processes and will be briefly reviewed. More extensive consideration of these and other factors is available elsewhere^{13, 14, 16, 411-413}.

Number and Location of Microorganisms

All other conditions remaining constant, the larger the number of microbes, the more time a germicide needs to destroy all of them. Spaulding illustrated this relation when he employed identical test conditions and demonstrated that it took 30 minutes to kill 10 *B. atrophaeus* (formerly *Bacillus subtilis*) spores but 3 hours to kill 100,000 *Bacillus atrophaeus* spores. This reinforces the need for scrupulous cleaning of medical instruments before disinfection and sterilization. Reducing the number of microorganisms that must be inactivated through meticulous cleaning, increases the margin of safety when the germicide is used according to the labeling and shortens the exposure time required to kill the entire microbial load. Researchers also have shown that aggregated or clumped cells are more difficult to inactivate than monodispersed cells⁴¹⁴.

The location of microorganisms also must be considered when factors affecting the efficacy of germicides are assessed. Medical instruments with multiple pieces must be disassembled and equipment such as endoscopes that have crevices, joints, and channels are more difficult to disinfect than are flat-surface equipment because penetration of the disinfectant of all parts of the equipment is more difficult. Only surfaces that directly contact the germicide will be disinfected, so there must be no air pockets and the equipment must be completely immersed for the entire exposure period. Manufacturers should be encouraged to produce equipment engineered for ease of cleaning and disinfection.

Innate Resistance of Microorganisms

Microorganisms vary greatly in their resistance to chemical germicides and sterilization processes (Figure 1)³⁴². Intrinsic resistance mechanisms in microorganisms to disinfectants vary. For example, spores are resistant to disinfectants because the spore coat and cortex act as a barrier, mycobacteria have a waxy cell wall that prevents disinfectant entry, and gram-negative bacteria possess an outer membrane that acts as a barrier to the uptake of disinfectants^{341, 343-345}. Implicit in all disinfection strategies is the consideration that the most resistant microbial subpopulation controls the sterilization or disinfection time. That is, to destroy the most resistant types of microorganisms (i.e., bacterial spores), the user needs to employ exposure times and a concentration of germicide needed to achieve complete destruction. Except for prions, bacterial spores possess the highest innate resistance to chemical germicides, followed by coccidia (e.g., *Cryptosporidium*), mycobacteria (e.g., *M. tuberculosis*), nonlipid or small viruses (e.g., poliovirus, and coxsackievirus), fungi (e.g., *Aspergillus*, and *Candida*), vegetative bacteria (e.g., *Staphylococcus*, and *Pseudomonas*) and lipid or medium-size viruses (e.g., herpes, and HIV). The germicidal resistance exhibited by the gram-positive and gram-negative bacteria is similar with some exceptions (e.g., *P. aeruginosa* which shows greater resistance to some disinfectants)^{369, 415, 416}. *P. aeruginosa* also is significantly more resistant to a variety of disinfectants in its "naturally occurring" state than are cells subcultured on laboratory media^{415, 417}. *Rickettsiae*, *Chlamydiae*, and mycoplasma cannot be placed in this scale of relative resistance because information about the efficacy of germicides against these agents is limited⁴¹⁸. Because these microorganisms contain lipid and are similar in structure and composition to other bacteria, they can be predicted to be inactivated by the same germicides that destroy lipid viruses and vegetative bacteria. A known exception to this supposition is *Coxiella burnetti*, which has demonstrated resistance to disinfectants⁴¹⁹.

Concentration and Potency of Disinfectants

With other variables constant, and with one exception (iodophors), the more concentrated the

disinfectant, the greater its efficacy and the shorter the time necessary to achieve microbial kill. Generally not recognized, however, is that all disinfectants are not similarly affected by concentration adjustments. For example, quaternary ammonium compounds and phenol have a concentration exponent of 1 and 6, respectively; thus, halving the concentration of a quaternary ammonium compound requires doubling its disinfecting time, but halving the concentration of a phenol solution requires a 64-fold (i.e., 2^6) increase in its disinfecting time^{365, 413, 420}.

Considering the length of the disinfection time, which depends on the potency of the germicide, also is important. This was illustrated by Spaulding who demonstrated using the mucin-loop test that 70% isopropyl alcohol destroyed 10^4 *M. tuberculosis* in 5 minutes, whereas a simultaneous test with 3% phenolic required 2–3 hours to achieve the same level of microbial kill¹⁴.

Physical and Chemical Factors

Several physical and chemical factors also influence disinfectant procedures: temperature, pH, relative humidity, and water hardness. For example, the activity of most disinfectants increases as the temperature increases, but some exceptions exist. Furthermore, too great an increase in temperature causes the disinfectant to degrade and weakens its germicidal activity and thus might produce a potential health hazard.

An increase in pH improves the antimicrobial activity of some disinfectants (e.g., glutaraldehyde, quaternary ammonium compounds) but decreases the antimicrobial activity of others (e.g., phenols, hypochlorites, and iodine). The pH influences the antimicrobial activity by altering the disinfectant molecule or the cell surface⁴¹³.

Relative humidity is the single most important factor influencing the activity of gaseous disinfectants/sterilants, such as EtO, chlorine dioxide, and formaldehyde.

Water hardness (i.e., high concentration of divalent cations) reduces the rate of kill of certain disinfectants because divalent cations (e.g., magnesium, calcium) in the hard water interact with the disinfectant to form insoluble precipitates^{13, 421}.

Organic and Inorganic Matter

Organic matter in the form of serum, blood, pus, or fecal or lubricant material can interfere with the antimicrobial activity of disinfectants in at least two ways. Most commonly, interference occurs by a chemical reaction between the germicide and the organic matter resulting in a complex that is less germicidal or nongermicidal, leaving less of the active germicide available for attacking microorganisms. Chlorine and iodine disinfectants, in particular, are prone to such interaction. Alternatively, organic material can protect microorganisms from attack by acting as a physical barrier^{422, 423}.

The effects of inorganic contaminants on the sterilization process were studied during the 1950s and 1960s^{424, 425}. These and other studies show the protection by inorganic contaminants of microorganisms to all sterilization processes results from occlusion in salt crystals^{426, 427}. This further emphasizes the importance of meticulous cleaning of medical devices before any sterilization or disinfection procedure because both organic and inorganic soils are easily removed by washing⁴²⁶.

Duration of Exposure

Items must be exposed to the germicide for the appropriate minimum contact time. Multiple investigators have demonstrated the effectiveness of low-level disinfectants against vegetative bacteria (e.g., *Listeria*, *E. coli*, *Salmonella*, VRE, MRSA), yeasts (e.g., *Candida*), mycobacteria (e.g., *M. tuberculosis*), and viruses (e.g., poliovirus) at exposure times of 30–60 seconds⁴⁶⁻⁶⁴. By law, all applicable label instructions on EPA-registered products must be followed. If the user selects exposure conditions that differ from those on the EPA-registered product label, the user assumes liability for any injuries resulting from off-label use and is potentially subject to enforcement action under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA)

All lumens and channels of endoscopic instruments must contact the disinfectant. Air pockets interfere with the disinfection process, and items that float on the disinfectant will not be disinfected. The disinfectant must be introduced reliably into the internal channels of the device. The exact times for disinfecting medical items are somewhat elusive because of the effect of the aforementioned factors on disinfection efficacy. Certain contact times have proved reliable (Table 1), but, in general, longer contact times are more effective than shorter contact times.

Biofilms

Microorganisms may be protected from disinfectants by production of thick masses of cells⁴²⁸ and extracellular materials, or biofilms⁴²⁹⁻⁴³⁵. Biofilms are microbial communities that are tightly attached to surfaces and cannot be easily removed. Once these masses form, microbes within them can be resistant to disinfectants by multiple mechanisms, including physical characteristics of older biofilms, genotypic variation of the bacteria, microbial production of neutralizing enzymes, and physiologic gradients within the biofilm (e.g., pH). Bacteria within biofilms are up to 1,000 times more resistant to antimicrobials than are the same bacteria in suspension⁴³⁶. Although new decontamination methods⁴³⁷ are being investigated for removing biofilms, chlorine and monochloramines can effectively inactivate biofilm bacteria^{431 438}. Investigators have hypothesized that the glycocalyx-like cellular masses on the interior walls of polyvinyl chloride pipe would protect embedded organisms from some disinfectants and be a reservoir for continuous contamination^{429, 430, 439}. Biofilms have been found in whirlpools⁴⁴⁰, dental unit waterlines⁴⁴¹, and numerous medical devices (e.g., contact lenses, pacemakers, hemodialysis systems, urinary catheters, central venous catheters, endoscopes)^{434, 436, 438, 442}. Their presence can have serious implications for immunocompromised patients and patients who have indwelling medical devices. Some enzymes^{436, 443, 444} and detergents⁴³⁶ can degrade biofilms or reduce numbers of viable bacteria within a biofilm, but no products are EPA-registered or FDA-cleared for this purpose.

CLEANING

Cleaning is the removal of foreign material (e.g., soil, and organic material) from objects and is normally accomplished using water with detergents or enzymatic products. Thorough cleaning is required before high-level disinfection and sterilization because inorganic and organic materials that remain on the surfaces of instruments interfere with the effectiveness of these processes. Also, if soiled materials dry or bake onto the instruments, the removal process becomes more difficult and the disinfection or sterilization process less effective or ineffective. Surgical instruments should be presoaked or rinsed to prevent drying of blood and to soften or remove blood from the instruments.

Cleaning is done manually in use areas without mechanical units (e.g., ultrasonic cleaners or washer-disinfectors) or for fragile or difficult-to-clean instruments. With manual cleaning, the two essential components are friction and fluidics. Friction (e.g., rubbing/scrubbing the soiled area with a brush) is an old and dependable method. Fluidics (i.e., fluids under pressure) is used to remove soil and debris from internal channels after brushing and when the design does not allow passage of a brush through a channel⁴⁴⁵. When a washer-disinfector is used, care should be taken in loading instruments: hinged instruments should be opened fully to allow adequate contact with the detergent solution; stacking of instruments in washers should be avoided; and instruments should be disassembled as much as possible.

The most common types of mechanical or automatic cleaners are ultrasonic cleaners, washer-decontaminators, washer-disinfectors, and washer-sterilizers. Ultrasonic cleaning removes soil by cavitation and implosion in which waves of acoustic energy are propagated in aqueous solutions to disrupt the bonds that hold particulate matter to surfaces. Bacterial contamination can be present in used ultrasonic cleaning solutions (and other used detergent solutions) because these solutions generally do not make antibacterial label claims⁴⁴⁶. Even though ultrasound alone does not significantly inactivate bacteria, sonication can act synergistically to increase the cidal efficacy of a disinfectant⁴⁴⁷. Users of ultrasonic cleaners should be aware that the cleaning fluid could result in endotoxin contamination of surgical instruments, which could cause severe inflammatory reactions⁴⁴⁸. Washer-sterilizers are modified steam sterilizers that clean by filling the chamber with water and detergent through which steam passes to provide agitation. Instruments are subsequently rinsed and subjected to a short steam-sterilization cycle. Another washer-sterilizer employs rotating spray arms for a wash cycle followed by a steam sterilization cycle at 285°F^{449, 450}. Washer-decontaminators/disinfectors act like a dishwasher that uses a combination of water circulation and detergents to remove soil. These units sometimes have a cycle that subjects the instruments to a heat process (e.g., 93°C for 10 minutes)⁴⁵¹. Washer-disinfectors are generally computer-controlled units for cleaning, disinfecting, and drying solid and hollow surgical and medical equipment. In one study, cleaning (measured as 5–6 log₁₀ reduction) was achieved on surfaces that had adequate contact with the water flow in the machine⁴⁵². Detailed information about cleaning and preparing supplies for terminal sterilization is provided by professional organizations^{453, 454} and books⁴⁵⁵. Studies have shown that manual and mechanical cleaning of endoscopes achieves approximately a 4-log₁₀ reduction of contaminating organisms^{83, 104, 456, 457}. Thus, cleaning alone effectively reduces the number of microorganisms on contaminated equipment. In a quantitative analysis of residual protein contamination of reprocessed surgical instruments, median levels of residual protein contamination per instrument for five trays were 267, 260, 163, 456, and 756 µg⁴⁵⁸. In another study, the median amount of protein from reprocessed surgical instruments from different hospitals ranged from 8 µg to 91 µg⁴⁵⁹. When manual methods were compared with automated methods for cleaning reusable accessory devices used for minimally invasive surgical procedures, the automated method was more efficient for cleaning biopsy forceps and ported and nonported laparoscopic devices and achieved a >99% reduction in soil parameters (i.e., protein, carbohydrate, hemoglobin) in the ported and nonported laparoscopic devices^{460, 461}.

For instrument cleaning, a neutral or near-neutral pH detergent solution commonly is used because such solutions generally provide the best material compatibility profile and good soil removal.

Enzymes, usually proteases, sometimes are added to neutral pH solutions to assist in removing organic material. Enzymes in these formulations attack proteins that make up a large portion of common soil (e.g., blood, pus). Cleaning solutions also can contain lipases (enzymes active on fats) and amylases (enzymes active on starches). Enzymatic cleaners are not disinfectants, and proteinaceous enzymes can be inactivated by germicides. As with all chemicals, enzymes must be rinsed from the equipment or adverse reactions (e.g., fever, residual amounts of high-level disinfectants, proteinaceous residue) could result^{462, 463}. Enzyme solutions should be used in accordance with manufacturer's instructions, which include proper dilution of the enzymatic detergent and contact with equipment for the amount of time specified on the label⁴⁶³. Detergent enzymes can result in asthma or other allergic effects in users. Neutral pH detergent solutions that contain enzymes are compatible with metals and other materials used in medical instruments and are the best choice for cleaning delicate medical instruments, especially flexible endoscopes⁴⁵⁷. Alkaline-based cleaning agents are used for processing medical devices because they efficiently dissolve protein and fat residues⁴⁶⁴; however, they can be corrosive⁴⁵⁷. Some data demonstrate that enzymatic cleaners are more effective than neutral detergents^{465, 466} in removing microorganisms from surfaces but two more recent studies found no difference in cleaning efficiency between enzymatic and alkaline-based cleaners^{443, 464}. Another study found no significant difference between enzymatic and non-enzymatic cleaners in terms of microbial cleaning efficacy⁴⁶⁷. A new non-enzyme, hydrogen peroxide-based formulation (not FDA-cleared) was as effective as enzymatic cleaners in removing protein, blood, carbohydrate, and endotoxin from surface test carriers⁴⁶⁸. In addition, this product effected a 5- \log_{10} reduction in microbial loads with a 3-minute exposure at room temperature⁴⁶⁸.

Although the effectiveness of high-level disinfection and sterilization mandates effective cleaning, no "real-time" tests exist that can be employed in a clinical setting to verify cleaning. If such tests were commercially available they could be used to ensure an adequate level of cleaning⁴⁶⁹⁻⁴⁷². The only way to ensure adequate cleaning is to conduct a reprocessing verification test (e.g., microbiologic sampling), but this is not routinely recommended⁴⁷³. Validation of the cleaning processes in a laboratory-testing program is possible by microorganism detection, chemical detection for organic contaminants, radionuclide tagging, and chemical detection for specific ions^{426, 471}. During the past few years, data have been published describing use of an artificial soil, protein, endotoxin, X-ray contrast medium, or blood to verify the manual or automated cleaning process^{169, 452, 474-478} and adenosine triphosphate bioluminescence and microbiologic sampling to evaluate the effectiveness of environmental surface cleaning^{170, 479}. At a minimum, all instruments should be individually inspected and be visibly clean.

DISINFECTION

Many disinfectants are used alone or in combinations (e.g., hydrogen peroxide and peracetic acid) in the health-care setting. These include alcohols, chlorine and chlorine compounds, formaldehyde, glutaraldehyde, *ortho*-phthalaldehyde, hydrogen peroxide, iodophors, peracetic acid, phenolics, and quaternary ammonium compounds. Commercial formulations based on these chemicals are considered unique products and must be registered with EPA or cleared by FDA. In most instances, a given product is designed for a specific purpose and is to be used in a certain manner. Therefore, users should read labels carefully to ensure the correct product is selected for the intended use and applied efficiently.

Disinfectants are not interchangeable, and incorrect concentrations and inappropriate disinfectants can result in excessive costs. Because occupational diseases among cleaning personnel have been associated with use of several disinfectants (e.g., formaldehyde, glutaraldehyde, and chlorine), precautions (e.g., gloves and proper ventilation) should be used to minimize exposure^{318, 480, 481}. Asthma and reactive airway disease can occur in sensitized persons exposed to any airborne chemical, including germicides. Clinically important asthma can occur at levels below ceiling levels regulated by OSHA or recommended by NIOSH. The preferred method of control is elimination of the chemical (through engineering controls or substitution) or relocation of the worker.

The following overview of the performance characteristics of each provides users with sufficient information to select an appropriate disinfectant for any item and use it in the most efficient way.

Chemical Disinfectants

Alcohol

Overview. In the healthcare setting, “alcohol” refers to two water-soluble chemical compounds—ethyl alcohol and isopropyl alcohol—that have generally underrated germicidal characteristics⁴⁸². FDA has not cleared any liquid chemical sterilant or high-level disinfectant with alcohol as the main active ingredient. These alcohols are rapidly bactericidal rather than bacteriostatic against vegetative forms of bacteria; they also are tuberculocidal, fungicidal, and virucidal but do not destroy bacterial spores. Their cidal activity drops sharply when diluted below 50% concentration, and the optimum bactericidal concentration is 60%–90% solutions in water (volume/volume)^{483, 484}.

Mode of Action. The most feasible explanation for the antimicrobial action of alcohol is denaturation of proteins. This mechanism is supported by the observation that absolute ethyl alcohol, a dehydrating agent, is less bactericidal than mixtures of alcohol and water because proteins are denatured more quickly in the presence of water^{484, 485}. Protein denaturation also is consistent with observations that alcohol destroys the dehydrogenases of *Escherichia coli*⁴⁸⁶, and that ethyl alcohol increases the lag phase of *Enterobacter aerogenes*⁴⁸⁷ and that the lag phase effect could be reversed by adding certain amino acids. The bacteriostatic action was believed caused by inhibition of the production of metabolites essential for rapid cell division.

Microbicidal Activity. Methyl alcohol (methanol) has the weakest bactericidal action of the alcohols and thus seldom is used in healthcare⁴⁸⁸. The bactericidal activity of various concentrations of ethyl alcohol (ethanol) was examined against a variety of microorganisms in exposure periods ranging from 10 seconds to 1 hour⁴⁸³. *Pseudomonas aeruginosa* was killed in 10 seconds by all concentrations of ethanol from 30% to 100% (v/v), and *Serratia marcescens*, *E. coli* and *Salmonella typhosa* were killed in 10 seconds by all concentrations of ethanol from 40% to 100%. The gram-positive organisms *Staphylococcus aureus* and *Streptococcus pyogenes* were slightly more resistant, being killed in 10 seconds by ethyl alcohol concentrations of 60%–95%. Isopropyl alcohol (isopropanol) was slightly more bactericidal than ethyl alcohol for *E. coli* and *S. aureus*⁴⁸⁹.

Ethyl alcohol, at concentrations of 60%–80%, is a potent virucidal agent inactivating all of the lipophilic viruses (e.g., herpes, vaccinia, and influenza virus) and many hydrophilic viruses (e.g.,

adenovirus, enterovirus, rhinovirus, and rotaviruses but not hepatitis A virus (HAV)⁵⁸ or poliovirus⁴⁹. Isopropyl alcohol is not active against the nonlipid enteroviruses but is fully active against the lipid viruses⁷². Studies also have demonstrated the ability of ethyl and isopropyl alcohol to inactivate the hepatitis B virus (HBV)^{224, 225} and the herpes virus,⁴⁹⁰ and ethyl alcohol to inactivate human immunodeficiency virus (HIV)²²⁷, rotavirus, echovirus, and astrovirus⁴⁹¹.

In tests of the effect of ethyl alcohol against *M. tuberculosis*, 95% ethanol killed the tubercle bacilli in sputum or water suspension within 15 seconds⁴⁹². In 1964, Spaulding stated that alcohols were the germicide of choice for tuberculocidal activity, and they should be the standard by which all other tuberculocides are compared. For example, he compared the tuberculocidal activity of iodophor (450 ppm), a substituted phenol (3%), and isopropanol (70%/volume) using the mucin-loop test (10⁶ *M. tuberculosis* per loop) and determined the contact times needed for complete destruction were 120–180 minutes, 45–60 minutes, and 5 minutes, respectively. The mucin-loop test is a severe test developed to produce long survival times. Thus, these figures should not be extrapolated to the exposure times needed when these germicides are used on medical or surgical material⁴⁸².

Ethyl alcohol (70%) was the most effective concentration for killing the tissue phase of *Cryptococcus neoformans*, *Blastomyces dermatitidis*, *Coccidioides immitis*, and *Histoplasma capsulatum* and the culture phases of the latter three organisms aerosolized onto various surfaces. The culture phase was more resistant to the action of ethyl alcohol and required about 20 minutes to disinfect the contaminated surface, compared with <1 minute for the tissue phase^{493, 494}.

Isopropyl alcohol (20%) is effective in killing the cysts of *Acanthamoeba culbertsoni* (560) as are chlorhexidine, hydrogen peroxide, and thimerosal⁴⁹⁶.

Uses. Alcohols are not recommended for sterilizing medical and surgical materials principally because they lack sporicidal action and they cannot penetrate protein-rich materials. Fatal postoperative wound infections with *Clostridium* have occurred when alcohols were used to sterilize surgical instruments contaminated with bacterial spores⁴⁹⁷. Alcohols have been used effectively to disinfect oral and rectal thermometers^{498, 499}, hospital pagers⁵⁰⁰, scissors⁵⁰¹, and stethoscopes⁵⁰². Alcohols have been used to disinfect fiberoptic endoscopes^{503, 504} but failure of this disinfectant have lead to infection^{280, 505}. Alcohol towelettes have been used for years to disinfect small surfaces such as rubber stoppers of multiple-dose medication vials or vaccine bottles. Furthermore, alcohol occasionally is used to disinfect external surfaces of equipment (e.g., stethoscopes, ventilators, manual ventilation bags)⁵⁰⁶, CPR manikins⁵⁰⁷, ultrasound instruments⁵⁰⁸ or medication preparation areas. Two studies demonstrated the effectiveness of 70% isopropyl alcohol to disinfect reusable transducer heads in a controlled environment^{509, 510}. In contrast, three bloodstream infection outbreaks have been described when alcohol was used to disinfect transducer heads in an intensive-care setting⁵¹¹.

The documented shortcomings of alcohols on equipment are that they damage the shellac mountings of lensed instruments, tend to swell and harden rubber and certain plastic tubing after prolonged and repeated use, bleach rubber and plastic tiles⁴⁸² and damage tonometer tips (by deterioration of the glue) after the equivalent of 1 working year of routine use⁵¹². Tonometer biprisms soaked in alcohol for 4 days developed rough front surfaces that potentially could cause corneal damage; this appeared to be caused by weakening of the cementing substances used to fabricate the biprisms⁵¹³. Corneal opacification has been reported when tonometer tips were swabbed with alcohol immediately before measurement of intraocular pressure⁵¹⁴. Alcohols are flammable and consequently must be stored in a cool, well-ventilated area. They also evaporate rapidly, making extended exposure time difficult to achieve unless the items are immersed.

Chlorine and Chlorine Compounds

Overview. Hypochlorites, the most widely used of the chlorine disinfectants, are available as liquid (e.g., sodium hypochlorite) or solid (e.g., calcium hypochlorite). The most prevalent chlorine

products in the United States are aqueous solutions of 5.25%–6.15% sodium hypochlorite (see glossary), usually called household bleach. They have a broad spectrum of antimicrobial activity, do not leave toxic residues, are unaffected by water hardness, are inexpensive and fast acting³²⁸, remove dried or fixed organisms and biofilms from surfaces⁴⁶⁵, and have a low incidence of serious toxicity⁵¹⁵⁻⁵¹⁷. Sodium hypochlorite at the concentration used in household bleach (5.25-6.15%) can produce ocular irritation or oropharyngeal, esophageal, and gastric burns^{318, 518-522}. Other disadvantages of hypochlorites include corrosiveness to metals in high concentrations (>500 ppm), inactivation by organic matter, discoloring or “bleaching” of fabrics, release of toxic chlorine gas when mixed with ammonia or acid (e.g., household cleaning agents)⁵²³⁻⁵²⁵, and relative stability³²⁷. The microbicidal activity of chlorine is attributed largely to undissociated hypochlorous acid (HOCl). The dissociation of HOCl to the less microbicidal form (hypochlorite ion OCl⁻) depends on pH. The disinfecting efficacy of chlorine decreases with an increase in pH that parallels the conversion of undissociated HOCl to OCl⁻^{329, 526}. A potential hazard is production of the carcinogen bis(chloromethyl) ether when hypochlorite solutions contact formaldehyde⁵²⁷ and the production of the animal carcinogen trihalomethane when hot water is hyperchlorinated⁵²⁸. After reviewing environmental fate and ecologic data, EPA has determined the currently registered uses of hypochlorites will not result in unreasonable adverse effects to the environment⁵²⁹.

Alternative compounds that release chlorine and are used in the health-care setting include demand-release chlorine dioxide, sodium dichloroisocyanurate, and chloramine-T. The advantage of these compounds over the hypochlorites is that they retain chlorine longer and so exert a more prolonged bactericidal effect. Sodium dichloroisocyanurate tablets are stable, and for two reasons, the microbicidal activity of solutions prepared from sodium dichloroisocyanurate tablets might be greater than that of sodium hypochlorite solutions containing the same total available chlorine. First, with sodium dichloroisocyanurate, only 50% of the total available chlorine is free (HOCl and OCl⁻), whereas the remainder is combined (monochloroisocyanurate or dichloroisocyanurate), and as free available chlorine is used up, the latter is released to restore the equilibrium. Second, solutions of sodium dichloroisocyanurate are acidic, whereas sodium hypochlorite solutions are alkaline, and the more microbicidal type of chlorine (HOCl) is believed to predominate⁵³⁰⁻⁵³³. Chlorine dioxide-based disinfectants are prepared fresh as required by mixing the two components (base solution [citric acid with preservatives and corrosion inhibitors] and the activator solution [sodium chlorite]). In vitro suspension tests showed that solutions containing about 140 ppm chlorine dioxide achieved a reduction factor exceeding 10⁶ of *S. aureus* in 1 minute and of *Bacillus atrophaeus* spores in 2.5 minutes in the presence of 3 g/L bovine albumin. The potential for damaging equipment requires consideration because long-term use can damage the outer plastic coat of the insertion tube⁵³⁴. In another study, chlorine dioxide solutions at either 600 ppm or 30 ppm killed *Mycobacterium avium-intracellulare* within 60 seconds after contact but contamination by organic material significantly affected the microbicidal properties⁵³⁵.

The microbicidal activity of a new disinfectant, “superoxidized water,” has been examined. The concept of electrolyzing saline to create a disinfectant or antiseptics is appealing because the basic materials of saline and electricity are inexpensive and the end product (i.e., water) does not damage the environment. The main products of this water are hypochlorous acid (e.g., at a concentration of about 144 mg/L) and chlorine. As with any germicide, the antimicrobial activity of superoxidized water is strongly affected by the concentration of the active ingredient (available free chlorine)⁵³⁶. One manufacturer generates the disinfectant at the point of use by passing a saline solution over coated titanium electrodes at 9 amps. The product generated has a pH of 5.0–6.5 and an oxidation-reduction potential (redox) of >950 mV. Although superoxidized water is intended to be generated fresh at the point of use, when tested under clean conditions the disinfectant was effective within 5 minutes when 48 hours old⁵³⁷. Unfortunately, the equipment required to produce the product can be expensive because parameters such as pH, current, and redox potential must be closely monitored. The solution is nontoxic to biologic tissues. Although the United Kingdom manufacturer claims the solution is noncorrosive and nondamaging to endoscopes and processing equipment, one flexible endoscope manufacturer (Olympus Key-Med, United Kingdom) has voided the warranty on the endoscopes if superoxidized water is used to disinfect them⁵³⁸. As with any germicide formulation, the user should check with the device manufacturer for

compatibility with the germicide. Additional studies are needed to determine whether this solution could be used as an alternative to other disinfectants or antiseptics for hand washing, skin antiseptics, room cleaning, or equipment disinfection (e.g., endoscopes, dialyzers)^{400, 539, 540}. In October 2002, the FDA cleared superoxidized water as a high-level disinfectant (FDA, personal communication, September 18, 2002).

Mode of Action. The exact mechanism by which free chlorine destroys microorganisms has not been elucidated. Inactivation by chlorine can result from a number of factors: oxidation of sulfhydryl enzymes and amino acids; ring chlorination of amino acids; loss of intracellular contents; decreased uptake of nutrients; inhibition of protein synthesis; decreased oxygen uptake; oxidation of respiratory components; decreased adenosine triphosphate production; breaks in DNA; and depressed DNA synthesis^{329, 347}. The actual microbicidal mechanism of chlorine might involve a combination of these factors or the effect of chlorine on critical sites³⁴⁷.

Microbicidal Activity. Low concentrations of free available chlorine (e.g., HOCl, OCl⁻, and elemental chlorine-Cl₂) have a biocidal effect on mycoplasma (25 ppm) and vegetative bacteria (<5 ppm) in seconds in the absence of an organic load^{329, 418}. Higher concentrations (1,000 ppm) of chlorine are required to kill *M. tuberculosis* using the Association of Official Analytical Chemists (AOAC) tuberculocidal test⁷³. A concentration of 100 ppm will kill ≥99.9% of *B. atrophaeus* spores within 5 minutes^{541, 542} and destroy mycotic agents in <1 hour³²⁹. Acidified bleach and regular bleach (5,000 ppm chlorine) can inactivate 10⁶ *Clostridium difficile* spores in <10 minutes²⁶². One study reported that 25 different viruses were inactivated in 10 minutes with 200 ppm available chlorine⁷². Several studies have demonstrated the effectiveness of diluted sodium hypochlorite and other disinfectants to inactivate HIV⁶¹. Chlorine (500 ppm) showed inhibition of *Candida* after 30 seconds of exposure⁵⁴. In experiments using the AOAC Use-Dilution Method, 100 ppm of free chlorine killed 10⁶–10⁷ *S. aureus*, *Salmonella choleraesuis*, and *P. aeruginosa* in <10 minutes³²⁷. Because household bleach contains 5.25%–6.15% sodium hypochlorite, or 52,500–61,500 ppm available chlorine, a 1:1,000 dilution provides about 53–62 ppm available chlorine, and a 1:10 dilution of household bleach provides about 5250–6150 ppm.

Data are available for chlorine dioxide that support manufacturers' bactericidal, fungicidal, sporicidal, tuberculocidal, and virucidal label claims⁵⁴³⁻⁵⁴⁶. A chlorine dioxide generator has been shown effective for decontaminating flexible endoscopes⁵³⁴ but it is not currently FDA-cleared for use as a high-level disinfectant⁸⁵. Chlorine dioxide can be produced by mixing solutions, such as a solution of chlorine with a solution of sodium chlorite³²⁹. In 1986, a chlorine dioxide product was voluntarily removed from the market when its use caused leakage of cellulose-based dialyzer membranes, which allowed bacteria to migrate from the dialysis fluid side of the dialyzer to the blood side⁵⁴⁷.

Sodium dichloroisocyanurate at 2,500 ppm available chlorine is effective against bacteria in the presence of up to 20% plasma, compared with 10% plasma for sodium hypochlorite at 2,500 ppm⁵⁴⁸.

“Superoxidized water” has been tested against bacteria, mycobacteria, viruses, fungi, and spores^{537, 539, 549}. Freshly generated superoxidized water is rapidly effective (<2 minutes) in achieving a 5-log₁₀ reduction of pathogenic microorganisms (i.e., *M. tuberculosis*, *M. chelonae*, poliovirus, HIV, multidrug-resistant *S. aureus*, *E. coli*, *Candida albicans*, *Enterococcus faecalis*, *P. aeruginosa*) in the absence of organic loading. However, the biocidal activity of this disinfectant decreased substantially in the presence of organic material (e.g., 5% horse serum)^{537, 549, 550}. No bacteria or viruses were detected on artificially contaminated endoscopes after a 5-minute exposure to superoxidized water⁵⁵¹ and HBV-DNA was not detected from any endoscope experimentally contaminated with HBV-positive mixed sera after a disinfectant exposure time of 7 minutes⁵⁵².

Uses. Hypochlorites are widely used in healthcare facilities in a variety of settings.³²⁸ Inorganic chlorine solution is used for disinfecting tonometer heads¹⁸⁸ and for spot-disinfection of countertops and floors. A 1:10–1:100 dilution of 5.25%–6.15% sodium hypochlorite (i.e., household bleach)^{22, 228, 553, 554} or

an EPA-registered tuberculocidal disinfectant¹⁷ has been recommended for decontaminating blood spills. For small spills of blood (i.e., drops of blood) on noncritical surfaces, the area can be disinfected with a 1:100 dilution of 5.25%–6.15% sodium hypochlorite or an EPA-registered tuberculocidal disinfectant. Because hypochlorites and other germicides are substantially inactivated in the presence of blood^{63, 548, 555, 556}, large spills of blood require that the surface be cleaned before an EPA-registered disinfectant or a 1:10 (final concentration) solution of household bleach is applied⁵⁵⁷. If a sharps injury is possible, the surface initially should be decontaminated^{69, 318}, then cleaned and disinfected (1:10 final concentration)⁶³. Extreme care always should be taken to prevent percutaneous injury. At least 500 ppm available chlorine for 10 minutes is recommended for decontaminating CPR training manikins⁵⁵⁸. Full-strength bleach has been recommended for self-disinfection of needles and syringes used for illicit-drug injection when needle-exchange programs are not available. The difference in the recommended concentrations of bleach reflects the difficulty of cleaning the interior of needles and syringes and the use of needles and syringes for parenteral injection⁵⁵⁹. Clinicians should not alter their use of chlorine on environmental surfaces on the basis of testing methodologies that do not simulate actual disinfection practices^{560, 561}. Other uses in healthcare include as an irrigating agent in endodontic treatment⁵⁶² and as a disinfectant for manikins, laundry, dental appliances, hydrotherapy tanks^{23, 41}, regulated medical waste before disposal³²⁸, and the water distribution system in hemodialysis centers and hemodialysis machines⁵⁶³.

Chlorine long has been used as the disinfectant in water treatment. Hyperchlorination of a *Legionella*-contaminated hospital water system²³ resulted in a dramatic decrease (from 30% to 1.5%) in the isolation of *L. pneumophila* from water outlets and a cessation of healthcare-associated Legionnaires' disease in an affected unit^{528, 564}. Water disinfection with monochloramine by municipal water-treatment plants substantially reduced the risk for healthcare-associated Legionnaires disease^{565, 566}. Chlorine dioxide also has been used to control *Legionella* in a hospital water supply.⁵⁶⁷ Chloramine T⁵⁶⁸ and hypochlorites⁴¹ have been used to disinfect hydrotherapy equipment.

Hypochlorite solutions in tap water at a pH >8 stored at room temperature (23°C) in closed, opaque plastic containers can lose up to 40%–50% of their free available chlorine level over 1 month. Thus, if a user wished to have a solution containing 500 ppm of available chlorine at day 30, he or she should prepare a solution containing 1,000 ppm of chlorine at time 0. Sodium hypochlorite solution does not decompose after 30 days when stored in a closed brown bottle³²⁷.

The use of powders, composed of a mixture of a chlorine-releasing agent with highly absorbent resin, for disinfecting spills of body fluids has been evaluated by laboratory tests and hospital ward trials. The inclusion of acrylic resin particles in formulations markedly increases the volume of fluid that can be soaked up because the resin can absorb 200–300 times its own weight of fluid, depending on the fluid consistency. When experimental formulations containing 1%, 5%, and 10% available chlorine were evaluated by a standardized surface test, those containing 10% demonstrated bactericidal activity. One problem with chlorine-releasing granules is that they can generate chlorine fumes when applied to urine⁵⁶⁹.

Formaldehyde

Overview. Formaldehyde is used as a disinfectant and sterilant in both its liquid and gaseous states. Liquid formaldehyde will be considered briefly in this section, and the gaseous form is reviewed elsewhere⁵⁷⁰. Formaldehyde is sold and used principally as a water-based solution called formalin, which is 37% formaldehyde by weight. The aqueous solution is a bactericide, tuberculocide, fungicide, virucide and sporicide^{72, 82, 571-573}. OSHA indicated that formaldehyde should be handled in the workplace as a potential carcinogen and set an employee exposure standard for formaldehyde that limits an 8-hour time-weighted average exposure concentration of 0.75 ppm^{574, 575}. The standard includes a second permissible exposure limit in the form of a short-term exposure limit (STEL) of 2 ppm that is the maximum exposure allowed during a 15-minute period⁵⁷⁶. Ingestion of formaldehyde can be fatal, and long-term exposure to low levels in the air or on the skin can cause asthma-like respiratory problems and skin irritation, such as dermatitis and itching. For these reasons, employees should have limited direct contact

with formaldehyde, and these considerations limit its role in sterilization and disinfection processes. Key provisions of the OSHA standard that protects workers from exposure to formaldehyde appear in Title 29 of the Code of Federal Regulations (CFR) Part 1910.1048 (and equivalent regulations in states with OSHA-approved state plans)⁵⁷⁷.

Mode of Action. Formaldehyde inactivates microorganisms by alkylating the amino and sulfhydryl groups of proteins and ring nitrogen atoms of purine bases³⁷⁶.

Microbicidal Activity. Varying concentrations of aqueous formaldehyde solutions destroy a wide range of microorganisms. Inactivation of poliovirus in 10 minutes required an 8% concentration of formalin, but all other viruses tested were inactivated with 2% formalin⁷². Four percent formaldehyde is a tuberculocidal agent, inactivating 10^4 *M. tuberculosis* in 2 minutes⁸², and 2.5% formaldehyde inactivated about 10^7 *Salmonella* Typhi in 10 minutes in the presence of organic matter⁵⁷². The sporicidal action of formaldehyde was slower than that of glutaraldehyde in comparative tests with 4% aqueous formaldehyde and 2% glutaraldehyde against the spores of *B. anthracis*⁸². The formaldehyde solution required 2 hours of contact to achieve an inactivation factor of 10^4 , whereas glutaraldehyde required only 15 minutes.

Uses. Although formaldehyde-alcohol is a chemical sterilant and formaldehyde is a high-level disinfectant, the health-care uses of formaldehyde are limited by its irritating fumes and its pungent odor even at very low levels (<1 ppm). For these reasons and others—such as its role as a suspected human carcinogen linked to nasal cancer and lung cancer⁵⁷⁸, this germicide is excluded from Table 1. When it is used, direct exposure to employees generally is limited; however, excessive exposures to formaldehyde have been documented for employees of renal transplant units^{574, 579}, and students in a gross anatomy laboratory⁵⁸⁰. Formaldehyde is used in the health-care setting to prepare viral vaccines (e.g., poliovirus and influenza); as an embalming agent; and to preserve anatomic specimens; and historically has been used to sterilize surgical instruments, especially when mixed with ethanol. A 1997 survey found that formaldehyde was used for reprocessing hemodialyzers by 34% of U.S. hemodialysis centers—a 60% decrease from 1983^{249, 581}. If used at room temperature, a concentration of 4% with a minimum exposure of 24 hours is required to disinfect disposable hemodialyzers reused on the same patient^{582, 583}. Aqueous formaldehyde solutions (1%–2%) also have been used to disinfect the internal fluid pathways of dialysis machines⁵⁸³. To minimize a potential health hazard to dialysis patients, the dialysis equipment must be thoroughly rinsed and tested for residual formaldehyde before use.

Paraformaldehyde, a solid polymer of formaldehyde, can be vaporized by heat for the gaseous decontamination of laminar flow biologic safety cabinets when maintenance work or filter changes require access to the sealed portion of the cabinet.

Glutaraldehyde

Overview. Glutaraldehyde is a saturated dialdehyde that has gained wide acceptance as a high-level disinfectant and chemical sterilant¹⁰⁷. Aqueous solutions of glutaraldehyde are acidic and generally in this state are not sporicidal. Only when the solution is “activated” (made alkaline) by use of alkalinizing agents to pH 7.5–8.5 does the solution become sporicidal. Once activated, these solutions have a shelf-life of minimally 14 days because of the polymerization of the glutaraldehyde molecules at alkaline pH levels. This polymerization blocks the active sites (aldehyde groups) of the glutaraldehyde molecules that are responsible for its biocidal activity.

Novel glutaraldehyde formulations (e.g., glutaraldehyde-phenol-sodium phenate, potentiated acid glutaraldehyde, stabilized alkaline glutaraldehyde) produced in the past 30 years have overcome the problem of rapid loss of activity (e.g., use-life 28–30 days) while generally maintaining excellent microbicidal activity^{584–588}. However, antimicrobial activity depends not only on age but also on use conditions, such as dilution and organic stress. Manufacturers' literature for these preparations suggests the neutral or alkaline glutaraldehydes possess microbicidal and anticorrosion properties superior to

those of acid glutaraldehydes, and a few published reports substantiate these claims^{542, 589, 590}. However, two studies found no difference in the microbicidal activity of alkaline and acid glutaraldehydes^{73, 591}. The use of glutaraldehyde-based solutions in health-care facilities is widespread because of their advantages, including excellent biocidal properties; activity in the presence of organic matter (20% bovine serum); and noncorrosive action to endoscopic equipment, thermometers, rubber, or plastic equipment (Tables 4 and 5).

Mode of Action. The biocidal activity of glutaraldehyde results from its alkylation of sulfhydryl, hydroxyl, carboxyl, and amino groups of microorganisms, which alters RNA, DNA, and protein synthesis. The mechanism of action of glutaraldehydes are reviewed extensively elsewhere^{592, 593}.

Microbicidal Activity. The in vitro inactivation of microorganisms by glutaraldehydes has been extensively investigated and reviewed^{592, 593}. Several investigators showed that $\geq 2\%$ aqueous solutions of glutaraldehyde, buffered to pH 7.5–8.5 with sodium bicarbonate effectively killed vegetative bacteria in <2 minutes; *M. tuberculosis*, fungi, and viruses in <10 minutes; and spores of *Bacillus* and *Clostridium* species in 3 hours^{542, 592-597}. Spores of *C. difficile* are more rapidly killed by 2% glutaraldehyde than are spores of other species of *Clostridium* and *Bacillus*^{79, 265, 266}. Microorganisms with substantial resistance to glutaraldehyde have been reported, including some mycobacteria (*M. chelonae*, *Mycobacterium avium-intracellulare*, *M. xenopi*)⁵⁹⁸⁻⁶⁰¹, *Methylobacterium mesophilicum*⁶⁰², *Trichosporon*, fungal ascospores (e.g., *Microascus cinereus*, *Cheatomium globosum*), and *Cryptosporidium*^{271, 603}. *M. chelonae* persisted in a 0.2% glutaraldehyde solution used to store porcine prosthetic heart valves⁶⁰⁴.

Two percent alkaline glutaraldehyde solution inactivated 10^5 *M. tuberculosis* cells on the surface of penicylinders within 5 minutes at 18°C⁵⁸⁹. However, subsequent studies⁸² questioned the mycobactericidal prowess of glutaraldehydes. Two percent alkaline glutaraldehyde has slow action (20 to >30 minutes) against *M. tuberculosis* and compares unfavorably with alcohols, formaldehydes, iodine, and phenol⁸². Suspensions of *M. avium*, *M. intracellulare*, and *M. gordonae* were more resistant to inactivation by a 2% alkaline glutaraldehyde (estimated time to complete inactivation: ~60 minutes) than were virulent *M. tuberculosis* (estimated time to complete inactivation ~25 minutes)⁶⁰⁵. The rate of kill was directly proportional to the temperature, and a standardized suspension of *M. tuberculosis* could not be sterilized within 10 minutes⁸⁴. An FDA-cleared chemical sterilant containing 2.5% glutaraldehyde uses increased temperature (35°C) to reduce the time required to achieve high-level disinfection (5 minutes)^{85, 606}, but its use is limited to automatic endoscope reprocessors equipped with a heater. In another study employing membrane filters for measurement of mycobactericidal activity of 2% alkaline glutaraldehyde, complete inactivation was achieved within 20 minutes at 20°C when the test inoculum was 10^6 *M. tuberculosis* per membrane⁸¹. Several investigators^{55, 57, 73, 76, 80, 81, 84, 605} have demonstrated that glutaraldehyde solutions inactivate 2.4 to >5.0 log₁₀ of *M. tuberculosis* in 10 minutes (including multidrug-resistant *M. tuberculosis*) and 4.0–6.4 log₁₀ of *M. tuberculosis* in 20 minutes. On the basis of these data and other studies, 20 minutes at room temperature is considered the minimum exposure time needed to reliably kill *Mycobacteria* and other vegetative bacteria with $\geq 2\%$ glutaraldehyde^{17, 19, 27, 57, 83, 94, 108, 111, 117-121, 607}.

Glutaraldehyde is commonly diluted during use, and studies showed a glutaraldehyde concentration decline after a few days of use in an automatic endoscope washer^{608, 609}. The decline occurs because instruments are not thoroughly dried and water is carried in with the instrument, which increases the solution's volume and dilutes its effective concentration⁶¹⁰. This emphasizes the need to ensure that semicritical equipment is disinfected with an acceptable concentration of glutaraldehyde. Data suggest that 1.0%–1.5% glutaraldehyde is the minimum effective concentration for >2% glutaraldehyde solutions when used as a high-level disinfectant^{76, 589, 590, 609}. Chemical test strips or liquid chemical monitors^{610, 611} are available for determining whether an effective concentration of glutaraldehyde is present despite repeated use and dilution. The frequency of testing should be based on how frequently the solutions are used (e.g., used daily, test daily; used weekly, test before use; used 30 times per day, test each 10th use), but the strips should not be used to extend the use life beyond the expiration date. Data suggest the chemicals in the test strip deteriorate with time⁶¹² and a

manufacturer's expiration date should be placed on the bottles. The bottle of test strips should be dated when opened and used for the period of time indicated on the bottle (e.g., 120 days). The results of test strip monitoring should be documented. The glutaraldehyde test kits have been preliminarily evaluated for accuracy and range⁶¹² but the reliability has been questioned⁶¹³. To ensure the presence of minimum effective concentration of the high-level disinfectant, manufacturers of some chemical test strips recommend the use of quality-control procedures to ensure the strips perform properly. If the manufacturer of the chemical test strip recommends a quality-control procedure, users should comply with the manufacturer's recommendations. The concentration should be considered unacceptable or unsafe when the test indicates a dilution below the product's minimum effective concentration (MEC) (generally to $\leq 1.0\%$ – 1.5% glutaraldehyde) by the indicator not changing color.

A 2.0% glutaraldehyde–7.05% phenol–1.20% sodium phenate product that contained 0.125% glutaraldehyde–0.44% phenol–0.075% sodium phenate when diluted 1:16 is not recommended as a high-level disinfectant because it lacks bactericidal activity in the presence of organic matter and lacks tuberculocidal, fungicidal, virucidal, and sporicidal activity^{49, 55, 56, 71, 73-79, 614}. In December 1991, EPA issued an order to stop the sale of all batches of this product because of efficacy data showing the product is not effective against spores and possibly other microorganisms or inanimate objects as claimed on the label⁶¹⁵. FDA has cleared a glutaraldehyde–phenol/phenate concentrate as a high-level disinfectant that contains 1.12% glutaraldehyde with 1.93% phenol/phenate at its use concentration. Other FDA cleared glutaraldehyde sterilants that contain 2.4%–3.4% glutaraldehyde are used undiluted⁶⁰⁶.

Uses. Glutaraldehyde is used most commonly as a high-level disinfectant for medical equipment such as endoscopes^{69, 107, 504}, spirometry tubing, dialyzers⁶¹⁶, transducers, anesthesia and respiratory therapy equipment⁶¹⁷, hemodialysis proportioning and dialysate delivery systems^{249, 618}, and reuse of laparoscopic disposable plastic trocars⁶¹⁹. Glutaraldehyde is noncorrosive to metal and does not damage lensed instruments, rubber, or plastics. Glutaraldehyde should not be used for cleaning noncritical surfaces because it is too toxic and expensive.

Colitis believed caused by glutaraldehyde exposure from residual disinfecting solution in endoscope solution channels has been reported and is preventable by careful endoscope rinsing^{318, 620-630}. One study found that residual glutaraldehyde levels were higher and more variable after manual disinfection (<0.2 mg/L to 159.5 mg/L) than after automatic disinfection (0.2–6.3 mg/L)⁶³¹. Similarly, keratopathy and corneal decompensation were caused by ophthalmic instruments that were inadequately rinsed after soaking in 2% glutaraldehyde^{632, 633}.

Healthcare personnel can be exposed to elevated levels of glutaraldehyde vapor when equipment is processed in poorly ventilated rooms, when spills occur, when glutaraldehyde solutions are activated or changed,⁶³⁴ or when open immersion baths are used. Acute or chronic exposure can result in skin irritation or dermatitis, mucous membrane irritation (eye, nose, mouth), or pulmonary symptoms^{318, 635-639}. Epistaxis, allergic contact dermatitis, asthma, and rhinitis also have been reported in healthcare workers exposed to glutaraldehyde^{636, 640-647}.

Glutaraldehyde exposure should be monitored to ensure a safe work environment. Testing can be done by four techniques: a silica gel tube/gas chromatography with a flame ionization detector, dinitrophenylhydrazine (DNPH)-impregnated filter cassette/high-performance liquid chromatography (HPLC) with an ultraviolet (UV) detector, a passive badge/HPLC, or a handheld glutaraldehyde air monitor⁶⁴⁸. The silica gel tube and the DNPH-impregnated cassette are suitable for monitoring the 0.05 ppm ceiling limit. The passive badge, with a 0.02 ppm limit of detection, is considered marginal at the American Council of Governmental Industrial Hygienists (ACGIH) ceiling level. The ceiling level is considered too close to the glutaraldehyde meter's 0.03 ppm limit of detection to provide confidence in the readings⁶⁴⁸. ACGIH does not require a specific monitoring schedule for glutaraldehyde; however, a monitoring schedule is needed to ensure the level is less than the ceiling limit. For example, monitoring

should be done initially to determine glutaraldehyde levels, after procedural or equipment changes, and in response to worker complaints⁶⁴⁹. In the absence of an OSHA permissible exposure limit, if the glutaraldehyde level is higher than the ACGIH ceiling limit of 0.05 ppm, corrective action and repeat monitoring would be prudent⁶⁴⁹.

Engineering and work-practice controls that can be used to resolve these problems include ducted exhaust hoods, air systems that provide 7–15 air exchanges per hour, ductless fume hoods with absorbents for the glutaraldehyde vapor, tight-fitting lids on immersion baths, personal protection (e.g., nitrile or butyl rubber gloves but not natural latex gloves, goggles) to minimize skin or mucous membrane contact, and automated endoscope processors^{7, 650}. If engineering controls fail to maintain levels below the ceiling limit, institutions can consider the use of respirators (e.g., a half-face respirator with organic vapor cartridge⁶⁴⁰ or a type "C" supplied air respirator with a full facepiece operated in a positive pressure mode)⁶⁵¹. In general, engineering controls are preferred over work-practice and administrative controls because they do not require active participation by the health-care worker. Even though enforcement of the OSHA ceiling limit was suspended in 1993 by the U.S. Court of Appeals⁵⁷⁷, limiting employee exposure to 0.05 ppm (according to ACGIH) is prudent because, at this level, glutaraldehyde can irritate the eyes, throat, and nose^{318, 577, 639, 652}. If glutaraldehyde disposal through the sanitary sewer system is restricted, sodium bisulfate can be used to neutralize the glutaraldehyde and make it safe for disposal.

Hydrogen Peroxide

Overview. The literature contains several accounts of the properties, germicidal effectiveness, and potential uses for stabilized hydrogen peroxide in the health-care setting. Published reports ascribe good germicidal activity to hydrogen peroxide and attest to its bactericidal, virucidal, sporicidal, and fungicidal properties⁶⁵³⁻⁶⁵⁵. (Tables 4 and 5) The FDA website lists cleared liquid chemical sterilants and high-level disinfectants containing hydrogen peroxide and their cleared contact conditions.

Mode of Action. Hydrogen peroxide works by producing destructive hydroxyl free radicals that can attack membrane lipids, DNA, and other essential cell components. Catalase, produced by aerobic organisms and facultative anaerobes that possess cytochrome systems, can protect cells from metabolically produced hydrogen peroxide by degrading hydrogen peroxide to water and oxygen. This defense is overwhelmed by the concentrations used for disinfection^{653, 654}.

Microbicidal Activity. Hydrogen peroxide is active against a wide range of microorganisms, including bacteria, yeasts, fungi, viruses, and spores^{78, 654}. A 0.5% accelerated hydrogen peroxide demonstrated bactericidal and virucidal activity in 1 minute and mycobactericidal and fungicidal activity in 5 minutes⁶⁵⁶. Bactericidal effectiveness and stability of hydrogen peroxide in urine has been demonstrated against a variety of health-care-associated pathogens; organisms with high cellular catalase activity (e.g., *S. aureus*, *S. marcescens*, and *Proteus mirabilis*) required 30–60 minutes of exposure to 0.6% hydrogen peroxide for a 10^8 reduction in cell counts, whereas organisms with lower catalase activity (e.g., *E. coli*, *Streptococcus* species, and *Pseudomonas* species) required only 15 minutes' exposure⁶⁵⁷. In an investigation of 3%, 10%, and 15% hydrogen peroxide for reducing spacecraft bacterial populations, a complete kill of 10^6 spores (i.e., *Bacillus* species) occurred with a 10% concentration and a 60-minute exposure time. A 3% concentration for 150 minutes killed 10^6 spores in six of seven exposure trials⁶⁵⁸. A 10% hydrogen peroxide solution resulted in a 10^3 decrease in *B. atrophaeus* spores, and a $\geq 10^5$ decrease when tested against 13 other pathogens in 30 minutes at 20°C^{659, 660}. A 3.0% hydrogen peroxide solution was ineffective against VRE after 3 and 10 minutes exposure times⁶⁶¹ and caused only a 2-log₁₀ reduction in the number of *Acanthamoeba* cysts in approximately 2 hours⁶⁶². A 7% stabilized hydrogen peroxide proved to be sporicidal (6 hours of exposure), mycobactericidal (20 minutes), fungicidal (5 minutes) at full strength, virucidal (5 minutes) and bactericidal (3 minutes) at a 1:16 dilution when a quantitative carrier test was used⁶⁵⁵. The 7% solution of hydrogen peroxide, tested after 14 days of stress (in the form of germ-loaded carriers and respiratory therapy equipment), was sporicidal (>7 log₁₀ reduction in 6 hours), mycobactericidal (>6.5 log₁₀ reduction in 25

minutes), fungicidal (>5 log₁₀ reduction in 20 minutes), bactericidal (>6 log₁₀ reduction in 5 minutes) and virucidal (5 log₁₀ reduction in 5 minutes)⁶⁶³. Synergistic sporicidal effects were observed when spores were exposed to a combination of hydrogen peroxide (5.9%–23.6%) and peracetic acid⁶⁶⁴. Other studies demonstrated the antiviral activity of hydrogen peroxide against rhinovirus⁶⁶⁵. The time required for inactivating three serotypes of rhinovirus using a 3% hydrogen peroxide solution was 6–8 minutes; this time increased with decreasing concentrations (18–20 minutes at 1.5%, 50–60 minutes at 0.75%).

Concentrations of hydrogen peroxide from 6% to 25% show promise as chemical sterilants. The product marketed as a sterilant is a premixed, ready-to-use chemical that contains 7.5% hydrogen peroxide and 0.85% phosphoric acid (to maintain a low pH)⁶⁹. The mycobactericidal activity of 7.5% hydrogen peroxide has been corroborated in a study showing the inactivation of >10⁵ multidrug-resistant *M. tuberculosis* after a 10-minute exposure⁶⁶⁶. Thirty minutes were required for >99.9% inactivation of poliovirus and HAV⁶⁶⁷. Three percent and 6% hydrogen peroxide were unable to inactivate HAV in 1 minute in a carrier test⁵⁸. When the effectiveness of 7.5% hydrogen peroxide at 10 minutes was compared with 2% alkaline glutaraldehyde at 20 minutes in manual disinfection of endoscopes, no significant difference in germicidal activity was observed⁶⁶⁸. No complaints were received from the nursing or medical staff regarding odor or toxicity. In one study, 6% hydrogen peroxide (unused product was 7.5%) was more effective in the high-level disinfection of flexible endoscopes than was the 2% glutaraldehyde solution⁴⁵⁶. A new, rapid-acting 13.4% hydrogen peroxide formulation (that is not yet FDA-cleared) has demonstrated sporicidal, mycobactericidal, fungicidal, and virucidal efficacy. Manufacturer data demonstrate that this solution sterilizes in 30 minutes and provides high-level disinfection in 5 minutes⁶⁶⁹. This product has not been used long enough to evaluate material compatibility to endoscopes and other semicritical devices, and further assessment by instrument manufacturers is needed.

Under normal conditions, hydrogen peroxide is extremely stable when properly stored (e.g., in dark containers). The decomposition or loss of potency in small containers is less than 2% per year at ambient temperatures⁶⁷⁰.

Uses. Commercially available 3% hydrogen peroxide is a stable and effective disinfectant when used on inanimate surfaces. It has been used in concentrations from 3% to 6% for disinfecting soft contact lenses (e.g., 3% for 2–3 hrs)^{653, 671, 672}, tonometer biphisms⁵¹³, ventilators⁶⁷³, fabrics³⁹⁷, and endoscopes⁴⁵⁶. Hydrogen peroxide was effective in spot-disinfecting fabrics in patients' rooms³⁹⁷. Corneal damage from a hydrogen peroxide-soaked tonometer tip that was not properly rinsed has been reported⁶⁷⁴. Hydrogen peroxide also has been instilled into urinary drainage bags in an attempt to eliminate the bag as a source of bladder bacteriuria and environmental contamination⁶⁷⁵. Although the instillation of hydrogen peroxide into the bag reduced microbial contamination of the bag, this procedure did not reduce the incidence of catheter-associated bacteriuria⁶⁷⁵.

A chemical irritation resembling pseudomembranous colitis caused by either 3% hydrogen peroxide or a 2% glutaraldehyde has been reported⁶²¹. An epidemic of pseudomembrane-like enteritis and colitis in seven patients in a gastrointestinal endoscopy unit also has been associated with inadequate rinsing of 3% hydrogen peroxide from the endoscope⁶⁷⁶.

As with other chemical sterilants, dilution of the hydrogen peroxide must be monitored by regularly testing the minimum effective concentration (i.e., 7.5%–6.0%). Compatibility testing by Olympus America of the 7.5% hydrogen peroxide found both cosmetic changes (e.g., discoloration of black anodized metal finishes)⁶⁹ and functional changes with the tested endoscopes (Olympus, written communication, October 15, 1999).

Iodophors

Overview. Iodine solutions or tinctures long have been used by health professionals primarily as antiseptics on skin or tissue. Iodophors, on the other hand, have been used both as antiseptics and

disinfectants. FDA has not cleared any liquid chemical sterilant or high-level disinfectants with iodophors as the main active ingredient. An iodophor is a combination of iodine and a solubilizing agent or carrier; the resulting complex provides a sustained-release reservoir of iodine and releases small amounts of free iodine in aqueous solution. The best-known and most widely used iodophor is povidone-iodine, a compound of polyvinylpyrrolidone with iodine. This product and other iodophors retain the germicidal efficacy of iodine but unlike iodine generally are nonstaining and relatively free of toxicity and irritancy^{677, 678}.

Several reports that documented intrinsic microbial contamination of antiseptic formulations of povidone-iodine and poloxamer-iodine⁶⁷⁹⁻⁶⁸¹ caused a reappraisal of the chemistry and use of iodophors⁶⁸². “Free” iodine (I₂) contributes to the bactericidal activity of iodophors and dilutions of iodophors demonstrate more rapid bactericidal action than does a full-strength povidone-iodine solution. The reason for the observation that dilution increases bactericidal activity is unclear, but dilution of povidone-iodine might weaken the iodine linkage to the carrier polymer with an accompanying increase of free iodine in solution⁶⁸⁰. Therefore, iodophors must be diluted according to the manufacturers' directions to achieve antimicrobial activity.

Mode of Action. Iodine can penetrate the cell wall of microorganisms quickly, and the lethal effects are believed to result from disruption of protein and nucleic acid structure and synthesis.

Microbicidal Activity. Published reports on the in vitro antimicrobial efficacy of iodophors demonstrate that iodophors are bactericidal, mycobactericidal, and virucidal but can require prolonged contact times to kill certain fungi and bacterial spores^{14, 71-73, 290, 683-686}. Three brands of povidone-iodine solution have demonstrated more rapid kill (seconds to minutes) of *S. aureus* and *M. chelonae* at a 1:100 dilution than did the stock solution⁶⁸³. The virucidal activity of 75–150 ppm available iodine was demonstrated against seven viruses⁷². Other investigators have questioned the efficacy of iodophors against poliovirus in the presence of organic matter⁶⁸⁵ and rotavirus SA-11 in distilled or tapwater²⁹⁰. Manufacturers' data demonstrate that commercial iodophors are not sporicidal, but they are tuberculocidal, fungicidal, virucidal, and bactericidal at their recommended use-dilution.

Uses. Besides their use as an antiseptic, iodophors have been used for disinfecting blood culture bottles and medical equipment, such as hydrotherapy tanks, thermometers, and endoscopes. Antiseptic iodophors are not suitable for use as hard-surface disinfectants because of concentration differences. Iodophors formulated as antiseptics contain less free iodine than do those formulated as disinfectants³⁷⁶. Iodine or iodine-based antiseptics should not be used on silicone catheters because they can adversely affect the silicone tubing⁶⁸⁷.

Ortho-phthalaldehyde (OPA)

Overview. Ortho-phthalaldehyde is a high-level disinfectant that received FDA clearance in October 1999. It contains 0.55% 1,2-benzenedicarboxaldehyde (OPA). OPA solution is a clear, pale-blue liquid with a pH of 7.5. (Tables 4 and 5)

Mode of Action. Preliminary studies on the mode of action of OPA suggest that both OPA and glutaraldehyde interact with amino acids, proteins, and microorganisms. However, OPA is a less potent cross-linking agent. This is compensated for by the lipophilic aromatic nature of OPA that is likely to assist its uptake through the outer layers of mycobacteria and gram-negative bacteria⁶⁸⁸⁻⁶⁹⁰. OPA appears to kill spores by blocking the spore germination process⁶⁹¹.

Microbicidal Activity. Studies have demonstrated excellent microbicidal activity in vitro^{69, 100, 271, 400, 692-703}. For example, OPA has superior mycobactericidal activity (5-log₁₀ reduction in 5 minutes) to glutaraldehyde. The mean times required to produce a 6-log₁₀ reduction for *M. bovis* using 0.21% OPA was 6 minutes, compared with 32 minutes using 1.5% glutaraldehyde⁶⁹³. OPA showed good activity against the mycobacteria tested, including the glutaraldehyde-resistant strains, but 0.5% OPA was not sporicidal with 270 minutes of exposure. Increasing the pH from its unadjusted level (about 6.5) to pH 8 improved the sporicidal activity of OPA⁶⁹⁴. The level of biocidal activity was directly related to the

temperature. A greater than 5- \log_{10} reduction of *B. atrophaeus* spores was observed in 3 hours at 35°C, than in 24 hours at 20°C. Also, with an exposure time \leq 5 minutes, biocidal activity decreased with increasing serum concentration. However, efficacy did not differ when the exposure time was \geq 10 minutes⁶⁹⁷. In addition, OPA is effective ($>5\text{-}\log_{10}$ reduction) against a wide range of microorganisms, including glutaraldehyde-resistant mycobacteria and *B. atrophaeus* spores⁶⁹⁴.

The influence of laboratory adaptation of test strains, such as *P. aeruginosa*, to 0.55% OPA has been evaluated. Resistant and multiresistant strains increased substantially in susceptibility to OPA after laboratory adaptation (\log_{10} reduction factors increased by 0.54 and 0.91 for resistant and multiresistant strains, respectively)⁷⁰⁴. Other studies have found naturally occurring cells of *P. aeruginosa* were more resistant to a variety of disinfectants than were subcultured cells⁷⁰⁵.

Uses. OPA has several potential advantages over glutaraldehyde. It has excellent stability over a wide pH range (pH 3–9), is not a known irritant to the eyes and nasal passages⁷⁰⁶, does not require exposure monitoring, has a barely perceptible odor, and requires no activation. OPA, like glutaraldehyde, has excellent material compatibility. A potential disadvantage of OPA is that it stains proteins gray (including unprotected skin) and thus must be handled with caution⁶⁹. However, skin staining would indicate improper handling that requires additional training and/or personal protective equipment (e.g., gloves, eye and mouth protection, and fluid-resistant gowns). OPA residues remaining on inadequately water-rinsed transesophageal echo probes can stain the patient's mouth⁷⁰⁷. Meticulous cleaning, using the correct OPA exposure time (e.g., 12 minutes) and copious rinsing of the probe with water should eliminate this problem. The results of one study provided a basis for a recommendation that rinsing of instruments disinfected with OPA will require at least 250 mL of water per channel to reduce the chemical residue to a level that will not compromise patient or staff safety (<1 ppm)⁷⁰⁸. Personal protective equipment should be worn when contaminated instruments, equipment, and chemicals are handled⁴⁰⁰. In addition, equipment must be thoroughly rinsed to prevent discoloration of a patient's skin or mucous membrane.

In April 2004, the manufacturer of OPA disseminated information to users about patients who reportedly experienced an anaphylaxis-like reaction after cystoscopy where the scope had been reprocessed using OPA. Of approximately 1 million urologic procedures performed using instruments reprocessed using OPA, 24 cases (17 cases in the United States, six in Japan, one in the United Kingdom) of anaphylaxis-like reactions have been reported after repeated cystoscopy (typically after four to nine treatments). Preventive measures include removal of OPA residues by thorough rinsing and not using OPA for reprocessing urologic instrumentation used to treat patients with a history of bladder cancer (Nevine Erian, personal communication, June 4, 2004; Product Notification, Advanced Sterilization Products, April 23, 2004)⁷⁰⁹.

A few OPA clinical studies are available. In a clinical-use study, OPA exposure of 100 endoscopes for 5 minutes resulted in a $>5\text{-}\log_{10}$ reduction in bacterial load. Furthermore, OPA was effective over a 14-day use cycle¹⁰⁰. Manufacturer data show that OPA will last longer in an automatic endoscope reprocessor before reaching its MEC limit (MEC after 82 cycles) than will glutaraldehyde (MEC after 40 cycles)⁴⁰⁰. High-pressure liquid chromatography confirmed that OPA levels are maintained above 0.3% for at least 50 cycles^{706, 710}. OPA must be disposed in accordance with local and state regulations. If OPA disposal through the sanitary sewer system is restricted, glycine (25 grams/gallon) can be used to neutralize the OPA and make it safe for disposal.

The high-level disinfectant label claims for OPA solution at 20°C vary worldwide (e.g., 5 minutes in Europe, Asia, and Latin America; 10 minutes in Canada and Australia; and 12 minutes in the United States). These label claims differ worldwide because of differences in the test methodology and requirements for licensure. In an automated endoscope reprocessor with an FDA-cleared capability to maintain solution temperatures at 25°C, the contact time for OPA is 5 minutes.

Peracetic Acid

Overview. Peracetic, or peroxyacetic, acid is characterized by rapid action against all microorganisms. Special advantages of peracetic acid are that it lacks harmful decomposition products (i.e., acetic acid, water, oxygen, hydrogen peroxide), enhances removal of organic material⁷¹¹, and leaves no residue. It remains effective in the presence of organic matter and is sporicidal even at low temperatures (Tables 4 and 5). Peracetic acid can corrode copper, brass, bronze, plain steel, and galvanized iron but these effects can be reduced by additives and pH modifications. It is considered unstable, particularly when diluted; for example, a 1% solution loses half its strength through hydrolysis in 6 days, whereas 40% peracetic acid loses 1%–2% of its active ingredients per month⁶⁵⁴.

Mode of Action. Little is known about the mechanism of action of peracetic acid, but it is believed to function similarly to other oxidizing agents—that is, it denatures proteins, disrupts the cell wall permeability, and oxidizes sulfhydryl and sulfur bonds in proteins, enzymes, and other metabolites⁶⁵⁴.

Microbicidal Activity. Peracetic acid will inactivate gram-positive and gram-negative bacteria, fungi, and yeasts in <5 minutes at <100 ppm. In the presence of organic matter, 200–500 ppm is required. For viruses, the dosage range is wide (12–2250 ppm), with poliovirus inactivated in yeast extract in 15 minutes with 1,500–2,250 ppm. In one study, 3.5% peracetic acid was ineffective against HAV after 1-minute exposure using a carrier test⁵⁸. Peracetic acid (0.26%) was effective (log₁₀ reduction factor >5) against all test strains of mycobacteria (*M. tuberculosis*, *M. avium-intracellulare*, *M. chelonae*, and *M. fortuitum*) within 20–30 minutes in the presence or absence of an organic load^{607, 712}. With bacterial spores, 500–10,000 ppm (0.05%–1%) inactivates spores in 15 seconds to 30 minutes using a spore suspension test^{654, 659, 713-715}.

Uses. An automated machine using peracetic acid to chemically sterilize medical (e.g., endoscopes, arthroscopes), surgical, and dental instruments is used in the United States⁷¹⁶⁻⁷¹⁸. As previously noted, dental handpieces should be steam sterilized. The sterilant, 35% peracetic acid, is diluted to 0.2% with filtered water at 50°C. Simulated-use trials have demonstrated excellent microbicidal activity^{111, 718-722}, and three clinical trials have demonstrated both excellent microbial killing and no clinical failures leading to infection^{90, 723, 724}. The high efficacy of the system was demonstrated in a comparison of the efficacies of the system with that of ethylene oxide. Only the peracetic acid system completely killed 6 log₁₀ of *M. chelonae*, *E. faecalis*, and *B. atrophaeus* spores with both an organic and inorganic challenge⁷²². An investigation that compared the costs, performance, and maintenance of urologic endoscopic equipment processed by high-level disinfection (with glutaraldehyde) with those of the peracetic acid system reported no clinical differences between the two systems. However, the use of this system led to higher costs than the high-level disinfection, including costs for processing (\$6.11 vs. \$0.45 per cycle), purchasing and training (\$24,845 vs. \$16), installation (\$5,800 vs. \$0), and endoscope repairs (\$6,037 vs. \$445)⁹⁰. Furthermore, three clusters of infection using the peracetic acid automated endoscope reprocessor were linked to inadequately processed bronchoscopes when inappropriate channel connectors were used with the system⁷²⁵. These clusters highlight the importance of training, proper model-specific endoscope connector systems, and quality-control procedures to ensure compliance with endoscope manufacturer recommendations and professional organization guidelines. An alternative high-level disinfectant available in the United Kingdom contains 0.35% peracetic acid. Although this product is rapidly effective against a broad range of microorganisms^{466, 726, 727}, it tarnishes the metal of endoscopes and is unstable, resulting in only a 24-hour use life⁷²⁷.

Peracetic Acid and Hydrogen Peroxide

Overview. Two chemical sterilants are available that contain peracetic acid plus hydrogen peroxide (i.e., 0.08% peracetic acid plus 1.0% hydrogen peroxide [no longer marketed]; and 0.23% peracetic acid plus 7.35% hydrogen peroxide (Tables 4 and 5).

Microbicidal Activity. The bactericidal properties of peracetic acid and hydrogen peroxide have been demonstrated⁷²⁸. Manufacturer data demonstrated this combination of peracetic acid and

hydrogen peroxide inactivated all microorganisms except bacterial spores within 20 minutes. The 0.08% peracetic acid plus 1.0% hydrogen peroxide product effectively inactivated glutaraldehyde-resistant mycobacteria⁷²⁹.

Uses. The combination of peracetic acid and hydrogen peroxide has been used for disinfecting hemodialyzers⁷³⁰. The percentage of dialysis centers using a peracetic acid-hydrogen peroxide-based disinfectant for reprocessing dialyzers increased from 5% in 1983 to 56% in 1997²⁴⁹. Olympus America does not endorse use of 0.08% peracetic acid plus 1.0% hydrogen peroxide (Olympus America, personal communication, April 15, 1998) on any Olympus endoscope because of cosmetic and functional damage and will not assume liability for chemical damage resulting from use of this product. This product is not currently available. FDA has cleared a newer chemical sterilant with 0.23% peracetic acid and 7.35% hydrogen peroxide (Tables 4 and 5). After testing the 7.35% hydrogen peroxide and 0.23% peracetic acid product, Olympus America concluded it was not compatible with the company's flexible gastrointestinal endoscopes; this conclusion was based on immersion studies where the test insertion tubes had failed because of swelling and loosening of the black polymer layer of the tube (Olympus America, personal communication, September 13, 2000).

Phenolics

Overview. Phenol has occupied a prominent place in the field of hospital disinfection since its initial use as a germicide by Lister in his pioneering work on antiseptic surgery. In the past 30 years, however, work has concentrated on the numerous phenol derivatives or phenolics and their antimicrobial properties. Phenol derivatives originate when a functional group (e.g., alkyl, phenyl, benzyl, halogen) replaces one of the hydrogen atoms on the aromatic ring. Two phenol derivatives commonly found as constituents of hospital disinfectants are *ortho*-phenylphenol and *ortho*-benzyl-*para*-chlorophenol. The antimicrobial properties of these compounds and many other phenol derivatives are much improved over those of the parent chemical. Phenolics are absorbed by porous materials, and the residual disinfectant can irritate tissue. In 1970, depigmentation of the skin was reported to be caused by phenolic germicidal detergents containing *para*-tertiary butylphenol and *para*-tertiary amyphenol⁷³¹.

Mode of Action. In high concentrations, phenol acts as a gross protoplasmic poison, penetrating and disrupting the cell wall and precipitating the cell proteins. Low concentrations of phenol and higher molecular-weight phenol derivatives cause bacterial death by inactivation of essential enzyme systems and leakage of essential metabolites from the cell wall⁷³².

Microbicidal Activity. Published reports on the antimicrobial efficacy of commonly used phenolics showed they were bactericidal, fungicidal, virucidal, and tuberculocidal^{14, 61, 71, 73, 227, 416, 573, 732-738}. One study demonstrated little or no virucidal effect of a phenolic against coxsackie B4, echovirus 11, and poliovirus 1⁷³⁶. Similarly, 12% *ortho*-phenylphenol failed to inactivate any of the three hydrophilic viruses after a 10-minute exposure time, although 5% phenol was lethal for these viruses⁷². A 0.5% dilution of a phenolic (2.8% *ortho*-phenylphenol and 2.7% *ortho*-benzyl-*para*-chlorophenol) inactivated HIV²²⁷ and a 2% solution of a phenolic (15% *ortho*-phenylphenol and 6.3% *para*-tertiary-amyphenol) inactivated all but one of 11 fungi tested⁷¹.

Manufacturers' data using the standardized AOAC methods demonstrate that commercial phenolics are not sporicidal but are tuberculocidal, fungicidal, virucidal, and bactericidal at their recommended use-dilution. Attempts to substantiate the bactericidal label claims of phenolics using the AOAC Use-Dilution Method occasionally have failed^{416, 737}. However, results from these same studies have varied dramatically among laboratories testing identical products.

Uses. Many phenolic germicides are EPA-registered as disinfectants for use on environmental surfaces (e.g., bedside tables, bedrails, and laboratory surfaces) and noncritical medical devices. Phenolics are not FDA-cleared as high-level disinfectants for use with semicritical items but could be used to preclean or decontaminate critical and semicritical devices before terminal sterilization or high-

level disinfection.

The use of phenolics in nurseries has been questioned because of hyperbilirubinemia in infants placed in bassinets where phenolic detergents were used⁷³⁹. In addition, bilirubin levels were reported to increase in phenolic-exposed infants, compared with nonphenolic-exposed infants, when the phenolic was prepared according to the manufacturers' recommended dilution⁷⁴⁰. If phenolics are used to clean nursery floors, they must be diluted as recommended on the product label. Phenolics (and other disinfectants) should not be used to clean infant bassinets and incubators while occupied. If phenolics are used to terminally clean infant bassinets and incubators, the surfaces should be rinsed thoroughly with water and dried before reuse of infant bassinets and incubators¹⁷.

Quaternary Ammonium Compounds

Overview. The quaternary ammonium compounds are widely used as disinfectants. Health-care-associated infections have been reported from contaminated quaternary ammonium compounds used to disinfect patient-care supplies or equipment, such as cystoscopes or cardiac catheters^{741, 742}. The quaternaries are good cleaning agents, but high water hardness⁷⁴³ and materials such as cotton and gauze pads can make them less microbicidal because of insoluble precipitates or cotton and gauze pads absorb the active ingredients, respectively. One study showed a significant decline (~40%–50% lower at 1 hour) in the concentration of quaternaries released when cotton rags or cellulose-based wipers were used in the open-bucket system, compared with the nonwoven spunlace wipers in the closed-bucket system⁷⁴⁴. As with several other disinfectants (e.g., phenolics, iodophors) gram-negative bacteria can survive or grow in them⁴⁰⁴.

Chemically, the quaternaries are organically substituted ammonium compounds in which the nitrogen atom has a valence of 5, four of the substituent radicals (R1-R4) are alkyl or heterocyclic radicals of a given size or chain length, and the fifth (X⁻) is a halide, sulfate, or similar radical⁷⁴⁵. Each compound exhibits its own antimicrobial characteristics, hence the search for one compound with outstanding antimicrobial properties. Some of the chemical names of quaternary ammonium compounds used in healthcare are alkyl dimethyl benzyl ammonium chloride, alkyl didecyl dimethyl ammonium chloride, and dialkyl dimethyl ammonium chloride. The newer quaternary ammonium compounds (i.e., fourth generation), referred to as twin-chain or dialkyl quaternaries (e.g. didecyl dimethyl ammonium bromide and dioctyl dimethyl ammonium bromide), purportedly remain active in hard water and are tolerant of anionic residues⁷⁴⁶.

A few case reports have documented occupational asthma as a result of exposure to benzalkonium chloride⁷⁴⁷.

Mode of Action. The bactericidal action of the quaternaries has been attributed to the inactivation of energy-producing enzymes, denaturation of essential cell proteins, and disruption of the cell membrane⁷⁴⁶. Evidence exists that supports these and other possibilities^{745 748}.

Microbicidal Activity. Results from manufacturers' data sheets and from published scientific literature indicate that the quaternaries sold as hospital disinfectants are generally fungicidal, bactericidal, and virucidal against lipophilic (enveloped) viruses; they are not sporicidal and generally not tuberculocidal or virucidal against hydrophilic (nonenveloped) viruses^{14, 54-56, 58, 59, 61, 71, 73, 186, 297, 748, 749}. The poor mycobactericidal activities of quaternary ammonium compounds have been demonstrated^{55, 73}. Quaternary ammonium compounds (as well as 70% isopropyl alcohol, phenolic, and a chlorine-containing wipe [80 ppm]) effectively (>95%) remove and/or inactivate contaminants (i.e., multidrug-resistant *S. aureus*, vancomycin-resistant *Enterococcus*, *P. aeruginosa*) from computer keyboards with a 5-second application time. No functional damage or cosmetic changes occurred to the computer keyboards after 300 applications of the disinfectants⁴⁵.

Attempts to reproduce the manufacturers' bactericidal and tuberculocidal claims using the AOAC

tests with a limited number of quaternary ammonium compounds occasionally have failed^{73, 416, 737}. However, test results have varied extensively among laboratories testing identical products^{416, 737}.

Uses. The quaternaries commonly are used in ordinary environmental sanitation of noncritical surfaces, such as floors, furniture, and walls. EPA-registered quaternary ammonium compounds are appropriate to use for disinfecting medical equipment that contacts intact skin (e.g., blood pressure cuffs).

MISCELLANEOUS INACTIVATING AGENTS

Other Germicides

Several compounds have antimicrobial activity but for various reasons have not been incorporated into the armamentarium of health-care disinfectants. These include mercurials, sodium hydroxide, β -propiolactone, chlorhexidine gluconate, cetrimide-chlorhexidine, glycols (triethylene and propylene), and the Tego disinfectants. Two authoritative references examine these agents in detail^{16,412}.

A peroxygen-containing formulation had marked bactericidal action when used as a 1% weight/volume solution and virucidal activity at 3%⁴⁹, but did not have mycobactericidal activity at concentrations of 2.3% and 4% and exposure times ranging from 30 to 120 minutes⁷⁵⁰. It also required 20 hours to kill *B. atrophaeus* spores⁷⁵¹. A powder-based peroxygen compound for disinfecting contaminated spill was strongly and rapidly bactericidal⁷⁵².

In preliminary studies, nanoemulsions (composed of detergents and lipids in water) showed activity against vegetative bacteria, enveloped viruses and *Candida*. This product represents a potential agent for use as a topical biocidal agent.⁷⁵³⁻⁷⁵⁵

New disinfectants that require further evaluation include glucoprotamin⁷⁵⁶, tertiary amines⁷⁰³, and a light-activated antimicrobial coating⁷⁵⁷. Several other disinfection technologies might have potential applications in the healthcare setting⁷⁵⁸.

Metals as Microbicides

Comprehensive reviews of antiseptics⁷⁵⁹, disinfection⁴²¹, and anti-infective chemotherapy⁷⁶⁰ barely mention the antimicrobial activity of heavy metals^{761,762}. Nevertheless, the anti-infective activity of some heavy metals has been known since antiquity. Heavy metals such as silver have been used for prophylaxis of conjunctivitis of the newborn, topical therapy for burn wounds, and bonding to indwelling catheters, and the use of heavy metals as antiseptics or disinfectants is again being explored⁷⁶³. Inactivation of bacteria on stainless steel surfaces by zeolite ceramic coatings containing silver and zinc ions has also been demonstrated^{764,765}.

Metals such as silver, iron, and copper could be used for environmental control, disinfection of water, or reusable medical devices or incorporated into medical devices (e.g., intravascular catheters)^{400,761-763,766-770}. A comparative evaluation of six disinfectant formulations for residual antimicrobial activity demonstrated that only the silver disinfectant demonstrated significant residual activity against *S. aureus* and *P. aeruginosa*⁷⁶³. Preliminary data suggest metals are effective against a wide variety of microorganisms.

Clinical uses of other heavy metals include copper-8-quinolinolate as a fungicide against *Aspergillus*, copper-silver ionization for *Legionella* disinfection⁷⁷¹⁻⁷⁷⁴, organic mercurials as an antiseptic (e.g., mercurochrome) and preservative/disinfectant (e.g., thimerosal [currently being removed from vaccines]) in pharmaceuticals and cosmetics⁷⁶².

Ultraviolet Radiation (UV)

The wavelength of UV radiation ranges from 328 nm to 210 nm (3280 Å to 2100 Å). Its maximum bactericidal effect occurs at 240–280 nm. Mercury vapor lamps emit more than 90% of their radiation at 253.7 nm, which is near the maximum microbicidal activity⁷⁷⁵. Inactivation of microorganisms results from destruction of nucleic acid through induction of thymine dimers. UV radiation has been employed in the disinfection of drinking water⁷⁷⁶, air⁷⁷⁵, titanium implants⁷⁷⁷, and contact lenses⁷⁷⁸. Bacteria and viruses are more easily killed by UV light than are bacterial spores⁷⁷⁵. UV radiation has several potential applications, but unfortunately its germicidal effectiveness and use is influenced by organic matter; wavelength; type of suspension; temperature; type of microorganism; and UV intensity, which is affected by distance and dirty tubes⁷⁷⁹. The application of UV radiation in the health-care environment (i.e.,

operating rooms, isolation rooms, and biologic safety cabinets) is limited to destruction of airborne organisms or inactivation of microorganisms on surfaces. The effect of UV radiation on postoperative wound infections was investigated in a double-blind, randomized study in five university medical centers. After following 14,854 patients over a 2-year period, the investigators reported the overall wound infection rate was unaffected by UV radiation, although postoperative infection in the “refined clean” surgical procedures decreased significantly (3.8%–2.9%)⁷⁸⁰. No data support the use of UV lamps in isolation rooms, and this practice has caused at least one epidemic of UV-induced skin erythema and keratoconjunctivitis in hospital patients and visitors⁷⁸¹.

Pasteurization

Pasteurization is not a sterilization process; its purpose is to destroy all pathogenic microorganisms. However, pasteurization does not destroy bacterial spores. The time-temperature relation for hot-water pasteurization is generally ~70°C (158°F) for 30 minutes. The water temperature and time should be monitored as part of a quality-assurance program⁷⁸². Pasteurization of respiratory therapy^{783, 784} and anesthesia equipment⁷⁸⁵ is a recognized alternative to chemical disinfection. The efficacy of this process has been tested using an inoculum that the authors believed might simulate contamination by an infected patient. Use of a large inoculum (10^7) of *P. aeruginosa* or *Acinetobacter calcoaceticus* in sets of respiratory tubing before processing demonstrated that machine-assisted chemical processing was more efficient than machine-assisted pasteurization with a disinfection failure rate of 6% and 83%, respectively⁷⁸³. Other investigators found hot water disinfection to be effective (inactivation factor $>5 \log_{10}$) against multiple bacteria, including multidrug-resistant bacteria, for disinfecting reusable anesthesia or respiratory therapy equipment⁷⁸⁴⁻⁷⁸⁶.

Flushing- and Washer-Disinfectors

Flushing- and washer-disinfectors are automated and closed equipment that clean and disinfect objects from bedpans and washbowls to surgical instruments and anesthesia tubes. Items such as bedpans and urinals can be cleaned and disinfected in flushing-disinfectors. They have a short cycle of a few minutes. They clean by flushing with warm water, possibly with a detergent, and then disinfect by flushing the items with hot water or with steam. Because this machine empties, cleans, and disinfects, manual cleaning is eliminated, fewer disposable items are needed, and fewer chemical germicides are used. A microbiologic evaluation of one washer/disinfector demonstrated complete inactivation of suspensions of *E. faecalis* or poliovirus⁷⁸⁷. Other studies have shown that strains of *Enterococcus faecium* can survive the British Standard for heat disinfection of bedpans (80°C for 1 minute). The significance of this finding with reference to the potential for enterococci to survive and disseminate in the health-care environment is debatable⁷⁸⁸⁻⁷⁹⁰. These machines are available and used in many European countries.

Surgical instruments and anesthesia equipment are more difficult to clean. They are run in washer-disinfectors on a longer cycle of approximately 20–30 minutes with a detergent. These machines also disinfect by hot water at approximately 90°C⁷⁹¹.

THE REGULATORY FRAMEWORK FOR DISINFECTANTS AND STERILANTS

Before using the guidance provided in this document, health-care workers should be aware of the federal laws and regulations that govern the sale, distribution, and use of disinfectants and sterilants. In particular, health-care workers need to know what requirements pertain to them when they apply these products. Finally, they should understand the relative roles of EPA, FDA, and CDC so the context for the guidance provided in this document is clear.

EPA and FDA

In the United States, chemical germicides formulated as sanitizers, disinfectants, or sterilants are regulated in interstate commerce by the Antimicrobials Division, Office of Pesticides Program, EPA, under the authority of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) of 1947, as amended⁷⁹². Under FIFRA, any substance or mixture of substances intended to prevent, destroy, repel, or mitigate any pest (including microorganisms but excluding those in or on living humans or animals) must be registered before sale or distribution. To obtain a registration, a manufacturer must submit specific data about the safety and effectiveness of each product. For example, EPA requires manufacturers of sanitizers, disinfectants, or chemical sterilants to test formulations by using accepted methods for microbiocidal activity, stability, and toxicity to animals and humans. The manufacturers submit these data to EPA along with proposed labeling. If EPA concludes the product can be used without causing “unreasonable adverse effects,” then the product and its labeling are registered, and the manufacturer can sell and distribute the product in the United States.

FIFRA also requires users of products to follow explicitly the labeling directions on each product. The following standard statement appears on all labels under the “Directions for Use” heading: “It is a violation of federal law to use this product in a manner inconsistent with its labeling.” This statement means a health-care worker must follow the safety precautions and use directions on the labeling of each registered product. Failure to follow the specified use-dilution, contact time, method of application, or any other condition of use is considered a misuse of the product and potentially subject to enforcement action under FIFRA.

In general, EPA regulates disinfectants and sterilants used on environmental surfaces, and not those used on critical or semicritical medical devices; the latter are regulated by FDA. In June 1993, FDA and EPA issued a “Memorandum of Understanding” that divided responsibility for review and surveillance of chemical germicides between the two agencies. Under the agreement, FDA regulates liquid chemical sterilants used on critical and semicritical devices, and EPA regulates disinfectants used on noncritical surfaces and gaseous sterilants⁷⁹³. In 1996, Congress passed the Food Quality Protection Act (FQPA). This act amended FIFRA in regard to several types of products regulated by both EPA and FDA. One provision of FQPA removed regulation of liquid chemical sterilants used on critical and semicritical medical devices from EPA’s jurisdiction, and it now rests solely with FDA^{792, 794}. EPA continues to register nonmedical chemical sterilants. FDA and EPA have considered the impact of FQPA, and in January 2000, FDA published its final guidance document on product submissions and labeling. Antiseptics are considered antimicrobial drugs used on living tissue and thus are regulated by FDA under the Food, Drug and Cosmetic Act. FDA regulates liquid chemical sterilants and high-level disinfectants intended to process critical and semicritical devices. FDA has published recommendations on the types of test methods that manufacturers should submit to FDA for 510[k] clearance for such agents.

CDC

At CDC, the mission of the Coordinating Center for Infections Diseases is to guide the public on how to prevent and respond to infectious diseases in both health-care settings and at home. With respect to disinfectants and sterilants, part of CDC’s role is to inform the public (in this case healthcare personnel) of current scientific evidence pertaining to these products, to comment about their safety and efficacy, and to recommend which chemicals might be most appropriate or effective for specific microorganisms and settings.

Test Methods

The methods EPA has used for registration are standardized by the AOAC International; however, a survey of scientific literature reveals a number of problems with these tests that were reported during 1987–1990^{58, 76, 80, 428, 736, 737, 795-800} that cause them to be neither accurate nor reproducible^{416, 737}.

As part of their regulatory authority, EPA and FDA support development and validation of methods for assessing disinfection claims⁸⁰¹⁻⁸⁰³. For example, EPA has supported the work of Dr. Syed Sattar and coworkers who have developed a two-tier quantitative carrier test to assess sporicidal, mycobactericidal, bactericidal, fungicidal, virucidal, and protozoacidal activity of chemical germicides^{701, 803}. EPA is accepting label claims against hepatitis B virus (HBV) using a surrogate organism, the duck HBV, to quantify disinfectant activity^{124, 804}. EPA also is accepting labeling claims against hepatitis C virus using the bovine viral diarrhea virus as a surrogate.

For nearly 30 years, EPA also performed intramural preregistration and postregistration efficacy testing of some chemical disinfectants in its own laboratories. In 1982, this was stopped, reportedly for budgetary reasons. At that time, manufacturers did not need to have microbiologic activity claims verified by EPA or an independent testing laboratory when registering a disinfectant or chemical sterilant⁸⁰⁵. This occurred when the frequency of contaminated germicides and infections secondary to their use had increased⁴⁰⁴. Investigations demonstrating that interlaboratory reproducibility of test results was poor and manufacturers' label claims were not verifiable^{416, 737} and symposia sponsored by the American Society for Microbiology⁸⁰⁰ heightened awareness of these problems and reconfirmed the need to improve the AOAC methods and reinstate a microbiologic activity verification program. A General Accounting Office report entitled *Disinfectants: EPA Lacks Assurance They Work*⁸⁰⁶ seemed to provide the necessary impetus for EPA to initiate corrective measures, including cooperative agreements to improve the AOAC methods and independent verification testing for all products labeled as sporicidal and disinfectants labeled as tuberculocidal. For example, of 26 sterilant products tested by EPA, 15 were canceled because of product failure. A list of products registered with EPA and labeled for use as sterilants or tuberculocides or against HIV and/or HBV is available through EPA's website at <http://www.epa.gov/oppad001/chemregindex.htm>. Organizations (e.g., Organization for Economic Cooperation and Development) are working to standardize requirements for germicide testing and registration.

Neutralization of Germicides

One of the difficulties associated with evaluating the bactericidal activity of disinfectants is prevention of bacteriostasis from disinfectant residues carried over into the subculture media. Likewise, small amounts of disinfectants on environmental surfaces can make an accurate bacterial count difficult to get when sampling of the health-care environment as part of an epidemiologic or research investigation. One way these problems may be overcome is by employing neutralizers that inactivate residual disinfectants⁸⁰⁷⁻⁸⁰⁹. Two commonly used neutralizing media for chemical disinfectants are Lethen Media and D/E Neutralizing Media. The former contains lecithin to neutralize quaternaries and polysorbate 80 (Tween 80) to neutralize phenolics, hexachlorophene, formalin, and, with lecithin, ethanol. The D/E Neutralizing media will neutralize a broad spectrum of antiseptic and disinfectant chemicals, including quaternary ammonium compounds, phenols, iodine and chlorine compounds, mercurials, formaldehyde, and glutaraldehyde⁸¹⁰. A review of neutralizers used in germicide testing has been published⁸⁰⁸.

STERILIZATION

Most medical and surgical devices used in healthcare facilities are made of materials that are heat stable and therefore undergo heat, primarily steam, sterilization. However, since 1950, there has been an increase in medical devices and instruments made of materials (e.g., plastics) that require low-temperature sterilization. Ethylene oxide gas has been used since the 1950s for heat- and moisture-sensitive medical devices. Within the past 15 years, a number of new, low-temperature sterilization systems (e.g., hydrogen peroxide gas plasma, peracetic acid immersion, ozone) have been developed and are being used to sterilize medical devices. This section reviews sterilization technologies used in healthcare and makes recommendations for their optimum performance in the processing of medical devices^{1, 18, 811-820}.

Sterilization destroys all microorganisms on the surface of an article or in a fluid to prevent disease transmission associated with the use of that item. While the use of inadequately sterilized critical items represents a high risk of transmitting pathogens, documented transmission of pathogens associated with an inadequately sterilized critical item is exceedingly rare^{821, 822}. This is likely due to the wide margin of safety associated with the sterilization processes used in healthcare facilities. The concept of what constitutes "sterile" is measured as a probability of sterility for each item to be sterilized. This probability is commonly referred to as the sterility assurance level (SAL) of the product and is defined as the probability of a single viable microorganism occurring on a product after sterilization. SAL is normally expressed as a 10^{-n} . For example, if the probability of a spore surviving were one in one million, the SAL would be 10^{-6} ^{823, 824}. In short, a SAL is an estimate of lethality of the entire sterilization process and is a conservative calculation. Dual SALs (e.g., 10^{-3} SAL for blood culture tubes, drainage bags; 10^{-6} SAL for scalpels, implants) have been used in the United States for many years and the choice of a 10^{-6} SAL was strictly arbitrary and not associated with any adverse outcomes (e.g., patient infections)⁸²³.

Medical devices that have contact with sterile body tissues or fluids are considered critical items. These items should be sterile when used because any microbial contamination could result in disease transmission. Such items include surgical instruments, biopsy forceps, and implanted medical devices. If these items are heat resistant, the recommended sterilization process is steam sterilization, because it has the largest margin of safety due to its reliability, consistency, and lethality. However, reprocessing heat- and moisture-sensitive items requires use of a low-temperature sterilization technology (e.g., ethylene oxide, hydrogen peroxide gas plasma, peracetic acid)⁸²⁵. A summary of the advantages and disadvantages for commonly used sterilization technologies is presented in Table 6.

Steam Sterilization

Overview. Of all the methods available for sterilization, moist heat in the form of saturated steam under pressure is the most widely used and the most dependable. Steam sterilization is nontoxic, inexpensive⁸²⁶, rapidly microbicidal, sporicidal, and rapidly heats and penetrates fabrics (Table 6)⁸²⁷. Like all sterilization processes, steam sterilization has some deleterious effects on some materials, including corrosion and combustion of lubricants associated with dental handpieces²¹²; reduction in ability to transmit light associated with laryngoscopes⁸²⁸; and increased hardening time (5.6 fold) with plaster-cast⁸²⁹.

The basic principle of steam sterilization, as accomplished in an autoclave, is to expose each item to direct steam contact at the required temperature and pressure for the specified time. Thus, there are four parameters of steam sterilization: steam, pressure, temperature, and time. The ideal steam for sterilization is dry saturated steam and entrained water (dryness fraction $\geq 97\%$)^{813, 819}. Pressure serves as a means to obtain the high temperatures necessary to quickly kill microorganisms. Specific temperatures must be obtained to ensure the microbicidal activity. The two common steam-sterilizing temperatures are 121°C (250°F) and 132°C (270°F). These temperatures (and other high temperatures)⁸³⁰ must be maintained for a minimal time to kill microorganisms. Recognized minimum exposure periods for sterilization of wrapped healthcare supplies are 30 minutes at 121°C (250°F) in a gravity displacement

sterilizer or 4 minutes at 132°C (270°C) in a prevacuum sterilizer (Table 7). At constant temperatures, sterilization times vary depending on the type of item (e.g., metal versus rubber, plastic, items with lumens), whether the item is wrapped or unwrapped, and the sterilizer type.

The two basic types of steam sterilizers (autoclaves) are the gravity displacement autoclave and the high-speed prevacuum sterilizer. In the former, steam is admitted at the top or the sides of the sterilizing chamber and, because the steam is lighter than air, forces air out the bottom of the chamber through the drain vent. The gravity displacement autoclaves are primarily used to process laboratory media, water, pharmaceutical products, regulated medical waste, and nonporous articles whose surfaces have direct steam contact. For gravity displacement sterilizers the penetration time into porous items is prolonged because of incomplete air elimination. This point is illustrated with the decontamination of 10 lbs of microbiological waste, which requires at least 45 minutes at 121°C because the entrapped air remaining in a load of waste greatly retards steam permeation and heating efficiency^{831, 832}. The high-speed prevacuum sterilizers are similar to the gravity displacement sterilizers except they are fitted with a vacuum pump (or ejector) to ensure air removal from the sterilizing chamber and load before the steam is admitted. The advantage of using a vacuum pump is that there is nearly instantaneous steam penetration even into porous loads. The Bowie-Dick test is used to detect air leaks and inadequate air removal and consists of folded 100% cotton surgical towels that are clean and preconditioned. A commercially available Bowie-Dick-type test sheet should be placed in the center of the pack. The test pack should be placed horizontally in the front, bottom section of the sterilizer rack, near the door and over the drain, in an otherwise empty chamber and run at 134°C for 3.5 minutes^{813, 819}. The test is used each day the vacuum-type steam sterilizer is used, before the first processed load. Air that is not removed from the chamber will interfere with steam contact. Smaller disposable test packs (or process challenge devices) have been devised to replace the stack of folded surgical towels for testing the efficacy of the vacuum system in a prevacuum sterilizer.⁸³³ These devices are “designed to simulate product to be sterilized and to constitute a defined challenge to the sterilization process”^{819, 834}. They should be representative of the load and simulate the greatest challenge to the load⁸³⁵. Sterilizer vacuum performance is acceptable if the sheet inside the test pack shows a uniform color change. Entrapped air will cause a spot to appear on the test sheet, due to the inability of the steam to reach the chemical indicator. If the sterilizer fails the Bowie-Dick test, do not use the sterilizer until it is inspected by the sterilizer maintenance personnel and passes the Bowie-Dick test^{813, 819, 836}.

Another design in steam sterilization is a steam flush-pressure pulsing process, which removes air rapidly by repeatedly alternating a steam flush and a pressure pulse above atmospheric pressure. Air is rapidly removed from the load as with the prevacuum sterilizer, but air leaks do not affect this process because the steam in the sterilizing chamber is always above atmospheric pressure. Typical sterilization temperatures and times are 132°C to 135°C with 3 to 4 minutes exposure time for porous loads and instruments^{827, 837}.

Like other sterilization systems, the steam cycle is monitored by mechanical, chemical, and biological monitors. Steam sterilizers usually are monitored using a printout (or graphically) by measuring temperature, the time at the temperature, and pressure. Typically, chemical indicators are affixed to the outside and incorporated into the pack to monitor the temperature or time and temperature. The effectiveness of steam sterilization is monitored with a biological indicator containing spores of *Geobacillus stearothermophilus* (formerly *Bacillus stearothermophilus*). Positive spore test results are a relatively rare event⁸³⁸ and can be attributed to operator error, inadequate steam delivery⁸³⁹, or equipment malfunction.

Portable (table-top) steam sterilizers are used in outpatient, dental, and rural clinics⁸⁴⁰. These sterilizers are designed for small instruments, such as hypodermic syringes and needles and dental instruments. The ability of the sterilizer to reach physical parameters necessary to achieve sterilization should be monitored by mechanical, chemical, and biological indicators.

Microbicidal Activity. The oldest and most recognized agent for inactivation of microorganisms is heat. D-values (time to reduce the surviving population by 90% or 1 log₁₀) allow a direct comparison of the heat resistance of microorganisms. Because a D-value can be determined at various temperatures, a subscript is used to designate the exposure temperature (i.e., D_{121C}). D_{121C}-values for *Geobacillus stearothermophilus* used to monitor the steam sterilization process range from 1 to 2 minutes. Heat-resistant nonspore-forming bacteria, yeasts, and fungi have such low D_{121C} values that they cannot be experimentally measured⁸⁴¹.

Mode of Action. Moist heat destroys microorganisms by the irreversible coagulation and denaturation of enzymes and structural proteins. In support of this fact, it has been found that the presence of moisture significantly affects the coagulation temperature of proteins and the temperature at which microorganisms are destroyed.

Uses. Steam sterilization should be used whenever possible on all critical and semicritical items that are heat and moisture resistant (e.g., steam sterilizable respiratory therapy and anesthesia equipment), even when not essential to prevent pathogen transmission. Steam sterilizers also are used in healthcare facilities to decontaminate microbiological waste and sharps containers^{831, 832, 842} but additional exposure time is required in the gravity displacement sterilizer for these items.

Flash Sterilization

Overview. “Flash” steam sterilization was originally defined by Underwood and Perkins as sterilization of an unwrapped object at 132°C for 3 minutes at 27-28 lbs. of pressure in a gravity displacement sterilizer⁸⁴³. Currently, the time required for flash sterilization depends on the type of sterilizer and the type of item (i.e., porous vs non-porous items)(see Table 8). Although the wrapped method of sterilization is preferred for the reasons listed below, correctly performed flash sterilization is an effective process for the sterilization of critical medical devices^{844, 845}. Flash sterilization is a modification of conventional steam sterilization (either gravity, prevacuum, or steam-flush pressure-pulse) in which the flashed item is placed in an open tray or is placed in a specially designed, covered, rigid container to allow for rapid penetration of steam. Historically, it is not recommended as a routine sterilization method because of the lack of timely biological indicators to monitor performance, absence of protective packaging following sterilization, possibility for contamination of processed items during transportation to the operating rooms, and the sterilization cycle parameters (i.e., time, temperature, pressure) are minimal. To address some of these concerns, many healthcare facilities have done the following: placed equipment for flash sterilization in close proximity to operating rooms to facilitate aseptic delivery to the point of use (usually the sterile field in an ongoing surgical procedure); extended the exposure time to ensure lethality comparable to sterilized wrapped items (e.g., 4 minutes at 132°C)^{846, 847}; used biological indicators that provide results in 1 hour for flash-sterilized items^{846, 847}; and used protective packaging that permits steam penetration^{812, 817-819, 845, 848}. Further, some rigid, reusable sterilization container systems have been designed and validated by the container manufacturer for use with flash cycles. When sterile items are open to air, they will eventually become contaminated. Thus, the longer a sterile item is exposed to air, the greater the number of microorganisms that will settle on it. Sterilization cycle parameters for flash sterilization are shown in Table 8.

A few adverse events have been associated with flash sterilization. When evaluating an increased incidence of neurosurgical infections, the investigators noted that surgical instruments were flash sterilized between cases and 2 of 3 craniotomy infections involved plate implants that were flash sterilized⁸⁴⁹. A report of two patients who received burns during surgery from instruments that had been flash sterilized reinforced the need to develop policies and educate staff to prevent the use of instruments hot enough to cause clinical burns⁸⁵⁰. Staff should use precautions to prevent burns with potentially hot instruments (e.g., transport tray using heat-protective gloves). Patient burns may be prevented by either air-cooling the instruments or immersion in sterile liquid (e.g., saline).

Uses. Flash sterilization is considered acceptable for processing cleaned patient-care items that

cannot be packaged, sterilized, and stored before use. It also is used when there is insufficient time to sterilize an item by the preferred package method. Flash sterilization should not be used for reasons of convenience, as an alternative to purchasing additional instrument sets, or to save time⁸¹⁷. Because of the potential for serious infections, flash sterilization is not recommended for implantable devices (i.e., devices placed into a surgically or naturally formed cavity of the human body); however, flash sterilization may be unavoidable for some devices (e.g., orthopedic screw, plates). If flash sterilization of an implantable device is unavoidable, recordkeeping (i.e., load identification, patient's name/hospital identifier, and biological indicator result) is essential for epidemiological tracking (e.g., of surgical site infection, tracing results of biological indicators to patients who received the item to document sterility), and for an assessment of the reliability of the sterilization process (e.g., evaluation of biological monitoring records and sterilization maintenance records noting preventive maintenance and repairs with dates).

Low-Temperature Sterilization Technologies

Ethylene oxide (ETO) has been widely used as a low-temperature sterilant since the 1950s. It has been the most commonly used process for sterilizing temperature- and moisture-sensitive medical devices and supplies in healthcare institutions in the United States. Two types of ETO sterilizers are available, mixed gas and 100% ETO. Until 1995, ethylene oxide sterilizers combined ETO with a chlorofluorocarbon (CFC) stabilizing agent, most commonly in a ratio of 12% ETO mixed with 88% CFC (referred to as 12/88 ETO).

For several reasons, healthcare personnel have been exploring the use of new low-temperature sterilization technologies^{825, 851}. First, CFCs were phased out in December 1995 under provisions of the Clean Air Act⁸⁵². CFCs were classified as a Class I substance under the Clean Air Act because of scientific evidence linking them to destruction of the earth's ozone layer. Second, some states (e.g., California, New York, Michigan) require the use of ETO abatement technology to reduce the amount of ETO being released into ambient air from 90 to 99.9% depending on the state. Third, OSHA regulates the acceptable vapor levels of ETO (i.e., 1 ppm averaged over 8 hours) due to concerns that ETO exposure represents an occupational hazard³¹⁸. These constraints have led to the development of alternative technologies for low-temperature sterilization in the healthcare setting.

Alternative technologies to ETO with chlorofluorocarbon that are currently available and cleared by the FDA for medical equipment include 100% ETO; ETO with a different stabilizing gas, such as carbon dioxide or hydrochlorofluorocarbons (HCFC); immersion in peracetic acid; hydrogen peroxide gas plasma; and ozone. Technologies under development for use in healthcare facilities, but not cleared by the FDA, include vaporized hydrogen peroxide, vapor phase peracetic acid, gaseous chlorine dioxide, ionizing radiation, or pulsed light^{400, 758, 853}. However, there is no guarantee that these new sterilization technologies will receive FDA clearance for use in healthcare facilities.

These new technologies should be compared against the characteristics of an ideal low-temperature (<60°C) sterilant (Table 9).⁸⁵¹ While it is apparent that all technologies will have limitations (Table 9), understanding the limitations imposed by restrictive device designs (e.g., long, narrow lumens) is critical for proper application of new sterilization technology⁸⁵⁴. For example, the development of increasingly small and complex endoscopes presents a difficult challenge for current sterilization processes. This occurs because microorganisms must be in direct contact with the sterilant for inactivation to occur. Several peer-reviewed scientific publications have data demonstrating concerns about the efficacy of several of the low-temperature sterilization processes (i.e., gas plasma, vaporized hydrogen peroxide, ETO, peracetic acid), particularly when the test organisms are challenged in the presence of serum and salt and a narrow lumen vehicle^{469, 721, 825, 855, 856}. Factors shown to affect the efficacy of sterilization are shown in Table 10.

Ethylene Oxide "Gas" Sterilization

Overview. ETO is a colorless gas that is flammable and explosive. The four essential

parameters (operational ranges) are: gas concentration (450 to 1200 mg/l); temperature (37 to 63°C); relative humidity (40 to 80%)(water molecules carry ETO to reactive sites); and exposure time (1 to 6 hours). These influence the effectiveness of ETO sterilization^{814, 857, 858}. Within certain limitations, an increase in gas concentration and temperature may shorten the time necessary for achieving sterilization.

The main disadvantages associated with ETO are the lengthy cycle time, the cost, and its potential hazards to patients and staff; the main advantage is that it can sterilize heat- or moisture-sensitive medical equipment without deleterious effects on the material used in the medical devices (Table 6). Acute exposure to ETO may result in irritation (e.g., to skin, eyes, gastrointestinal or respiratory tracts) and central nervous system depression⁸⁵⁹⁻⁸⁶². Chronic inhalation has been linked to the formation of cataracts, cognitive impairment, neurologic dysfunction, and disabling polyneuropathies^{860, 861, 863-866}. Occupational exposure in healthcare facilities has been linked to hematologic changes⁸⁶⁷ and an increased risk of spontaneous abortions and various cancers^{318, 868-870}. ETO should be considered a known human carcinogen⁸⁷¹.

The basic ETO sterilization cycle consists of five stages (i.e., preconditioning and humidification, gas introduction, exposure, evacuation, and air washes) and takes approximately 2 1/2 hrs excluding aeration time. Mechanical aeration for 8 to 12 hours at 50 to 60°C allows desorption of the toxic ETO residual contained in exposed absorbent materials. Most modern ETO sterilizers combine sterilization and aeration in the same chamber as a continuous process. These ETO models minimize potential ETO exposure during door opening and load transfer to the aerator. Ambient room aeration also will achieve desorption of the toxic ETO but requires 7 days at 20°C. There are no federal regulations for ETO sterilizer emission; however, many states have promulgated emission-control regulations⁸¹⁴.

The use of ETO evolved when few alternatives existed for sterilizing heat- and moisture-sensitive medical devices; however, favorable properties (Table 6) account for its continued widespread use⁸⁷². Two ETO gas mixtures are available to replace ETO-chlorofluorocarbon (CFC) mixtures for large capacity, tank-supplied sterilizers. The ETO-carbon dioxide (CO₂) mixture consists of 8.5% ETO and 91.5% CO₂. This mixture is less expensive than ETO-hydrochlorofluorocarbons (HCFC), but a disadvantage is the need for pressure vessels rated for steam sterilization, because higher pressures (28-psi gauge) are required. The other mixture, which is a drop-in CFC replacement, is ETO mixed with HCFC. HCFCs are approximately 50-fold less damaging to the earth's ozone layer than are CFCs. The EPA will begin regulation of HCFC in the year 2015 and will terminate production in the year 2030. Two companies provide ETO-HCFC mixtures as drop-in replacement for CFC-12; one mixture consists of 8.6% ETO and 91.4% HCFC, and the other mixture is composed of 10% ETO and 90% HCFC⁸⁷². An alternative to the pressurized mixed gas ETO systems is 100% ETO. The 100% ETO sterilizers using unit-dose cartridges eliminate the need for external tanks.

ETO is absorbed by many materials. For this reason, following sterilization the item must undergo aeration to remove residual ETO. Guidelines have been promulgated regarding allowable ETO limits for devices that depend on how the device is used, how often, and how long in order to pose a minimal risk to patients in normal product use⁸¹⁴.

ETO toxicity has been established in a variety of animals. Exposure to ETO can cause eye pain, sore throat, difficulty breathing and blurred vision. Exposure can also cause dizziness, nausea, headache, convulsions, blisters and vomiting and coughing⁸⁷³. In a variety of *in vitro* and animal studies, ETO has been demonstrated to be carcinogenic. ETO has been linked to spontaneous abortion, genetic damage, nerve damage, peripheral paralysis, muscle weakness, and impaired thinking and memory⁸⁷³. Occupational exposure in healthcare facilities has been linked to an increased risk of spontaneous abortions and various cancers³¹⁸. Injuries (e.g., tissue burns) to patients have been associated with ETO residues in implants used in surgical procedures⁸⁷⁴. Residual ETO in capillary flow dialysis membranes has been shown to be neurotoxic *in vitro*⁸⁷⁵. OSHA has established a PEL of 1 ppm airborne ETO in the workplace, expressed as a TWA for an 8-hour work shift in a 40-hour work week. The "action level" for ETO is 0.5 ppm, expressed as an 8-hour TWA, and the short-term excursion limit is 5 ppm, expressed as

a 15-minute TWA⁸¹⁴. For details of the requirements in OSHA's ETO standard for occupational exposures, see Title 29 of the Code of Federal Regulations (CFR) Part 1910.1047⁸⁷³. Several personnel monitoring methods (e.g., charcoal tubes and passive sampling devices) are in use⁸¹⁴. OSHA has established a PEL of 5 ppm for ethylene chlorohydrin (a toxic by-product of ETO) in the workplace⁸⁷⁶. Additional information regarding use of ETO in health care facilities is available from NIOSH.

Mode of Action. The microbicidal activity of ETO is considered to be the result of alkylation of protein, DNA, and RNA. Alkylation, or the replacement of a hydrogen atom with an alkyl group, within cells prevents normal cellular metabolism and replication⁸⁷⁷.

Microbicidal Activity. The excellent microbicidal activity of ETO has been demonstrated in several studies^{469, 721, 722, 856, 878, 879} and summarized in published reports⁸⁷⁷. ETO inactivates all microorganisms although bacterial spores (especially *B. atrophaeus*) are more resistant than other microorganisms. For this reason *B. atrophaeus* is the recommended biological indicator.

Like all sterilization processes, the effectiveness of ETO sterilization can be altered by lumen length, lumen diameter, inorganic salts, and organic materials^{469, 721, 722, 855, 856, 879}. For example, although ETO is not used commonly for reprocessing endoscopes²⁸, several studies have shown failure of ETO in inactivating contaminating spores in endoscope channels⁸⁵⁵ or lumen test units^{469, 721, 879} and residual ETO levels averaging 66.2 ppm even after the standard degassing time⁴⁵⁶. Failure of ETO also has been observed when dental handpieces were contaminated with *Streptococcus mutans* and exposed to ETO⁸⁸⁰. It is recommended that dental handpieces be steam sterilized.

Uses. ETO is used in healthcare facilities to sterilize critical items (and sometimes semicritical items) that are moisture or heat sensitive and cannot be sterilized by steam sterilization.

Hydrogen Peroxide Gas Plasma

Overview. New sterilization technology based on plasma was patented in 1987 and marketed in the United States in 1993. Gas plasmas have been referred to as the fourth state of matter (i.e., liquids, solids, gases, and gas plasmas). Gas plasmas are generated in an enclosed chamber under deep vacuum using radio frequency or microwave energy to excite the gas molecules and produce charged particles, many of which are in the form of free radicals. A free radical is an atom with an unpaired electron and is a highly reactive species. The proposed mechanism of action of this device is the production of free radicals within a plasma field that are capable of interacting with essential cell components (e.g., enzymes, nucleic acids) and thereby disrupt the metabolism of microorganisms. The type of seed gas used and the depth of the vacuum are two important variables that can determine the effectiveness of this process.

In the late 1980s the first hydrogen peroxide gas plasma system for sterilization of medical and surgical devices was field-tested. According to the manufacturer, the sterilization chamber is evacuated and hydrogen peroxide solution is injected from a cassette and is vaporized in the sterilization chamber to a concentration of 6 mg/l. The hydrogen peroxide vapor diffuses through the chamber (50 minutes), exposes all surfaces of the load to the sterilant, and initiates the inactivation of microorganisms. An electrical field created by a radio frequency is applied to the chamber to create a gas plasma. Microbicidal free radicals (e.g., hydroxyl and hydroperoxyl) are generated in the plasma. The excess gas is removed and in the final stage (i.e., vent) of the process the sterilization chamber is returned to atmospheric pressure by introduction of high-efficiency filtered air. The by-products of the cycle (e.g., water vapor, oxygen) are nontoxic and eliminate the need for aeration. Thus, the sterilized materials can be handled safely, either for immediate use or storage. The process operates in the range of 37-44°C and has a cycle time of 75 minutes. If any moisture is present on the objects the vacuum will not be achieved and the cycle aborts^{856, 881-883}.

A newer version of the unit improves sterilizer efficacy by using two cycles with a hydrogen

peroxide diffusion stage and a plasma stage per sterilization cycle. This revision, which is achieved by a software modification, reduces total processing time from 73 to 52 minutes. The manufacturer believes that the enhanced activity obtained with this system is due in part to the pressure changes that occur during the injection and diffusion phases of the process and to the fact that the process consists of two equal and consecutive half cycles, each with a separate injection of hydrogen peroxide.^{856, 884, 885} This system and a smaller version^{400, 882} have received FDA 510[k] clearance with limited application for sterilization of medical devices (Table 6). The biological indicator used with this system is *Bacillus atrophaeus* spores⁸⁵¹. The newest version of the unit, which employs a new vaporization system that removes most of the water from the hydrogen peroxide, has a cycle time from 28-38 minutes (see manufacturer's literature for device dimension restrictions).

Penetration of hydrogen peroxide vapor into long or narrow lumens has been addressed outside the United States by the use of a diffusion enhancer. This is a small, breakable glass ampoule of concentrated hydrogen peroxide (50%) with an elastic connector that is inserted into the device lumen and crushed immediately before sterilization^{470, 885}. The diffusion enhancer has been shown to sterilize bronchoscopes contaminated with *Mycobacteria tuberculosis*⁸⁸⁶. At the present time, the diffusion enhancer is not FDA cleared.

Another gas plasma system, which differs from the above in several important ways, including the use of peracetic acid-acetic acid-hydrogen peroxide vapor, was removed from the marketplace because of reports of corneal destruction to patients when ophthalmic surgery instruments had been processed in the sterilizer^{887, 888}. In this investigation, exposure of potentially wet ophthalmologic surgical instruments with small bores and brass components to the plasma gas led to degradation of the brass to copper and zinc^{888, 889}. The experimenters showed that when rabbit eyes were exposed to the rinsates of the gas plasma-sterilized instruments, corneal decompensation was documented. This toxicity is highly unlikely with the hydrogen peroxide gas plasma process since a toxic, soluble form of copper would not form (LA Feldman, written communication, April 1998).

Mode of Action. This process inactivates microorganisms primarily by the combined use of hydrogen peroxide gas and the generation of free radicals (hydroxyl and hydroperoxyl free radicals) during the plasma phase of the cycle.

Microbicidal Activity. This process has the ability to inactivate a broad range of microorganisms, including resistant bacterial spores. Studies have been conducted against vegetative bacteria (including mycobacteria), yeasts, fungi, viruses, and bacterial spores^{469, 721, 856, 881-883, 890-893}. Like all sterilization processes, the effectiveness can be altered by lumen length, lumen diameter, inorganic salts, and organic materials^{469, 721, 855, 856, 890, 891, 893}.

Uses. Materials and devices that cannot tolerate high temperatures and humidity, such as some plastics, electrical devices, and corrosion-susceptible metal alloys, can be sterilized by hydrogen peroxide gas plasma. This method has been compatible with most (>95%) medical devices and materials tested^{884, 894, 895}.

Peracetic Acid Sterilization

Overview. Peracetic acid is a highly biocidal oxidizer that maintains its efficacy in the presence of organic soil. Peracetic acid removes surface contaminants (primarily protein) on endoscopic tubing^{711, 717}. An automated machine using peracetic acid to sterilize medical, surgical, and dental instruments chemically (e.g., endoscopes, arthroscopes) was introduced in 1988. This microprocessor-controlled, low-temperature sterilization method is commonly used in the United States¹⁰⁷. The sterilant, 35% peracetic acid, and an anticorrosive agent are supplied in a single-dose container. The container is punctured at the time of use, immediately prior to closing the lid and initiating the cycle. The concentrated peracetic acid is diluted to 0.2% with filtered water (0.2 μm) at a temperature of approximately 50°C. The diluted peracetic acid is circulated within the chamber of the machine and

pumped through the channels of the endoscope for 12 minutes, decontaminating exterior surfaces, lumens, and accessories. Interchangeable trays are available to permit the processing of up to three rigid endoscopes or one flexible endoscope. Connectors are available for most types of flexible endoscopes for the irrigation of all channels by directed flow. Rigid endoscopes are placed within a lidded container, and the sterilant fills the lumens either by immersion in the circulating sterilant or by use of channel connectors to direct flow into the lumen(s) (see below for the importance of channel connectors). The peracetic acid is discarded via the sewer and the instrument rinsed four times with filtered water. Concern has been raised that filtered water may be inadequate to maintain sterility⁸⁹⁶. Limited data have shown that low-level bacterial contamination may follow the use of filtered water in an AER but no data has been published on AERs using the peracetic acid system¹⁶¹. Clean filtered air is passed through the chamber of the machine and endoscope channels to remove excess water⁷¹⁹. As with any sterilization process, the system can only sterilize surfaces that can be contacted by the sterilant. For example, bronchoscopy-related infections occurred when bronchoscopes were processed using the wrong connector^{155, 725}. Investigation of these incidents revealed that bronchoscopes were inadequately reprocessed when inappropriate channel connectors were used and when there were inconsistencies between the reprocessing instructions provided by the manufacturer of the bronchoscope and the manufacturer of the automatic endoscope reprocessor¹⁵⁵. The importance of channel connectors to achieve sterilization was also shown for rigid lumen devices^{137, 856}.

The manufacturers suggest the use of biological monitors (*G. stearothermophilus* spore strips) both at the time of installation and routinely to ensure effectiveness of the process. The manufacturer's clip must be used to hold the strip in the designated spot in the machine as a broader clamp will not allow the sterilant to reach the spores trapped under it⁸⁹⁷. One investigator reported a 3% failure rate when the appropriate clips were used to hold the spore strip within the machine⁷¹⁸. The use of biological monitors designed to monitor either steam sterilization or ETO for a liquid chemical sterilizer has been questioned for several reasons including spore wash-off from the filter paper strips which may cause less valid monitoring⁸⁹⁸⁻⁹⁰¹. The processor is equipped with a conductivity probe that will automatically abort the cycle if the buffer system is not detected in a fresh container of the peracetic acid solution. A chemical monitoring strip that detects that the active ingredient is >1500 ppm is available for routine use as an additional process control.

Mode of Action. Only limited information is available regarding the mechanism of action of peracetic acid, but it is thought to function as other oxidizing agents, i.e., it denatures proteins, disrupts cell wall permeability, and oxidizes sulfhydryl and sulfur bonds in proteins, enzymes, and other metabolites^{654, 726}.

Microbicidal Activity. Peracetic acid will inactivate gram-positive and gram-negative bacteria, fungi, and yeasts in <5 minutes at <100 ppm. In the presence of organic matter, 200-500 ppm is required. For viruses, the dosage range is wide (12-2250 ppm), with poliovirus inactivated in yeast extract in 15 minutes with 1500 to 2250 ppm. Bacterial spores in suspension are inactivated in 15 seconds to 30 minutes with 500 to 10,000 ppm (0.05 to 1%)⁶⁵⁴.

Simulated-use trials have demonstrated microbicidal activity^{111, 718-722} and three clinical trials have demonstrated both microbial killing and no clinical failures leading to infection^{90, 723, 724}. Alfa and co-workers, who compared the peracetic acid system with ETO, demonstrated the high efficacy of the system. Only the peracetic acid system was able to completely kill 6-log₁₀ of *Mycobacterium chelonae*, *Enterococcus faecalis*, and *B. atrophaeus* spores with both an organic and inorganic challenge⁷²². Like other sterilization processes, the efficacy of the process can be diminished by soil challenges⁹⁰² and test conditions⁸⁵⁶.

Uses. This automated machine is used to chemically sterilize medical (e.g., GI endoscopes) and surgical (e.g., flexible endoscopes) instruments in the United States. Lumened endoscopes must be connected to an appropriate channel connector to ensure that the sterilant has direct contact with the contaminated lumen.^{137, 856, 903} Olympus America has not listed this system as a compatible product for

use in reprocessing Olympus bronchoscopes and gastrointestinal endoscopes (Olympus America, January 30, 2002, written communication).

Microbicidal Activity of Low-Temperature Sterilization Technologies

Sterilization processes used in the United States must be cleared by FDA, and they require that sterilizer microbicidal performance be tested under simulated-use conditions⁹⁰⁴. FDA requires that the test article be inoculated with 10^6 colony-forming units of the most resistant test organism and prepared with organic and inorganic test loads as would occur after actual use. FDA requires manufacturers to use organic soil (e.g., 5% fetal calf serum), dried onto the device with the inoculum, to represent soil remaining on the device following marginal cleaning. However, 5% fetal calf serum as a measure of marginal cleaning has not been validated by measurements of protein load on devices following use and the level of protein removal by various cleaning methods. The inocula must be placed in various locations of the test articles, including those least favorable to penetration and contact with the sterilant (e.g., lumens). Cleaning before sterilization is not allowed in the demonstration of sterilization efficacy⁹⁰⁴. Several studies have evaluated the relative microbicidal efficacy of these low-temperature sterilization technologies (Table 11). These studies have either tested the activity of a sterilization process against specific microorganisms^{892, 905, 906}, evaluated the microbicidal activity of a singular technology^{711, 719, 724, 855, 879, 882-884, 890, 891, 907} or evaluated the comparative effectiveness of several sterilization technologies^{271, 426, 469, 721, 722, 856, 908, 909}. Several test methodologies use stainless steel or porcelain carriers that are inoculated with a test organism. Commonly used test organisms include vegetative bacteria, mycobacteria, and spores of *Bacillus* species. The available data demonstrate that low-temperature sterilization technologies are able to provide a 6-log₁₀ reduction of microbes when inoculated onto carriers in the absence of salt and serum. However, tests can be constructed such that all of the available sterilization technologies are unable to reliably achieve complete inactivation of a microbial load.^{425, 426, 469, 721, 856, 909} For example, almost all of the sterilization processes will fail to reliably inactivate the microbial load in the presence of salt and serum^{469, 721, 909}.

The effect of salts and serums on the sterilization process were studied initially in the 1950s and 1960s^{424, 910}. These studies showed that a high concentration of crystalline-type materials and a low protein content provided greater protection to spores than did serum with a high protein content⁴²⁶. A study by Doyle and Ernst demonstrated resistance of spores by crystalline material applied not only to low-temperature sterilization technology but also to steam and dry heat⁴²⁵. These studies showed that occlusion of *Bacillus atrophaeus* spores in calcium carbonate crystals dramatically increased the time required for inactivation as follows: 10 seconds to 150 minutes for steam (121°C), 3.5 hours to 50 hours for dry heat (121°C), 30 seconds to >2 weeks for ETO (54°C). Investigators have corroborated and extended these findings^{469, 470, 721, 855, 908, 909}. While soils containing both organic and inorganic materials impair microbial killing, soils that contain a high inorganic salt-to-protein ratio favor crystal formation and impair sterilization by occlusion of organisms^{425, 426, 881}.

Alfa and colleagues demonstrated a 6-log₁₀ reduction of the microbial inoculum of porcelain penicylinders using a variety of vegetative and spore-forming organisms (Table 11)⁴⁶⁹. However, if the bacterial inoculum was in tissue-culture medium supplemented with 10% serum, only the ETO 12/88 and ETO-HCFC sterilization mixtures could sterilize 95% to 97% of the penicylinder carriers. The plasma and 100% ETO sterilizer demonstrated significantly reduced activity (Table 11). For all sterilizers evaluated using penicylinder carriers (i.e., ETO 12/88, 100% ETO, hydrogen peroxide gas plasma), there was a 3- to 6-log₁₀ reduction of inoculated bacteria even in the presence of serum and salt. For each sterilizer evaluated, the ability to inactivate microorganisms in the presence of salt and serum was reduced even further when the inoculum was placed in a narrow-lumen test object (3 mm diameter by 125 cm long). Although there was a 2- to 4-log₁₀ reduction in microbial kill, less than 50% of the lumen test objects were sterile when processed using any of the sterilization methods evaluated except the peracetic acid immersion system (Table 11)⁷²¹. Complete killing (or removal) of 6-log₁₀ of *Enterococcus faecalis*, *Mycobacterium chelonae*, and *Bacillus atrophaeus* spores in the presence of salt and serum and lumen test objects was observed only for the peracetic acid immersion system.

With respect to the results by Alfa and coworkers⁴⁶⁹, Jacobs showed that the use of the tissue culture media created a technique-induced sterilization failure⁴²⁶. Jacobs et al. showed that microorganisms mixed with tissue culture media, used as a surrogate body fluid, formed physical crystals that protected the microorganisms used as a challenge. If the carriers were exposed for 60 sec to nonflowing water, the salts dissolved and the protective effect disappeared. Since any device would be exposed to water for a short period of time during the washing procedure, these protective effects would have little clinical relevance⁴²⁶.

Narrow lumens provide a challenge to some low-temperature sterilization processes. For example, Rutala and colleagues showed that, as lumen size decreased, increased failures occurred with some low-temperature sterilization technologies. However, some low-temperature processes such as ETO-HCFC and the hydrogen peroxide gas plasma process remained effective even when challenged by a lumen as small as 1 mm in the absence of salt and serum⁸⁵⁶.

The importance of allowing the sterilant to come into contact with the inoculated carrier is demonstrated by comparing the results of two investigators who studied the peracetic acid immersion system. Alfa and coworkers demonstrated excellent activity of the peracetic acid immersion system against three test organisms using a narrow-lumen device. In these experiments, the lumen test object was connected to channel irrigators, which ensured that the sterilant had direct contact with the contaminated carriers⁷²². This effectiveness was achieved through a combination of organism wash-off and peracetic acid sterilant killing the test organisms⁷²². The data reported by Rutala et al. demonstrated failure of the peracetic acid immersion system to eliminate *Geobacillus stearothermophilus* spores from a carrier placed in a lumen test object. In these experiments, the lumen test unit was not connected to channel irrigators. The authors attributed the failure of the peracetic acid immersion system to eliminate the high levels of spores from the center of the test unit to the inability of the peracetic acid to diffuse into the center of 40-cm long, 3-mm diameter tubes. This may be caused by an air lock or air bubbles formed in the lumen, impeding the flow of the sterilant through the long and narrow lumen and limiting complete access to the *Bacillus* spores^{137, 856}. Experiments using a channel connector specifically designed for 1-, 2-, and 3-mm lumen test units with the peracetic acid immersion system were completely effective in eliminating an inoculum of 10^6 *Geobacillus stearothermophilus* spores⁷. The restricted diffusion environment that exists in the test conditions would not exist with flexible scopes processed in the peracetic acid immersion system, because the scopes are connected to channel irrigators to ensure that the sterilant has direct contact with contaminated surfaces. Alfa and associates attributed the efficacy of the peracetic acid immersion system to the ability of the liquid chemical process to dissolve salts and remove protein and bacteria due to the flushing action of the fluid⁷²².

Bioburden of Surgical Devices

In general, used medical devices are contaminated with a relatively low bioburden of organisms^{179, 911, 912}. Nystrom evaluated medical instruments used in general surgical, gynecological, orthopedic, and ear-nose-throat operations and found that 62% of the instruments were contaminated with $<10^1$ organisms after use, 82% with $<10^2$, and 91% with $<10^3$. After being washed in an instrument washer, more than 98% of the instruments had $<10^1$ organisms, and none $>10^2$ organisms⁹¹¹. Other investigators have published similar findings^{179, 912}. For example, after a standard cleaning procedure, 72% of 50 surgical instruments contained $<10^1$ organisms, 86% $<10^2$, and only 6% had $>3 \times 10^{2912}$. In another study of rigid-lumen medical devices, the bioburden on both the inner and outer surface of the lumen ranged from 10^1 to 10^4 organisms per device. After cleaning, 83% of the devices had a bioburden $\leq 10^2$ organisms¹⁷⁹. In all of these studies, the contaminating microflora consisted mainly of vegetative bacteria, usually of low pathogenicity (e.g., coagulase-negative *Staphylococcus*)^{179, 911, 912}.

An evaluation of the microbial load on used critical medical devices such as spinal anesthesia needles and angiographic catheters and sheaths demonstrated that mesophilic microorganisms were detected at levels of 10^1 to 10^2 in only two of five needles. The bioburden on used angiographic

catheters and sheath introducers exceeded 10^3 CFUs on 14% (3 of 21) and 21% (6 of 28), respectively⁹⁰⁷.

Effect of Cleaning on Sterilization Efficacy

The effect of salt and serum on the efficacy of low-temperature sterilization technologies has raised concern regarding the margin of safety of these technologies. Experiments have shown that salts have the greatest impact on protecting microorganisms from killing^{426, 469}. However, other studies have suggested that these concerns may not be clinically relevant. One study evaluated the relative rate of removal of inorganic salts, organic soil, and microorganisms from medical devices to better understand the dynamics of the cleaning process⁴²⁶. These tests were conducted by inoculating Alfa soil (tissue-culture media and 10% fetal bovine serum)⁴⁶⁹ containing 10^6 *G. stearothermophilus* spores onto the surface of a stainless-steel scalpel blade. After drying for 30 minutes at 35°C followed by 30 minutes at room temperature, the samples were placed in water at room temperature. The blades were removed at specified times, and the concentration of total protein and chloride ion was measured. The results showed that soaking in deionized water for 60 seconds resulted in a >95% release rate of chloride ion from NaCl solution in 20 seconds, Alfa soil in 30 seconds, and fetal bovine serum in 120 seconds. Thus, contact with water for short periods, even in the presence of protein, rapidly leads to dissolution of salt crystals and complete inactivation of spores by a low-temperature sterilization process (Table 10). Based on these experimental data, cleaning procedures would eliminate the detrimental effect of high salt content on a low-temperature sterilization process.

These articles^{426, 469, 721} assessing low-temperature sterilization technology reinforce the importance of meticulous cleaning before sterilization. These data support the critical need for healthcare facilities to develop rigid protocols for cleaning contaminated objects before sterilization⁴⁷². Sterilization of instruments and medical devices is compromised if the process is not preceded by meticulous cleaning.

The cleaning of any narrow-lumen medical device used in patient care presents a major challenge to reprocessing areas. While attention has been focused on flexible endoscopes, cleaning issues related to other narrow-lumen medical devices such as sphinctertomes have been investigated⁹¹³. This study compared manual cleaning with that of automated cleaning with a narrow-lumen cleaner and found that only retro-flushing with the narrow lumen cleaner provided adequate cleaning of the three channels. If reprocessing was delayed for more than 24 hours, retro-flush cleaning was no longer effective and ETO sterilization failure was detected when devices were held for 7 days⁹¹³. In another study involving simulated-use cleaning of laparoscopic devices, Alfa found that minimally the use of retro-flushing should be used during cleaning of non-ported laparoscopic devices⁹¹⁴.

Other Sterilization Methods

Ionizing Radiation. Sterilization by ionizing radiation, primarily by cobalt 60 gamma rays or electron accelerators, is a low-temperature sterilization method that has been used for a number of medical products (e.g., tissue for transplantation, pharmaceuticals, medical devices). There are no FDA-cleared ionizing radiation sterilization processes for use in healthcare facilities. Because of high sterilization costs, this method is an unfavorable alternative to ETO and plasma sterilization in healthcare facilities but is suitable for large-scale sterilization. Some deleterious effects on patient-care equipment associated with gamma radiation include induced oxidation in polyethylene⁹¹⁵ and delamination and cracking in polyethylene knee bearings⁹¹⁶. Several reviews^{917, 918} dealing with the sources, effects, and application of ionizing radiation may be referred to for more detail.

Dry-Heat Sterilizers. This method should be used only for materials that might be damaged by moist heat or that are impenetrable to moist heat (e.g., powders, petroleum products, sharp instruments). The advantages for dry heat include the following: it is nontoxic and does not harm the environment; a dry heat cabinet is easy to install and has relatively low operating costs; it penetrates materials; and it is noncorrosive for metal and sharp instruments. The disadvantages for dry heat are the slow rate of heat penetration and microbial killing makes this a time-consuming method. In addition, the high temperatures

are not suitable for most materials⁹¹⁹. The most common time-temperature relationships for sterilization with hot air sterilizers are 170°C (340°F) for 60 minutes, 160°C (320°F) for 120 minutes, and 150°C (300°F) for 150 minutes. *B. atrophaeus* spores should be used to monitor the sterilization process for dry heat because they are more resistant to dry heat than are *G. stearothermophilus* spores. The primary lethal process is considered to be oxidation of cell constituents.

There are two types of dry-heat sterilizers: the static-air type and the forced-air type. The static-air type is referred to as the oven-type sterilizer as heating coils in the bottom of the unit cause the hot air to rise inside the chamber via gravity convection. This type of dry-heat sterilizer is much slower in heating, requires longer time to reach sterilizing temperature, and is less uniform in temperature control throughout the chamber than is the forced-air type. The forced-air or mechanical convection sterilizer is equipped with a motor-driven blower that circulates heated air throughout the chamber at a high velocity, permitting a more rapid transfer of energy from the air to the instruments⁹²⁰.

Liquid Chemicals. Several FDA-cleared liquid chemical sterilants include indications for sterilization of medical devices (Tables 4 and 5)⁶⁹. The indicated contact times range from 3 hours to 12 hours. However, except for a few of the products, the contact time is based only on the conditions to pass the AOAC Sporicidal Test as a sterilant and not on simulated use testing with devices. These solutions are commonly used as high-level disinfectants when a shorter processing time is required. Generally, chemical liquid sterilants cannot be monitored using a biological indicator to verify sterility^{899, 900}.

The survival kinetics for thermal sterilization methods, such as steam and dry heat, have been studied and characterized extensively, whereas the kinetics for sterilization with liquid sterilants are less well understood⁹²¹. The information that is available in the literature suggests that sterilization processes based on liquid chemical sterilants, in general, may not convey the same sterility assurance level as sterilization achieved using thermal or physical methods⁸²³. The data indicate that the survival curves for liquid chemical sterilants may not exhibit log-linear kinetics and the shape of the survivor curve may vary depending of the formulation, chemical nature and stability of the liquid chemical sterilant. In addition, the design of the AOAC Sporicidal Test does not provide quantification of the microbial challenge. Therefore, sterilization with a liquid chemical sterilant may not convey the same sterility assurance as other sterilization methods.

One of the differences between thermal and liquid chemical processes for sterilization of devices is the accessibility of microorganisms to the sterilant. Heat can penetrate barriers, such as biofilm, tissue, and blood, to attain organism kill, whereas liquids cannot adequately penetrate these barriers. In addition, the viscosity of some liquid chemical sterilants impedes their access to organisms in the narrow lumens and mated surfaces of devices⁹²². Another limitation to sterilization of devices with liquid chemical germicides is the post-processing environment of the device. Devices cannot be wrapped or adequately contained during processing in a liquid chemical sterilant to maintain sterility following processing and during storage. Furthermore, devices may require rinsing following exposure to the liquid chemical sterilant with water that typically is not sterile. Therefore, due to the inherent limitations of using liquid chemical sterilants, their use should be restricted to reprocessing critical devices that are heat-sensitive and incompatible with other sterilization methods.

Several published studies compare the sporicidal effect of liquid chemical germicides against spores of *Bacillus* and *Clostridium*^{78, 659, 660, 715}.

Performic Acid. Performic acid is a fast-acting sporicide that was incorporated into an automated endoscope reprocessing system⁴⁰⁰. Systems using performic acid are not currently FDA cleared.

Filtration. Although filtration is not a lethality-based process and is not an FDA-cleared sterilization method, this technology is used to remove bacteria from thermolabile pharmaceutical fluids

that cannot be purified by any other means. In order to remove bacteria, the membrane pore size (e.g., 0.22 μm) must be smaller than the bacteria and uniform throughout⁹²³. Some investigators have appropriately questioned whether the removal of microorganisms by filtration really is a sterilization method because of slight bacterial passage through filters, viral passage through filters, and transference of the sterile filtrate into the final container under aseptic conditions entail a risk of contamination⁹²⁴.

Microwave. Microwaves are used in medicine for disinfection of soft contact lenses, dental instruments, dentures, milk, and urinary catheters for intermittent self-catheterization⁹²⁵⁻⁹³¹. However, microwaves must only be used with products that are compatible (e.g., do not melt)⁹³¹. Microwaves are radio-frequency waves, which are usually used at a frequency of 2450 MHz. The microwaves produce friction of water molecules in an alternating electrical field. The intermolecular friction derived from the vibrations generates heat and some authors believe that the effect of microwaves depends on the heat produced while others postulate a nonthermal lethal effect⁹³²⁻⁹³⁴. The initial reports showed microwaves to be an effective microbicide. The microwaves produced by a "home-type" microwave oven (2.45 GHz) completely inactivate bacterial cultures, mycobacteria, viruses, and *G. stearothermophilus* spores within 60 seconds to 5 minutes depending on the challenge organism^{933, 935-937}. Another study confirmed these results but also found that higher power microwaves in the presence of water may be needed for sterilization⁹³². Complete destruction of *Mycobacterium bovis* was obtained with 4 minutes of microwave exposure (600W, 2450 MHz)⁹³⁷. The effectiveness of microwave ovens for different sterilization and disinfection purposes should be tested and demonstrated as test conditions affect the results (e.g., presence of water, microwave power). Sterilization of metal instruments can be accomplished but requires certain precautions.⁹²⁶ Of concern is that home-type microwave ovens may not have even distribution of microwave energy over the entire dry device (there may be hot and cold spots on solid medical devices); hence there may be areas that are not sterilized or disinfected. The use of microwave ovens to disinfect intermittent-use catheters also has been suggested. Researchers found that test bacteria (e.g., *E. coli*, *Klebsiella pneumoniae*, *Candida albicans*) were eliminated from red rubber catheters within 5 minutes⁹³¹. Microwaves used for sterilization of medical devices have not been FDA cleared.

Glass Bead "Sterilizer". Glass bead "sterilization" uses small glass beads (1.2-1.5 mm diameter) and high temperature (217 °C -232°C) for brief exposure times (e.g., 45 seconds) to inactivate microorganisms. These devices have been used for several years in the dental profession⁹³⁸⁻⁹⁴⁰. FDA believes there is a risk of infection with this device because of potential failure to sterilize dental instruments and their use should be discontinued until the device has received FDA clearance.

Vaporized Hydrogen Peroxide (VHP®). Hydrogen peroxide solutions have been used as chemical sterilants for many years. However, the VHP® was not developed for the sterilization of medical equipment until the mid-1980s. One method for delivering VHP to the reaction site uses a deep vacuum to pull liquid hydrogen peroxide (30-35% concentration) from a disposable cartridge through a heated vaporizer and then, following vaporization, into the sterilization chamber. A second approach to VHP delivery is the flow-through approach in which the VHP is carried into the sterilization chamber by a carrier gas such as air using either a slight negative pressure (vacuum) or slight positive pressure. Applications of this technology include vacuum systems for industrial sterilization of medical devices and atmospheric systems for decontaminating for large and small areas⁸⁵³. VHP offers several appealing features that include rapid cycle time (e.g., 30-45 minutes); low temperature; environmentally safe by-products (H_2O , oxygen [O_2]); good material compatibility; and ease of operation, installation and monitoring. VHP has limitations including that cellulose cannot be processed; nylon becomes brittle; and VHP penetration capabilities are less than those of ETO. VHP has not been cleared by FDA for sterilization of medical devices in healthcare facilities.

The feasibility of utilizing vapor-phase hydrogen peroxide as a surface decontaminant and sterilizer was evaluated in a centrifuge decontamination application. In this study, vapor-phase hydrogen peroxide was shown to possess significant sporicidal activity⁹⁴¹. In preliminary studies, hydrogen

peroxide vapor decontamination has been found to be a highly effective method of eradicating MRSA, *Serratia marcescens*, *Clostridium botulinum* spores and *Clostridium difficile* from rooms, furniture, surfaces and/or equipment; however, further investigation of this method to demonstrate both safety and effectiveness in reducing infection rates are required⁹⁴²⁻⁹⁴⁵.

Ozone. Ozone has been used for years as a drinking water disinfectant. Ozone is produced when O₂ is energized and split into two monatomic (O₁) molecules. The monatomic oxygen molecules then collide with O₂ molecules to form ozone, which is O₃. Thus, ozone consists of O₂ with a loosely bonded third oxygen atom that is readily available to attach to, and oxidize, other molecules. This additional oxygen atom makes ozone a powerful oxidant that destroys microorganisms but is highly unstable (i.e., half-life of 22 minutes at room temperature).

A new sterilization process, which uses ozone as the sterilant, was cleared by FDA in August 2003 for processing reusable medical devices. The sterilizer creates its own sterilant internally from USP grade oxygen, steam-quality water and electricity; the sterilant is converted back to oxygen and water vapor at the end of the cycle by a passing through a catalyst before being exhausted into the room. The duration of the sterilization cycle is about 4 h and 15 m, and it occurs at 30-35°C. Microbial efficacy has been demonstrated by achieving a SAL of 10⁻⁶ with a variety of microorganisms to include the most resistant microorganism, *Geobacillus stearothermophilus*.

The ozone process is compatible with a wide range of commonly used materials including stainless steel, titanium, anodized aluminum, ceramic, glass, silica, PVC, Teflon, silicone, polypropylene, polyethylene and acrylic. In addition, rigid lumen devices of the following diameter and length can be processed: internal diameter (ID): > 2 mm, length ≤ 25 cm; ID > 3 mm, length ≤ 47 cm; and ID > 4 mm, length ≤ 60 cm.

The process should be safe for use by the operator because there is no handling of the sterilant, no toxic emissions, no residue to aerate, and low operating temperature means there is no danger of an accidental burn. The cycle is monitored using a self-contained biological indicator and a chemical indicator. The sterilization chamber is small, about 4 ft³ (Written communication, S Dufresne, July 2004).

A gaseous ozone generator was investigated for decontamination of rooms used to house patients colonized with MRSA. The results demonstrated that the device tested would be inadequate for the decontamination of a hospital room⁹⁴⁶.

Formaldehyde Steam. Low-temperature steam with formaldehyde is used as a low-temperature sterilization method in many countries, particularly in Scandinavia, Germany, and the United Kingdom. The process involves the use of formalin, which is vaporized into a formaldehyde gas that is admitted into the sterilization chamber. A formaldehyde concentration of 8-16 mg/l is generated at an operating temperature of 70-75°C. The sterilization cycle consists of a series of stages that include an initial vacuum to remove air from the chamber and load, followed by steam admission to the chamber with the vacuum pump running to purge the chamber of air and to heat the load, followed by a series of pulses of formaldehyde gas, followed by steam. Formaldehyde is removed from the sterilizer and load by repeated alternate evacuations and flushing with steam and air. This system has some advantages, e.g., the cycle time for formaldehyde gas is faster than that for ETO and the cost per cycle is relatively low. However, ETO is more penetrating and operates at lower temperatures than do steam/formaldehyde sterilizers. Low-temperature steam formaldehyde sterilization has been found effective against vegetative bacteria, mycobacteria, *B. atrophaeus* and *G. stearothermophilus* spores and *Candida albicans*⁹⁴⁷⁻⁹⁴⁹.

Formaldehyde vapor cabinets also may be used in healthcare facilities to sterilize heat-sensitive medical equipment⁹⁵⁰. Commonly, there is no circulation of formaldehyde and no temperature and humidity controls. The release of gas from paraformaldehyde tablets (placed on the lower tray) is slow and produces a low partial pressure of gas. The microbicidal quality of this procedure is unknown⁹⁵¹.

Reliable sterilization using formaldehyde is achieved when performed with a high concentration of gas, at a temperature between 60° and 80°C and with a relative humidity of 75 to 100%.

Studies indicate that formaldehyde is a mutagen and a potential human carcinogen, and OSHA regulates formaldehyde. The permissible exposure limit for formaldehyde in work areas is 0.75 ppm measured as a 8-hour TWA. The OSHA standard includes a 2 ppm STEL (i.e., maximum exposure allowed during a 15-minute period). As with the ETO standard, the formaldehyde standard requires that the employer conduct initial monitoring to identify employees who are exposed to formaldehyde at or above the action level or STEL. If this exposure level is maintained, employers may discontinue exposure monitoring until there is a change that could affect exposure levels or an employee reports formaldehyde-related signs and symptoms^{269, 578}. The formaldehyde steam sterilization system has not been FDA cleared for use in healthcare facilities.

Gaseous chlorine dioxide. A gaseous chlorine dioxide system for sterilization of healthcare products was developed in the late 1980s^{853, 952, 953}. Chlorine dioxide is not mutagenic or carcinogenic in humans. As the chlorine dioxide concentration increases, the time required to achieve sterilization becomes progressively shorter. For example, only 30 minutes were required at 40 mg/l to sterilize the 10⁶ *B. atrophaeus* spores at 30° to 32°C⁹⁵⁴. Currently, no gaseous chlorine dioxide system is FDA cleared.

Vaporized Peracetic Acid. The sporicidal activity of peracetic acid vapor at 20, 40, 60, and 80% relative humidity and 25°C was determined on *Bacillus atrophaeus* spores on paper and glass surfaces. Appreciable activity occurred within 10 minutes of exposure to 1 mg of peracetic acid per liter at 40% or higher relative humidity⁹⁵⁵. No vaporized peracetic acid system is FDA cleared.

Infrared radiation. An infrared radiation prototype sterilizer was investigated and found to destroy *B. atrophaeus* spores. Some of the possible advantages of infrared technology include short cycle time, low energy consumption, no cycle residuals, and no toxicologic or environmental effects. This may provide an alternative technology for sterilization of selected heat-resistant instruments but there are no FDA-cleared systems for use in healthcare facilities⁹⁵⁶.

The other sterilization technologies mentioned above may be used for sterilization of critical medical items if cleared by the FDA and ideally, the microbicidal effectiveness of the technology has been published in the scientific literature. The selection and use of disinfectants, chemical sterilants and sterilization processes in the healthcare field is dynamic, and products may become available that are not in existence when this guideline was written. As newer disinfectants and sterilization processes become available, persons or committees responsible for selecting disinfectants and sterilization processes should be guided by products cleared by FDA and EPA as well as information in the scientific literature.

Sterilizing Practices

Overview. The delivery of sterile products for use in patient care depends not only on the effectiveness of the sterilization process but also on the unit design, decontamination, disassembling and packaging of the device, loading the sterilizer, monitoring, sterilant quality and quantity, and the appropriateness of the cycle for the load contents, and other aspects of device reprocessing. Healthcare personnel should perform most cleaning, disinfecting, and sterilizing of patient-care supplies in a central processing department in order to more easily control quality. The aim of central processing is the orderly processing of medical and surgical instruments to protect patients from infections while minimizing risks to staff and preserving the value of the items being reprocessed⁹⁵⁷. Healthcare facilities should promote the same level of efficiency and safety in the preparation of supplies in other areas (e.g., operating room, respiratory therapy) as is practiced in central processing.

Ensuring consistency of sterilization practices requires a comprehensive program that ensures operator competence and proper methods of cleaning and wrapping instruments, loading the sterilizer,

operating the sterilizer, and monitoring of the entire process. Furthermore, care must be consistent from an infection prevention standpoint in all patient-care settings, such as hospital and outpatient facilities.

Sterilization Cycle Verification. A sterilization process should be verified before it is put into use in healthcare settings. All steam, ETO, and other low-temperature sterilizers are tested with biological and chemical indicators upon installation, when the sterilizer is relocated, redesigned, after major repair and after a sterilization failure has occurred to ensure they are functioning prior to placing them into routine use. Three consecutive empty steam cycles are run with a biological and chemical indicator in an appropriate test package or tray. Each type of steam cycle used for sterilization (e.g., vacuum-assisted, gravity) is tested separately. In a prevacuum steam sterilizer three consecutive empty cycles are also run with a Bowie-Dick test. The sterilizer is not put back into use until all biological indicators are negative and chemical indicators show a correct end-point response^{811-814, 819, 958}.

Biological and chemical indicator testing is also done for ongoing quality assurance testing of representative samples of actual products being sterilized and product testing when major changes are made in packaging, wraps, or load configuration. Biological and chemical indicators are placed in products, which are processed in a full load. When three consecutive cycles show negative biological indicators and chemical indicators with a correct end point response, you can put the change made into routine use^{811-814, 958}. Items processed during the three evaluation cycles should be quarantined until the test results are negative.

Physical Facilities. The central processing area(s) ideally should be divided into at least three areas: decontamination, packaging, and sterilization and storage. Physical barriers should separate the decontamination area from the other sections to contain contamination on used items. In the decontamination area reusable contaminated supplies (and possibly disposable items that are reused) are received, sorted, and decontaminated. The recommended airflow pattern should contain contaminants within the decontamination area and minimize the flow of contaminants to the clean areas. The American Institute of Architects⁹⁵⁹ recommends negative pressure and no fewer than six air exchanges per hour in the decontamination area (AAMI recommends 10 air changes per hour) and 10 air changes per hour with positive pressure in the sterilizer equipment room. The packaging area is for inspecting, assembling, and packaging clean, but not sterile, material. The sterile storage area should be a limited access area with a controlled temperature (may be as high as 75°F) and relative humidity (30-60% in all works areas except sterile storage, where the relative humidity should not exceed 70%)⁸¹⁹. The floors and walls should be constructed of materials capable of withstanding chemical agents used for cleaning or disinfecting. Ceilings and wall surfaces should be constructed of non-shedding materials. Physical arrangements of processing areas are presented schematically in four references^{811, 819, 920, 957}.

Cleaning. As repeatedly mentioned, items must be cleaned using water with detergents or enzymatic cleaners^{465, 466, 468} before processing. Cleaning reduces the bioburden and removes foreign material (i.e., organic residue and inorganic salts) that interferes with the sterilization process by acting as a barrier to the sterilization agent^{179, 426, 457, 911, 912}. Surgical instruments are generally presoaked or prerinsed to prevent drying of blood and tissue. Precleaning in patient-care areas may be needed on items that are heavily soiled with feces, sputum, blood, or other material. Items sent to central processing without removing gross soil may be difficult to clean because of dried secretions and excretions. Cleaning and decontamination should be done as soon as possible after items have been used.

Several types of mechanical cleaning machines (e.g., utensil washer-sanitizer, ultrasonic cleaner, washer-sterilizer, dishwasher, washer-disinfector) may facilitate cleaning and decontamination of most items. This equipment often is automated and may increase productivity, improve cleaning effectiveness, and decrease worker exposure to blood and body fluids. Delicate and intricate objects and heat- or moisture-sensitive articles may require careful cleaning by hand. All used items sent to the central processing area should be considered contaminated (unless decontaminated in the area of origin), handled with gloves (forceps or tongs are sometimes needed to avoid exposure to sharps), and decontaminated by one of the aforementioned methods to render them safer to handle. Items composed

of more than one removable part should be disassembled. Care should be taken to ensure that all parts are kept together, so that reassembly can be accomplished efficiently⁸¹¹.

Investigators have described the degree of cleanliness by visual and microscopic examination. One study found 91% of the instruments to be clean visually but, when examined microscopically, 84% of the instruments had residual debris. Sites that contained residual debris included junctions between insulating sheaths and activating mechanisms of laparoscopic instruments and articulations and grooves of forceps. More research is needed to understand the clinical significance of these findings⁹⁶⁰ and how to ensure proper cleaning.

Personnel working in the decontamination area should wear household-cleaning-type rubber or plastic gloves when handling or cleaning contaminated instruments and devices. Face masks, eye protection such as goggles or full-length faceshields, and appropriate gowns should be worn when exposure to blood and contaminated fluids may occur (e.g., when manually cleaning contaminated devices)⁹⁶¹. Contaminated instruments are a source of microorganisms that could inoculate personnel through nonintact skin on the hands or through contact with the mucous membranes of eyes, nose, or mouth^{214, 811, 813}. Reusable sharps that have been in contact with blood present a special hazard. Employees must not reach with their gloved hands into trays or containers that hold these sharps to retrieve them²¹⁴. Rather, employees should use engineering controls (e.g., forceps) to retrieve these devices.

Packaging. Once items are cleaned, dried, and inspected, those requiring sterilization must be wrapped or placed in rigid containers and should be arranged in instrument trays/baskets according to the guidelines provided by the AAMI and other professional organizations^{454, 811-814, 819, 836, 962}. These guidelines state that hinged instruments should be opened; items with removable parts should be disassembled unless the device manufacturer or researchers provide specific instructions or test data to the contrary¹⁸¹; complex instruments should be prepared and sterilized according to device manufacturer's instructions and test data; devices with concave surfaces should be positioned to facilitate drainage of water; heavy items should be positioned not to damage delicate items; and the weight of the instrument set should be based on the design and density of the instruments and the distribution of metal mass^{811, 962}. While there is no longer a specified sterilization weight limit for surgical sets, heavy metal mass is a cause of wet packs (i.e., moisture inside the case and tray after completion of the sterilization cycle)⁹⁶³. Other parameters that may influence drying are the density of the wraps and the design of the set⁹⁶⁴.

There are several choices in methods to maintain sterility of surgical instruments, including rigid containers, peel-open pouches (e.g., self-sealed or heat-sealed plastic and paper pouches), roll stock or reels (i.e., paper-plastic combinations of tubing designed to allow the user to cut and seal the ends to form a pouch)⁴⁵⁴ and sterilization wraps (woven and nonwoven). Healthcare facilities may use all of these packaging options. The packaging material must allow penetration of the sterilant, provide protection against contact contamination during handling, provide an effective barrier to microbial penetration, and maintain the sterility of the processed item after sterilization⁹⁶⁵. An ideal sterilization wrap would successfully address barrier effectiveness, penetrability (i.e., allows sterilant to penetrate), aeration (e.g., allows ETO to dissipate), ease of use, drapeability, flexibility, puncture resistance, tear strength, toxicity, odor, waste disposal, linting, cost, and transparency⁹⁶⁶. Unacceptable packaging for use with ETO (e.g., foil, polyvinylchloride, and polyvinylidene chloride [kitchen-type transparent wrap])⁸¹⁴ or hydrogen peroxide gas plasma (e.g., linens and paper) should not be used to wrap medical items.

In central processing, double wrapping can be done sequentially or nonsequentially (i.e., simultaneous wrapping). Wrapping should be done in such a manner to avoid tenting and gapping. The sequential wrap uses two sheets of the standard sterilization wrap, one wrapped after the other. This procedure creates a package within a package. The nonsequential process uses two sheets wrapped at the same time so that the wrapping needs to be performed only once. This latter method provides

multiple layers of protection of surgical instruments from contamination and saves time since wrapping is done only once. Multiple layers are still common practice due to the rigors of handling within the facility even though the barrier efficacy of a single sheet of wrap has improved over the years⁹⁶⁶. Written and illustrated procedures for preparation of items to be packaged should be readily available and used by personnel when packaging procedures are performed⁴⁵⁴.

Loading. All items to be sterilized should be arranged so all surfaces will be directly exposed to the sterilizing agent. Thus, loading procedures must allow for free circulation of steam (or another sterilant) around each item. Historically, it was recommended that muslin fabric packs should not exceed the maximal dimensions, weight, and density of 12 inches wide x 12 inches high x 20 inches long, 12 lbs, and 7.2 lbs per cubic foot, respectively. Due to the variety of textiles and metal/plastic containers on the market, the textile and metal/plastic container manufacturer and the sterilizer manufacturers should be consulted for instructions on pack preparation and density parameters⁸¹⁹.

There are several important basic principles for loading a sterilizer: allow for proper sterilant circulation; perforated trays should be placed so the tray is parallel to the shelf; nonperforated containers should be placed on their edge (e.g., basins); small items should be loosely placed in wire baskets; and peel packs should be placed on edge in perforated or mesh bottom racks or baskets^{454, 811, 836}.

Storage. Studies in the early 1970s suggested that wrapped surgical trays remained sterile for varying periods depending on the type of material used to wrap the trays. Safe storage times for sterile packs vary with the porosity of the wrapper and storage conditions (e.g., open versus closed cabinets). Heat-sealed, plastic peel-down pouches and wrapped packs sealed in 3-mil (3/1000 inch) polyethylene overwrap have been reported to be sterile for as long as 9 months after sterilization. The 3-mil polyethylene is applied after sterilization to extend the shelf life for infrequently used items⁹⁶⁷. Supplies wrapped in double-thickness muslin comprising four layers, or equivalent, remain sterile for at least 30 days. Any item that has been sterilized should not be used after the expiration date has been exceeded or if the sterilized package is wet, torn, or punctured.

Although some hospitals continue to date every sterilized product and use the time-related shelf-life practice, many hospitals have switched to an event-related shelf-life practice. This latter practice recognizes that the product should remain sterile until some event causes the item to become contaminated (e.g., tear in packaging, packaging becomes wet, seal is broken)⁹⁶⁸. Event-related factors that contribute to the contamination of a product include bioburden (i.e., the amount of contamination in the environment), air movement, traffic, location, humidity, insects, vermin, flooding, storage area space, open/closed shelving, temperature, and the properties of the wrap material^{966, 969}. There are data that support the event-related shelf-life practice⁹⁷⁰⁻⁹⁷². One study examined the effect of time on the sterile integrity of paper envelopes, peel pouches, and nylon sleeves. The most important finding was the absence of a trend toward an increased rate of contamination over time for any pack when placed in covered storage⁹⁷¹. Another evaluated the effectiveness of event-related outdating by microbiologically testing sterilized items. During the 2-year study period, all of the items tested were sterile⁹⁷². Thus, contamination of a sterile item is event-related and the probability of contamination increases with increased handling⁹⁷³.

Following the sterilization process, medical and surgical devices must be handled using aseptic technique in order to prevent contamination. Sterile supplies should be stored far enough from the floor (8 to 10 inches), the ceiling (5 inches unless near a sprinkler head [18 inches from sprinkler head]), and the outside walls (2 inches) to allow for adequate air circulation, ease of cleaning, and compliance with local fire codes (e.g., supplies must be at least 18 inches from sprinkler heads). Medical and surgical supplies should not be stored under sinks or in other locations where they can become wet. Sterile items that become wet are considered contaminated because moisture brings with it microorganisms from the air and surfaces. Closed or covered cabinets are ideal but open shelving may be used for storage. Any package that has fallen or been dropped on the floor must be inspected for damage to the packaging and

contents (if the items are breakable). If the package is heat-sealed in impervious plastic and the seal is still intact, the package should be considered not contaminated. If undamaged, items packaged in plastic need not be reprocessed.

Monitoring. The sterilization procedure should be monitored routinely by using a combination of mechanical, chemical, and biological indicators to evaluate the sterilizing conditions and indirectly the microbiologic status of the processed items. The mechanical monitors for steam sterilization include the daily assessment of cycle time and temperature by examining the temperature record chart (or computer printout) and an assessment of pressure via the pressure gauge. The mechanical monitors for ETO include time, temperature, and pressure recorders that provide data via computer printouts, gauges, and/or displays⁸¹⁴. Generally, two essential elements for ETO sterilization (i.e., the gas concentration and humidity) cannot be monitored in healthcare ETO sterilizers.

Chemical indicators are convenient, are inexpensive, and indicate that the item has been exposed to the sterilization process. In one study, chemical indicators were more likely than biological indicators to inaccurately indicate sterilization at marginal sterilization times (e.g., 2 minutes)⁸⁴⁷. Chemical indicators should be used in conjunction with biological indicators, but based on current studies should not replace them because they indicate sterilization at marginal sterilization time and because only a biological indicator consisting of resistant spores can measure the microbial killing power of the sterilization process.^{847, 974} Chemical indicators are affixed on the outside of each pack to show that the package has been processed through a sterilization cycle, but these indicators do not prove sterilization has been achieved. Preferably, a chemical indicator also should be placed on the inside of each pack to verify sterilant penetration. Chemical indicators usually are either heat- or chemical-sensitive inks that change color when one or more sterilization parameters (e.g., steam-time, temperature, and/or saturated steam; ETO-time, temperature, relative humidity and/or ETO concentration) are present. Chemical indicators have been grouped into five classes based on their ability to monitor one or multiple sterilization parameters^{813, 819}. If the internal and/or external indicator suggests inadequate processing, the item should not be used⁸¹⁵. An air-removal test (Bowie-Dick Test) must be performed daily in an empty dynamic-air-removal sterilizer (e.g., prevacuum steam sterilizer) to ensure air removal.

Biological indicators are recognized by most authorities as being closest to the ideal monitors of the sterilization process^{974, 975} because they measure the sterilization process directly by using the most resistant microorganisms (i.e., *Bacillus* spores), and not by merely testing the physical and chemical conditions necessary for sterilization. Since the *Bacillus* spores used in biological indicators are more resistant and present in greater numbers than are the common microbial contaminants found on patient-care equipment, the demonstration that the biological indicator has been inactivated strongly implies that other potential pathogens in the load have been killed⁸⁴⁴.

An ideal biological monitor of the sterilization process should be easy to use, be inexpensive, not be subject to exogenous contamination, provide positive results as soon as possible after the cycle so that corrective action may be accomplished, and provide positive results only when the sterilization parameters (e.g., steam-time, temperature, and/or saturated steam; ETO-time, temperature, relative humidity and/or ETO concentration) are inadequate to kill microbial contaminants⁸⁴⁷.

Biological indicators are the only process indicators that directly monitor the lethality of a given sterilization process. Spores used to monitor a sterilization process have demonstrated resistance to the sterilizing agent and are more resistant than the bioburden found on medical devices^{179, 911, 912}. *B. atropheus* spores (10^6) are used to monitor ETO and dry heat, and *G. stearothermophilus* spores (10^5) are used to monitor steam sterilization, hydrogen peroxide gas plasma, and liquid peracetic acid sterilizers. *G. stearothermophilus* is incubated at 55-60°C, and *B. atropheus* is incubated at 35-37°C. Steam and low temperature sterilizers (e.g., hydrogen peroxide gas plasma, peracetic acid) should be monitored at least weekly with the appropriate commercial preparation of spores. If a sterilizer is used frequently (e.g., several loads per day), daily use of biological indicators allows earlier discovery of

equipment malfunctions or procedural errors and thus minimizes the extent of patient surveillance and product recall needed in the event of a positive biological indicator⁸¹¹. Each load should be monitored if it contains implantable objects. If feasible, implantable items should not be used until the results of spore tests are known to be negative.

Originally, spore-strip biological indicators required up to 7 days of incubation to detect viable spores from marginal cycles (i.e., when few spores remained viable). The next generation of biological indicator was self-contained in plastic vials containing a spore-coated paper strip and a growth media in a crushable glass ampoule. This indicator had a maximum incubation of 48 hours but significant failures could be detected in ≤ 24 hours. A rapid-readout biological indicator that detects the presence of enzymes of *G. stearothermophilus* by reading a fluorescent product produced by the enzymatic breakdown of a nonfluorescent substrate has been marketed for the more than 10 years. Studies demonstrate that the sensitivity of rapid-readout tests for steam sterilization (1 hour for 132°C gravity sterilizers, 3 hrs for 121°C gravity and 132°C vacuum sterilizers) parallels that of the conventional sterilization-specific biological indicators^{846, 847, 976, 977} and the fluorescent rapid readout results reliably predict 24- and 48-hour and 7-day growth⁹⁷⁸. The rapid-readout biological indicator is a dual indicator system as it also detects acid metabolites produced during growth of the *G. stearothermophilus* spores. This system is different from the indicator system consisting of an enzyme system of bacterial origin without spores. Independent comparative data using suboptimal sterilization cycles (e.g., reduced time or temperature) with the enzyme-based indicator system have not been published⁹⁷⁹.

A new rapid-readout ETO biological indicator has been designed for rapid and reliable monitoring of ETO sterilization processes. The indicator has been cleared by the FDA for use in the United States⁴⁰⁰. The rapid-readout ETO biological indicator detects the presence of *B. atrophaeus* by detecting a fluorescent signal indicating the activity of an enzyme present within the *B. atrophaeus* organism, beta-glucosidase. The fluorescence indicates the presence of an active spore-associated enzyme and a sterilization process failure. This indicator also detects acid metabolites produced during growth of the *B. atrophaeus* spore. Per manufacturer's data, the enzyme always was detected whenever viable spores were present. This was expected because the enzyme is relatively ETO resistant and is inactivated at a slightly longer exposure time than the spore. The rapid-readout ETO biological indicator can be used to monitor 100% ETO, and ETO-HCFC mixture sterilization cycles. It has not been tested in ETO-CO₂ mixture sterilization cycles.

The standard biological indicator used for monitoring full-cycle steam sterilizers does not provide reliable monitoring flash sterilizers⁹⁸⁰. Biological indicators specifically designed for monitoring flash sterilization are now available, and studies comparing them have been published^{846, 847, 981}.

Since sterilization failure can occur (about 1% for steam)⁹⁸², a procedure to follow in the event of positive spore tests with steam sterilization has been provided by CDC and the Association of periOperative Registered Nurses (AORN). The 1981 CDC recommendation is that "objects, other than implantable objects, do not need to be recalled because of a single positive spore test unless the steam sterilizer or the sterilization procedure is defective." The rationale for this recommendation is that single positive spore tests in sterilizers occur sporadically. They may occur for reasons such as slight variation in the resistance of the spores⁹⁸³, improper use of the sterilizer, and laboratory contamination during culture (uncommon with self-contained spore tests). If the mechanical (e.g., time, temperature, pressure in the steam sterilizer) and chemical (internal and/or external) indicators suggest that the sterilizer was functioning properly, a single positive spore test probably does not indicate sterilizer malfunction but the spore test should be repeated immediately⁹⁸³. If the spore tests remain positive, use of the sterilizer should be discontinued until it is serviced¹. Similarly, AORN states that a single positive spore test does not necessarily indicate a sterilizer failure. If the test is positive, the sterilizer should immediately be rechallenged for proper use and function. Items, other than implantable ones, do not necessarily need to be recalled unless a sterilizer malfunction is found. If a sterilizer malfunction is discovered, the items must be considered nonsterile, and the items from the suspect load(s) should be recalled, insofar as

possible, and reprocessed⁹⁸⁴. A suggested protocol for management of positive biological indicators is shown in Table 12⁸³⁹. A more conservative approach also has been recommended⁸¹³ in which any positive spore test is assumed to represent sterilizer malfunction and requires that all materials processed in that sterilizer, dating from the sterilization cycle having the last negative biologic indicator to the next cycle showing satisfactory biologic indicator challenge results, must be considered nonsterile and retrieved, if possible, and reprocessed. This more conservative approach should be used for sterilization methods other than steam (e.g., ETO, hydrogen peroxide gas plasma). However, no action is necessary if there is strong evidence for the biological indicator being defective⁹⁸³ or the growth medium contained a *Bacillus* contaminant⁹⁸⁵.

If patient-care items were used before retrieval, the infection control professional should assess the risk of infection in collaboration with central processing, surgical services, and risk management staff. The factors that should be considered include the chemical indicator result (e.g., nonreactive chemical indicator may indicate temperature not achieved); the results of other biological indicators that followed the positive biological indicator (e.g., positive on Tuesday, negative on Wednesday); the parameters of the sterilizer associated with the positive biological indicator (e.g., reduced time at correct temperature); the time-temperature chart (or printout); and the microbial load associated with decontaminated surgical instruments (e.g., 85% of decontaminated surgical instruments have less than 100 CFU). The margin of safety in steam sterilization is sufficiently large that there is minimal infection risk associated with items in a load that show spore growth, especially if the item was properly cleaned and the temperature was achieved (e.g., as shown by acceptable chemical indicator or temperature chart). There are no published studies that document disease transmission via a nonretrieved surgical instrument following a sterilization cycle with a positive biological indicator.

False-positive biological indicators may occur from improper testing or faulty indicators. The latter may occur from improper storage, processing, product contamination, material failure, or variation in resistance of spores. Gram stain and subculture of a positive biological indicator may determine if a contaminant has created a false-positive result^{839, 986}. However, in one incident, the broth used as growth medium contained a contaminant, *B. coagulans*, which resulted in broth turbidity at 55°C⁹⁸⁵. Testing of paired biological indicators from different manufacturers can assist in assessing a product defect⁸³⁹. False-positive biological indicators due to extrinsic contamination when using self-contained biological indicators should be uncommon. A biological indicator should not be considered a false-positive indicator until a thorough analysis of the entire sterilization process shows this to be likely.

The size and composition of the biological indicator test pack should be standardized to create a significant challenge to air removal and sterilant penetration and to obtain interpretable results. There is a standard 16-towel pack recommended by AAMI for steam sterilization^{813, 819, 987} consisting of 16 clean, preconditioned, reusable huck or absorbent surgical towels each of which is approximately 16 inches by 26 inches. Each towel is folded lengthwise into thirds and then folded widthwise in the middle. One or more biological indicators are placed between the eight and ninth towels in the approximate geometric center of the pack. When the towels are folded and placed one on top of another, to form a stack (approximately 6 inch height) it should weigh approximately 3 pounds and should have a density of approximately 11.3 pounds per cubic foot⁸¹³. This test pack has not gained universal use as a standard pack that simulates the actual in-use conditions of steam sterilizers. Commercially available disposable test packs that have been shown to be equivalent to the AAMI 16 towel test pack also may be used. The test pack should be placed flat in an otherwise fully loaded sterilizer chamber, in the area least favorable to sterilization (i.e., the area representing the greatest challenge to the biological indicator). This area is normally in the front, bottom section of the sterilizer, near the drain^{811, 813}. A control biological indicator from the lot used for testing should be left unexposed to the sterilant, and then incubated to verify the presterilization viability of the test spores and proper incubation. The most conservative approach would be to use a control for each run; however, less frequent use may be adequate (e.g., weekly). There also is a routine test pack for ETO where a biological indicator is placed in a plastic syringe with plunger, then placed in the folds of a clean surgical towel, and wrapped. Alternatively, commercially available disposal

test packs that have been shown to be equivalent to the AAMI test pack may be used. The test pack is placed in the center of the sterilizer load⁸¹⁴. Sterilization records (mechanical, chemical, and biological) should be retained for a time period in compliance with standards (e.g., Joint Commission for the Accreditation of Healthcare Facilities requests 3 years) and state and federal regulations.

In Europe, biological monitors are not used routinely to monitor the sterilization process. Instead, release of sterilizer items is based on monitoring the physical conditions of the sterilization process that is termed “parametric release.” Parametric release requires that there is a defined quality system in place at the facility performing the sterilization and that the sterilization process be validated for the items being sterilized. At present in Europe, parametric release is accepted for steam, dry heat, and ionizing radiation processes, as the physical conditions are understood and can be monitored directly⁹⁸⁸. For example, with steam sterilizers the load could be monitored with probes that would yield data on temperature, time, and humidity at representative locations in the chamber and compared to the specifications developed during the validation process.

Periodic infection control rounds to areas using sterilizers to standardize the sterilizer’s use may identify correctable variances in operator competence; documentation of sterilization records, including chemical and biological indicator test results; sterilizer maintenance and wrapping; and load numbering of packs. These rounds also may identify improvement activities to ensure that operators are adhering to established standards⁹⁸⁹.

REUSE OF SINGLE-USE MEDICAL DEVICES

The reuse of single-use medical devices began in the late 1970s. Before this time most devices were considered reusable. Reuse of single-use devices increased as a cost-saving measure. Approximately 20 to 30% of U.S. hospitals reported that they reuse at least one type of single-use device. Reuse of single-use devices involves regulatory, ethical, medical, legal and economic issues and has been extremely controversial for more than two decades⁹⁹⁰. The U.S. public has expressed increasing concern regarding the risk of infection and injury when reusing medical devices intended and labeled for single use. Although some investigators have demonstrated it is safe to reuse disposable medical devices such as cardiac electrode catheters,⁹⁹¹⁻⁹⁹³ additional studies are needed to define the risks⁹⁹⁴ and document the benefits. In August 2000, FDA released a guidance document on single-use devices reprocessed by third parties or hospitals⁹⁹⁵. In this guidance document, FDA states that hospitals or third-party reproducers will be considered “manufacturers” and regulated in the same manner. A reused single-use device will have to comply with the same regulatory requirements of the device when it was originally manufactured. This document presents FDA’s intent to enforce premarket submission requirements within 6 months (February 2001) for class III devices (e.g., cardiovascular intra-aortic balloon pump, transluminal coronary angioplasty catheter); 12 months (August 2001) for class II devices (e.g., blood pressure cuff, bronchoscope biopsy forceps); and 18 months (February 2002) for class I devices (e.g., disposable medical scissors, ophthalmic knife). FDA uses two types of premarket requirements for nonexempt class I and II devices, a 510(k) submission that may have to show that the device is as safe and effective as the same device when new, and a premarket approval application. The 510(k) submission must provide scientific evidence that the device is safe and effective for its intended use. FDA allowed hospitals a year to comply with the nonpremarket requirements (registration and listing, reporting adverse events associated with medical devices, quality system regulations, and proper labeling). The options for hospitals are to stop reprocessing single-use devices, comply with the rule, or outsource to a third-party reproducer. FDA guidance document does not apply to permanently implantable pacemakers, hemodialyzers, opened but unused single-use devices, or healthcare settings other than acute-care hospitals. The reuse of single use medical devices continues to be an evolving area of regulations. For this reason, healthcare workers should refer to FDA for the latest guidance (www.fda.gov)⁹⁹⁶.

CONCLUSION

When properly used, disinfection and sterilization can ensure the safe use of invasive and non-invasive medical devices. However, current disinfection and sterilization guidelines must be strictly followed.

WED-BASED DISINFECTION AND STERILIZATION RESOURCES

Additional information about disinfection and sterilization is available at the following dedicated websites:

Food and Drug Administration, Rockville, Maryland
<http://www.fda.gov/dcrh/ode/germlab.html>

Environmental Protection Agency, Washington, D.C.
<http://www.epa.gov/oppad001/chemregindex.htm>

Centers for Disease Control and Prevention, Atlanta, Georgia
<http://www.cdc.gov/ncidod/dhqp/sterile.html>

University of North Carolina, Chapel Hill, North Carolina
<http://www.disinfectionandsterilization.org>

RECOMMENDATIONS FOR DISINFECTION AND STERILIZATION IN HEALTHCARE FACILITIES

A. Rationale

The ultimate goal of the Recommendations for Disinfection and Sterilization in Health-Care Facilities, 2008, is to reduce rates of health-care–associated infections through appropriate use of both disinfection and sterilization. Each recommendation is categorized according to scientific evidence, theoretical rationale, applicability, and federal regulations. Examples are included in some recommendations to aid the reader; however, these examples are not intended to define the only method of implementing the recommendation. The CDC system for categorizing recommendations is defined in the following (Rankings) section.

B. Rankings

Category IA. Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiologic studies.

Category IB. Strongly recommended for implementation and supported by some experimental, clinical, or epidemiologic studies, and by a strong theoretical rationale.

Category IC. Required by state or federal regulations. Because of state differences, readers should not assume that the absence of an *IC* recommendation implies the absence of state regulations.

Category II. Suggested for implementation and supported by suggestive clinical or epidemiologic studies or by a theoretical rationale.

No recommendation. Unresolved issue. These include practices for which insufficient evidence or no consensus exists regarding efficacy.

C. Recommendations

1. **Occupational Health and Exposure**

- a. Inform each worker of the possible health effects of his or her exposure to infectious agents (e.g., hepatitis B virus [HBV], hepatitis C virus, human immunodeficiency virus [HIV]), and/or chemicals (e.g., EtO, formaldehyde). The information should be consistent with Occupational Safety and Health Administration (OSHA) requirements and identify the areas and tasks in which potential exists for exposure. *Category II, IC*^{214, 320, 959, 997, 998}
- b. Educate health-care workers in the selection and proper use of personal protective equipment (PPE). *Category II, IC*
- c. Ensure that workers wear appropriate PPE to preclude exposure to infectious agents or chemicals through the respiratory system, skin, or mucous membranes of the eyes, nose, or mouth. PPE can include gloves, gowns, masks, and eye protection. The exact type of PPE depends on the infectious or chemical agent and the anticipated duration of exposure. The employer is responsible for making such equipment and training available. *Category II, IC.*^{214, 997-999}
- d. Establish a program for monitoring occupational exposure to regulated chemicals (e.g., formaldehyde, EtO) that adheres to state and federal regulations. *Category II, IC.*^{997, 1000, 1001}
- e. Exclude healthcare workers with weeping dermatitis of hands from direct contact with patient-care equipment. *Category IB.*^{1002, 1003}

2. **Cleaning of Patient-Care Devices**

- a. In hospitals, perform most cleaning, disinfection, and sterilization of patient-care devices in a central processing department in order to more easily control quality. *Category II.*^{454, 836, 959}
- b. Meticulously clean patient-care items with water and detergent, or with water and enzymatic cleaners before high-level disinfection or sterilization procedures. *Category IB.*^{6, 83, 101, 104-106, 124, 179, 424-426, 436, 465, 471, 911-913, 1004}
 - i. Remove visible organic residue (e.g., residue of blood and tissue) and inorganic salts with cleaning. Use cleaning agents that are capable of removing visible organic and inorganic residues. *Category IB.*^{424-426, 466, 468, 469, 471, 908, 910}

- ii. Clean medical devices as soon as practical after use (e.g., at the point of use) because soiled materials become dried onto the instruments. Dried or baked materials on the instrument make the removal process more difficult and the disinfection or sterilization process less effective or ineffective. *Category IB.*^{55, 56, 59, 291, 465, 1005, 1006}
 - c. Perform either manual cleaning (i.e., using friction) or mechanical cleaning (e.g., with ultrasonic cleaners, washer-disinfector, washer-sterilizers). *Category IB.*^{426, 456, 471, 999}
 - d. If using an automatic washer/disinfector, ensure that the unit is used in accordance with the manufacturer's recommendations. *Category IB.*^{7, 133, 155, 725}
 - e. Ensure that the detergents or enzymatic cleaners selected are compatible with the metals and other materials used in medical instruments. Ensure that the rinse step is adequate for removing cleaning residues to levels that will not interfere with subsequent disinfection/sterilization processes. *Category II.*^{836, 1004}
 - f. Inspect equipment surfaces for breaks in integrity that would impair either cleaning or disinfection/sterilization. Discard or repair equipment that no longer functions as intended or cannot be properly cleaned, and disinfected or sterilized. *Category II.*⁸⁸⁸
 - g.
- 3. **Indications for Sterilization, High-Level Disinfection, and Low-Level Disinfection**
 - a. Before use on each patient, sterilize critical medical and surgical devices and instruments that enter normally sterile tissue or the vascular system or through which a sterile body fluid flows (e.g., blood). See recommendation 7g for exceptions. *Category IA.*^{179, 497, 821, 822, 907, 911, 912}
 - b. Provide, at a minimum, high-level disinfection for semicritical patient-care equipment (e.g., gastrointestinal endoscopes, endotracheal tubes, anesthesia breathing circuits, and respiratory therapy equipment) that touches either mucous membranes or nonintact skin. *Category IA.*^{6-8, 17, 20, 99, 101, 108, 113-115, 129, 138, 139, 147, 152-154, 471, 1007}
 - c. Perform low-level disinfection for noncritical patient-care surfaces (e.g., bedrails, over-the-bed table) and equipment (e.g., blood pressure cuff) that touch intact skin (see Recommendation 5g). *Category II.*^{17, 46-48, 50-52, 67, 68, 372, 373, 378, 382, 401}
- 4. **Selection and Use of Low-Level Disinfectants for Noncritical Patient-Care Devices**
 - a. Process noncritical patient-care devices using a disinfectant and the concentration of germicide listed in Table 1. *Category IB.*^{17, 46-48, 50-52, 67, 68, 378, 382, 401}
 - b. Disinfect noncritical medical devices (e.g., blood pressure cuff) with an EPA-registered hospital disinfectant using the label's safety precautions and use directions. Most EPA-registered hospital disinfectants have a label contact time of 10 minutes. However, multiple scientific studies have demonstrated the efficacy of hospital disinfectants against pathogens with a contact time of at least 1 minute. By law, all applicable label instructions on EPA-registered products must be followed. If the user selects exposure conditions that differ from those on the EPA-registered product label, the user assumes liability from any injuries resulting from off-label use and is potentially subject to enforcement action under FIFRA. *Category IB.*^{17, 47, 48, 50, 51, 53-57, 59, 60, 62-64, 355, 378, 382}
 - c. Ensure that, at a minimum, noncritical patient-care devices are disinfected when visibly soiled and on a regular basis (such as after use on each patient or once daily or once weekly). *Category II.*^{378, 380, 1008}
 - d. If dedicated, disposable devices are not available, disinfect noncritical patient-care equipment after using it on a patient who is on contact precautions before using this equipment on another patient. *Category IB.*^{47, 67, 391, 1009}
- 5. **Cleaning and Disinfecting Environmental Surfaces in Healthcare Facilities**
 - a. Clean housekeeping surfaces (e.g., floors, tabletops) on a regular basis, when spills occur, and when these surfaces are visibly soiled. *Category II.*^{23, 378, 380, 382, 1008, 1010}
 - b. Disinfect (or clean) environmental surfaces on a regular basis (e.g., daily, three times per week) and when surfaces are visibly soiled. *Category II.*^{378, 380, 402, 1008}
 - c. Follow manufacturers' instructions for proper use of disinfecting (or detergent) products --- such as recommended use-dilution, material compatibility, storage, shelf-life, and safe use and

- disposal. *Category II.* ^{327, 365, 404}
- d. Clean walls, blinds, and window curtains in patient-care areas when these surfaces are visibly contaminated or soiled. *Category II.* ¹⁰¹¹
 - e. Prepare disinfecting (or detergent) solutions as needed and replace these with fresh solution frequently (e.g., replace floor mopping solution every three patient rooms, change no less often than at 60-minute intervals), according to the facility's policy. *Category IB.* ^{68, 379}
 - f. Decontaminate mop heads and cleaning cloths regularly to prevent contamination (e.g., launder and dry at least daily). *Category II.* ^{68, 402, 403}
 - g. Use a one-step process and an EPA-registered hospital disinfectant designed for housekeeping purposes in patient care areas where 1) uncertainty exists about the nature of the soil on the surfaces (e.g., blood or body fluid contamination versus routine dust or dirt); or 2) uncertainty exists about the presence of multidrug resistant organisms on such surfaces. See 5n for recommendations requiring cleaning and disinfecting blood-contaminated surfaces. *Category II.* ^{23, 47, 48, 51, 214, 378, 379, 382, 416, 1012}
 - h. Detergent and water are adequate for cleaning surfaces in nonpatient-care areas (e.g., administrative offices). *Category II.* ²³
 - i. Do not use high-level disinfectants/liquid chemical sterilants for disinfection of non-critical surfaces. *Category IB.* ^{23, 69, 318}
 - j. Wet-dust horizontal surfaces regularly (e.g., daily, three times per week) using clean cloths moistened with an EPA-registered hospital disinfectant (or detergent). Prepare the disinfectant (or detergent) as recommended by the manufacturer. *Category II.* ^{68, 378, 380, 402, 403, 1008}
 - k. Disinfect noncritical surfaces with an EPA-registered hospital disinfectant according to the label's safety precautions and use directions. Most EPA-registered hospital disinfectants have a label contact time of 10 minutes. However, many scientific studies have demonstrated the efficacy of hospital disinfectants against pathogens with a contact time of at least 1 minute. By law, the user must follow all applicable label instructions on EPA-registered products. If the user selects exposure conditions that differ from those on the EPA-registered product label, the user assumes liability for any injuries resulting from off-label use and is potentially subject to enforcement action under FIFRA. *Category II, IC.* ^{17, 47, 48, 50, 51, 53-57, 59, 60, 62-64, 355, 378, 382}
 - l. Do not use disinfectants to clean infant bassinets and incubators while these items are occupied. If disinfectants (e.g., phenolics) are used for the terminal cleaning of infant bassinets and incubators, thoroughly rinse the surfaces of these items with water and dry them before these items are reused. *Category IB.* ^{17, 739, 740}
 - m. Promptly clean and decontaminate spills of blood and other potentially infectious materials. Discard blood-contaminated items in compliance with federal regulations. *Category IB, IC.* ²¹⁴
 - n. For site decontamination of spills of blood or other potentially infectious materials (OPIM), implement the following procedures. Use protective gloves and other PPE (e.g., when sharps are involved use forceps to pick up sharps, and discard these items in a puncture-resistant container) appropriate for this task. Disinfect areas contaminated with blood spills using an EPA-registered tuberculocidal agent, a registered germicide on the EPA Lists D and E (i.e., products with specific label claims for HIV or HBV or freshly diluted hypochlorite solution. *Category II, IC.* ^{214, 215, 557, 1013} If sodium hypochlorite solutions are selected use a 1:100 dilution (e.g., 1:100 dilution of a 5.25-6.15% sodium hypochlorite provides 525-615 ppm available chlorine) to decontaminate nonporous surfaces after a small spill (e.g., <10 mL) of either blood or OPIM. If a spill involves large amounts (e.g., >10 mL) of blood or OPIM, or involves a culture spill in the laboratory, use a 1:10 dilution for the first application of hypochlorite solution before cleaning in order to reduce the risk of infection during the cleaning process in the event of a sharp injury. Follow this decontamination process with a terminal disinfection, using a 1:100 dilution of sodium hypochlorite. *Category IB, IC.* ^{63, 215, 557}
 - o. If the spill contains large amounts of blood or body fluids, clean the visible matter with disposable absorbent material, and discard the contaminated materials in appropriate, labeled containment. *Category II, IC.* ^{44, 214}
 - p. Use protective gloves and other PPE appropriate for this task. *Category II, IC.* ^{44, 214}

- q. In units with high rates of endemic *Clostridium difficile* infection or in an outbreak setting, use dilute solutions of 5.25%–6.15% sodium hypochlorite (e.g., 1:10 dilution of household bleach) for routine environmental disinfection. Currently, no products are EPA-registered specifically for inactivating *C. difficile* spores. *Category II.*²⁵⁷⁻²⁵⁹
- r. If chlorine solution is not prepared fresh daily, it can be stored at room temperature for up to 30 days in a capped, opaque plastic bottle with a 50% reduction in chlorine concentration after 30 days of storage (e.g., 1000 ppm chlorine [approximately a 1:50 dilution] at day 0 decreases to 500 ppm chlorine by day 30). *Category IB.*^{327, 1014}
- s. An EPA-registered sodium hypochlorite product is preferred, but if such products are not available, generic versions of sodium hypochlorite solutions (e.g., household chlorine bleach) can be used. *Category II.*⁴⁴

6. **Disinfectant Fogging**

- a. Do not perform disinfectant fogging for routine purposes in patient-care areas. *Category II.*^{23, 228}

7. **High-Level Disinfection of Endoscopes**

- a. To detect damaged endoscopes, test each flexible endoscope for leaks as part of each reprocessing cycle. Remove from clinical use any instrument that fails the leak test, and repair this instrument. *Category II.*^{113, 115, 116}
- b. Immediately after use, meticulously clean the endoscope with an enzymatic cleaner that is compatible with the endoscope. Cleaning is necessary before both automated and manual disinfection. *Category IA.*^{83, 101, 104-106, 113, 115, 116, 124, 126, 456, 465, 466, 471, 1015}
- c. Disconnect and disassemble endoscopic components (e.g., suction valves) as completely as possible and completely immerse all components in the enzymatic cleaner. Steam sterilize these components if they are heat stable. *Category IB.*^{115, 116, 139, 465, 466}
- d. Flush and brush all accessible channels to remove all organic (e.g., blood, tissue) and other residue. Clean the external surfaces and accessories of the devices by using a soft cloth or sponge or brushes. Continue brushing until no debris appears on the brush. *Category IA.*^{6, 17, 108, 113, 115, 116, 137, 145, 147, 725, 856, 903}
- e. Use cleaning brushes appropriate for the size of the endoscope channel or port (e.g., bristles should contact surfaces). Cleaning items (e.g., brushes, cloth) should be disposable or, if they are not disposable, they should be thoroughly cleaned and either high-level disinfected or sterilized after each use. *Category II.*^{113, 115, 116, 1016}
- f. Discard enzymatic cleaners (or detergents) after each use because they are not microbicidal and, therefore, will not retard microbial growth. *Category IB.*^{38, 113, 115, 116, 466}
- g. Process endoscopes (e.g., arthroscopes, cystoscope, laparoscopes) that pass through normally sterile tissues using a sterilization procedure before each use; if this is not feasible, provide at least high-level disinfection. High-level disinfection of arthroscopes, laparoscopes, and cystoscopes should be followed by a sterile water rinse. *Category IB.*^{1, 17, 31, 32, 35, 89, 90, 113, 554}
- h. Phase out endoscopes that are critical items (e.g., arthroscopes, laparoscopes) but cannot be steam sterilized. Replace these endoscopes with steam sterilizable instruments when feasible. *Category II.*
- i. Mechanically clean reusable accessories inserted into endoscopes (e.g., biopsy forceps or other cutting instruments) that break the mucosal barrier (e.g., ultrasonically clean biopsy forceps) and then sterilize these items between each patient. *Category IA.*^{1, 6, 8, 17, 108, 113, 115, 116, 138, 145, 147, 153, 278}
- j. Use ultrasonic cleaning of reusable endoscopic accessories to remove soil and organic material from hard-to-clean areas. *Category II.*^{116, 145, 148}
- k. Process endoscopes and accessories that contact mucous membranes as semicritical items, and use at least high-level disinfection after use on each patient. *Category IA.*^{1, 6, 8, 17, 108, 113, 115, 116, 129, 138, 145-148, 152-154, 278}
- l. Use an FDA-cleared sterilant or high-level disinfectant for sterilization or high-level disinfection (Table 1). *Category IA.*^{1, 6-8, 17, 85, 108, 113, 115, 116, 147}
- m. After cleaning, use formulations containing glutaraldehyde, glutaraldehyde with phenol/phenate,

- ortho-phthalaldehyde, hydrogen peroxide, and both hydrogen peroxide and peracetic acid to achieve high-level disinfection followed by rinsing and drying (see Table 1 for recommended concentrations). *Category IB.* ^{1, 6-8, 17, 38, 85, 108, 113, 145-148}
- n. Extend exposure times beyond the minimum effective time for disinfecting semicritical patient-care equipment cautiously and conservatively because extended exposure to a high-level disinfectant is more likely to damage delicate and intricate instruments such as flexible endoscopes. The exposure times vary among the Food and Drug Administration (FDA)-cleared high-level disinfectants (Table 2). *Category IB.* ^{17, 69, 73, 76, 78, 83}
 - o. Federal regulations are to follow the FDA-cleared label claim for high-level disinfectants. The FDA-cleared labels for high-level disinfection with >2% glutaraldehyde at 25°C range from 20-90 minutes, depending upon the product based on three tier testing which includes AOAC sporicidal tests, simulated use testing with mycobacteria and in-use testing. *Category IC.*
 - p. Several scientific studies and professional organizations support the efficacy of >2% glutaraldehyde for 20 minutes at 20°C; that efficacy assumes adequate cleaning prior to disinfection, whereas the FDA-cleared label claim incorporates an added margin of safety to accommodate possible lapses in cleaning practices. Facilities that have chosen to apply the 20 minute duration at 20°C have done so based on the IA recommendation in the July 2003 SHEA position paper, "Multi-society Guideline for Reprocessing Flexible Gastrointestinal Endoscopes" ^{12, 17, 19, 26, 27, 49, 55, 57, 58, 60, 73, 76, 79-81, 83-85, 93, 94, 104-106, 110, 111, 115-121, 124, 125, 233, 235, 236, 243, 265, 266, 609}
 - q. When using FDA-cleared high-level disinfectants, use manufacturers' recommended exposure conditions. Certain products may require a shorter exposure time (e.g., 0.55% ortho-phthalaldehyde for 12 minutes at 20°C, 7.35% hydrogen peroxide plus 0.23% peracetic acid for 15 minutes at 20°C) than glutaraldehyde at room temperature because of their rapid inactivation of mycobacteria or reduced exposure time because of increased mycobactericidal activity at elevated temperature (e.g., 2.5% glutaraldehyde at 5 minutes at 35°C). *Category IB.* ^{83, 100, 689, 693, 694, 700}
 - r. Select a disinfectant or chemical sterilant that is compatible with the device that is being reprocessed. Avoid using reprocessing chemicals on an endoscope if the endoscope manufacturer warns against using these chemicals because of functional damage (with or without cosmetic damage). *Category IB.* ^{69, 113, 116}
 - s. Completely immerse the endoscope in the high-level disinfectant, and ensure all channels are perfused. As soon as is feasible, phase out nonimmersible endoscopes. *Category IB.* ^{108, 113-116, 137, 725, 856, 882}
 - t. After high-level disinfection, rinse endoscopes and flush channels with sterile water, filtered water, or tapwater to prevent adverse effects on patients associated with disinfectant retained in the endoscope (e.g., disinfectant induced colitis). Follow this water rinse with a rinse with 70% - 90% ethyl or isopropyl alcohol. *Category IB.* ^{17, 31-35, 38, 39, 108, 113, 115, 116, 134, 145-148, 620-622, 624-630, 1017}
 - u. After flushing all channels with alcohol, purge the channels using forced air to reduce the likelihood of contamination of the endoscope by waterborne pathogens and to facilitate drying. *Category IB.* ^{39, 113, 115, 116, 145, 147}
 - v. Hang endoscopes in a vertical position to facilitate drying. *Category II.* ^{17, 108, 113, 115, 116, 145, 815}
 - w. Store endoscopes in a manner that will protect them from damage or contamination. *Category II.* ^{17, 108, 113, 115, 116, 145}
 - x. Sterilize or high-level disinfect both the water bottle used to provide intraprocedural flush solution and its connecting tube at least once daily. After sterilizing or high-level disinfecting the water bottle, fill it with sterile water. *Category IB.* ^{10, 31-35, 113, 116, 1017}
 - y. Maintain a log for each procedure and record the following: patient's name and medical record number (if available), procedure, date, endoscopist, system used to reprocess the endoscope (if more than one system could be used in the reprocessing area), and serial number or other identifier of the endoscope used. *Category II.* ^{108, 113, 115, 116}
 - z. Design facilities where endoscopes are used and disinfected to provide a safe environment for healthcare workers and patients. Use air-exchange equipment (e.g., the ventilation system, out-exhaust ducts) to minimize exposure of all persons to potentially toxic vapors (e.g.,

glutaraldehyde vapor). Do not exceed the allowable limits of the vapor concentration of the chemical sterilant or high-level disinfectant (e.g., those of ACGIH and OSHA). *Category IB, IC.* 116, 145, 318, 322, 577, 652

- aa. Routinely test the liquid sterilant/high-level disinfectant to ensure minimal effective concentration of the active ingredient. Check the solution each day of use (or more frequently) using the appropriate chemical indicator (e.g., glutaraldehyde chemical indicator to test minimal effective concentration of glutaraldehyde) and document the results of this testing. Discard the solution if the chemical indicator shows the concentration is less than the minimum effective concentration. Do not use the liquid sterilant/high-level disinfectant beyond the reuse-life recommended by the manufacturer (e.g., 14 days for *ortho*-phthalaldehyde). *Category IA.* 76, 108, 113, 115, 116, 608, 609
 - bb. Provide personnel assigned to reprocess endoscopes with device-specific reprocessing instructions to ensure proper cleaning and high-level disinfection or sterilization. Require competency testing on a regular basis (e.g., beginning of employment, annually) of all personnel who reprocess endoscopes. *Category IA.* 6-8, 108, 113, 115, 116, 145, 148, 155
 - cc. Educate all personnel who use chemicals about the possible biologic, chemical, and environmental hazards of performing procedures that require disinfectants. *Category IB, IC.* 116, 997, 998, 1018, 1019
 - dd. Make PPE (e.g., gloves, gowns, eyewear, face mask or shields, respiratory protection devices) available and use these items appropriately to protect workers from exposure to both chemicals and microorganisms (e.g., HBV). *Category IB, IC.* 115, 116, 214, 961, 997, 998, 1020, 1021
 - ee. If using an automated endoscope reprocessor (AER), place the endoscope in the reprocessor and attach all channel connectors according to the AER manufacturer's instructions to ensure exposure of all internal surfaces to the high-level disinfectant/chemical sterilant. *Category IB.* 7, 8, 115, 116, 155, 725, 903
 - ff. If using an AER, ensure the endoscope can be effectively reprocessed in the AER. Also, ensure any required manual cleaning/disinfecting steps are performed (e.g., elevator wire channel of duodenoscopes might not be effectively disinfected by most AERs). *Category IB.* 7, 8, 115, 116, 155, 725
 - gg. Review the FDA advisories and the scientific literature for reports of deficiencies that can lead to infection because design flaws and improper operation and practices have compromised the effectiveness of AERs. *Category II.* 7, 98, 133, 134, 155, 725
 - hh. Develop protocols to ensure that users can readily identify an endoscope that has been properly processed and is ready for patient use. *Category II.*
 - ii. Do not use the carrying case designed to transport clean and reprocessed endoscopes outside of the healthcare environment to store an endoscope or to transport the instrument within the healthcare environment. *Category II.*
 - jj. No recommendation is made about routinely performing microbiologic testing of either endoscopes or rinse water for quality assurance purposes. *Unresolved Issue.* 116, 164
 - kk. If environmental microbiologic testing is conducted, use standard microbiologic techniques. *Category II.* 23, 116, 157, 161, 167
 - ll. If a cluster of endoscopy-related infections occurs, investigate potential routes of transmission (e.g., person-to-person, common source) and reservoirs. *Category IA.* 8, 1022
 - mm. Report outbreaks of endoscope-related infections to persons responsible for institutional infection control and risk management and to FDA. *Category IB.* 6, 7, 113, 116, 1023 Notify the local and the state health departments, CDC, and the manufacturer(s). *Category II.*
 - nn. No recommendation is made regarding the reprocessing of an endoscope again immediately before use if that endoscope has been processed after use according to the recommendations in this guideline. *Unresolved issue.* 157
 - oo. Compare the reprocessing instructions provided by both the endoscope's and the AER's manufacturer's instructions and resolve any conflicting recommendations. *Category IB.* 116, 155
- 8. Management of Equipment and Surfaces in Dentistry**
- a. Dental instruments that penetrate soft tissue or bone (e.g., extraction forceps, scalpel blades, bone chisels, periodontal scalers, and surgical burs) are classified as critical and should be

sterilized after each use or discarded. In addition, after each use, sterilize dental instruments that are not intended to penetrate oral soft tissue or bone (e.g., amalgam condensers, air-water syringes) but that might contact oral tissues and are heat-tolerant, although classified as semicritical. Clean and, at a minimum, high-level disinfect heat-sensitive semicritical items.

Category IA. ^{43, 209-211}

- b. Noncritical clinical contact surfaces, such as uncovered operatory surfaces (e.g., countertops, switches, light handles), should be barrier-protected or disinfected between patients with an intermediate-disinfectant (i.e., EPA-registered hospital disinfectant with a tuberculocidal claim) or low-level disinfectant (i.e., EPA-registered hospital disinfectant with HIV and HBV claim).

Category IB. ^{43, 209-211}

- c. Barrier protective coverings can be used for noncritical clinical contact surfaces that are touched frequently with gloved hands during the delivery of patient care, that are likely to become contaminated with blood or body substances, or that are difficult to clean. Change these coverings when they are visibly soiled, when they become damaged, and on a routine basis (e.g., between patients). Disinfect protected surfaces at the end of the day or if visibly soiled. *Category II.* ^{43, 210}

9. Processing Patient-Care Equipment Contaminated with Bloodborne Pathogens (HBV, Hepatitis C Virus, HIV), Antibiotic-Resistant Bacteria (e.g., Vancomycin-Resistant Enterococci, Methicillin-Resistant Staphylococcus aureus, Multidrug Resistant Tuberculosis), or Emerging Pathogens (e.g., Cryptosporidium, Helicobacter pylori, Escherichia coli O157:H7, Clostridium difficile, Mycobacterium tuberculosis, Severe Acute Respiratory Syndrome Coronavirus), or Bioterrorist Agents

- a. Use standard sterilization and disinfection procedures for patient-care equipment (as recommended in this guideline), because these procedures are adequate to sterilize or disinfect instruments or devices contaminated with blood or other body fluids from persons infected with bloodborne pathogens or emerging pathogens, with the exception of prions. No changes in these procedures for cleaning, disinfecting, or sterilizing are necessary for removing bloodborne and emerging pathogens other than prions. *Category IA.* ^{22, 53, 60-62, 73, 79-81, 105, 118-121, 125, 126, 221, 224-234, 236, 244, 265, 266, 271-273, 279, 282, 283, 354-357, 666}

10. Disinfection Strategies for Other Semicritical Devices

- a. Even if probe covers have been used, clean and high-level disinfect other semicritical devices such as rectal probes, vaginal probes, and cryosurgical probes with a product that is not toxic to staff, patients, probes, and retrieved germ cells (if applicable). Use a high-level disinfectant at the FDA-cleared exposure time. (See Recommendations 7o and 11e for exceptions.) *Category IB.* ^{6-8, 17, 69}
- b. When probe covers are available, use a probe cover or condom to reduce the level of microbial contamination. *Category II.* ¹⁹⁷⁻²⁰¹ Do not use a lower category of disinfection or cease to follow the appropriate disinfectant recommendations when using probe covers because these sheaths and condoms can fail. *Category IB.* ¹⁹⁷⁻²⁰¹
- c. After high-level disinfection, rinse all items. Use sterile water, filtered water or tapwater followed by an alcohol rinse for semicritical equipment that will have contact with mucous membranes of the upper respiratory tract (e.g., nose, pharynx, esophagus). *Category II.* ^{10, 31-35, 1017}
- d. There is no recommendation to use sterile or filtered water rather than tapwater for rinsing semicritical equipment that contact the mucous membranes of the rectum (e.g., rectal probes, anoscope) or vagina (e.g., vaginal probes). *Unresolved issue.* ¹¹
- e. Wipe clean tonometer tips and then disinfect them by immersing for 5-10 minutes in either 5000 ppm chlorine or 70% ethyl alcohol. None of these listed disinfectant products are FDA-cleared high-level disinfectants. *Category II.* ^{49, 95, 185, 188, 293}

11. Disinfection by Healthcare Personnel in Ambulatory Care and Home Care

- a. Follow the same classification scheme described above (i.e., that critical devices require sterilization, semicritical devices require high-level disinfection, and noncritical equipment

requires low-level disinfection) in the ambulatory-care (outpatient medical/surgical facilities) setting because risk for infection in this setting is similar to that in the hospital setting (see Table 1). *Category IB.*^{6-8, 17, 330}

- b. When performing care in the home, clean and disinfect reusable objects that touch mucous membranes (e.g., tracheostomy tubes) by immersing these objects in a 1:50 dilution of 5.25%-6.15% sodium hypochlorite (household bleach) (3 minutes), 70% isopropyl alcohol (5 minutes), or 3% hydrogen peroxide (30 minutes) because the home environment is, in most instances, safer than either hospital or ambulatory care settings because person-to-person transmission is less likely. *Category II.*^{327, 328, 330, 331}
- c. Clean noncritical items that would not be shared between patients (e.g., crutches, blood pressure cuffs) in the home setting with a detergent or commercial household disinfectant. *Category II.*^{53, 330}

12. **Microbial Contamination of Disinfectants**

- a. Institute the following control measures to reduce the occurrence of contaminated disinfectants: 1) prepare the disinfectant correctly to achieve the manufacturer's recommended use-dilution; and 2) prevent common sources of extrinsic contamination of germicides (e.g., container contamination or surface contamination of the healthcare environment where the germicide are prepared and/or used). *Category IB.*^{404, 406, 1024}

13. **Flash Sterilization**

- a. Do not flash sterilize implanted surgical devices unless doing so is unavoidable. *Category IB.*^{849, 850}
- b. Do not use flash sterilization for convenience, as an alternative to purchasing additional instrument sets, or to save time. *Category II.*^{817, 962}
- c. When using flash sterilization, make sure the following parameters are met: 1) clean the item before placing it in the sterilizing container (that are FDA cleared for use with flash sterilization) or tray; 2) prevent exogenous contamination of the item during transport from the sterilizer to the patient; and 3) monitor sterilizer function with mechanical, chemical, and biologic monitors. *Category IB.*^{812, 819, 846, 847, 962}
- d. Do not use packaging materials and containers in flash sterilization cycles unless the sterilizer and the packaging material/container are designed for this use. *Category IB.*^{812, 819, 1025}
- e. When necessary, use flash sterilization for patient-care items that will be used immediately (e.g., to reprocess an inadvertently dropped instrument). *Category IB.*^{812, 817, 819, 845}
- f. When necessary, use flash sterilization for processing patient-care items that cannot be packaged, sterilized, and stored before use. *Category IB.*^{812, 819}

14. **Methods of Sterilization**

- a. Steam is the preferred method for sterilizing critical medical and surgical instruments that are not damaged by heat, steam, pressure, or moisture. *Category IA.*^{181, 271, 425, 426, 827, 841, 1026, 1027}
- b. Cool steam- or heat-sterilized items before they are handled or used in the operative setting. *Category IB.*⁸⁵⁰
- c. Follow the sterilization times, temperatures, and other operating parameters (e.g., gas concentration, humidity) recommended by the manufacturers of the instruments, the sterilizer, and the container or wrap used, and that are consistent with guidelines published by government agencies and professional organizations. *Category IB.*^{811-814, 819, 825, 827, 841, 1026-1028}
- d. Use low-temperature sterilization technologies (e.g., EtO, hydrogen peroxide gas plasma) for reprocessing critical patient-care equipment that is heat or moisture sensitive. *Category IA.*^{469, 721, 825, 856, 858, 878, 879, 881, 882, 890, 891, 1027}
- e. Completely aerate surgical and medical items that have been sterilized in the EtO sterilizer (e.g., polyvinylchloride tubing requires 12 hours at 50°C, 8 hours at 60°C) before using these items in patient care. *Category IB.*⁸¹⁴
- f. Sterilization using the peracetic acid immersion system can be used to sterilize heat-sensitive

immersible medical and surgical items. *Category IB.*^{90, 717-719, 721-724}

- g. Critical items that have been sterilized by the peracetic acid immersion process must be used immediately (i.e., items are not completely protected from contamination, making long-term storage unacceptable). *Category II.*^{817, 825}
- h. Dry-heat sterilization (e.g., 340°F for 60 minutes) can be used to sterilize items (e.g., powders, oils) that can sustain high temperatures. *Category IB.*^{815, 827}
- i. Comply with the sterilizer manufacturer's instructions regarding the sterilizer cycle parameters (e.g., time, temperature, concentration). *Category IB.*^{155, 725, 811-814, 819}
- j. Because narrow-lumen devices provide a challenge to all low-temperature sterilization technologies and direct contact is necessary for the sterilant to be effective, ensure that the sterilant has direct contact with contaminated surfaces (e.g., scopes processed in peracetic acid must be connected to channel irrigators). *Category IB.*^{137, 725, 825, 856, 890, 891, 1029}

15. **Packaging**

- a. Ensure that packaging materials are compatible with the sterilization process and have received FDA 510[k] clearance. *Category IB.*^{811-814, 819, 966}
- b. Ensure that packaging is sufficiently strong to resist punctures and tears to provide a barrier to microorganisms and moisture. *Category IB.*^{454, 811-814, 819, 966}

16. **Monitoring of Sterilizers**

- a. Use mechanical, chemical, and biologic monitors to ensure the effectiveness of the sterilization process. *Category IB.*^{811-815, 819, 846, 847, 975-977}
- b. Monitor each load with mechanical (e.g., time, temperature, pressure) and chemical (internal and external) indicators. If the internal chemical indicator is visible, an external indicator is not needed. *Category II.*^{811-815, 819, 846, 847, 975-977, 980}
- c. Do not use processed items if the mechanical (e.g., time, temperature, pressure) or chemical (internal and/or external) indicators suggest inadequate processing. *Category IB.*^{811-814, 819}
- d. Use biologic indicators to monitor the effectiveness of sterilizers at least weekly with an FDA-cleared commercial preparation of spores (e.g., *Geobacillus stearothermophilus* for steam) intended specifically for the type and cycle parameters of the sterilizer. *Category IB.*^{1, 811, 813-815, 819, 846, 847, 976, 977}
- e. After a single positive biologic indicator used with a method other than steam sterilization, treat as nonsterile all items that have been processed in that sterilizer, dating from the sterilization cycle having the last negative biologic indicator to the next cycle showing satisfactory biologic indicator results. These nonsterile items should be retrieved if possible and reprocessed. *Category II.*¹
- f. After a positive biologic indicator with steam sterilization, objects other than implantable objects do not need to be recalled because of a single positive spore test unless the sterilizer or the sterilization procedure is defective as determined by maintenance personnel or inappropriate cycle settings. If additional spore tests remain positive, consider the items nonsterile and recall and reprocess the items from the implicated load(s). *Category II.*¹
- g. Use biologic indicators for every load containing implantable items and quarantine items, whenever possible, until the biologic indicator is negative. *Category IB.*^{811-814, 819}

17. **Load Configuration.**

- a. Place items correctly and loosely into the basket, shelf, or cart of the sterilizer so as not to impede the penetration of the sterilant. *Category IB.*^{445, 454, 811, 813, 819, 836}

18. **Storage of Sterile Items**

- a. Ensure the sterile storage area is a well-ventilated area that provides protection against dust, moisture, insects, and temperature and humidity extremes. *Category II.*^{454, 819, 836, 969}
- b. Store sterile items so the packaging is not compromised (e.g., punctured, bent). *Category II.*^{454, 816, 819, 968, 969, 1030}

- c. Label sterilized items with a load number that indicates the sterilizer used, the cycle or load number, the date of sterilization, and, if applicable, the expiration date. *Category IB.*^{811, 812, 814, 816, 819}
- d. The shelf life of a packaged sterile item depends on the quality of the wrapper, the storage conditions, the conditions during transport, the amount of handling, and other events (moisture) that compromise the integrity of the package. If event-related storage of sterile items is used, then packaged sterile items can be used indefinitely unless the packaging is compromised (see f and g below). *Category IB.*^{816, 819, 836, 968, 973, 1030, 1031}
- e. Evaluate packages before use for loss of integrity (e.g., torn, wet, punctured). The pack can be used unless the integrity of the packaging is compromised. *Category II.*^{819, 968}
- f. If the integrity of the packaging is compromised (e.g., torn, wet, or punctured), repack and reprocess the pack before use. *Category II.*^{819, 1032}
- g. If time-related storage of sterile items is used, label the pack at the time of sterilization with an expiration date. Once this date expires, reprocess the pack. *Category II.*^{819, 968}

19. Quality Control

- a. Provide comprehensive and intensive training for all staff assigned to reprocess semicritical and critical medical/surgical instruments to ensure they understand the importance of reprocessing these instruments. To achieve and maintain competency, train each member of the staff that reprocesses semicritical and/or critical instruments as follows: 1) provide hands-on training according to the institutional policy for reprocessing critical and semicritical devices; 2) supervise all work until competency is documented for each reprocessing task; 3) conduct competency testing at beginning of employment and regularly thereafter (e.g., annually); and 4) review the written reprocessing instructions regularly to ensure they comply with the scientific literature and the manufacturers' instructions. *Category IB.*^{6-8, 108, 114, 129, 155, 725, 813, 819}
- b. Compare the reprocessing instructions (e.g., for the appropriate use of endoscope connectors, the capping/noncapping of specific lumens) provided by the instrument manufacturer and the sterilizer manufacturer and resolve any conflicting recommendations by communicating with both manufacturers. *Category IB.*^{155, 725}
- c. Conduct infection control rounds periodically (e.g., annually) in high-risk reprocessing areas (e.g., the Gastroenterology Clinic, Central Processing); ensure reprocessing instructions are current and accurate and are correctly implemented. Document all deviations from policy. All stakeholders should identify what corrective actions will be implemented. *Category IB.*^{6-8, 129}
- d. Include the following in a quality control program for sterilized items: a sterilizer maintenance contract with records of service; a system of process monitoring; air-removal testing for prevacuum steam sterilizers; visual inspection of packaging materials; and traceability of load contents. *Category II*^{811-814, 819}
- e. For each sterilization cycle, record the type of sterilizer and cycle used; the load identification number; the load contents; the exposure parameters (e.g., time and temperature); the operator's name or initials; and the results of mechanical, chemical, and biological monitoring. *Category II*^{811-814, 819}
- f. Retain sterilization records (mechanical, chemical, and biological) for a time period that complies with standards (e.g., 3 years), statutes of limitations, and state and federal regulations. *Category II, IC.*¹⁰³³
- g. Prepare and package items to be sterilized so that sterility can be achieved and maintained to the point of use. Consult the Association for the Advancement of Medical Instrumentation or the manufacturers of surgical instruments, sterilizers, and container systems for guidelines for the density of wrapped packages. *Category II.*^{811-814, 819}
- h. Periodically review policies and procedures for sterilization. *Category II.*¹⁰³³
- i. Perform preventive maintenance on sterilizers by qualified personnel who are guided by the manufacturer's instruction. *Category II.*^{811-814, 819}

20. Reuse of Single-Use Medical Devices

- a. Adhere to the FDA enforcement document for single-use devices reprocessed by hospitals. FDA considers the hospital that reprocesses a single-use device as the manufacturer of the device and regulates the hospital using the same standards by which it regulates the original equipment manufacturer. *Category II, IC.*⁹⁹⁵

PERFORMANCE INDICATORS

1. Monitor adherence to high-level disinfection and/or sterilization guidelines for endoscopes on a regular basis. This monitoring should include ensuring the proper training of persons performing reprocessing and their adherence to all endoscope reprocessing steps, as demonstrated by competency testing at commencement of employment and annually.
2. Develop a mechanism for the occupational health service to report all adverse health events potentially resulting from exposure to disinfectants and sterilants; review such exposures; and implement engineering, work practice, and PPE to prevent future exposures.
3. Monitor possible sterilization failures that resulted in instrument recall. Assess whether additional training of personnel or equipment maintenance is required.

ACKNOWLEDGEMENTS

The authors gratefully acknowledge Eva P. Clontz, M.S., for her assistance in referencing this guideline. The Healthcare Infection Control Practices Advisory Committee thanks the following experts for reviewing a draft of this guideline: Martin S. Favero, Ph.D., Syed A. Sattar, Ph.D., A. Denver Russell, D.Sc., and Martin Exner, M.D. The opinions of the reviewers might not be reflected in all the recommendations contained in this document.

GLOSSARY

Action level: concentration of a regulated substance (e.g., ethylene oxide, formaldehyde) within the employee breathing zone, above which OSHA requirements apply.

Activation of a sterilant: process of mixing the contents of a chemical sterilant that come in two containers (small vial with the activator solution; container of the chemical) Keeping the two chemicals separate until use extends the shelf life of the chemicals.

Aeration: method by which ethylene oxide (EtO) is removed from EtO-sterilized items by warm air circulation in an enclosed cabinet specifically designed for this purpose.

Antimicrobial agent: any agent that kills or suppresses the growth of microorganisms.

Antiseptic: substance that prevents or arrests the growth or action of microorganisms by inhibiting their activity or by destroying them. The term is used especially for preparations applied topically to living tissue.

Asepsis: prevention of contact with microorganisms.

Autoclave: device that sterilizes instruments or other objects using steam under pressure. The length of time required for sterilization depends on temperature, vacuum, and pressure.

Bacterial count: method of estimating the number of bacteria per unit sample. The term also refers to the estimated number of bacteria per unit sample, usually expressed as number of colony-forming units.

Bactericide: agent that kills bacteria.

Bioburden: number and types of viable microorganisms with which an item is contaminated; also called *bioload* or *microbial load*.

Biofilm: accumulated mass of bacteria and extracellular material that is tightly adhered to a surface and cannot be easily removed.

Biologic indicator: device for monitoring the sterilization process. The device consists of a standardized, viable population of microorganisms (usually bacterial spores) known to be resistant to the sterilization process being monitored. Biologic indicators are intended to demonstrate whether conditions were adequate to achieve sterilization. A negative biologic indicator does not prove that all items in the load are sterile or that they were all exposed to adequate sterilization conditions.

Bleach: Household bleach (5.25% or 6.00%–6.15% sodium hypochlorite depending on manufacturer) usually diluted in water at 1:10 or 1:100. Approximate dilutions are 1.5 cups of bleach in a gallon of water for a 1:10 dilution (~6,000 ppm) and 0.25 cup of bleach in a gallon of water for a 1:100 dilution (~600 ppm). Sodium hypochlorite products that make pesticidal claims, such as sanitization or disinfection, must be registered by EPA and be labeled with an EPA Registration Number.

Bleach Solution	Dilution	Chlorine (ppm)
5.25-6.15%	None	52,500-61,500
	1:10	5,250-6,150
	1:100	525-615
	1:1000	53-62

Bowie-Dick test: diagnostic test of a sterilizer's ability to remove air from the chamber of a prevacuum steam sterilizer. The air-removal or Bowie-Dick test is not a test for sterilization.

Ceiling limit: concentration of an airborne chemical contaminant that should not be exceeded during any part of the workday. If instantaneous monitoring is not feasible, the ceiling must be assessed as a 15-minute time-weighted average exposure.

Centigrade or Celsius: a temperature scale (0°C = freezing point of water; 100°C = boiling point of water at sea level). Equivalents mentioned in the guideline are as follows: $20^{\circ}\text{C} = 68^{\circ}\text{F}$; $25^{\circ}\text{C} = 77^{\circ}\text{F}$; $121^{\circ}\text{C} = 250^{\circ}\text{F}$; $132^{\circ}\text{C} = 270^{\circ}\text{F}$; $134^{\circ}\text{C} = 273^{\circ}\text{F}$. For other temperatures the formula is: $F^{\circ} = (C^{\circ} \times 9/5) + 32$ or $C^{\circ} = (F^{\circ} - 32) \times 5/9$.

Central processing or Central service department: the department within a health-care facility that processes, issues, and controls professional supplies and equipment, both sterile and nonsterile, for some or all patient-care areas of the facility.

Challenge test pack: pack used in installation, qualification, and ongoing quality assurance testing of health-care facility sterilizers.

Chemical indicator: device for monitoring a sterilization process. The device is designed to respond with a characteristic chemical or physical change to one or more of the physical conditions within the sterilizing chamber. Chemical indicators are intended to detect potential sterilization failures that could result from incorrect packaging, incorrect loading of the sterilizer, or malfunctions of the sterilizer. The "pass" response of a chemical indicator does not prove the item accompanied by the indicator is necessarily sterile. The Association for the Advancement of Medical Instrumentation has defined five classes of chemical indicators: Class 1 (process indicator); Class 2 (Bowie-Dick test indicator); Class 3 (single-parameter indicator); Class 4 (multi-parameter indicator); and Class 5 (integrating indicator).

Contact time: time a disinfectant is in direct contact with the surface or item to be disinfected. For surface disinfection, this period is framed by the application to the surface until complete drying has occurred.

Container system, rigid container: sterilization containment device designed to hold medical devices for sterilization, storage, transportation, and aseptic presentation of contents.

Contaminated: state of having actual or potential contact with microorganisms. As used in health care, the term generally refers to the presence of microorganisms that could produce disease or infection.

Control, positive: biologic indicator, from the same lot as a test biologic indicator, that is left unexposed to the sterilization cycle and then incubated to verify the viability of the test biologic indicator.

Cleaning: removal, usually with detergent and water or enzyme cleaner and water, of adherent visible soil, blood, protein substances, microorganisms and other debris from the surfaces, crevices, serrations, joints, and lumens of instruments, devices, and equipment by a manual or mechanical process that prepares the items for safe handling and/or further decontamination.

Culture: growth of microorganisms in or on a nutrient medium; to grow microorganisms in or on such a medium.

Culture medium: substance or preparation used to grow and cultivate microorganisms.

Cup: 8 fluid ounces.

Decontamination: according to OSHA, “the use of physical or chemical means to remove, inactivate, or destroy bloodborne pathogens on a surface or item to the point where they are no longer capable of transmitting infectious particles and the surface or item is rendered safe for handling, use, or disposal” [29 CFR 1910.1030]. In health-care facilities, the term generally refers to all pathogenic organisms.

Decontamination area: area of a health-care facility designated for collection, retention, and cleaning of soiled and/or contaminated items.

Detergent: cleaning agent that makes no antimicrobial claims on the label. They comprise a hydrophilic component and a lipophilic component and can be divided into four types: anionic, cationic, amphoteric, and non-ionic detergents.

Disinfectant: usually a chemical agent (but sometimes a physical agent) that destroys disease-causing pathogens or other harmful microorganisms but might not kill bacterial spores. It refers to substances applied to inanimate objects. EPA groups disinfectants by product label claims of “limited,” “general,” or “hospital” disinfection.

Disinfection: thermal or chemical destruction of pathogenic and other types of microorganisms. Disinfection is less lethal than sterilization because it destroys most recognized pathogenic microorganisms but not necessarily all microbial forms (e.g., bacterial spores).

D value: time or radiation dose required to inactivate 90% of a population of the test microorganism under stated exposure conditions.

Endoscope: an instrument that allows examination and treatment of the interior of the body canals and hollow organs.

Enzyme cleaner: a solution used before disinfecting instruments to improve removal of organic material (e.g., proteases to assist in removing protein).

EPA Registration Number or EPA Reg. No.: a hyphenated, two- or three-part number assigned by EPA to identify each germicidal product registered within the United States. The first number is the company identification number, the second is the specific product number, and the third (when present) is the company identification number for a supplemental registrant.

Exposure time: period in a sterilization process during which items are exposed to the sterilant at the specified sterilization parameters. For example, in a steam sterilization process, exposure time is the period during which items are exposed to saturated steam at the specified temperature.

Flash sterilization: process designed for the steam sterilization of unwrapped patient-care items for immediate use (or placed in a specially designed, covered, rigid container to allow for rapid penetration of steam).

Fungicide: agent that destroys fungi (including yeasts) and/or fungal spores pathogenic to humans or other animals in the inanimate environment.

General disinfectant: EPA-registered disinfectant labeled for use against both gram-negative and gram-positive bacteria. Efficacy is demonstrated against both *Salmonella choleraesuis* and *Staphylococcus aureus*. Also called *broad-spectrum disinfectant*.

Germicide: agent that destroys microorganisms, especially pathogenic organisms.

Germicidal detergent: detergent that also is EPA-registered as a disinfectant.

High-level disinfectant: agent capable of killing bacterial spores when used in sufficient concentration under suitable conditions. It therefore is expected to kill all other microorganisms.

Hospital disinfectant: disinfectant registered for use in hospitals, clinics, dental offices, and any other medical-related facility. Efficacy is demonstrated against *Salmonella choleraesuis*, *Staphylococcus aureus*, and *Pseudomonas aeruginosa*. EPA has registered approximately 1,200 hospital disinfectants.

Huck towel: all-cotton surgical towel with a honey-comb weave; both warp and fill yarns are tightly twisted. Huck towels can be used to prepare biologic indicator challenge test packs.

Implantable device: according to FDA, “device that is placed into a surgically or naturally formed cavity of the human body if it is intended to remain there for a period of 30 days or more” [21 CFR 812.3(d)].

Inanimate surface: nonliving surface (e.g., floors, walls, furniture).

Incubator: apparatus for maintaining a constant and suitable temperature for the growth and cultivation of microorganisms.

Infectious microorganisms: microorganisms capable of producing disease in appropriate hosts.

Inorganic and organic load: naturally occurring or artificially placed inorganic (e.g., metal salts) or organic (e.g., proteins) contaminants on a medical device before exposure to a microbicidal process.

Intermediate-level disinfectant: agent that destroys all vegetative bacteria, including tubercle bacilli, lipid and some nonlipid viruses, and fungi, but not bacterial spores.

Limited disinfectant: disinfectant registered for use against a specific major group of organisms (gram-negative or gram-positive bacteria). Efficacy has been demonstrated in laboratory tests against either *Salmonella choleraesuis* or *Staphylococcus aureus* bacteria.

Lipid virus: virus surrounded by an envelope of lipoprotein in addition to the usual core of nucleic acid surrounded by a coat of protein. This type of virus (e.g., HIV) is generally easily inactivated by many types of disinfectants. Also called *enveloped* or *lipophilic virus*.

Low-level disinfectant: agent that destroys all vegetative bacteria (except tubercle bacilli), lipid viruses, some nonlipid viruses, and some fungi, but not bacterial spores.

Mechanical indicator: devices that monitor the sterilization process (e.g., graphs, gauges, printouts).

Medical device: instrument, apparatus, material, or other article, whether used alone or in combination, including software necessary for its application, intended by the manufacturer to be used for human beings for

- diagnosis, prevention, monitoring treatment, or alleviation of disease;
- diagnosis, monitoring, treatment, or alleviation of or compensation for an injury or handicap;
- investigation, replacement, or modification of the anatomy or of a physiologic process; or
- control of conception

and that does not achieve its primary intended action in or on the human body by pharmacologic, immunologic, or metabolic means but might be assisted in its function by such means.

Microbicide: any substance or mixture of substances that effectively kills microorganisms.

Microorganisms: animals or plants of microscopic size. As used in health care, generally refers to bacteria, fungi, viruses, and bacterial spores.

Minimum effective concentration (MEC): the minimum concentration of a liquid chemical germicide needed to achieve the claimed microbicidal activity as determined by dose-response testing. Sometimes used interchangeably with *minimum recommended concentration*.

Muslin: loosely woven (by convention, 140 threads per square inch), 100% cotton cloth. Formerly used as a wrap for sterile packs or a surgical drape. Fabric wraps used currently consist of a cotton-polyester blend.

Mycobacteria: bacteria with a thick, waxy coat that makes them more resistant to chemical germicides than other types of vegetative bacteria.

Nonlipid viruses: generally considered more resistant to inactivation than lipid viruses. Also called nonenveloped or hydrophilic viruses.

One-step disinfection process: simultaneous cleaning and disinfection of a noncritical surface or item.

Pasteurization: process developed by Louis Pasteur of heating milk, wine, or other liquids to 65–77°C (or the equivalent) for approximately 30 minutes to kill or markedly reduce the number of pathogenic and spoilage organisms other than bacterial spores.

Parametric release: declaration that a product is sterile on the basis of physical and/or chemical process data rather than on sample testing or biologic indicator results.

Penicylinder: carriers inoculated with the test bacteria for in vitro tests of germicides. Can be constructed of stainless steel, porcelain, glass, or other materials and are approximately 8 x 10 mm in diameter.

Permissible exposure limit (PEL): time-weighted average maximum concentration of an air contaminant to which a worker can be exposed, according to OSHA standards. Usually calculated over 8 hours, with exposure considered over a 40-hour work week.

Personal protective equipment (PPE): specialized clothing or equipment worn by an employee for protection against a hazard. General work clothes (e.g., uniforms, pants, shirts) not intended to function as protection against a hazard are not considered to be PPE.

Parts per million (ppm): common measurement for concentrations by volume of trace contaminant gases in the air (or chemicals in a liquid); 1 volume of contaminated gas per 1 million volumes of contaminated air or 1¢ in \$10,000 both equal 1 ppm. Parts per million = µg/mL or mg/L.

Prions: transmissible pathogenic agents that cause a variety of neurodegenerative diseases of humans and animals, including sheep and goats, bovine spongiform encephalopathy in cattle, and Creutzfeldt-Jakob disease in humans. They are unlike any other infectious pathogens because they are composed of an abnormal conformational isoform of a normal cellular protein, the prion protein (PrP). Prions are extremely resistant to inactivation by sterilization processes and disinfecting agents.

Process challenge device (PCD): item designed to simulate product to be sterilized and to constitute a defined challenge to the sterilization process and used to assess the effective performance of the process. A PCD is a challenge test pack or test tray that contains a biologic indicator, a Class 5 integrating indicator, or an enzyme-only indicator.

QUAT: abbreviation for *quaternary ammonium compound*, a surface-active, water-soluble disinfecting

substance that has four carbon atoms linked to a nitrogen atom through covalent bonds.

Recommended exposure limit (REL): occupational exposure limit recommended by NIOSH as being protective of worker health and safety over a working lifetime. Frequently expressed as a 40-hour time-weighted-average exposure for up to 10 hours per day during a 40-work week.

Reprocess: method to ensure proper disinfection or sterilization; can include: cleaning, inspection, wrapping, sterilizing, and storing.

Sanitizer: agent that reduces the number of bacterial contaminants to safe levels as judged by public health requirements. Commonly used with substances applied to inanimate objects. According to the protocol for the official sanitizer test, a sanitizer is a chemical that kills 99.999% of the specific test bacteria in 30 seconds under the conditions of the test.

Shelf life: length of time an undiluted or use dilution of a product can remain active and effective. Also refers to the length of time a sterilized product (e.g., sterile instrument set) is expected to remain sterile.

Spaulding classification: strategy for reprocessing contaminated medical devices. The system classifies a medical device as critical, semicritical, or noncritical on the basis of risk to patient safety from contamination on a device. The system also established three levels of germicidal activity (sterilization, high-level disinfection, and low-level disinfection) for strategies with the three classes of medical devices (critical, semicritical, and noncritical).

Spore: relatively water-poor round or elliptical resting cell consisting of condensed cytoplasm and nucleus surrounded by an impervious cell wall or coat. Spores are relatively resistant to disinfectant and sterilant activity and drying conditions (specifically in the genera *Bacillus* and *Clostridium*).

Spore strip: paper strip impregnated with a known population of spores that meets the definition of biological indicators.

Steam quality: steam characteristic reflecting the dryness fraction (weight of dry steam in a mixture of dry saturated steam and entrained water) and the level of noncondensable gas (air or other gas that will not condense under the conditions of temperature and pressure used during the sterilization process). The dryness fraction (i.e., the proportion of completely dry steam in the steam being considered) should not fall below 97%.

Steam sterilization: sterilization process that uses saturated steam under pressure for a specified exposure time and at a specified temperature, as the sterilizing agent.

Steam sterilization, dynamic air removal type: one of two types of sterilization cycles in which air is removed from the chamber and the load by a series of pressure and vacuum excursions (prevacuum cycle) or by a series of steam flushes and pressure pulses above atmospheric pressure (steam-flush-pressure-pulse cycle).

Sterile or Sterility: state of being free from all living microorganisms. In practice, usually described as a probability function, e.g., as the probability of a microorganism surviving sterilization being one in one million.

Sterility assurance level (SAL): probability of a viable microorganism being present on a product unit after sterilization. Usually expressed as 10^{-6} ; a SAL of 10^{-6} means $\leq 1/1$ million chance that a single viable microorganism is present on a sterilized item. A SAL of 10^{-6} generally is accepted as appropriate for items intended to contact compromised tissue (i.e., tissue that has lost the integrity of the natural body barriers). The sterilizer manufacturer is responsible for ensuring the sterilizer can achieve the desired SAL. The

user is responsible for monitoring the performance of the sterilizer to ensure it is operating in conformance to the manufacturer's recommendations.

Sterilization: validated process used to render a product free of all forms of viable microorganisms. In a sterilization process, the presence of microorganisms on any individual item can be expressed in terms of probability. Although this probability can be reduced to a very low number, it can never be reduced to zero.

Sterilization area: area of a health-care facility designed to house sterilization equipment, such as steam ethylene oxide, hydrogen peroxide gas plasma, or ozone sterilizers.

Sterilizer: apparatus used to sterilize medical devices, equipment, or supplies by direct exposure to the sterilizing agent.

Sterilizer, gravity-displacement type: type of steam sterilizer in which incoming steam displaces residual air through a port or drain in or near the bottom (usually) of the sterilizer chamber. Typical operating temperatures are 121–123°C (250–254°F) and 132–135°C (270–275°F).

Sterilizer, prevacuum type: type of steam sterilizer that depends on one or more pressure and vacuum excursions at the beginning of the cycle to remove air. This method of operation results in shorter cycle times for wrapped items because of the rapid removal of air from the chamber and the load by the vacuum system and because of the usually higher operating temperature (132–135°C [270–275°F]; 141–144°C [285–291°F]). This type of sterilizer generally provides for shorter exposure time and accelerated drying of fabric loads by pulling a further vacuum at the end of the sterilizing cycle.

Sterilizer, steam-flush pressure-pulse type: type of sterilizer in which a repeated sequence consisting of a steam flush and a pressure pulse removes air from the sterilizing chamber and processed materials using steam at above atmospheric pressure (no vacuum is required). Like a prevacuum sterilizer, a steam-flush pressure-pulse sterilizer rapidly removes air from the sterilizing chamber and wrapped items; however, the system is not susceptible to air leaks because air is removed with the sterilizing chamber pressure at above atmospheric pressure. Typical operating temperatures are 121–123°C (250–254°F), 132–135°C (270–275°F), and 141–144°C (285–291°F).

Surfactant: agent that reduces the surface tension of water or the tension at the interface between water and another liquid; a wetting agent found in many sterilants and disinfectants.

Tabletop steam sterilizer: a compact gravity-displacement steam sterilizer that has a chamber volume of not more than 2 cubic feet and that generates its own steam when distilled or deionized water is added.

Time-weighted average (TWA): an average of all the concentrations of a chemical to which a worker has been exposed during a specific sampling time, reported as an average over the sampling time. For example, the permissible exposure limit for ethylene oxide is 1 ppm as an 8-hour TWA. Exposures above the ppm limit are permitted if they are compensated for by equal or longer exposures below the limit during the 8-hour workday as long as they do not exceed the ceiling limit; short-term exposure limit; or, in the case of ethylene oxide, excursion limit of 5 ppm averaged over a 15-minute sampling period.

Tuberculocide: an EPA-classified hospital disinfectant that also kills *Mycobacterium tuberculosis* (tubercle bacilli). EPA has registered approximately 200 tuberculocides. Such agents also are called *mycobactericides*.

Use-life: the length of time a diluted product can remain active and effective. The stability of the chemical and the storage conditions (e.g., temperature and presence of air, light, organic matter, or metals)

determine the use-life of antimicrobial products.

Vegetative bacteria: bacteria that are devoid of spores and usually can be readily inactivated by many types of germicides.

Virucide: an agent that kills viruses to make them noninfective.

Adapted from Association for the Advancement of Medical Instrumentation;^{811-814, 819} Association of periOperating Registered Nurses (AORN),⁸¹⁵ American Hospital Association,³¹⁹ and Block.^{16, 1034}

Table 1. Methods of sterilization and disinfection.

Object	Sterilization		Disinfection		
	Procedure	Exposure time	High-level (semicritical items; [except dental] will come in contact with mucous membrane or nonintact skin)	Intermediate-level (some semicritical items ¹ and noncritical items)	Low-level (noncritical items; will come in contact with intact skin)
			Critical items (will enter tissue or vascular system or blood will flow through them)	Procedure (exposure time 12-30 min at $\geq 20^{\circ}\text{C}$) ^{2,3}	Procedure (exposure time ≥ 1 m) ⁹
Smooth, hard Surface ^{1,4}	A	MR	D	K	K
	B	MR	E	L ⁵	L
	C	MR	F	M	M
	D	10 h at 20-25°C	H	N	N
	F	6 h	I ⁶		O
	G	12 m at 50-56°C	J		
	H	3-8 h			
Rubber tubing and catheters ^{3,4}	A	MR	D		
	B	MR	E		
	C	MR	F		
	D	10 h at 20-25°C	H		
	F	6 h	I ⁶		
	G	12 m at 50-56°C	J		
	H	3-8 h			
Polyethylene tubing and catheters ^{3,4,7}	A	MR	D		
	B	MR	E		
	C	MR	F		
	D	10 h at 20-25°C	H		
	F	6 h	I ⁶		
	G	12 m at 50-56°C	J		
	H	3-8 h			
Lensed instruments ⁴	A	MR	D		
	B	MR	E		
	C	MR	F		
	D	10 h at 20-25°C	H		
	F	6 h	J		
	G	12 m at 50-56°C			
	H	3-8 h			
Thermometers (oral and rectal) ⁸					K ⁸
Hinged instruments ⁴	A	MR	D		
	B	MR	E		
	C	MR	F		
	D	10 h at 20-25°C	H		
	F	6 h	I ⁶		
	G	12 m at 50-56°C	J		
	H	3-8 h			

Modified from Rutala and Simmons.^{15, 17, 18, 421} The selection and use of disinfectants in the healthcare field is dynamic, and products may become available that are not in existence when this guideline was written. As newer disinfectants become available, persons or committees responsible for selecting disinfectants and sterilization processes should be guided by products cleared by the FDA and the EPA as well as information in the scientific literature.

- A, Heat sterilization, including steam or hot air (see manufacturer's recommendations, steam sterilization processing time from 3-30 minutes)
- B, Ethylene oxide gas (see manufacturer's recommendations, generally 1-6 hours processing time plus aeration time of 8-12 hours at 50-60°C)
- C, Hydrogen peroxide gas plasma (see manufacturer's recommendations for internal diameter and length restrictions, processing time between 45-72 minutes).
- D, Glutaraldehyde-based formulations ($\geq 2\%$ glutaraldehyde, caution should be exercised with all glutaraldehyde formulations when further in-use dilution is anticipated); glutaraldehyde (1.12%) and 1.93% phenol/phenate. One glutaraldehyde-based product has a high-level disinfection claim of 5 minutes at 35°C.
- E, Ortho-phthalaldehyde (OPA) 0.55%
- F, Hydrogen peroxide 7.5% (will corrode copper, zinc, and brass)
- G, Peracetic acid, concentration variable but 0.2% or greater is sporicidal. Peracetic acid immersion system operates at 50-56°C.
- H, Hydrogen peroxide (7.35%) and 0.23% peracetic acid; hydrogen peroxide 1% and peracetic acid 0.08% (will corrode metal instruments)
- I, Wet pasteurization at 70°C for 30 minutes with detergent cleaning
- J, Hypochlorite, single use chlorine generated on-site by electrolyzing saline containing >650-675 active free chlorine; (will corrode metal instruments)
- K, Ethyl or isopropyl alcohol (70-90%)
- L, Sodium hypochlorite (5.25-6.15% household bleach diluted 1:500 provides >100 ppm available chlorine)
- M, Phenolic germicidal detergent solution (follow product label for use-dilution)
- N, Iodophor germicidal detergent solution (follow product label for use-dilution)
- O, Quaternary ammonium germicidal detergent solution (follow product label for use-dilution)
- MR, Manufacturer's recommendations
- NA, Not applicable

¹ See text for discussion of hydrotherapy.

² The longer the exposure to a disinfectant, the more likely it is that all microorganisms will be eliminated. Follow the FDA-cleared high-level disinfection claim. Ten-minute exposure is not adequate to disinfect many objects, especially those that are difficult to clean because they have narrow channels or other areas that can harbor organic material and bacteria. Twenty-minute exposure at 20°C is the minimum time needed to reliably kill *M. tuberculosis* and nontuberculous mycobacteria with a 2% glutaraldehyde. Some high-level disinfectants have a reduced exposure time (e.g., ortho-phthalaldehyde at 12 minutes at 20°C) because of their rapid activity against mycobacteria or reduced exposure time due to increased mycobactericidal activity at elevated temperature (e.g., 2.5% glutaraldehyde at 5 minutes at 35°C, 0.55% OPA at 5 min at 25°C in automated endoscope reprocessor).

³ Tubing must be completely filled for high-level disinfection and liquid chemical sterilization; care must be taken to avoid entrapment of air bubbles during immersion.

⁴ Material compatibility should be investigated when appropriate.

⁵ A concentration of 1000 ppm available chlorine should be considered where cultures or concentrated preparations of microorganisms have spilled (5.25% to 6.15% household bleach diluted 1:50 provides > 1000 ppm available chlorine). This solution may corrode some surfaces.

⁶ Pasteurization (washer-disinfector) of respiratory therapy or anesthesia equipment is a recognized alternative to high-level disinfection. Some data challenge the efficacy of some pasteurization units.

⁷ Thermostability should be investigated when appropriate.

⁸ Do not mix rectal and oral thermometers at any stage of handling or processing.

⁹ By law, all applicable label instructions on EPA-registered products must be followed. If the user selects exposure conditions that differ from those on the EPA-registered products label, the user assumes liability from any injuries resulting from off-label use and is potentially subject to enforcement action under FIFRA.

Table 2. Properties of an ideal disinfectant.

Broad spectrum: should have a wide antimicrobial spectrum
Fast acting: should produce a rapid kill
Not affected by environmental factors: should be active in the presence of organic matter (e.g., blood, sputum, feces) and compatible with soaps, detergents, and other chemicals encountered in use
Nontoxic: should not be harmful to the user or patient
Surface compatibility: should not corrode instruments and metallic surfaces and should not cause the deterioration of cloth, rubber, plastics, and other materials
Residual effect on treated surfaces: should leave an antimicrobial film on the treated surface
Easy to use with clear label directions
Odorless: should have a pleasant odor or no odor to facilitate its routine use
Economical: should not be prohibitively high in cost
Solubility: should be soluble in water
Stability: should be stable in concentrate and use-dilution
Cleaner: should have good cleaning properties
Environmentally friendly: should not damage the environment on disposal

Modified from Molinari¹⁰³⁵.

Table 3. Epidemiologic evidence associated with the use of surface disinfectants or detergents on noncritical environmental surfaces.

Justification for Use of Disinfectants for Noncritical Environmental Surfaces

Surfaces may contribute to transmission of epidemiologically important microbes (e.g., vancomycin-resistant Enterococci, methicillin-resistant *S. aureus*, viruses)

Disinfectants are needed for surfaces contaminated by blood and other potentially infective material

Disinfectants are more effective than detergents in reducing microbial load on floors

Detergents become contaminated and result in seeding the patient's environment with bacteria

Disinfection of noncritical equipment and surfaces is recommended for patients on isolation precautions by the Centers for Disease Control and Prevention.

Advantage of using a single product for decontamination of noncritical surfaces, both floors and equipment

Some newer disinfectants have persistent antimicrobial activity

Justification for Using a Detergent on Noncritical Environmental Surfaces

Noncritical surfaces contribute minimally to endemic healthcare-associated infections

No difference in healthcare-associated infection rates when floors are cleaned with detergent versus disinfectant

No environmental impact (aquatic or terrestrial) issues with disposal

No occupational health exposure issues

Lower costs

Use of antiseptics/disinfectants selects for antibiotic-resistant bacteria (?)

More aesthetically pleasing floor

Modified from Rutala³⁷⁸.

Figure 1. Decreasing order of resistance of microorganisms to disinfection and sterilization and the level of disinfection or sterilization.

Resistant	Level
Prions (Creutzfeldt-Jakob Disease)	Prion reprocessing
Bacterial spores (<i>Bacillus atrophaeus</i>)	Sterilization
Coccidia (<i>Cryptosporidium</i>)	
Mycobacteria (<i>M. tuberculosis</i> , <i>M. terrae</i>)	High
Nonlipid or small viruses (polio, coxsackie)	Intermediate
Fungi (<i>Aspergillus</i> , <i>Candida</i>)	
Vegetative bacteria (<i>S. aureus</i> , <i>P. aeruginosa</i>)	Low
↓ Lipid or medium-sized viruses (HIV, herpes, hepatitis B)	

Susceptible

Modified from Russell and Favero^{13, 344}.

Table 4. Comparison of the characteristics of selected chemicals used as high-level disinfectants or chemical sterilants.

	HP (7.5%)	PA (0.2%)	Glut ($\geq 2.0\%$)	OPA (0.55%)	HP/PA (7.35%/0.23%)
HLD Claim	30 m @ 20°C	NA	20-90 m @ 20°-25°C	12 m @ 20°C, 5 m @ 25°C in AER	15m @ 20°C
Sterilization Claim	6 h @ 20°	12m @ 50-56°C	10 h @ 20°-25°C	None	3 h @ 20°C
Activation	No	No	Yes (alkaline glut)	No	No
Reuse Life ¹	21d	Single use	14-30 d	14d	14d
Shelf Life Stability ²	2 y	6 mo	2 y	2 y	2 y
Disposal Restrictions	None	None	Local ³	Local ³	None
Materials Compatibility	Good	Good	Excellent	Excellent	No data
Monitor MEC ⁴	Yes (6%)	No	Yes (1.5% or higher)	Yes (0.3% OPA)	No
Safety	Serious eye damage (safety glasses)	Serious eye and skin damage (conc soln) ⁵	Respiratory	Eye irritant, stains skin	Eye damage
Processing	Manual or automated	Automated	Manual or automated	Manual or automated	Manual
Organic material resistance	Yes	Yes	Yes	Yes	Yes
OSHA exposure limit	1 ppm TWA	None	None ⁶	None	HP-1 ppm TWA
Cost profile (per cycle) ⁷	+ (manual), ++ (automated)	+++++ (automated)	+ (manual), ++ (automated)	++ (manual)	++ (manual)

Modified from Rutala⁶⁹.

Abbreviations: HLD=high-level disinfectant; HP=hydrogen peroxide; PA=peracetic acid; glut=glutaraldehyde; PA/HP=peracetic acid and hydrogen peroxide; OPA =ortho-phthalaldehyde (FDA cleared as a high-level disinfectant, included for comparison to other chemical agents used for high-level disinfection); m=minutes; h=hours; NA=not applicable; TWA=time-weighted average for a conventional 8-hour workday.

¹number of days a product can be reused as determined by re-use protocol

²time a product can remain in storage (unused)

³no U.S. EPA regulations but some states and local authorities have additional restrictions

⁴MEC=minimum effective concentration is the lowest concentration of active ingredients at which the product is still effective

⁵Conc soln=concentrated solution

⁶The ceiling limit recommended by the American Conference of Governmental Industrial Hygienists is 0.05 ppm.

⁷per cycle cost profile considers cost of the processing solution (suggested list price to healthcare facilities in August 2001) and assumes maximum use life (e.g., 21 days for hydrogen peroxide, 14 days for glutaraldehyde), 5 reprocessing cycles per day, 1-gallon basin for manual processing, and 4-gallon tank for automated processing. + = least expensive; +++++ = most expensive

Table 5. Summary of advantages and disadvantages of chemical agents used as chemical sterilants ¹ or as high-level disinfectants.		
Sterilization Method	Advantages	Disadvantages
Peracetic Acid/Hydrogen Peroxide	<ul style="list-style-type: none"> No activation required Odor or irritation not significant 	<ul style="list-style-type: none"> Materials compatibility concerns (lead, brass, copper, zinc) both cosmetic and functional Limited clinical experience Potential for eye and skin damage
Glutaraldehyde	<ul style="list-style-type: none"> Numerous use studies published Relatively inexpensive Excellent materials compatibility 	<ul style="list-style-type: none"> Respiratory irritation from glutaraldehyde vapor Pungent and irritating odor Relatively slow mycobactericidal activity Coagulates blood and fixes tissue to surfaces Allergic contact dermatitis Glutaraldehyde vapor monitoring recommended
Hydrogen Peroxide	<ul style="list-style-type: none"> No activation required May enhance removal of organic matter and organisms No disposal issues No odor or irritation issues Does not coagulate blood or fix tissues to surfaces Inactivates <i>Cryptosporidium</i> Use studies published 	<ul style="list-style-type: none"> Material compatibility concerns (brass, zinc, copper, and nickel/silver plating) both cosmetic and functional Serious eye damage with contact
Ortho-phthalaldehyde	<ul style="list-style-type: none"> Fast acting high-level disinfectant No activation required Odor not significant Excellent materials compatibility claimed Does not coagulate blood or fix tissues to surfaces claimed 	<ul style="list-style-type: none"> Stains skin, mucous membranes, clothing, and environmental surfaces Repeated exposure may result in hypersensitivity in some patients with bladder cancer More expensive than glutaraldehyde Eye irritation with contact Slow sporicidal activity
Peracetic Acid	<ul style="list-style-type: none"> Rapid sterilization cycle time (30-45 minutes) Low temperature (50-55°C) liquid immersion sterilization Environmental friendly by-products (acetic acid, O₂, H₂O) Fully automated Single-use system eliminates need for concentration testing Standardized cycle May enhance removal of organic material and endotoxin No adverse health effects to operators under normal operating conditions Compatible with many materials and instruments Does not coagulate blood or fix tissues to surfaces Sterilant flows through scope facilitating salt, protein, and microbe removal Rapidly sporicidal Provides procedure standardization (constant dilution, perfusion of channel, temperatures, exposure) 	<ul style="list-style-type: none"> Potential material incompatibility (e.g., aluminum anodized coating becomes dull) Used for immersible instruments only Biological indicator may not be suitable for routine monitoring One scope or a small number of instruments can be processed in a cycle More expensive (endoscope repairs, operating costs, purchase costs) than high-level disinfection Serious eye and skin damage (concentrated solution) with contact Point-of-use system, no sterile storage

Modified from Rutala⁶⁹.

¹All products effective in presence of organic soil, relatively easy to use, and have a broad spectrum of antimicrobial activity (bacteria, fungi, viruses, bacterial spores, and mycobacteria). The above characteristics are documented in the literature; contact the manufacturer of the instrument and sterilant for additional information. All products listed above are FDA-cleared as chemical sterilants except OPA, which is an FDA-cleared high-level disinfectant.

Table 6. Summary of advantages and disadvantages of commonly used sterilization technologies.

Sterilization Method	Advantages	Disadvantages
Steam	<ul style="list-style-type: none"> · Nontoxic to patient, staff, environment · Cycle easy to control and monitor · Rapidly microbicidal · Least affected by organic/inorganic soils among sterilization processes listed · Rapid cycle time · Penetrates medical packing, device lumens 	<ul style="list-style-type: none"> · Deleterious for heat-sensitive instruments · Microsurgical instruments damaged by repeated exposure · May leave instruments wet, causing them to rust • Potential for burns
Hydrogen Peroxide Gas Plasma	<ul style="list-style-type: none"> · Safe for the environment · Leaves no toxic residuals · Cycle time is 28-75 minutes (varies with model type) and no aeration necessary · Used for heat- and moisture-sensitive items since process temperature <50°C · Simple to operate, install (208 V outlet), and monitor · Compatible with most medical devices · Only requires electrical outlet 	<ul style="list-style-type: none"> · Cellulose (paper), linens and liquids cannot be processed · Sterilization chamber size from 1.8-9.4 ft³ total volume (varies with model type) · Some endoscopes or medical devices with long or narrow lumens cannot be processed at this time in the United States (see manufacturer's recommendations for internal diameter and length restrictions) · Requires synthetic packaging (polypropylene wraps, polyolefin pouches) and special container tray • Hydrogen peroxide may be toxic at levels greater than 1 ppmTWA
100% Ethylene Oxide (ETO)	<ul style="list-style-type: none"> · Penetrates packaging materials, device lumens · Single-dose cartridge and negative- pressure chamber minimizes the potential for gas leak and ETO exposure · Simple to operate and monitor · Compatible with most medical materials 	<ul style="list-style-type: none"> · Requires aeration time to remove ETO residue · Sterilization chamber size from 4.0-7.9 ft³ total volume (varies with model type) · ETO is toxic, a carcinogen, and flammable · ETO emission regulated by states but catalytic cell removes 99.9% of ETO and converts it to CO₂ and H₂O · ETO cartridges should be stored in flammable liquid storage cabinet · Lengthy cycle/aeration time
ETO Mixtures 8.6% ETO/91.4% HCFC 10% ETO/90% HCFC 8.5% ETO/91.5% CO ₂	<ul style="list-style-type: none"> · Penetrates medical packaging and many plastics · Compatible with most medical materials · Cycle easy to control and monitor 	<ul style="list-style-type: none"> · Some states (e.g., CA, NY, MI) require ETO emission reduction of 90-99.9% · CFC (inert gas that eliminates explosion hazard) banned in 1995 · Potential hazards to staff and patients · Lengthy cycle/aeration time · ETO is toxic, a carcinogen, and flammable
Peracetic Acid	<ul style="list-style-type: none"> · Rapid cycle time (30-45 minutes) · Low temperature (50-55°C liquid immersion sterilization) · Environmental friendly by-products · Sterilant flows through endoscope which facilitates salt, protein and microbe removal 	<ul style="list-style-type: none"> · Point-of-use system, no sterile storage · Biological indicator may not be suitable for routine monitoring · Used for immersible instruments only · Some material incompatibility (e.g., aluminum anodized coating becomes dull) · One scope or a small number of instruments processed in a cycle • Potential for serious eye and skin damage (concentrated solution) with contact

Modified from Rutala.⁸²⁵

Abbreviations: CFC=chlorofluorocarbon, HCFC=hydrochlorofluorocarbon.

Table 7. Minimum cycle times for steam sterilization cycles

Type of sterilizer	Item	Exposure time at 250°F (121°C)	Exposure time at 270°F (132°C)	Drying time
Gravity displacement	Wrapped instruments	30 min	15 min	15-30 min
	Textile packs	30 min	25 min	15 min
	Wrapped utensils	30 min	15 min	15-30 min
Dynamic-air-removal (e.g., prevacuum)	Wrapped instruments		4 min	20-30 min
	Textile packs		4 min	5-20 min
	Wrapped utensils		4 min	20 min

Modified from Association for the Advancement of Medical Instrumentation.^{813, 819}

Table 8. Examples of flash steam sterilization parameters.

Type of sterilizer	Load configuration	Temperature	Time
Gravity displacement	Nonporous items only (i.e., routine metal instruments, no lumens)	132°C (270°F)	3 minutes
	Nonporous and porous items (e.g., rubber or plastic items, items with lumens) sterilized together	132°C (270°F)	10 minutes
Prevacuum	Nonporous items only (i.e., routine metal instruments, no lumens)	132°C (270°F)	3 minutes
	Nonporous and porous items (e.g., rubber or plastic items, items with lumens) sterilized together	132°C (270°F)	4 minutes
Steam-flush pressure-pulse	Nonporous or mixed nonporous/porous items	132° (270°F) Manufacturers' instruction	4 minutes

Modified from Association for the Advancement of Medical Instrumentation. ^{812, 819}

Table 9. Characteristics of an ideal low-temperature sterilization process.

High efficacy: the agent should be virucidal, bactericidal, tuberculocidal, fungicidal and sporicidal
Rapid activity: ability to quickly achieve sterilization
Strong penetrability: ability to penetrate common medical-device packaging materials and penetrate into the interior of device lumens
Material compatibility: produces only negligible changes in the appearance or the function of processed items and packaging materials even after repeated cycling
Nontoxic: presents no toxic health risk to the operator or the patient and poses no hazard to the environment
Organic material resistance: withstands reasonable organic material challenge without loss of efficacy
Adaptability: suitable for large or small (point of use) installations
Monitoring capability: monitored easily and accurately with physical, chemical, and biological process monitors
Cost effectiveness: reasonable cost for installation and for routine operation

Modified from Schneider.⁸⁵¹

Table 10. Factors affecting the efficacy of sterilization.

Factors	Effect
Cleaning ¹	Failure to adequately clean instrument results in higher bioburden, protein load, and salt concentration. These will decrease sterilization efficacy.
Bioburden ¹	The natural bioburden of used surgical devices is 10 ⁰ to 10 ³ organisms (primarily vegetative bacteria), which is substantially below the 10 ⁵ -10 ⁶ spores used with biological indicators.
Pathogen type	Spore-forming organisms are most resistant to sterilization and are the test organisms required for FDA clearance. However, the contaminating microflora on used surgical instruments consists mainly of vegetative bacteria.
Protein ¹	Residual protein decreases efficacy of sterilization. However, cleaning appears to rapidly remove protein load.
Salt ¹	Residual salt decreases efficacy of sterilization more than does protein load. However, cleaning appears to rapidly remove salt load.
Biofilm accumulation ¹	Biofilm accumulation reduces efficacy of sterilization by impairing exposure of the sterilant to the microbial cell.
Lumen length	Increasing lumen length impairs sterilant penetration. May require forced flow through lumen to achieve sterilization.
Lumen diameter	Decreasing lumen diameter impairs sterilant penetration. May require forced flow through lumen to achieve sterilization.
Restricted flow	Sterilant must come into contact with microorganisms. Device designs that prevent or inhibit this contact (e.g., sharp bends, blind lumens) will decrease sterilization efficacy.
Device design and construction	Materials used in construction may affect compatibility with different sterilization processes and affect sterilization efficacy. Design issues (e.g., screws, hinges) will also affect sterilization efficacy.

Modified from Alfa and Rutala.^{470, 825}

¹ Factor only relevant for reused surgical/medical devices

Table 11. Comparative evaluation of the microbicidal activity of low-temperature sterilization technology.

Challenge	Carriers Sterilized by Various Low-Temperature Sterilization Technologies						Reference
	ETO 12/88	100% ETO	HCFC-ETO	HPGP 100	HPGP 100S	PA	
No salt or serum ¹	100%	100%	96%	100%	ND	ND	Alfa ⁷²¹
10% serum and 0.65% salt ²	97%	60%	95%	37%	ND	ND	Alfa ⁷²¹
Lumen (125 cm long x 3 mm wide) without serum or salt ¹	ND	96%	96%	ND	ND	ND	Alfa ⁷²¹
Lumen (125 cm long x 3 mm wide) with 10% serum and 0.65% salt ²	44%	40%	49%	35%	ND	100% ¹	Alfa ⁷²¹
Lumen (40 cm long x 3 mm wide) ³	ND	ND	100%	95%	100%	8%	Rutala ⁸⁵⁶
Lumen (40 cm long x 2 mm wide) ³	ND	ND	100%	93%	100%	ND	Rutala ⁸⁵⁶
Lumen (40 cm long x 1 mm wide) ³	ND	ND	100%	26%	100%	ND	Rutala ⁸⁵⁶
Lumen (40 cm long x 3 mm wide) ⁴	ND	ND	100%	100%	100%	ND	Rutala ⁸⁵⁶

—

Modified from Rutala.⁸²⁵

Abbreviations: ETO=ethylene oxide; HCFC=hydrochlorofluorocarbon; ND=no data; HPGP=hydrogen peroxide gas plasma; PA=peracetic acid.

¹Test organisms included *Enterococcus faecalis*, *Mycobacterium chelonae*, and *Bacillus atrophaeus* spores.

²Test organisms included *E. faecalis*, *P. aeruginosa*, *E. coli*, *M. chelonae*, *B. atrophaeus* spores, *G. stearothermophilus* spores, and *B. circulans* spores.

³Test organism was *G. stearothermophilus* spores. The lumen test units had a removable 5 cm center piece (1.2 cm diameter) of stainless steel sealed to the narrower steel tubing by hard rubber septums.

⁴Test organism was *G. stearothermophilus* spores. The lumen test unit was a straight stainless steel tube.

Table 12. Suggested protocol for management of positive biological indicator in a steam sterilizer.

1. Take the sterilizer out of service. Notify area supervisor and infection control department.
2. Objects, other than implantable objects, do not need to be recalled because of a single positive spore test unless the sterilizer or the sterilization procedure is defective. As soon as possible, repeat biological indicator test in three consecutive sterilizer cycles. If additional spore tests remain positive, the items should be considered nonsterile, and supplies processed since the last acceptable (negative) biological indicator should be recalled. The items from the suspect load(s) should be recalled and reprocessed.
3. Check to ensure the sterilizer was used correctly (e.g., verify correct time and temperature setting). If not, repeat using appropriate settings and recall and reprocess all inadequately processed items.
4. Check with hospital maintenance for irregularities (e.g., electrical) or changes in the hospital steam supply (i.e., from standard $\geq 97\%$ steam, $< 3\%$ moisture). Any abnormalities should be reported to the person who performs sterilizer maintenance (e.g., medical engineering, sterilizer manufacturer).
5. Check to ensure the correct biological indicator was used and appropriately interpreted. If not, repeat using appropriate settings.

If steps 1 through 5 resolve the problem

6. If all three repeat biological indicators from three consecutive sterilizer cycles (step 2 above) are negative, put the sterilizer back in service.

If one or both biological indicators are positive, do one or more of the following until problem is resolved.

7.
 - A. Request an inspection of the equipment by sterilizer maintenance personnel.
 - B. Have hospital maintenance inspect the steam supply lines.
 - C. Discuss the abnormalities with the sterilizer manufacturer.
 - D. Repeat the biological indicator using a different manufacturer's indicator.

If step 7 does not resolve the problem

Close sterilizer down until the manufacturer can assure that it is operating properly. Retest at that time with biological indicators in three consecutive sterilizer cycles.

Modified from Bryce.⁸³⁹

Disclosure of Financial Interests and Relationships (2000- July 2004)

William A. Rutala: Honoraria from Advanced Sterilization Products, Kimberly-Clark; consultation with Advanced Sterilization Products, Aesculap, Clorox, 3M, SC Johnson, Intelligent Biocides, Metrex; and an educational grant from Consumer Specialty Products Association, Kimberly-Clark.

David J. Weber: Honoraria from Consumer Specialty Products Association; consultation with Clorox; and educational grant from Consumer Specialty Products Association.

REFERENCES

1. Garner JS, Favero MS. CDC Guideline for handwashing and hospital environmental control, 1985. *Infect. Control* 1986;7:231-43.
2. Centers for Disease Control. Ambulatory and inpatient procedures in the United States, 1996. Atlanta, GA, 1998:1-39.
3. Uttley AH, Simpson RA. Audit of bronchoscope disinfection: a survey of procedures in England and Wales and incidents of mycobacterial contamination. *J. Hosp. Infect.* 1994;26:301-8.
4. Zaidi M, Angulo M, Sifuentes-Osornio J. Disinfection and sterilization practices in Mexico. *J. Hosp. Infect.* 1995;31:25-32.
5. McCarthy GM, Koval JJ, John MA, MacDonald JK. Infection control practices across Canada: do dentists follow the recommendations? *J. Can. Dent. Assoc.* 1999;65:506-11.
6. Spach DH, Silverstein FE, Stamm WE. Transmission of infection by gastrointestinal endoscopy and bronchoscopy. *Ann. Intern. Med.* 1993;118:117-28.
7. Weber DJ, Rutala WA. Lessons from outbreaks associated with bronchoscopy. *Infect. Control Hosp. Epidemiol.* 2001;22:403-8.
8. Weber DJ, Rutala WA, DiMarino AJ, Jr. The prevention of infection following gastrointestinal endoscopy: the importance of prophylaxis and reprocessing. In: DiMarino AJ, Jr, Benjamin SB, eds. *Gastrointestinal diseases: an endoscopic approach*. Thorofare, NJ: Slack Inc., 2002:87-106.
9. Meyers H, Brown-Elliott BA, Moore D, et al. An outbreak of *Mycobacterium chelonae* infection following liposuction. *Clin. Infect. Dis.* 2002;34:1500-7.
10. Lowry PW, Jarvis WR, Oberle AD, et al. *Mycobacterium chelonae* causing otitis media in an ear-nose-and-throat practice. *N. Engl. J. Med.* 1988;319:978-82.
11. Centers for Disease Control and Prevention. *Pseudomonas aeruginosa* infections associated with transrectal ultrasound-guided prostate biopsies--Georgia, 2005. *MMWR CDC Surveill. Summ.* 2006;55:776-7.
12. Mehta AC, Prakash UBS, Garland R, et al. Prevention of flexible bronchoscopy-associated infection. *Chest* 2006;128:1742-55.
13. Favero MS, Bond WW. Chemical disinfection of medical and surgical materials. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001:881-917.
14. Spaulding EH. Chemical disinfection of medical and surgical materials. In: Lawrence C, Block SS, eds. *Disinfection, sterilization, and preservation*. Philadelphia: Lea & Febiger, 1968:517-31.
15. Simmons BP. CDC guidelines for the prevention and control of nosocomial infections. Guideline for hospital environmental control. *Am. J. Infect. Control* 1983;11:97-120.
16. Block SS. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001.
17. Rutala WA, 1994, 1995, and 1996 APIC Guidelines Committee. APIC guideline for selection and use of disinfectants. Association for Professionals in Infection Control and Epidemiology, Inc. *Am. J. Infect. Control* 1996;24:313-42.
18. Rutala WA. Disinfection, sterilization and waste disposal. In: Wenzel RP, ed. *Prevention and control of nosocomial infections*. Baltimore: Williams and Wilkins, 1997:539-93.
19. Rutala WA. APIC guideline for selection and use of disinfectants. *Am. J. Infect. Control* 1990;18:99-117.
20. Association of peri-Operative Registered Nurses. Recommended practices for high-level disinfection. *AORN J.* 2005;81:402-12.
21. Garner JS, Favero MS. CDC guidelines for the prevention and control of nosocomial infections. Guideline for handwashing and hospital environmental control, 1985. Supersedes guideline for hospital environmental control published in 1981. *Am. J. Infect. Control* 1986;14:110-29.
22. Centers for Disease Control. Guidelines for prevention of transmission of human immunodeficiency virus

- and hepatitis B virus to health-care and public-safety workers. *MMWR* 1989;38:1-37.
23. Centers for Disease Control. Guidelines for Environmental Infection Control in Health-Care Facilities, 2003. *MMWR* 2003;52 (No. RR-10):1-44.
 24. Bhattachatyya M, Kepnes LJ. The effectiveness of immersion disinfection for flexible fiberoptic laryngoscopes. *Otolaryngol Head Neck* 2004;130:681-5.
 25. Hamasuna R, Nose K, Sueyoshi T, Nagano M, Hasui Y, Osada Y. High-level disinfection of cystoscopic equipment with ortho-phthalaldehyde solution. *J. Hosp. Infect.* 2004;57:346-8.
 26. Foliente RL KB, Aprecio RM, Bains HJ, Kettering JD, Chen YK. Efficacy of high-level disinfectants for reprocessing gastrointestinal endoscopes in simulated-use testing. *Gastrointest. Endosc.* 2001;53:456-62.
 27. Kovacs BJ, Chen YK, Kettering JD, Aprecio RM, Roy I. High-level disinfection of gastrointestinal endoscopes: are current guidelines adequate? *Am. J. Gastroenterol.* 1999;94:1546-50.
 28. Rutala WA, Clontz EP, Weber DJ, Hoffmann KK. Disinfection practices for endoscopes and other semicritical items. *Infect. Control Hosp. Epidemiol.* 1991;12:282-8.
 29. Phillips J, Hulka B, Hulka J, Keith D, Keith L. Laparoscopic procedures: The American Association of Gynecologic Laparoscopists' Membership Survey for 1975. *J. Reprod. Med.* 1977;18:227-32.
 30. Muscarella LF. Current instrument reprocessing practices: Results of a national survey. *Gastrointestinal Nursing* 2001;24:253-60.
 31. Wright EP, Collins CH, Yates MD. *Mycobacterium xenopi* and *Mycobacterium kansasii* in a hospital water supply. *J. Hosp. Infect.* 1985;6:175-8.
 32. Wallace RJ, Jr., Brown BA, Driffith DE. Nosocomial outbreaks/pseudo-outbreaks caused by nontuberculous mycobacteria. *Annu. Rev. Microbiol.* 1998;52:453-90.
 33. Mitchell DH, Hicks LJ, Chiew R, Montanaro JC, Chen SC. Pseudoepidemic of *Legionella pneumophila* serogroup 6 associated with contaminated bronchoscopes. *J. Hosp. Infect.* 1997;37:19-23.
 34. Meenhorst PL, Reingold AL, Groothuis DG, et al. Water-related nosocomial pneumonia caused by *Legionella pneumophila* serogroups 1 and 10. *J. Infect. Dis.* 1985;152:356-64.
 35. Atlas RM. *Legionella*: from environmental habitats to disease pathology, detection and control. *Environ. Microbiol.* 1999;1:283-93.
 36. Rutala WA, Weber DJ. Water as a reservoir of nosocomial pathogens. *Infect. Control Hosp. Epidemiol.* 1997;18:609-16.
 37. Weber DJ, Rutala WA. Environmental issues and nosocomial infections. In: Wenzel RP, ed. *Prevention and control of nosocomial infections*. Baltimore: Williams and Wilkins, 1997:491-514.
 38. Society of Gastroenterology Nurses and Associates. Standards for infection control and reprocessing of flexible gastrointestinal endoscopes. *Gastroenterol. Nurs.* 2000;23:172-9.
 39. Gerding DN, Peterson LR, Vennes JA. Cleaning and disinfection of fiberoptic endoscopes: evaluation of glutaraldehyde exposure time and forced-air drying. *Gastroenterology* 1982;83:613-8.
 40. Society of Gastroenterology Nurses and Associates. Guideline for the use of high-level disinfectants and sterilants in reprocessing of flexible gastrointestinal endoscopes.
 41. Turner AG, Higgins MM, Craddock JG. Disinfection of immersion tanks (Hubbard) in a hospital burn unit. *Arch. Environ. Health* 1974;28:101-4.
 42. Rutala DR, Rutala WA, Weber DJ, Thomann CA. Infection risks associated with spirometry. *Infect. Control Hosp. Epidemiol.* 1991;12:89-92.
 43. Kohn WG, Collins AS, Cleveland JL, Harte JA, Eklund KJ, Malvitz DM. Guidelines for infection control in dental health-care settings-2003. *MMWR* 2003;52 (no. RR-17):1-67.
 44. Sehulster L, Chinn RYW, Healthcare Infection Control Practices Advisory Committee. Guidelines for environmental infection control in health-care facilities. *MMWR* 2003;52:1-44.
 45. Rutala WA, White MS, Gergen MF, Weber DJ. Bacterial contamination of keyboards: Efficacy and functional impact of disinfectants. *Infect Control Hosp Epidemiol* 2006;27:372-7.
 46. Sattar SA, Lloyd-Evans N, Springthorpe VS, Nair RC. Institutional outbreaks of rotavirus diarrhoea: potential role of fomites and environmental surfaces as vehicles for virus transmission. *J. Hyg. (Lond)*. 1986;96:277-89.
 47. Weber DJ, Rutala WA. Role of environmental contamination in the transmission of vancomycin-resistant enterococci. *Infect. Control Hosp. Epidemiol.* 1997;18:306-9.
 48. Ward RL, Bernstein DI, Knowlton DR, et al. Prevention of surface-to-human transmission of rotaviruses by treatment with disinfectant spray. *J. Clin. Microbiol.* 1991;29:1991-6.

49. Tyler R, Ayliffe GA, Bradley C. Virucidal activity of disinfectants: studies with the poliovirus. *J. Hosp. Infect.* 1990;15:339-45.
50. Gwaltney JM, Jr., Hendley JO. Transmission of experimental rhinovirus infection by contaminated surfaces. *Am. J. Epidemiol.* 1982;116:828-33.
51. Sattar SA, Jacobsen H, Springthorpe VS, Cusack TM, Rubino JR. Chemical disinfection to interrupt transfer of rhinovirus type 14 from environmental surfaces to hands. *Appl. Environ. Microbiol.* 1993;59:1579-85.
52. Sattar SA, Jacobsen H, Rahman H, Cusack TM, Rubino JR. Interruption of rotavirus spread through chemical disinfection. *Infect. Control Hosp. Epidemiol.* 1994;15:751-6.
53. Rutala WA, Barbee SL, Aguiar NC, Sobsey MD, Weber DJ. Antimicrobial activity of home disinfectants and natural products against potential human pathogens. *Infect. Control Hosp. Epidemiol.* 2000;21:33-8.
54. Silverman J, Vazquez JA, Sobel JD, Zervos MJ. Comparative in vitro activity of antiseptics and disinfectants versus clinical isolates of *Candida* species. *Infect. Control Hosp. Epidemiol.* 1999;20:676-84.
55. Best M, Sattar SA, Springthorpe VS, Kennedy ME. Efficacies of selected disinfectants against *Mycobacterium tuberculosis*. *J. Clin. Microbiol.* 1990;28:2234-9.
56. Best M, Kennedy ME, Coates F. Efficacy of a variety of disinfectants against *Listeria* spp. *Appl. Environ. Microbiol.* 1990;56:377-80.
57. Best M, Springthorpe VS, Sattar SA. Feasibility of a combined carrier test for disinfectants: studies with a mixture of five types of microorganisms. *Am. J. Infect. Control* 1994;22:152-62.
58. Mbithi JN, Springthorpe VS, Sattar SA. Chemical disinfection of hepatitis A virus on environmental surfaces. *Appl. Environ. Microbiol.* 1990;56:3601-4.
59. Springthorpe VS, Grenier JL, Lloyd-Evans N, Sattar SA. Chemical disinfection of human rotaviruses: efficacy of commercially-available products in suspension tests. *J. Hyg. (Lond).* 1986;97:139-61.
60. Akamatsu T, Tabata K, Hironga M, Kawakami H, Uyeda M. Transmission of *Helicobacter pylori* infection via flexible fiberoptic endoscopy. *Am. J. Infect. Control* 1996;24:396-401.
61. Sattar SA, Springthorpe VS. Survival and disinfectant inactivation of the human immunodeficiency virus: a critical review. *Rev. Infect. Dis.* 1991;13:430-47.
62. Resnick L, Veren K, Salahuddin SZ, Tondreau S, Markham PD. Stability and inactivation of HTLV-III/LAV under clinical and laboratory environments. *JAMA* 1986;255:1887-91.
63. Weber DJ, Barbee SL, Sobsey MD, Rutala WA. The effect of blood on the antiviral activity of sodium hypochlorite, a phenolic, and a quaternary ammonium compound. *Infect. Control Hosp. Epidemiol.* 1999;20:821-7.
64. Rice EW, Clark RM, Johnson CH. Chlorine inactivation of *Escherichia coli* O157:H7. *Emerg. Infect. Dis.* 1999;5:461-3.
65. Pentella MA, Fisher T, Chandler S, Britt-Ohrmund T, Kwa BH, Yangco BG. Are disinfectants accurately prepared for use in hospital patient care areas? *Infect. Control Hosp. Epidemiol.* 2000;21:103.
66. Bhalla A, Pultz NJ, Gries DM, et al. Acquisition of nosocomial pathogens on hands after contact with environmental surfaces near hospitalized patients. *Infect. Control Hosp. Epidemiol.* 2004;25:164-7.
67. Ray AJ, Hoyen CK, Taub TF, Eckstein EC, Donskey CJ. Nosocomial transmission of vancomycin-resistant enterococci from surfaces. *JAMA* 2002;287:1400-1.
68. Westwood JC, Mitchell MA, Legace S. Hospital sanitation: the massive bacterial contamination of the wet mop. *Appl. Microbiol.* 1971;21:693-7.
69. Rutala WA, Weber DJ. Disinfection of endoscopes: review of new chemical sterilants used for high-level disinfection. *Infect. Control Hosp. Epidemiol.* 1999;20:69-76.
70. Russell AD. Bacterial spores and chemical sporicidal agents. *Clin. Microbiol. Rev.* 1990;3:99-119.
71. Terleckyj B, Axler DA. Quantitative neutralization assay of fungicidal activity of disinfectants. *Antimicrob. Agents Chemother.* 1987;31:794-8.
72. Klein M, DeForest A. The inactivation of viruses by germicides. *Chem. Specialists Manuf. Assoc. Proc.* 1963;49:116-8.
73. Rutala WA, Cole EC, Wannamaker NS, Weber DJ. Inactivation of *Mycobacterium tuberculosis* and *Mycobacterium bovis* by 14 hospital disinfectants. *Am. J. Med.* 1991;91:267S-271S.
74. Robison RA, Bodily HL, Robinson DF, Christensen RP. A suspension method to determine reuse life of chemical disinfectants during clinical use. *Appl. Environ. Microbiol.* 1988;54:158-64.
75. Isenberg HD, Giugliano ER, France K, Alperstein P. Evaluation of three disinfectants after in-use stress. *J.*

- Hosp. Infect. 1988;11:278-85.
76. Cole EC, Rutala WA, Nessen L, Wannamaker NS, Weber DJ. Effect of methodology, dilution, and exposure time on the tuberculocidal activity of glutaraldehyde-based disinfectants. *Appl. Environ. Microbiol.* 1990;56:1813-7.
 77. Power EG, Russell AD. Sporicidal action of alkaline glutaraldehyde: factors influencing activity and a comparison with other aldehydes. *J. Appl. Bacteriol.* 1990;69:261-8.
 78. Rutala WA, Gergen MF, Weber DJ. Sporicidal activity of chemical sterilants used in hospitals. *Infect. Control Hosp. Epidemiol.* 1993;14:713-8.
 79. Rutala WA, Gergen MF, Weber DJ. Inactivation of *Clostridium difficile* spores by disinfectants. *Infect. Control Hosp. Epidemiol.* 1993;14:36-9.
 80. Ascenzi JM, Ezzell RJ, Wendt TM. A more accurate method for measurement of tuberculocidal activity of disinfectants. *Appl. Environ. Microbiol.* 1987;53:2189-92.
 81. Collins FM. Use of membrane filters for measurement of mycobactericidal activity of alkaline glutaraldehyde solution. *Appl. Environ. Microbiol.* 1987;53:737-9.
 82. Rubbo SD, Gardner JF, Webb RL. Biocidal activities of glutaraldehyde and related compounds. *J. Appl. Bacteriol.* 1967;30:78-87.
 83. Rutala WA, Weber DJ. FDA labeling requirements for disinfection of endoscopes: a counterpoint. *Infect. Control Hosp. Epidemiol.* 1995;16:231-5.
 84. Collins FM. Kinetics of the tuberculocidal response by alkaline glutaraldehyde in solution and on an inert surface. *J. Appl. Bacteriol.* 1986;61:87-93.
 85. Food and Drug Administration. 2005. FDA-cleared sterilants and high-level disinfectants with general claims for processing reusable medical and dental devices, May 13, 2005. www.fda.gov/cdrh/ode/germlab.html.
 86. Crow S, Metcalf RW, Beck WC, Birnbaum D. Disinfection or sterilization? Four views on arthroscopes. *AORN J.* 1983;37:854-9, 862-8.
 87. Loffer FD. Disinfection vs. sterilization of gynecologic laparoscopy equipment. The experience of the Phoenix Surgicenter. *J. Reprod. Med.* 1980;25:263-6.
 88. Johnson LL, Shneider DA, Austin MD, Goodman FG, Bullock JM, DeBruin JA. Two per cent glutaraldehyde: a disinfectant in arthroscopy and arthroscopic surgery. *J. Bone Joint Surg.* 1982;64:237-9.
 89. Burns S, Edwards M, Jennings J, et al. Impact of variation in reprocessing invasive fiberoptic scopes on patient outcomes. *Infect. Control Hosp. Epidemiol.* 1996;17(suppl):P42.
 90. Fuselier HA, Jr., Mason C. Liquid sterilization versus high level disinfection in the urologic office. *Urology* 1997;50:337-40.
 91. Muscarella LF. High-level disinfection or "sterilization" of endoscopes? *Infect. Control Hosp. Epidemiol.* 1996;17:183-7.
 92. Miles RS. What standards should we use for the disinfection of large equipment? *J. Hosp. Infect.* 1991;18:264-73.
 93. Lee RM, Kozarek RA, Sumida SE, Raltz SL. Risk of contamination of sterile biopsy forceps in disinfected endoscopes. *Gastrointest. Endosc.* 1998;47:377-81.
 94. Kinney TP, Kozarek RA, Raltz S, Attia F. Contamination of single-use biopsy forceps: A prospective in vitro analysis. *Gastrointest. Endosc.* 2002;56:209-12.
 95. Centers for Disease Control. Recommendations for preventing possible transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus from tears. *MMWR* 1985;34:533-4.
 96. Lettau LA, Bond WW, McDougal JS. Hepatitis and diaphragm fitting. *JAMA* 1985;254:752.
 97. Schembre DB. Infectious complications associated with gastrointestinal endoscopy. *Gastrointest. Endosc. Clin. N. Am.* 2000;10:215-32.
 98. Nelson DB. Infectious disease complications of GI endoscopy: Part II, exogenous infections. *Gastrointest. Endosc.* 2003;57:695-711.
 99. Chu NS, Favero M. The microbial flora of the gastrointestinal tract and the cleaning of flexible endoscopes. *Gastrointest. Endosc. Clin. N. Am.* 2000;10:233-44.
 100. Alfa MJ, Sitter DL. In-hospital evaluation of orthophthalaldehyde as a high level disinfectant for flexible endoscopes. *J. Hosp. Infect.* 1994;26:15-26.
 101. Vesley D, Melson J, Stanley P. Microbial bioburden in endoscope reprocessing and an in-use evaluation of the high-level disinfection capabilities of Cidex PA. *Gastroenterol. Nurs.* 1999;22:63-8.

102. Chu NS, McAlister D, Antonoplos PA. Natural bioburden levels detected on flexible gastrointestinal endoscopes after clinical use and manual cleaning. *Gastrointest. Endosc.* 1998;48:137-42.
103. Rutala WA, Weber DJ. Reprocessing endoscopes: United States perspective. *J. Hosp. Infect.* 2004;56:S27-S39.
104. Hanson PJ, Gor D, Clarke JR, et al. Contamination of endoscopes used in AIDS patients. *Lancet* 1989;2:86-8.
105. Hanson PJ, Gor D, Clarke JR, et al. Recovery of the human immunodeficiency virus from fiberoptic bronchoscopes. *Thorax* 1991;46:410-2.
106. Chafour X, Deva AK, Vickery K, et al. Evaluation of disinfection and sterilization of reusable angioscopes with the duck hepatitis B model. *J. Vasc. Surg.* 1999;30:277-82.
107. Cheung RJ, Ortiz D, DiMarino AJ, Jr. GI endoscopic reprocessing practices in the United States. *Gastrointest. Endosc.* 1999;50:362-8.
108. American Society for Gastrointestinal Endoscopy. Position statement: reprocessing of flexible gastrointestinal endoscopes. *Gastrointest. Endosc.* 1996;43:541-6.
109. Food and Drug Administration. Content and format of premarket notification [510(k)] submissions for liquid chemical sterilants/high level disinfectants. www.fda.gov/cdrh/ode/397 2000.
110. Urayama S, Kozarek RA, Sumida S, Raltz S, Merriam L, Pethigal P. Mycobacteria and glutaraldehyde: is high-level disinfection of endoscopes possible? *Gastrointest. Endosc.* 1996;43:451-6.
111. Jackson J, Leggett JE, Wilson DA, Gilbert DN. *Mycobacterium gordonae* in fiberoptic bronchoscopes. *Am. J. Infect. Control* 1996;24:19-23.
112. Martiny H, Floss H, Zuhlsdorf B. The importance of cleaning for the overall results of processing endoscopes. *J. Hosp. Infect.* 2004;56:S16-S22.
113. Alvarado CJ, Reichelderfer M. APIC guideline for infection prevention and control in flexible endoscopy. Association for Professionals in Infection Control. *Am. J. Infect. Control* 2000;28:138-55.
114. Society of Gastroenterology Nurses and Associates. Guideline for the use of high-level disinfectants and sterilants for reprocessing of flexible gastrointestinal endoscopes. *Gastroenterol. Nurs.* 2000;23:180-7.
115. Society of Gastroenterology Nurses and Associates. Standards of infection control in reprocessing of flexible gastrointestinal endoscopes. *Gastroenterol. Nurs.* 2006;29:142-8.
116. Nelson DB, Jarvis WR, Rutala WA, et al. Multi-society guideline for reprocessing flexible gastrointestinal endoscopes. *Infect Control Hosp Epidemiol* 2003;24:532-537.
117. Martin MA, Reichelderfer M, 1991, and 1993 APIC Guidelines Committee. APIC guidelines for infection prevention and control in flexible endoscopy. *Am. J. Infect. Control* 1994;22:19-38.
118. Rey JF, Halfon P, Feryn JM, Khiri H, Masseyeff MF, Ouzan D. Risk of transmission of hepatitis C virus by digestive endoscopy. *Gastroenterol. Clin. Biol.* 1995;19:346-9.
119. Cronmiller JR, Nelson DK, Jackson DK, Kim CH. Efficacy of conventional endoscopic disinfection and sterilization methods against *Helicobacter pylori* contamination. *Helicobacter* 1999;4:198-203.
120. Sartor C, Charrel RN, de Lamballerie X, Sambuc R, De Micco P, Boubli L. Evaluation of a disinfection procedure for hysteroscopes contaminated by hepatitis C virus. *Infect. Control Hosp. Epidemiol.* 1999;20:434-6.
121. Hanson PJ, Chadwick MV, Gaya H, Collins JV. A study of glutaraldehyde disinfection of fiberoptic bronchoscopes experimentally contaminated with *Mycobacterium tuberculosis*. *J. Hosp. Infect.* 1992;22:137-42.
122. Merighi A, Contato E, Scagliarini R, et al. Quality improvement in gastrointestinal endoscopy: microbiologic surveillance of disinfection. *Gastrointest. Endosc.* 1996;43:457-62.
123. Bond WW. Endoscope reprocessing: Problems and solutions. In: Rutala WA, ed. *Disinfection, sterilization, and antisepsis in healthcare*. Champlain, New York: Polyscience Publications, 1998:151-163.
124. Deva AK, Vickery K, Zou J, West RH, Harris JP, Cossart YE. Establishment of an in-use testing method for evaluating disinfection of surgical instruments using the duck hepatitis B model. *J. Hosp. Infect.* 1996;33:119-30.
125. Hanson PJ, Gor D, Jeffries DJ, Collins JV. Elimination of high titre HIV from fiberoptic endoscopes. *Gut* 1990;31:657-9.
126. Wu MS, Wang JT, Yang JC, et al. Effective reduction of *Helicobacter pylori* infection after upper gastrointestinal endoscopy by mechanical washing of the endoscope. *Hepatogastroenterology.* 1996;43:1660-4.

127. Kirschke DL, Jones TF, Craig AS, et al. *Pseudomonas aeruginosa* and *Serratia marcescens* contamination associated with a manufacturing defect in bronchoscopes. *N. Engl. J. Med.* 2003;348:214-20.
128. Srinivasan A, Wolfenden LL, Song X, et al. An outbreak of *Pseudomonas aeruginosa* infections associated with flexible bronchoscopes. *N. Engl. J. Med.* 2003;348:221-7.
129. Kaczmarek RG, Moore RM, Jr., McCrohan J, et al. Multi-state investigation of the actual disinfection/sterilization of endoscopes in health care facilities. *Am. J. Med.* 1992;92:257-61.
130. Bradley CR, Babb JR. Endoscope decontamination: automated vs. manual. *J. Hosp. Infect.* 1995;30:537-42.
131. Muscarella LF. Advantages and limitations of automatic flexible endoscope reprocessors. *Am. J. Infect. Control* 1996;24:304-9.
132. Muscarella LF. Automatic flexible endoscope reprocessors. *Gastrointest. Endosc. Clin. N. Am.* 2000;10:245-57.
133. Alvarado CJ, Stolz SM, Maki DG. Nosocomial infections from contaminated endoscopes: a flawed automated endoscope washer. An investigation using molecular epidemiology. *Am. J. Med.* 1991;91:272S-280S.
134. Fraser VJ, Jones M, Murray PR, Medoff G, Zhang Y, Wallace RJ, Jr. Contamination of flexible fiberoptic bronchoscopes with *Mycobacterium chelonae* linked to an automated bronchoscope disinfection machine. *Am. Rev. Respir. Dis.* 1992;145:853-5.
135. Cooke RP, Whyman-Morris A, Umasankar RS, Goddard SV. Bacteria-free water for automatic washer-disinfectors: an impossible dream? *J. Hosp. Infect.* 1998;39:63-5.
136. Muscarella LF. Deja Vu...All over again? The importance of instrument drying. *Infect. Control Hosp. Epidemiol.* 2000;21:628-9.
137. Rutala WA, Weber DJ. Importance of lumen flow in liquid chemical sterilization. *Am. J. Infect. Control* 1999;20:458-9.
138. Dwyer DM, Klein EG, Istre GR, Robinson MG, Neumann DA, McCoy GA. *Salmonella newport* infections transmitted by fiberoptic colonoscopy. *Gastrointest. Endosc.* 1987;33:84-7.
139. Wheeler PW, Lancaster D, Kaiser AB. Bronchopulmonary cross-colonization and infection related to mycobacterial contamination of suction valves of bronchoscopes. *J. Infect. Dis.* 1989;159:954-8.
140. Bond WW. Virus transmission via fiberoptic endoscope: recommended disinfection. *JAMA* 1987;257:843-4.
141. Lynch DA, Porter C, Murphy L, Axon AT. Evaluation of four commercial automatic endoscope washing machines. *Endoscopy* 1992;24:766-70.
142. Bond WW. Disinfection and endoscopy: microbial considerations. *J. Gastroenterol. Hepatol.* 1991;6:31-6.
143. Nelson D. Newer technologies for endoscope disinfection: electrolyzed acid water and disposable-component endoscope systems. *Gastrointest. Endosc. Clin. N. Am.* 2000;10:319-28.
144. Silberman HD. Non-inflatable sterile sheath for introduction of the flexible nasopharyngolaryngoscope. *Ann Otol, Rhinol, Laryngol* 2001;110:385-7.
145. Kruse A, Rey JF. Guidelines on cleaning and disinfection in GI endoscopy. Update 1999. The European Society of Gastrointestinal Endoscopy. *Endoscopy* 2000;32:77-80.
146. British Thoracic Society. British Thoracic Society guidelines on diagnostic flexible bronchoscopy. *Thorax* 2001;56:1-21.
147. Association of Operating Room Nurses. Recommended practices for use and care of endoscopes. 2000 standards, recommended practices, and guidelines. Denver, CO: AORN, 2000:243-7.
148. British Society of Gastroenterology. Cleaning and disinfection of equipment for gastrointestinal endoscopy. Report of a working party of the British Society of Gastroenterology Endoscope Committee. *Gut* 1998;42:585-93.
149. Jackson FW, Ball MD. Correction of deficiencies in flexible fiberoptic sigmoidoscope cleaning and disinfection technique in family practice and internal medicine offices. *Arch. Fam. Med.* 1997;6:578-82.
150. Orsi GB, Filocamo A, Di Stefano L, Tittobello A. Italian National Survey of Digestive Endoscopy Disinfection Procedures. *Endoscopy* 1997;29:732-8; quiz 739-40.
151. Honeybourne D, Neumann CS. An audit of bronchoscopy practice in the United Kingdom: a survey of adherence to national guidelines. *Thorax* 1997;52:709-13.
152. Michele TM, Cronin WA, Graham NM, et al. Transmission of *Mycobacterium tuberculosis* by a fiberoptic bronchoscope. Identification by DNA fingerprinting. *JAMA* 1997;278:1093-5.

153. Bronowicki JP, Venard V, Botte C, et al. Patient-to-patient transmission of hepatitis C virus during colonoscopy. *N. Engl. J. Med.* 1997;337:237-40.
154. Agerton T, Valway S, Gore B, et al. Transmission of a highly drug-resistant strain (strain W1) of *Mycobacterium tuberculosis*. Community outbreak and nosocomial transmission via a contaminated bronchoscope. *JAMA* 1997;278:1073-7.
155. Food and Drug Administration, Centers for Disease Control and Prevention. FDA and CDC public health advisory: Infections from endoscopes inadequately reprocessed by an automated endoscope reprocessing system, Food and Drug Administration, Rockville, MD. 1999.
156. Nelson DB, Muscarella LF. Current issues in endoscope reprocessing and infection control during gastrointestinal endoscopy. *World J Gastroenterol* 2006;12:3953-64.
157. Riley R, Beanland C, Bos H. Establishing the shelf life of flexible colonoscopes. *Gastroenterol. Nurs.* 2002;25:114-9.
158. Rejchrt S, Cermak P, Pavlatova L, Mickova E, Bures J. Bacteriologic testing of endoscopes after high-level disinfection. *Gastrointest. Endosc.* 2004;60:76-8.
159. Willis C. Bacteria-free endoscopy rinse water - a realistic aim? *Epidemiol. Infect.* 2005;134:279-84.
160. Humphreys H, McGrath H, McCormick PA, Walsh C. Quality of final rinse water used in washer-disinfectors for endoscopes. *J. Hosp. Infect.* 2002;51:151-3.
161. Pang J, Perry P, Ross A, Forbes GM. Bacteria-free rinse water for endoscope disinfection. *Gastrointest. Endosc.* 2002;56:402-6.
162. Leung J, Vallero R, Wilson R. Surveillance cultures to monitor quality of gastrointestinal endoscope reprocessing. *Am. J. Gastroenterol.* 2003;98.
163. Moses FM, Lee J. Surveillance cultures to monitor quality of gastrointestinal endoscope reprocessing. *Am. J. Gastroenterol.* 2003;98:77-81.
164. Tunuguntla A, Sullivan MJ. Monitoring quality of flexible endoscopic disinfection by microbiologic surveillance cultures. *Tennessee Med* 2004;October:453-6.
165. Muscarella LF. Application of environmental sampling to flexible endoscope reprocessing: The importance of monitoring the rinse water. *Infect. Control Hosp. Epidemiol.* 2002;23:285-9.
166. Fraser TG, Reiner S, Malcznski M, Yarnold PR, Warren J, Noskin GA. Multidrug-resistant *Pseudomonas aeruginosa* cholangiopancreatography: Failure of routine endoscope cultures to prevent an outbreak. *Infect Control Hosp Epidemiol* 2004;25:856-9.
167. Bond WW, Hedrick ER. Microbiological culturing of environmental and medical-device surfaces. In: Isenberg HD, and M.J.R. Gilchrist, ed. *Clinical Microbiology Procedures Handbook*, Section 11, Epidemiologic and Infection Control Microbiology. Washington, DC: American Society for Microbiology, 1992:11.10.1-11.10.9.
168. Murray PR, Baron EJ, Pfaller MA, Jorgensen JH, Tenover FC, Tenover FC, eds. *Manual of Clinical Microbiology*. In: Murray PR, Baron EJ, Pfaller MA, Jorgensen JH, Tenover FC, Tenover FC, eds. Washington, D.C.: American Society for Microbiology Press, 2003.
169. Blob R, Kampf G. Test models to determine cleaning efficacy with different types of bioburden and its clinical correlation. *J. Hosp. Infect.* 2004;56 (suppl):S44-S48.
170. Obee PC, Griffith CJ, Cooper RA, Cooke RP, Bennion NE, Lewis M. Real-time monitoring in managing the decontamination of flexible gastrointestinal endoscopes. *Am. J. Infect. Control* 2005;33:202-6.
171. Sciortino CV, Xia EL, Mozee A. Assessment of a novel approach to evaluate the outcome of endoscope reprocessing. *Infect Control Hosp Epidemiol* 2004;25:284-90.
172. Murphy C. Inactivated glutaraldehyde: Lessons for infection control. *Am. J. Infect. Control* 1998;26:159-60.
173. Carsaw H, Debacker N. Recall of patients after use of inactive batch of Cidex disinfection solution in Belgian hospitals, Fifth International Conference of the Hospital Infection Society, Edinburgh, September 15-18, 2002. Hospital Infections Society.
174. Ad hoc Committee on Infection Control in the Handling of Endoscopic Equipment. Guidelines for preparation of laparoscopic instrumentation. *AORN J.* 1980;32:65-6, 70, 74, 76.
175. Taylor EW, Mehtar S, Cowan RE, Feneley RC. Endoscopy: disinfectants and health. Report of a meeting held at the Royal College of Surgeons of England, February 1993. *J. Hosp. Infect.* 1994;28:5-14.
176. Hulka JF, Wisler MG, Bruch C. A discussion: laparoscopic instrument sterilization. *Med. Instrum.* 1977;11:122-3.

177. Corson SL, Block S, Mintz C, Dole M, Wainwright A. Sterilization of laparoscopes. Is soaking sufficient? *J. Reprod. Med.* 1979;23:49-56.
178. Corson SL, Dole M, Kraus R, Richards L, Logan B. Studies in sterilization of the laparoscope: II. *J. Reprod. Med.* 1979;23:57-9.
179. Chan-Myers H, McAlister D, Antonoplos P. Natural bioburden levels detected on rigid lumened medical devices before and after cleaning. *Am. J. Infect. Control* 1997;25:471-6.
180. Rodrigues C, Mehta AC, Jha U, Bharucha M, Dastur FD, Udwardia TE. Nosocomial *Mycobacterium chelonae* infection in laparoscopic surgery. *Infect. Control Hosp. Epidemiol.* 2001;22:474-5.
181. Marshburn PB, Rutala WA, Wannamaker NS, Hulka JF. Gas and steam sterilization of assembled versus disassembled laparoscopic equipment. *Microbiologic studies. J. Reprod. Med.* 1991;36:483-7.
182. Bernhang AM. Clostridium pyoarthrosis following arthroscopy. *Arthroscopy* 1987;3:56-8.
183. D'Angelo GL, Ogilvie-Harris DJ. Septic arthritis following arthroscopy, with cost/benefit analysis of antibiotic prophylaxis. *Arthroscopy* 1988;4:10-4.
184. Weber DJ, Rutala WA. Nosocomial ocular infections. In: Mayhall CG, ed. *Infect. Control and Hosp. Epidemiol.* Philadelphia: Lippincott Williams & Wilkins, 1999:287-99.
185. Rutala WA, Peacock JE, Gergen MF, Sobsey MD, Weber DJ. Efficacy of hospital germicides against adenovirus 8, a common cause of epidemic keratoconjunctivitis in health care facilities. *Antimicrob. Agents Chemother.* 2006;50:1419-24.
186. Sattar SA, Springthorpe VS, Karim Y, Loro P. Chemical disinfection of non-porous inanimate surfaces experimentally contaminated with four human pathogenic viruses. *Epidemiol. Infect.* 1989;102:493-505.
187. Chronister CL. Structural damage to Schiottz tonometers after disinfection with solutions. *Optom. Vis. Sci.* 1997;74:164-6.
188. Nagington J, Sutehall GM, Whipp P. Tonometer disinfection and viruses. *Br. J. Ophthalmol.* 1983;67:674-6.
189. Craven ER, Butler SL, McCulley JP, Luby JP. Applanation tonometer tip sterilization for adenovirus type 8. *Ophthalmology* 1987;94:1538-40.
190. American Academy of Ophthalmology. Updated recommendations for ophthalmic practice in relation to the human immunodeficiency virus. American Academy of Ophthalmology, San Francisco, CA, 1988.
191. Pepose JS, Linette G, Lee SF, MacRae S. Disinfection of Goldmann tonometers against human immunodeficiency virus type 1. *Arch. Ophthalmol.* 1989;107:983-5.
192. Ventura LM, Dix RD. Viability of herpes simplex virus type 1 on the applanation tonometer. *Am. J. Ophthalmol.* 1987;103:48-52.
193. Koo D, Bouvier B, Wesley M, Courtright P, Reingold A. Epidemic keratoconjunctivitis in a university medical center ophthalmology clinic; need for re-evaluation of the design and disinfection of instruments. *Infect. Control Hosp. Epidemiol.* 1989;10:547-52.
194. Jernigan JA, Lowry BS, Hayden FG, et al. Adenovirus type 8 epidemic keratoconjunctivitis in an eye clinic: risk factors and control. *J. Infect. Dis.* 1993;167:1307-13.
195. Fritz S, Hust MH, Ochs C, Gratwohl I, Staiger M, Braun B. Use of a latex cover sheath for transesophageal echocardiography (TEE) instead of regular disinfection of the echoscope? *Clin. Cardiol.* 1993;16:737-40.
196. Lawrentschuk N, Chamberlain M. Sterile disposable sheath system for flexible cystoscopes. *Urology* 2005;66:1310-3.
197. Milki AA, Fisch JD. Vaginal ultrasound probe cover leakage: implications for patient care. *Fertil. Steril.* 1998;69:409-11.
198. Storment JM, Monga M, Blanco JD. Ineffectiveness of latex condoms in preventing contamination of the transvaginal ultrasound transducer head. *South. Med. J.* 1997;90:206-8.
199. Hignett M, Claman P. High rates of perforation are found in endovaginal ultrasound probe covers before and after oocyte retrieval for *in vitro* fertilization-embryo transfer. *J. Assist. Reprod. Genet.* 1995;12:606-9.
200. Amis S, Ruddy M, Kibbler CC, Economides DL, MacLean AB. Assessment of condoms as probe covers for transvaginal sonography. *J. Clin. Ultrasound* 2000;28:295-8.
201. Rooks VJ, Yancey MK, Elg SA, Brueske L. Comparison of probe sheaths for endovaginal sonography. *Obstet. Gynecol.* 1996;87:27-9.
202. Odwin CS, Fleischer AC, Kepple DM, Chiang DT. Probe covers and disinfectants for transvaginal transducers. *J. Diagnostic Med. Sonography* 1990;6:130-5.
203. Benson WG. Exposure to glutaraldehyde. *J. Soc. Occup. Med.* 1984;34:63-4.

204. Garland SM, de Crespigny L. Prevention of infection in obstetrical and gynaecological ultrasound practice. *Aust. N. Z. J. Obstet Gynaecol.* 1996;36:392-5.
205. Fowler C, McCracken D. US probes: risk of cross infection and ways to reduce it--comparison of cleaning methods. *Radiology* 1999;213:299-300.
206. Muradali D, Gold WL, Phillips A, Wilson S. Can ultrasound probes and coupling gel be a source of nosocomial infection in patients undergoing sonography? An in vivo and in vitro study. *AJR. Am. J. Roentgenol.* 1995;164:1521-4.
207. Lewis DL, Arens M, Appleton SS, et al. Cross-contamination potential with dental equipment. *Lancet* 1992;340:1252-4.
208. Lewis DL, Boe RK. Cross-infection risks associated with current procedures for using high-speed dental handpieces. *J. Clin. Microbiol.* 1992;30:401-6.
209. American Dental Association. Infection control recommendations for the dental office and the dental laboratory. *JADA* 1996;127:672-80.
210. Centers for Disease Control. Recommended Infection-Control Practices for Dentistry, 1993. *MMWR* 1993;41:1-12.
211. Department of Health and Human Services. Food and Drug Administration. Dental handpiece sterilization, Food and Drug Administration, Rockville, MD, 1992.
212. Silverstone SE, Hill DE. Evaluation of sterilization of dental handpieces by heating in synthetic compressor lubricant. *Gen. Dent.* 1999;47:158-60.
213. Goodman HS, Carpenter RD, Cox MR. Sterilization of dental instruments and devices: an update. *Am. J. Infect. Control* 1994;22:90-4.
214. Occupational Safety and Health Administration. Occupational exposure to bloodborne pathogens; final rule. *Fed. Regist.* 1991;56:64003-182.
215. Occupational Safety and Health Administration. OSHA Memorandum from Stephen Mallinger. EPA-registered disinfectants for HIV/HBV. Washington, DC, 1997.
216. Gurevich I, Dubin R, Cunha BA. Dental instrument and device sterilization and disinfection practices. *J. Hosp. Infect.* 1996;32:295-304.
217. Smith A, Dickson M, Aitken J, Bagg J. Contaminated dental instruments. *J. Hosp. Infect.* 2002;51:233-5.
218. Hastreiter RJ, Molinari JA, Falken MC, Roesch MH, Gleason MJ, Merchant VA. Effectiveness of dental office instrument sterilization procedures. *J. Am. Dent. Assoc.* 1991;122:51-6.
219. Andres MT, Tejerina JM, Fierro JF. Reliability of biologic indicators in a mail-return sterilization-monitoring service: a review of 3 years. *Quintessence Int.* 1995;26:865-70.
220. Miller CH, Sheldrake MA. The ability of biological indicators to detect sterilization failures. *Am. J. Dent.* 1994;7:95-7.
221. Sarin PS, Scheer DI, Kross RD. Inactivation of human T-cell lymphotropic retrovirus (HTLV-III) by LD. *N. Engl. J. Med.* 1985;313:1416.
222. Sarin PS, Scheer DI, Kross RD. Inactivation of human T-cell lymphotropic retrovirus. *Environ Microbiol* 1990;56:1423-8.
223. Ascenzi JM. Standardization of tuberculocidal testing of disinfectants. *J. Hosp. Infect.* 1991;18:256-63.
224. Bond WW, Favero MS, Petersen NJ, Ebert JW. Inactivation of hepatitis B virus by intermediate-to-high-level disinfectant chemicals. *J. Clin. Microbiol.* 1983;18:535-8.
225. Kobayashi H, Tsuzuki M. The effect of disinfectants and heat on hepatitis B virus. *J. Hosp. Infect.* 1984;5:93-4.
226. Spire B, Barre-Sinoussi F, Montagnier L, Chermann JC. Inactivation of lymphadenopathy associated virus by chemical disinfectants. *Lancet* 1984;2:899-901.
227. Martin LS, McDougal JS, Loskoski SL. Disinfection and inactivation of the human T lymphotropic virus type III/Lymphadenopathy-associated virus. *J. Infect. Dis.* 1985;152:400-3.
228. Centers for Disease Control. Recommendations for prevention of HIV transmission in health-care settings. *MMWR* 1987;36:S3-S18.
229. Prince DL, Prince HN, Thraenhart O, Muchmore E, Bonder E, Pugh J. Methodological approaches to disinfection of human hepatitis B virus. *J. Clin. Microbiol.* 1993;31:3296-304.
230. Prince DL, Prince RN, Prince HN. Inactivation of human immunodeficiency virus type 1 and herpes simplex virus type 2 by commercial hospital disinfectants. *Chemical Times and Trends* 1990;13:13-16.
231. Sattar SA, Springthorpe VS, Conway B, Xu Y. Inactivation of the human immunodeficiency virus: an

- update. Rev. Med. Microbiol. 1994;5:139-150.
232. Kaplan JC, Crawford DC, Durno AG, Schooley RT. Inactivation of human immunodeficiency virus by Betadine. Infect. Control 1987;8:412-4.
 233. Hanson PJ, Gor D, Jeffries DJ, Collins JV. Chemical inactivation of HIV on surfaces. Br. Med. J. 1989;298:862-4.
 234. Hanson PJ, Jeffries DJ, Collins JV. Viral transmission and fiberoptic endoscopy. J. Hosp. Infect. 1991;18:136-40.
 235. Payan C, Cottin J, Lemarie C, Ramont C. Inactivation of hepatitis B virus in plasma by hospital in-use chemical disinfectants assessed by a modified HepG2 cell culture. J. Hosp. Infect. 2001;47:282-87.
 236. Chanzy B, Duc-Bin DL, Rousset B, et al. Effectiveness of a manual disinfection procedure in eliminating hepatitis C virus from experimentally contaminated endoscopes. Gastrointest. Endosc. 1999;50:147-51.
 237. Druce JD, Russell JS, Birch CJ, Yates LA, Harper RW, Smolich JJ. A decontamination and sterilization protocol employed during reuse of cardiac electrophysiology catheters inactivates human immunodeficiency virus. Infect. Control Hosp. Epidemiol. 2003;24:184-90.
 238. Payan C, Pivert A, Kampf G, Ramont C, Cottin J, Lemarie C. Assessment of new chemical disinfectants for HBV virucidal activity in a cell culture model. J. Hosp. Infect. 2004;56 (suppl):S58-S63.
 239. Reynolds CD, Rhinehart E, Dreyer P, Goldmann DA. Variability in reprocessing policies and procedures for flexible fiberoptic endoscopes in Massachusetts hospitals. Am. J. Infect. Control 1992;20:283-90.
 240. Handsfield HH, Cummings MJ, Swenson PD. Prevalence of antibody to human immunodeficiency virus and hepatitis B surface antigen in blood samples submitted to a hospital laboratory. Implications for handling specimens. JAMA 1987;258:3395-7.
 241. Baker JL, Kelen GD, Sivertson KT, Quinn TC. Unsuspected human immunodeficiency virus in critically ill emergency patients. JAMA 1987;257:2609-11.
 242. Kelen GD, Fritz S, Qaqish B, et al. Unrecognized human immunodeficiency virus infection in emergency department patients. N. Engl. J. Med. 1988;318:1645-50.
 243. Ishino Y, Ido K, Sugano K. Contamination with hepatitis B virus DNA in gastrointestinal endoscope channels: Risk of infection on reuse after on-site cleaning. Endoscopy 2005;37:548-51.
 244. Agolini G, Russo A, Clementi M. Effect of phenolic and chlorine disinfectants on hepatitis C virus binding and infectivity. Am. J. Infect. Control 1999;27:236-9.
 245. Alter MJ, Tokars JI, Arduino MJ, Favero MS. Nosocomial infections with hemodialysis. In: Mayhall CG, ed. Infect. Control and Hosp. Epidemiol. Philadelphia: Lippincott Williams & Wilkins, 2004:1139-60.
 246. Centers for Disease Control. Recommendations for preventing transmission of infections among chronic hemodialysis patients. MMWR. 2001;50:1-43.
 247. Velandia M, Fridkin SK, Cardenas V, et al. Transmission of HIV in dialysis centre. Lancet 1995;345:1417-22.
 248. Guinto CH, Bottone EJ, Raffalli JT, Montecalvo MA, Wormser GP. Evaluation of dedicated stethoscopes as a potential source of nosocomial pathogens. Am. J. Infect. Control 2002;30:499-502.
 249. Tokars JI, Miller ER, Alter MJ, Arduino MJ. National surveillance of dialysis-associated diseases in the United States, 1997. Semin. Dialysis 2000;13:75-85.
 250. Amato RL, Curtis JM. The practical application of ozone in dialysis. Nephrol. News Issues 2002;September 27-9.
 251. Smeets E, Koonman J, van der Sande F, et al. Prevention of biofilm formation in dialysis water treatment systems. Kidney Int. 2003;63:1574-6.
 252. Finelli L, Miller JT, Tokars JI, Alter MJ, Arduino MJ. National surveillance of dialysis-associated diseases in the United States, 2002. Semin Dialysis 2005;18:52-61.
 253. Association for the Advancement of Medical Instrumentation. Reuse of hemodialyzers: Association for the Advancement of Medical Instrumentation, Arlington VA, 2002/2003:ANSI/AAMI RD47:2002 & RD47:2002/A1:2003; 1-32.
 254. Kim KH, Fekety R, Batts DH, et al. Isolation of *Clostridium difficile* from the environment and contacts of patients with antibiotic-associated colitis. J. Infect. Dis. 1981;143:42-50.
 255. Skoutelis AT, Westenfelder GO, Beckerdite M, Phair JP. Hospital carpeting and epidemiology of *Clostridium difficile*. Am. J. Infect. Control 1994;22:212-7.
 256. Wilcox MH, Fawley WN. Hospital disinfectants and spore formation by *Clostridium difficile*. Lancet 2000;356:1324.

257. Kaatz GW, Gitlin SD, Schaberg DR, et al. Acquisition of *Clostridium difficile* from the hospital environment. *Am. J. Epidemiol.* 1988;127:1289-94.
258. Wilcox MH, Fawley WN, Wigglesworth N, Parnell P, Verity P, Freeman J. Comparison of the effect of detergent versus hypochlorite cleaning on environmental contamination and incidence of *Clostridium difficile* infection. *J. Hosp. Infect.* 2003;54:109-14.
259. Mayfield JL, Leet T, Miller J, Mundy LM. Environmental control to reduce transmission of *Clostridium difficile*. *Clin. Infect. Dis.* 2000;31:995-1000.
260. Marler LM, Siders JA, Wolters LC, Pettigrew Y, Skitt BL, Allen SD. Comparison of five cultural procedures for isolation of *Clostridium difficile* from stools. *J. Clin. Microbiol.* 1992;30:514-6.
261. Brazier JG. The diagnosis of *Clostridium difficile*-associated disease. *J. Antimicrob. Chemother.* 1998;41 (suppl):29-40.
262. Perez J, Springthorpe S, Sattar SA. Activity of selected oxidizing microbicides against spores of *Clostridium difficile*: Relevance to environmental control. *Am. J. Infect. Control* 2005;33:320-5.
263. McFarland LV, Mulligan ME, Kwok RY, Stamm WE. Nosocomial acquisition of *Clostridium difficile* infection. *N. Engl. J. Med.* 1989;320:204-10.
264. Jernigan JA, Siegman-Igra Y, Guerrant RC, Farr BM. A randomized crossover study of disposable thermometers for prevention of *Clostridium difficile* and other nosocomial infections. *Infect Control Hosp Epidemiol* 1998;19:494-9.
265. Hughes CE, Gebhard RL, Peterson LR, Gerding DN. Efficacy of routine fiberoptic endoscope cleaning and disinfection for killing *Clostridium difficile*. *Gastrointest. Endosc.* 1986;32:7-9.
266. Dyas A, Das BC. The activity of glutaraldehyde against *Clostridium difficile*. *J. Hosp. Infect.* 1985;6:41-5.
267. Wullt M, Odenholt I, Walder M. Activity of three disinfectants and acidified nitrite against *Clostridium difficile* spores. *Infect Control Hosp Epidemiol* 2003;24:765-8.
268. Block C. The effect of Perasafe and sodium dichloroisocyanurate (NaDCC) against spores of *Clostridium difficile* and *Bacillus atrophaeus* on stainless steel and polyvinyl chloride surfaces. *J. Hosp. Infect.* 2004;57:144-8.
269. Occupational Safety and Health Administration. OSHA instruction CPL 2-2.44C. Office of Health Compliance Assistance. Washington, DC, 1992.
270. Rutala WA, Weber DJ. Infection control: the role of disinfection and sterilization. *J. Hosp. Infect.* 1999;43:S43-55.
271. Barbee SL, Weber DJ, Sobsey MD, Rutala WA. Inactivation of *Cryptosporidium parvum* oocyst infectivity by disinfection and sterilization processes. *Gastrointest. Endosc.* 1999;49:605-11.
272. Wilson JA, Margolin AB. The efficacy of three common hospital liquid germicides to inactivate *Cryptosporidium parvum* oocysts. *J. Hosp. Infect.* 1999;42:231-7.
273. Fayer R, Graczyk TK, Cranfield MR, Trout JM. Gaseous disinfection of *Cryptosporidium parvum* oocysts. *Appl. Environ. Microbiol.* 1996;62:3908-9.
274. Venkitanarayanan KS, Ezeike GO, Hung YC, Doyle MP. Inactivation of *Escherichia coli* O157:H7 and *Listeria monocytogenes* on plastic kitchen cutting boards by electrolyzed oxidizing water. *J. Food Prot.* 1999;62:857-60.
275. Taormina PJ, Beuchat LR. Behavior of enterohemorrhagic *Escherichia coli* O157:H7 on alfalfa sprouts during the sprouting process as influenced by treatments with various chemicals. *J. Food Prot.* 1999;62:850-6.
276. Taormina PJ, Beuchat LR. Comparison of chemical treatments to eliminate enterohemorrhagic *Escherichia coli* O157:H7 on alfalfa seeds. *J. Food Prot.* 1999;62:318-24.
277. Castillo A, Lucia LM, Kemp GK, Acuff GR. Reduction of *Escherichia coli* O157:H7 and *Salmonella typhimurium* on beef carcass surfaces using acidified sodium chlorite. *J. Food Prot.* 1999;62:580-4.
278. Graham DY, Osato MS. Disinfection of biopsy forceps and culture of *Helicobacter pylori* from gastric mucosal biopsies. *Am. J. Gastroenterol.* 1999;94:1422-3.
279. Kaneko H, Mitsuma T, Kotera H, Uchida K, Furusawa A, Morise K. Are routine cleaning methods sufficient to remove *Helicobacter pylori* from endoscopic equipment? *Endoscopy* 1993;25:435.
280. Langenberg W, Rauws EA, Oudbier JH, Tytgat GN. Patient-to-patient transmission of *Campylobacter pylori* infection by fiberoptic gastroduodenoscopy and biopsy. *J. Infect. Dis.* 1990;161:507-11.
281. Miyaji H, Kohli Y, Azuma T, et al. Endoscopic cross-infection with *Helicobacter pylori*. *Lancet* 1995;345:464.

282. Fantry GT, Zheng QX, James SP. Conventional cleaning and disinfection techniques eliminate the risk of endoscopic transmission of *Helicobacter pylori*. *Am. J. Gastroenterol.* 1995;90:227-32.
283. Shimada T, Terano A, Ota S, Takikawa H, Sumino S. Risk of iatrogenic transmission of *Helicobacter pylori* by gastroscopes. *Lancet* 1996;347:1342-3.
284. Roosendaal R, Kuipers EJ, van den Brule AJ, et al. Detection of *Helicobacter pylori* DNA by PCR in gastrointestinal equipment. *Lancet* 1993;341:900.
285. Johnson CH, Rice EW, Reasoner DJ. Inactivation of *Helicobacter pylori* by chlorination. *Appl. Environ. Microbiol.* 1997;63:4969-70.
286. Chapin M, Yatabe J, Cherry JD. An outbreak of rotavirus gastroenteritis on a pediatric unit. *Am. J. Infect. Control* 1983;11:88-91.
287. Keswick BH, Pickering LK, DuPont HL, Woodward WE. Survival and detection of rotaviruses on environmental surfaces in day care centers. *Appl. Environ. Microbiol.* 1983;46:813-16.
288. Ansari SA, Spingthorpe S, Sattar SA. Survival and vehicular spread of human rotaviruses: Possible relation to seasonality of outbreaks. *Rev. Infect. Dis.* 1991;13:448-61.
289. Ansari SA, Sattar SA, Springthorpe VS, Wells GA, Tostowaryk W. Rotavirus survival on human hands and transfer of infectious virus to animate and nonporous inanimate surfaces. *J. Clin. Microbiol.* 1988;26:1513-8.
290. Sattar SA, Raphael RA, Lochnan H, Springthorpe VS. Rotavirus inactivation by chemical disinfectants and antiseptics used in hospitals. *Can. J. Microbiol.* 1983;29:1464-9.
291. Lloyd-Evans N, Springthorpe VS, Sattar SA. Chemical disinfection of human rotavirus-contaminated inanimate surfaces. *J. Hyg. (Lond).* 1986;97:163-73.
292. Tan JA, Schnagl RD. Inactivation of a rotavirus by disinfectants. *Med. J. Aust.* 1981;1:19-23.
293. Sattar SA, Springthorpe VS, Karim Y, Loro P. Chemical disinfection of non-porous inanimate surfaces experimentally contaminated with four human pathogenic viruses. *Epidemiol Infect* 1989;102:493-505.
294. Green J, Wright PA, Gallimore CI, Mitchell O, Morgan-Capner P, Brown DWG. The role of environmental contamination with small round structured viruses in a hospital outbreak investigated by reverse-transcriptase polymerase chain reaction assay. *J. Hosp. Infect.* 1998;39:39-45.
295. Evans MR, Meldrum R, Lane W, et al. An outbreak of viral gastroenteritis following environmental contamination at a concert hall. *Epidemiol & Infect* 2002;129:355-360.
296. Marks PJ, Vipond IB, Regan FM, Wedgwood K, Fey RE, Caul EO. A school outbreak of Norwalk-like virus: Evidence for airborne transmission. *Epidemiol Infect* 2003;131:727-36.
297. Doultree JC, Druce JD, Birch CJ, Bowden DS, Marshall JA. Inactivation of feline calicivirus, a Norwalk virus surrogate. *J. Hosp. Infect.* 1999;41:51-7.
298. Sattar SA. Microbicides and the environmental control of nosocomial viral infections. *J. Hosp. Infect.* 2004;56 (suppl):S64-S69.
299. Jimenez L, Chiang M. Virucidal activity of a quaternary ammonium compound disinfectant against feline calicivirus: A surrogate for norovirus. *Am. J. Infect. Control* 2006;34:269-73.
300. Gehrke C, Steinmann J, Goroncy-Bermes P. Inactivation of feline calicivirus, a surrogate of norovirus (formerly Norwalk-like viruses), by different types of alcohol in vitro and in vivo. *J. Hosp. Infect.* 2004;56:49-55.
301. Centers for Disease Control and Prevention. Update: Severe acute respiratory syndrome - United States, May 14, 2003. *MMWR* 2003;52:436-8.
302. Saknimit M, Inatsuki I, Sugiyama Y, Yagami K. Virucidal efficacy of physico-chemical treatments against coronaviruses and parvoviruses of laboratory animals. *Jikken Dobutsu.* 1988;37:341-5.
303. Sizun J, Yu MW, Talbot PJ. Survival of human coronaviruses 229E and OC43 in suspension and after drying on surfaces: a possible source of hospital-acquired infections. *J. Hosp. Infect.* 2000;46:55-60.
304. Kariwa H, Fujii N, Takashima I. Inactivation of SARS coronavirus by means of povidone-iodine, physical conditions, and chemical reagents. *Jpn. J. Vet. Res.* 2004;52:105-12.
305. Greub G, Raoult D. Biocides currently used for bronchoscope decontamination are poorly effective against free-living amoebae. *Infect Control Hosp Epidemiol* 2003;24:784-6.
306. Leggiadro RJ. The threat of biological terrorism: A public health and infection control reality. *Infect. Control Hosp. Epidemiol.* 2000;21:53-6.
307. Henderson DA. The looming threat of bioterrorism. *Science* 1999;283:1279-82.
308. Centers for Disease Control. Biological and chemical terrorism: strategic plan for preparedness and

- response. MMWR 2000;49 (no. RR-4):1-14.
309. Weber DJ, Rutala WA. Disinfection and sterilization of potential bioterrorism agents. In: Rutala WA, ed. Disinfection, sterilization and antisepsis: Principles, practices and new research. Washington DC: Association for Professionals in Infection Control and Epidemiology, 2004:86-103.
 310. Ferrier A, Garin D, Crance JM. Rapid inactivation of vaccinia virus in suspension and dried on surfaces. J. Hosp. Infect. 2004;57:73-9.
 311. Butcher W, Ulaeto D. Contact inactivation of orthopoxviruses by household disinfectants. J. Appl. Microbiol. 2005;99:279-84.
 312. Brazis AR, Leslie JE, PW K, RL W. The inactivation of spores of *Bacillus globigii* and *Bacillus anthracis* by free available chlorine. Appl. Microbiol. 1958;6:338-342.
 313. Sattar SA, Springthorpe VS, Adegbonrin O. Is *Bacillus subtilis* (ATCC 19659) a suitable surrogate for evaluating microbicides against *Bacillus anthracis*., Association for Official Analytical Chemists International Annual Meeting, St. Louis, Missouri, 2004.
 314. Whitney EAS, Beatty ME, Taylor TH Jr, et al. Inactivation of *Bacillus anthracis* spores. Emerg. Infect. Dis. 2003;9:623-7.
 315. Weber DJ, Rutala WA. Risks and prevention of nosocomial transmission of rare zoonotic diseases. Clin. Infect. Dis. 2001;32:446-456.
 316. Chataigner D, Garnier R, Sans S, Efthymiou ML. [Acute accidental poisoning with hospital disinfectant. 45 cases of which 13 with fatal outcome]. Presse Med. 1991;20:741-3.
 317. Hess JA, Molinari JA, Gleason MJ, Radecki C. Epidermal toxicity of disinfectants. Am. J. Dent. 1991;4:51-6.
 318. Weber DJ, Rutala WA. Occupational risks associated with the use of selected disinfectants and sterilants. In: Rutala WA, ed. Disinfection, sterilization, and antisepsis in healthcare. Champlain, New York: Polyscience Publications, 1998:211-26.
 319. Cokendolpher JC, Haukos JF. The Practical Application of Disinfection and Sterilization in Health Care Facilities. Chicago: American Hospital Association, 1996.
 320. Rideout K, Teschke K, Dimich-Ward H, Kennedy SM. Considering risks to healthcare workers from glutaraldehyde alternatives in high-level disinfection. J. Hosp. Infect. 2005;59:4-11.
 321. Oie S, Kamiya A. Assessment of and intervention for the misuse of aldehyde disinfectants in Japan. Infect. Control Hosp. Epidemiol. 2002;23:98-9.
 322. American Conference of Governmental Industrial Hygienists (ACGIH). Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Cincinnati: ACGIH, 2001.
 323. Jordan SLP, Russo MR, Blessing RL, Grab LA. Glutaraldehyde safety: inactivation and disposal. Abstract. Am. J. Infect. Control 1997;25:154-55.
 324. Jordan SL. The correct use of glutaraldehyde in the healthcare environment. Gastroenterol. Nurs. 1995;18:143-5.
 325. Cheung HY, Brown MR. Evaluation of glycine as an inactivator of glutaraldehyde. J Pharmacy Pharmacol 1982;34:211-4.
 326. Daschner F. The hospital and pollution: Role of the hospital epidemiologist in protecting the environment. In: Wenzel RP, ed. Prevention and control of nosocomial infections. Baltimore: Williams and Wilkins, 1997:595-605.
 327. Rutala WA, Cole EC, Thomann CA, Weber DJ. Stability and bactericidal activity of chlorine solutions. Infect. Control Hosp. Epidemiol. 1998;19:323-7.
 328. Rutala WA, Weber DJ. Uses of inorganic hypochlorite (bleach) in health-care facilities. Clin. Microbiol. Rev. 1997;10:597-610.
 329. Dychdala GR. Chlorine and chlorine compounds. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lippincott Williams & Wilkins, 2001:135-157.
 330. Rutala WA, Weber DJ. Principles of disinfecting patient-care items. In: Rutala WA, ed. Disinfection, sterilization, and antisepsis in healthcare. Champlain, New York: Polyscience Publications, 1998:133-49.
 331. Luebbert P. Home care. In: Pfeiffer JA, ed. APIC text of infection control and epidemiology. Vol. 1. Washington: Association for Professionals in Infection control and epidemiology, 2000:44-7.
 332. Parnes CA. Efficacy of sodium hypochlorite bleach and "alternative" products in preventing transfer of bacteria to and from inanimate surfaces. Environ. Health 1997;59:14-20.
 333. Karapinar M, Gonul SA. Effects of sodium bicarbonate, vinegar, acetic and citric acids on growth and

- survival of *Yersinia enterocolitica*. Int. J. Food Microbiol. 1992;16:343-7.
334. McMurry LM, Oethinger M, Levy SB. Triclosan targets lipid synthesis. Nature 1998;394:531-2.
335. Moken MC, McMurry LM, Levy SB. Selection of multiple-antibiotic-resistant (*mar*) mutants of *Escherichia coli* by using the disinfectant pine oil: roles of the *mar* and *acrAB* loci. Antimicrob. Agents Chemother. 1997;41:2770-2.
336. Scott E, Bloomfield SF, Barlow CG. An investigation of microbial contamination in the home. J. Hyg. (Lond). 1982;89:279-93.
337. Rusin P, Orosz-Coughlin P, Gerba C. Reduction of faecal coliform, coliform and heterotrophic plate count bacteria in the household kitchen and bathroom by disinfection with hypochlorite cleaners. J. Appl. Microbiol. 1998;85:819-28.
338. Gilbert P, McBain AJ. Potential impact of increased use of biocides in consumer products on prevalence of antibiotic resistance. Clin Microbiol Reviews 2003;16:189-208.
339. Bueumer R, Bloomfield SF, Exner M, Fara G, Scott EA. The need for a home hygiene policy and guidelines on home hygiene. Ann. Ig. 1999;11:11-26.
340. International Scientific Forum on Home Hygiene. www.ifh-homehygiene.org.
341. Russell AD, Russell NJ. Biocides: activity, action and resistance. In: Hunter PA, Darby GK, Russell NJ, eds. Fifty years of antimicrobials: past perspectives and future trends. England: Cambridge University Press, 1995:327-65.
342. Russell AD. Bacterial resistance to disinfectants: Present knowledge and future problems. J. Hosp. Infect. 1998;43:S57-S68.
343. Russell AD. Plasmids and bacterial resistance to biocides. J. Appl. Microbiol. 1997;83:155-65.
344. Russell AD. Bacterial resistance to disinfectants: present knowledge and future problems. J. Hosp. Infect. 1998;43:S57-68.
345. Russell AD. Principles of antimicrobial activity and resistance. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lippincott Williams & Wilkins, 2001:31-55.
346. McDonnell G, Russell AD. Antiseptics and disinfectants: activity, action, and resistance. Clin. Microbiol. Rev. 1999;12:147-79.
347. Gerba CP, Rusin P. Relationship between the use of antiseptics/disinfectants and the development of antimicrobial resistance. In: Rutala WA, ed. Disinfection, sterilization and antisepsis: principles and practices in healthcare facilities. Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:187-94.
348. Townsend DE, Ashdown N, Greed LC, Grubb WB. Transposition of gentamicin resistance to staphylococcal plasmids encoding resistance to cationic agents. J. Antimicrob. Chemother. 1984;14:115-24.
349. Brumfitt W, Dixon S, Hamilton-Miller JM. Resistance to antiseptics in methicillin and gentamicin resistant *Staphylococcus aureus*. Lancet 1985;1:1442-3.
350. Al-Masaudi SB, Day MJ, Russell AD. Sensitivity of methicillin-resistant *Staphylococcus aureus* strains to some antibiotics, antiseptics and disinfectants. J. Appl. Bacteriol. 1988;65:329-37.
351. Tennent JM, Lyon BR, Midgley M, Jones IG, Purewal AS, Skurray RA. Physical and biochemical characterization of the *qacA* gene encoding antiseptic and disinfectant resistance in *Staphylococcus aureus*. J. Gen. Microbiol. 1989;135:1-10.
352. Kaulfers PM, Laufs R. [Transmissible formaldehyde resistance in *Serratia marcescens*]. Zentralblatt für Bakteriologie, Mikrobiologie und Hygiene - 1 - Abt - Originale B, Hygiene 1985;181:309-19.
353. Tennent JM, Lyon BR, Gillespie MT, May JW, Skurray RA. Cloning and expression of *Staphylococcus aureus* plasmid-mediated quaternary ammonium resistance in *Escherichia coli*. Antimicrob. Agents Chemother. 1985;27:79-83.
354. Rutala WA, Stiegel MM, Sarubbi FA, Weber DJ. Susceptibility of antibiotic-susceptible and antibiotic-resistant hospital bacteria to disinfectants. Infect. Control Hosp. Epidemiol. 1997;18:417-21.
355. Anderson RL, Carr JH, Bond WW, Favero MS. Susceptibility of vancomycin-resistant enterococci to environmental disinfectants. Infect. Control Hosp. Epidemiol. 1997;18:195-9.
356. Sakagami Y, Kajimura K. Bactericidal activities of disinfectants against vancomycin-resistant enterococci. J. Hosp. Infect. 2002;50:140-4.
357. Sehulster LM, Anderson RL. Susceptibility of glycopeptide-intermediate resistant *Staphylococcus aureus* (GISA) to surface disinfectants, hand washing chemicals, and a skin antiseptic. Abstract Y-3. 98th General

- Meeting of American Society for Microbiology, May, 1998:547.
358. Rutala WA, Weber DJ, Gergen MF. Studies on the disinfection of VRE-contaminated surfaces. *Infect. Control Hosp. Epidemiol.* 2000;21:548.
359. Byers KE, Durbin LJ, Simonton BM, Anglim AM, Adal KA, Farr BM. Disinfection of hospital rooms contaminated with vancomycin-resistant *Enterococcus faecium*. *Infect. Control Hosp. Epidemiol.* 1998;19:261-4.
360. Carling PC, Briggs JL, Perkins J, Highlander D. Improved cleaning of patient rooms using a new targeting method. *Clin Inf Dis* 2006;42:385-8.
361. Russell AD, Suller MT, Maillard JY. Do antiseptics and disinfectants select for antibiotic resistance? *J. Med. Microbiol.* 1999;48:613-5.
362. Russell AD. Bacterial adaptation and resistance to antiseptics, disinfectants and preservatives is not a new phenomenon. *J. Hosp. Infect.* 2004;57:97-104.
363. Levy SB. The challenge of antibiotic resistance. *Scientific Am.* 1998;278:46-53.
364. Jones RD, Jampani HB, Newman JL, Lee AS. Triclosan: a review of effectiveness and safety in health care settings. *Am. J. Infect. Control* 2000;28:184-96.
365. Russell AD, McDonnell G. Concentration: a major factor in studying biocidal action. *J. Hosp. Infect.* 2000;44:1-3.
366. Russell AD, Maillard JY. Reaction and response-relationship between antibiotic resistance and resistance to antiseptics and disinfectants. *Am. J. Infect. Control* 2000;28:204-6.
367. Murtough SM, Hiom SJ, Palmer M, Russell AD. Biocide rotation in the healthcare setting: is there a case for policy implementation? *J. Hosp. Infect.* 2001;48:1-6.
368. Murtough SM, Hiom SJ, Palmer M, Russell AD. A survey of rotational use of biocides in hospital pharmacy aseptic units. *J. Hosp. Infect.* 2002;50:228-31.
369. Gebel J, Sonntag H-G, Werner H-P, Vavata V, Exner M, Kistemann T. The higher disinfectant resistance of nosocomial isolates of *Klebsiella oxytoca*: How reliable are indicator organisms in disinfectant testing? *J. Hosp. Infect.* 2002;50:309-11.
370. Ruden H, Daschner F. Should we routinely disinfect floors? *J. Hosp. Infect.* 2002;51:309.
371. Rutala WA, DJ W. Should we routinely disinfect floors? Reply to Professor F. Daschner. *J. Hosp. Infect.* 2002;51:309-11.
372. Rutala WA, Weber DJ. The benefits of surface disinfection. *Am. J. Infect. Control* 2004;32:226-31.
373. Dettenkofer M, Wenzler S, Amthor S, Antes G, Motschall E, Daschner FD. Does disinfection of environmental surfaces influence nosocomial infection rates? A systematic review. *Am. J. Infect. Control* 2004;32:84-9.
374. Daschner F, Schuster A. Disinfection and the prevention of infectious disease-no adverse effects? *Am. J. Infect. Control* 2004;32:224-5.
375. Cozad A, Jones RD. Disinfection and the prevention of infectious disease. *Am. J. Infect. Control* 2003;31:243-54.
376. Favero MS, Bond WW. Chemical disinfection of medical and surgical materials. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lea & Febiger, 1991:617-41.
377. Rheinbaben FV, Schunemann S, Grob T, Wolff MH. Transmission of viruses via contact in a household setting: experiments using bacteriophage OX174 as a model virus. *J. Hosp. Infect.* 2000;46:61-66.
378. Rutala WA, Weber DJ. Surface disinfection: should we do it? *J. Hosp. Infect.* 2001;48 (supplement A):S64-S68.
379. Ayliffe GAJ, Collins DM, Lowbury EJJ. Cleaning and disinfection of hospital floors. *Brit. Med. J.* 1966;2:442-5.
380. Ayliffe GA, Collins BJ, Lowbury EJ, Babb JR, Lilly HA. Ward floors and other surfaces as reservoirs of hospital infection. *J. Hyg. (Lond)*. 1967;65:515-36.
381. Exner M, Vacata V, Hornei B, Dietlein E, Gebel J. Household cleaning and surface disinfection: New insights and strategies. *J. Hosp. Infect.* 2004;56 (suppl):S70-S75.
382. Dharan S, Mourouga P, Copin P, Bessmer G, Tschanz B, Pittet D. Routine disinfection of patients' environmental surfaces. Myth or reality? *J. Hosp. Infect.* 1999;42:113-7.
383. Engelhart S KL, Glasmacher A, Fischnaller E, Marklein G, Exner M. *Pseudomonas aeruginosa* outbreak in a haematology-oncology unit associated with contaminated surface cleaning equipment. *J. Hosp. Infect.* 2002;52:93-98.

384. Denton M, Wilcox MH, Parnell P, et al. Role of environmental cleaning in controlling an outbreak of *Acinetobacter baumannii* on a neurosurgical intensive care unit. *J. Hosp. Infect.* 2004;56:106-10.
385. Barker J, Vipond IB, Bloomfield SF. Effects of cleaning and disinfection in reducing the spread of Norovirus contamination via environmental surfaces. *J. Hosp. Infect.* 2004;58:42-9.
386. Garner JS. Guideline for isolation precautions in hospitals. The Hospital Infection Control Practices Advisory Committee. *Infect. Control Hosp. Epidemiol.* 1996;17:53-80.
387. Maki DG, Alvarado CJ, Hassemer CA, Zilz MA. Relation of the inanimate hospital environment to endemic nosocomial infection. *N. Engl. J. Med.* 1982;307:1562-6.
388. Daschner F, Rabbenstein G, Langmaack H. [Surface decontamination in the control of hospital infections: comparison of different methods (author's transl)]. *Dtsch. Med. Wochenschr.* 1980;105:325-9.
389. Danforth D, Nicolle LE, Hume K, Alfieri N, Sims H. Nosocomial infections on nursing units with floors cleaned with a disinfectant compared with detergent. *J. Hosp. Infect.* 1987;10:229-35.
390. Smith TL, Iwen PC, Olson SB, Rupp ME. Environmental contamination with vancomycin-resistant enterococci in an outpatient setting. *Infect. Control Hosp. Epidemiol.* 1998;19:515-8.
391. Boyce JM, Potter-Bynoe G, Chenevert C, King T. Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. *Infect. Control Hosp. Epidemiol.* 1997;18:622-7.
392. Bonten MJM, Hayden MJ, Nathan C, et al. Epidemiology of colonisation of patients and environment with vancomycin-resistant enterococci. *Lancet* 1996;348:1615-9.
393. Hardy KJ, Oppenheim BA, Gossain S, Gao F, Hawkey PM. A study of the relationship between environmental contamination with methicillin-resistant *Staphylococcus aureus* (MRSA) and patients' acquisition of MRSA. *Infect Control Hosp Epidemiol* 2006;27:127-32.
394. Hota B. Contamination, disinfection, and cross-contamination: Are hospital surfaces reservoirs for nosocomial infection? *Clin. Infect. Dis.* 2004;39:1182-9.
395. Neely AN, Maley MP. Survival of enterococci and staphylococci on hospital fabrics and plastic. *J. Clin. Microbiol.* 2000;38:724-6.
396. Wendt C, Wiensenthal B, Dietz E, Ruden H. Survival of enterococci on dry surfaces. *J. Clin. Microbiol.* 1998;36:3734-6.
397. Neely AN, Maley MP. The 1999 Lindberg award. 3% hydrogen peroxide for the gram-positive disinfection of fabrics. *J. Burn Care Rehabil.* 1999;20:471-7.
398. Griffith CJ, Cooper RA, Gilmore J, Davies C, Lewis M. An evaluation of hospital cleaning regimes and standards. *J. Hosp. Infect.* 2000;45:19-28.
399. Tiller JC, Liao CJ, Lewis K, Klivanov AM. Designing surfaces that kill bacteria on contact. *Proc. Natl. Acad. Sci.* 2001;98:5981-5.
400. Rutala WA, Weber DJ. New disinfection and sterilization methods. *Emerg. Inf. Dis.* 2001;7:348-53.
401. Whitby JL, Rampling A. *Pseudomonas aeruginosa* contamination in domestic and hospital environments. *Lancet* 1972;1:15-7.
402. Scott E, Bloomfield SF. The survival and transfer of microbial contamination via cloths, hand and utensils. *J. Appl. Bacteriol.* 1990;68:271-8.
403. Scott E, Bloomfield SF. Investigations of the effectiveness of detergent washing, drying and chemical disinfection on contamination of cleaning cloths. *J. Appl. Bacteriol.* 1990;68:279-83.
404. Rutala WA, Cole EC. Antiseptics and disinfectants--safe and effective? *Infect. Control* 1984;5:215-8.
405. Oie S, Huang Y, Kamiya A, Konishi H, Nakazawa T. Efficacy of disinfectants against biofilm cells of methicillin-resistant *Staphylococcus aureus*. *Microbios* 1996;85:223-30.
406. Sartor C, Jacomo V, Duvivier C, Tissot-Dupont H, Sambuc R, Drancourt M. Nosocomial *Serratia marcescens* infections associated with extrinsic contamination of a liquid nonmedicated soap. *Infect. Control Hosp. Epidemiol.* 2000;21:196-9.
407. Reiss I, Borkhardt A, Fussle R, Sziegoleit A, Gortner L. Disinfectant contaminated with *Klebsiella oxytoca* as a source of sepsis in babies. *Lancet* 2000;356:310.
408. O'Rourke E, Runyan D, O'Leary J, Stern J. Contaminated iodophor in the operating room. *Am. J. Infect. Control* 2003;31:255-6.
409. Chuanchuen R, Karkhoff-Schweizer RR, Schweizer HP. High-level triclosan resistance in *Pseudomonas aeruginosa* is solely a result of efflux. *Am. J. Infect. Control* 2003;31:124-7.
410. Newman KA, Tenney JH, Oken HA, Moody MR, Wharton R, Schimpff SC. Persistent isolation of an

- unusual *Pseudomonas* species from a phenolic disinfectant system. *Infect. Control* 1984;5:219-22.
411. Bean HS. Types and characteristics of disinfectants. *J. Appl. Bacteriol.* 1967;30:6-16.
412. Russell AD, Hugo WB, Ayliffe GAJ. *Principles and Practice of Disinfection, Preservation and Sterilization*. Oxford, England: Blackwell Scientific Publications, 1999.
413. Russell AD. Factors influencing the efficacy of germicides. In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research*. Washington DC: Association for Professionals in Infection Control and Epidemiology, 2004:162-70.
414. Gillis RJ, Schmidt WC. Scanning electron microscopy of spores on inoculated product surfaces. *MD* 1983;46-9.
415. Favero MS, Petersen NJ, Carson LA, Bond WW, Hindman SH. Gram-negative water bacteria in hemodialysis systems. *Health Lab. Sci.* 1975;12:321-34.
416. Rutala WA, Cole EC. Ineffectiveness of hospital disinfectants against bacteria: a collaborative study. *Infect. Control* 1987;8:501-6.
417. Favero MS. Naturally occurring microorganisms and their resistance to physical and chemical agents. In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research*. Washington DC: Association for Professionals in Infection Control and Epidemiology, 2004:1-14.
418. Lee DH, Miles RJ, Perry BF. The mycoplasmacidal properties of sodium hypochlorite. *J. Hyg. (Lond)*. 1985;95:243-53.
419. Scott GH, Williams JC. Susceptibility of *Coxiella burnetii* to chemical disinfectants. *Ann. N. Y. Acad. Sci.* 1990;590:291-6.
420. Russell AD. Factors influencing the efficacy of antimicrobial agents. In: Russell AD, Hugo WB, Ayliffe GAJ, eds. *Principles and practice of disinfection, preservation and sterilization*. Oxford: Blackwell Science, 1999:95-123.
421. Rutala WA. Selection and use of disinfectants in healthcare. In: Mayhall CG, ed. *Infect. Control and Hosp. Epidemiol.* Philadelphia: Lippincott Williams & Wilkins, 1999:1161-87.
422. Lewis DL, Arens M. Resistance of microorganisms to disinfection in dental and medical devices. *Nat. Med.* 1995;1:956-8.
423. Muscarella LF. Sterilizing dental equipment. *Nat. Med.* 1995;1:1223-5.
424. Abbott CF, Cockton J, Jones W. Resistance of crystalline substances to gas sterilization. *J. Pharm. Pharmacol.* 1956;8:709-20.
425. Doyle JE, Ernst RR. Resistance of *Bacillus subtilis* var. *niger* spores occluded in water-insoluble crystals to three sterilization agents. *Appl. Microbiol.* 1967;15:726-30.
426. Jacobs P. Cleaning: Principles, methods and benefits. In: Rutala WA, ed. *Disinfection, sterilization, and antisepsis in healthcare*. Champlain, New York: Polyscience Publications, 1998:165-81.
427. Gorham RA, Jacobs P, Roberts CG. Laboratory artifacts due to protein and salt crystals on the inactivation of *Bacillus stearothermophilus*. *J. Hosp. Infect.* 1998;40:abstract P.9.2.2.
428. Cole EC, Rutala WA, Carson JL, Alfano EM. *Pseudomonas* pellicle in disinfectant testing: electron microscopy, pellicle removal, and effect on test results. *Appl. Environ. Microbiol.* 1989;55:511-3.
429. Anderson RL, Holland BW, Carr JK, Bond WW, Favero MS. Effect of disinfectants on pseudomonads colonized on the interior surface of PVC pipes. *Am. J. Public Health* 1990;80:17-21.
430. Anderson RL, Vess RW, Carr JH, Bond WW, Panlilio AL, Favero MS. Investigations of intrinsic *Pseudomonas cepacia* contamination in commercially manufactured povidone-iodine. *Infect. Control Hosp. Epidemiol.* 1991;12:297-302.
431. LeChevallier MW, Cawthon CD, Lee RG. Inactivation of biofilm bacteria. *Appl. Environ. Microbiol.* 1988;54:2492-9.
432. LeChevallier MW, Cawthon CD, Lee RG. Factors promoting survival of bacteria in chlorinated water supplies. *Appl. Environ. Microbiol.* 1988;54:649-54.
433. Costerton JS, Stewart PS, Greenberg EP. Bacterial biofilms: a common cause of persistent infections. *Science* 1999;284:1318-22.
434. Donlan RM, Costerton JW. Biofilms: Survival mechanisms of clinically relevant microorganisms. *Clin. Microbiol. Rev.* 2002;15:167-93.
435. Dunne WM. Bacterial adhesion: Seen any good biofilms lately? *Clin. Microbiol. Rev.* 2002;15:155-66.
436. Vickery K, Pajkos A, Cossart Y. Removal of biofilm from endoscopes: Evaluation of detergent efficiency.

- Am. J. Infect. Control 2004;32:170-6.
437. Marion K, Freney J, James G, Bergeron E, Renaud FNR, Costerton JW. Using an efficient biofilm detaching agent: An essential step for the improvement of endoscope reprocessing protocols. *J. Hosp. Infect.* 2006;In press.
 438. Marion-Ferey K, Pasmore M, Stoodley P, Wilson S, Husson GP, Costerton JW. Biofilm removal from silicone tubing: an assessment of the efficacy of dialysis machine decontamination procedures using an in vitro model. *J. Hosp. Infect.* 2003;53:64-71.
 439. Brown ML, Aldrich HC, Gauthier JJ. Relationship between glycocalyx and povidone-iodine resistance in *Pseudomonas aeruginosa* (ATCC 27853) biofilms. *Appl. Environ. Microbiol.* 1995;61:187-93.
 440. Price D, Ahearn DG. Incidence and persistence of *Pseudomonas aeruginosa* in whirlpools. *J. Clin. Microbiol.* 1988;26:1650-4.
 441. Anonymous. Dental Unit Waterlines: Approaching the Year 2000. ADA Council on Scientific Affairs. *JADA* 1999;130:1653-64.
 442. Donlan RM. Biofilms: a source of infection? In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: principles and practices in healthcare facilities*. Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:219-26.
 443. Loukili NH, Zink E, Grandadam S, Bientz M, Meunier O. Effectiveness of detergent-disinfecting agents on *Escherichia coli* 54127 biofilm. *J. Hosp. Infect.* 2004;57:175-8.
 444. Johansen C, Fallholt P, Gram L. Enzymatic removal and disinfection of bacterial biofilms. *Appl. Environ. Microbiol.* 1997;63:3724-8.
 445. Reichert M. Preparation of supplies for terminal sterilization. In: Reichert M, Young JH, eds. *Sterilization technology for the health care facility*. Gaithersburg, MD: Aspen Publication, 1997:36-50.
 446. Miller CH, Riggen SD, Sheldrake MA, Neeb JM. Presence of microorganisms in used ultrasonic cleaning solutions. *Am. J. Dent.* 1993;6:27-31.
 447. Jatzwauk L, Schone H, Pietsch H. How to improve instrument disinfection by ultrasound. *J. Hosp. Infect.* 2001;48 (Supple):S80-S83.
 448. Richburg FA, Reidy JJ, Apple DJ, Olson RJ. Sterile hypopyon secondary to ultrasonic cleaning solution. *J. Cataract Refract. Surg.* 1986;12:248-51.
 449. Schultz JK. Decontamination alternative. *Infect. Control Hosp. Epidemiol.* 1990;11:8-9.
 450. Rutala WA, Shafer KM. General information on cleaning, disinfection, and sterilization. In: Pfeiffer JA, ed. *APIC infection control and applied epidemiology: principles and practice*,. St. Louis: Mosby, 1996:15.1-15.17.
 451. Leonard DL, Mills SE. Comparison of automated instrument cleaning: preliminary results. *Infect. Control Steril. Technol.* 1997;20-23, 26-28.
 452. Ransjo U, Engstrom L, Hakansson P, et al. A test for cleaning and disinfection processes in a washer-disinfector. *APMIS* 2001;109:299-304.
 453. American Society for Hospital Central Service Personnel. *Training manual for central service technicians*. Chicago: American Hospital Association, 2001:1-271.
 454. Ninemeier JD. *Central service technical manual*. Chicago: International Association of Healthcare Central Service Materiel Management, 1998.
 455. Reichert M, Young JH. *Sterilization technology for the health care facility*. Gaithersburg: Aspen Publication, 1997:307.
 456. Vesley D, Norlien KG, Nelson B, Ott B, Streifel AJ. Significant factors in the disinfection and sterilization of flexible endoscopes. *Am. J. Infect. Control* 1992;20:291-300.
 457. Roberts CG. Studies on the bioburden on medical devices and the importance of cleaning. In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: principles and practices in healthcare facilities*. Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:63-9.
 458. Baxter RL, Baxter HC, Campbell GA, et al. Quantitative analysis of residual protein contamination on reprocessed surgical instruments. *J. Hosp. Infect.* 2006;63:439-44.
 459. Murdoch H, Taylor D, Dickinson J, et al. Surface decontamination of surgical instruments: An ongoing dilemma. *J. Hosp. Infect.* 2006;63:432-8.
 460. Alfa MJ, Nemes R. Manual versus automated methods for cleaning reusable accessory devices used for minimally invasive surgical procedures. *J. Hosp. Infect.* 2004;58:50-8.
 461. Alfa MJ, Nemes R, Olson N, Mulaire A. Manual methods are suboptimal compared with automated

- methods for cleaning of single-use biopsy forceps. *Infect Control Hosp Epidemiol* 2006;27:841-6.
462. Lee CH, Cheng SM, Humar A, et al. Acute febrile reactions with hypotension temporally associated with the introduction of a concentrated bioenzyme preparation in the cleaning and sterilization process of endomyocardial biopones. *Infect. Control Hosp. Epidemiol.* 2000;21:102.
463. Hutchisson B, LeBlanc C. The truth and consequences of enzymatic detergents. *Gastroenterol. Nurs.* 2005;28:372-6.
464. Zuhlsdorf B EM, Floss H, Martiny H,. Cleaning efficacy of nine different cleaners in a washer-disinfector designed for flexible endoscopes. *J. Hosp. Infect.* 2002;52:206-11.
465. Merritt K, Hitchins VM, Brown SA. Safety and cleaning of medical materials and devices. *J. Biomed. Mater. Res.* 2000;53:131-6.
466. Babb JR, Bradley CR. Endoscope decontamination: where do we go from here? *J. Hosp. Infect.* 1995;30:543-51.
467. Zuhlsdorf B, Floss H, Martiny H. Efficacy of 10 different cleaning processes in a washer-disinfector for flexible endoscopes. *J. Hosp. Infect.* 2004;56:305-11.
468. Alfa MJ, Jackson M. A new hydrogen peroxide-based medical-device detergent with germicidal properties: Comparison with enzymatic cleaners. *Am. J. Infect. Control* 2001;29:168-77.
469. Alfa MJ, DeGagne P, Olson N, Puchalski T. Comparison of ion plasma, vaporized hydrogen peroxide and 100% ethylene oxide sterilizers to the 12/88 ethylene oxide gas sterilizer. *Infect. Control Hosp. Epidemiol.* 1996;17:92-100.
470. Alfa MJ. Flexible endoscope reprocessing. *Infect. Control Steril. Technol.* 1997;3:26-36.
471. Alfa MJ, Degagne P, Olson N. Worst-case soiling levels for patient-used flexible endoscopes before and after cleaning. *Am. J. Infect. Control* 1999;27:392-401.
472. Rutala WA, Weber DJ. Low-temperature sterilization technology: Do we need to redefine sterilization? *Infect. Control Hosp. Epidemiol.* 1996;17:89-91.
473. Dancer SJ. How do we assess hospital cleaning? A proposal for microbiological standards for surface hygiene in hospitals. *J. Hosp. Infect.* 2004;56:10-5.
474. Pfeifer M. Standardised test soil blood 1:Composition, preparation, application. *Zentr. Steril.* 1998;6:381-5.
475. Pfeifer M. Blood as a soil on surgical instruments: Chemical profile, cleaning, detection. *Zentr. Steril.* 1998;6:304-10.
476. Fengier TW, Pahike H, Bisson S, Michels W. Are processed surgical instruments free of protein? *Zentr. Steril.* 2001;9:20-32.
477. Takashina M. Application of a bioluminescent method for checking cleaning results. *Zentr. Steril.* 2001;9:248-58.
478. Lipscomb IP, Sihota AK, Botham M, Harris KL, Keevil CW. Rapid method for the sensitive detection of protein contamination on surgical instruments. *J. Hosp. Infect.* 2006;62:141-8.
479. Malik RE, Cooper RA, Griffith CJ. Use of audit tools to evaluate the efficacy of cleaning systems in hospitals. *Am. J. Infect. Control* 2003;31:181-7.
480. Hansen KS. Occupational dermatoses in hospital cleaning women. *Contact Dermatitis* 1983;9:343-51.
481. Melli MC, Giorgini S, Sertoli A. Sensitization from contact with ethyl alcohol. *Contact Dermatitis* 1986;14:315.
482. Spaulding EH. Alcohol as a surgical disinfectant. *AORN J.* 1964;2:67-71.
483. Morton HE. The relationship of concentration and germicidal efficiency of ethyl alcohol. *Ann N.Y. Acad. Sci.* 1950;53:191-96.
484. Ali Y, Dolan MJ, Fendler EJ, Larson EL. Alcohols. In: Block SS, ed. *Disinfection, sterilization, and preservation.* Philadelphia: Lippincott Williams & Wilkins, 2001:229-54.
485. Morton HE. Alcohols. In: Block SS, ed. *Disinfection, sterilization, and preservation.* Philadelphia: Lea & Febiger, 1983:225-239.
486. Sykes G. The influence of germicides on the dehydrogenases of *Bact. coli*. Part I. The succinic acid dehydrogenase of *Bact. coli*. *J. Hyg. (Camb)* 1939;39:463-69.
487. Dagley S, Dawes EA, Morrison GA. Inhibition of growth of *Aerobacter aerogenes*: the mode of action of phenols, alcohols, acetone and ethyl acetate. *J. Bacteriol.* 1950;60:369-78.
488. Tilley FW, Schaffer JM. Relation between the chemical constitution and germicidal activity of the monohydric alcohols and phenols. *J. Bacteriol.* 1926;12:303-9.
489. Coulthard CE, Sykes G. The germicidal effect of alcohol with special reference to its action on bacterial

- spores. *Pharmaceutical J.* 1936;137:79-81.
490. Tyler R, Ayliffe GA. A surface test for virucidal activity of disinfectants: preliminary study with herpes virus. *J. Hosp. Infect.* 1987;9:22-9.
491. Kurtz JB, Lee TW, Parsons AJ. The action of alcohols on rotavirus, astrovirus and enterovirus. *J. Hosp. Infect.* 1980;1:321-5.
492. Smith CR. Alcohol as a disinfectant against the tubercle bacillus. *Public Health Rep.* 1947;62:1285-95.
493. Kruse RH, Green TD, Chambers RC, Jones MW. Disinfection of aerosolized pathogenic fungi on laboratory surfaces. I. Tissue phase. *Appl. Microbiol.* 1963;11:436-45.
494. Kruse RH, Green TD, Chambers RC, Jones MW. Disinfection of aerosolized pathogenic fungi on laboratory surfaces. II Culture phase. *Appl. Microbiol.* 1964;12:155-60.
495. Connor CG, Hopkins SL, Salisbury RD. Effectivity of contact lens disinfection systems against *Acanthamoeba culbertsoni*. *Optom. Vis. Sci.* 1991;68:138-41.
496. Turner NA, Russell AD, Furr JR, Lloyd D. *Acanthamoeba* spp., antimicrobial agents and contact lenses. *Sci. Prog.* 1999;82:1-8.
497. Nye RN, Mallory TB. A note on the fallacy of using alcohol for the sterilization of surgical instruments. *Boston Med. Surg. J.* 1923;189:561-3.
498. Frobisher M, Sommermeyer L, Blackwell MJ. Studies on disinfection of clinical thermometers. I. Oral thermometers. *Appl. Microbiol.* 1973;1:187-94.
499. Sommermeyer L, Frobisher M. Laboratory studies on disinfection of rectal thermometers. *Nurs. Res.* 1973;2:85-9.
500. Singh D, Kaur H, Gardner WG, Treen LB. Bacterial contamination of hospital pagers. *Infect. Control Hosp. Epidemiol.* 2002;23:274-6.
501. Embil JM, Zhanel GG, Plourde J, Hoban D. Scissors: A potential source of nosocomial infection. *Infect. Control Hosp. Epidemiol.* 2002;23:147-51.
502. Zachary KC, Bayne PS, Morrison VJ, Ford DS, Silver LC, Hooper DC. Contamination of gowns, gloves, and stethoscopes with vancomycin-resistant enterococci. *Infect. Control Hosp. Epidemiol.* 2001;22:560-4.
503. Babb JR, Bradley CR, Deverill CE, Ayliffe GA, Melikian V. Recent advances in the cleaning and disinfection of fibrescopes. *J. Hosp. Infect.* 1981;2:329-40.
504. Garcia de Cabo A, Martinez Larriba PL, Checa Pinilla J, Guerra Sanz F. A new method of disinfection of the flexible fiberoptic bronchoscope. *Thorax* 1978;33:270-2.
505. Elson CO, Hattori K, Blackstone MO. Polymicrobial sepsis following endoscopic retrograde cholangiopancreatography. *Gastroenterology* 1975;69:507-10.
506. Weber DJ, Wilson MB, Rutala WA, Thomann CA. Manual ventilation bags as a source for bacterial colonization of intubated patients. *Am. Rev. Respir. Dis.* 1990;142:892-4.
507. Cavagnolo RZ. Inactivation of herpesvirus on CPR manikins utilizing a currently recommended disinfecting procedure. *Infect. Control* 1985;6:456-8.
508. Ohara T, Itoh Y, Itoh K. Ultrasound instruments as possible vectors of staphylococcal infection. *J. Hosp. Infect.* 1998;40:73-7.
509. Talbot GH, Skros M, Provencher M. 70% alcohol disinfection of transducer heads: experimental trials. *Infect. Control* 1985;6:237-9.
510. Platt R, Lehr JL, Marino S, Munoz A, Nash B, Raemer DB. Safe and cost-effective cleaning of pressure-monitoring transducers. *Infect. Control Hosp. Epidemiol.* 1988;9:409-16.
511. Beck-Sague CM, Jarvis WR. Epidemic bloodstream infections associated with pressure transducers: a persistent problem. *Infect. Control Hosp. Epidemiol.* 1989;10:54-9.
512. Chronister CL, Russo P. Effects of disinfecting solutions on tonometer tips. *Optom. Vis. Sci.* 1990;67:818-21.
513. Lingel NJ, Coffey B. Effects of disinfecting solutions recommended by the Centers for Disease Control on Goldmann tonometer biprisms. *J. Am. Optom. Assoc.* 1992;63:43-8.
514. Soukiasian SH, Asdourian GK, Weiss JS, Kachadoorian HA. A complication from alcohol-swabbed tonometer tips. *Am. J. Ophthalmol.* 1988;105:424-5.
515. Jakobsson SW, Rajs J, Jonsson JA, Persson H. Poisoning with sodium hypochlorite solution. Report of a fatal case, supplemented with an experimental and clinico-epidemiological study. *Am. J. Forensic Med. Pathol.* 1991;12:320-7.
516. Heidemann SM, Goetting MG. Treatment of acute hypoxemic respiratory failure caused by chlorine

- exposure. *Pediatr. Emerg. Care* 1991;7:87-8.
517. Hoy RH. Accidental systemic exposure to sodium hypochlorite (Clorox) during hemodialysis. *Am. J. Hosp. Pharm.* 1981;38:1512-4.
 518. Landau GD, Saunders WH. The effect of chlorine bleach on the esophagus. *Arch. Otolaryngol.* 1964;80:174-6.
 519. French RJ, Tabb HG, Rutledge LJ. Esophageal stenosis produced by ingestion of bleach: report of two cases. *South. Med. J.* 1970;63:1140-4.
 520. Ward MJ, Routledge PA. Hypernatraemia and hyperchloraemic acidosis after bleach ingestion. *Hum. Toxicol.* 1988;7:37-8.
 521. Ingram TA. Response of the human eye to accidental exposure to sodium hypochlorite. *J Endodontics* 1990;16:235-8.
 522. Haag JR, Gieser RG. Effects of swimming pool water on the cornea. *JAMA* 1983;249:2507-8.
 523. Mrvos R, Dean BS, Krenzelok EP. Home exposures to chlorine/chloramine gas: review of 216 cases. *South. Med. J.* 1993;86:654-7.
 524. Reisz GR, Gammon RS. Toxic pneumonitis from mixing household cleaners. *Chest* 1986;89:49-52.
 525. Gapany-Gapanavicius M, Yellin A, Almog S, Tirosh M. Pneumomediastinum. A complication of chlorine exposure from mixing household cleaning agents. *JAMA* 1982;248:349-50.
 526. Hoffman PN, Death JE, Coates D. The stability of sodium hypochlorite solutions. In: Collins CH, Allwood MC, Bloomfield SF, Fox A, eds. *Disinfectants: their use and evaluation of effectiveness.* London: Academic Press, 1981:77-83.
 527. Gamble MR. Hazard: formaldehyde and hypochlorites. *Lab. Anim.* 1977;11:61.
 528. Helms C, Massanari R, Wenzel R, et al. Control of epidemic nosocomial legionellosis: a 5 year progress report on continuous hyperchlorination of a water distribution system. Abstracts of 27th Interscience Conference of Antimicrobial Agents and Chemotherapy, 1987:349, p.158.
 529. Environmental Protection Agency. R.E.D. Facts sodium and calcium hypochlorite salts. 1991.
 530. Coates D. Comparison of sodium hypochlorite and sodium dichloroisocyanurate disinfectants: neutralization by serum. *J. Hosp. Infect.* 1988;11:60-7.
 531. Coates D. A comparison of sodium hypochlorite and sodium dichloroisocyanurate products. *J. Hosp. Infect.* 1985;6:31-40.
 532. Coates D, Wilson M. Use of sodium dichloroisocyanurate granules for spills of body fluids. *J. Hosp. Infect.* 1989;13:241-51.
 533. Bloomfield SF, Uso EE. The antibacterial properties of sodium hypochlorite and sodium dichloroisocyanurate as hospital disinfectants. *J. Hosp. Infect.* 1985;6:20-30.
 534. Coates D. An evaluation of the use of chlorine dioxide (Tristel One-Shot) in an automated washer/disinfector (Medivator) fitted with a chlorine dioxide generator for decontamination of flexible endoscopes. *J. Hosp. Infect.* 2001;48:55-65.
 535. Isomoto H, Urata M, Kawazoe K, et al. Endoscope disinfection using chlorine dioxide in an automated washer-disinfector. *J. Hosp. Infect.* 2006;63:298-305.
 536. Sampson MN MA. Not all super-oxidized waters are the same. *J. Hosp. Infect.* 2002;52:227-8.
 537. Selkon JB, Babb JR, Morris R. Evaluation of the antimicrobial activity of a new super-oxidized water, Sterilox®, for the disinfection of endoscopes. *J. Hosp. Infect.* 1999;41:59-70.
 538. Fraise AP. Choosing disinfectants. *J. Hosp. Infect.* 1999;43:255-64.
 539. Tanaka H, Hirakata Y, Kaku M, et al. Antimicrobial activity of superoxidized water. *J. Hosp. Infect.* 1996;34:43-9.
 540. Tanaka N, Fujisawa T, Daimon T, Fujiwara K, Yamamoto M, Abe T. The use of electrolyzed solutions for the cleaning and disinfecting of dialyzers. *Artif. Organs* 2000;24:921-8.
 541. Williams ND, Russell AD. The effects of some halogen-containing compounds on *Bacillus subtilis* endospores. *J. Appl. Bacteriol.* 1991;70:427-36.
 542. Babb JR, Bradley CR, Ayliffe GAJ. Sporicidal activity of glutaraldehydes and hypochlorites and other factors influencing their selection for the treatment of medical equipment. *J. Hosp. Infect.* 1980;1:63-75.
 543. Brown DG, Skyllis TP, Fekety FR. Comparison of chemical sterilant/disinfectant solutions against spores of *Clostridium difficile*. Abstracts of the American Society for Microbiology, 1983:Q39,267.
 544. Grant D, Venneman M, Burns RM. Mycobactericidal activity of Alcide an experimental liquid sterilant. Abstracts of the Annual Meeting of the American Society of Microbiology, 1982: Q101,226.

545. Korich DG, Mead JR, Madore MS, Sinclair NA, Sterling CR. Effects of ozone, chlorine dioxide, chlorine, and monochloramine on *Cryptosporidium parvum* oocyst viability. *Appl. Environ. Microbiol.* 1990;56:1423-8.
546. Griffiths PA, Babb JR, Fraise AP. Mycobactericidal activity of selected disinfectants using a quantitative suspension test. *J. Hosp. Infect.* 1999;41:111-21.
547. Centers for Disease Control. Bacteremia associated with reuse of disposable hollow-fiber hemodialyzers. *MMWR* 1986;35:417-8.
548. Bloomfield SF, Miller EA. A comparison of hypochlorite and phenolic disinfectants for disinfection of clean and soiled surfaces and blood spillages. *J. Hosp. Infect.* 1989;13:231-9.
549. Shetty N, Srinivasan S, Holton J, Ridgway GL. Evaluation of microbicidal activity of a new disinfectant: Sterilox® 2500 against *Clostridium difficile* spores, *Helicobacter pylori*, vancomycin resistant *Enterococcus* species, *Candida albicans* and several *Mycobacterium* species. *J. Hosp. Infect.* 1999;41:101-5.
550. Urata M, Isomot H, Murase K, et al. Comparison of the microbicidal activities of superoxidized and ozonated water in the disinfection of endoscopes. *J Intern Med Res* 2003;31:299-306.
551. Tsuji S, Kawano S, Oshita M, et al. Endoscope disinfection using acidic electrolytic water. *Endoscopy* 1999;31:528-35.
552. Lee JH, Rhee PL, Kim JH, et al. Efficacy of electrolyzed acid water in reprocessing patient-used flexible upper endoscopes: Comparison with 2% alkaline glutaraldehyde. *J. Gastroenterol. Hepatol.* 2004;19:897-904.
553. Centers for Disease Control. Acquired immune deficiency syndrome (AIDS): precautions for clinical and laboratory staffs. *MMWR* 1982;31:577-80.
554. Garner JS, Simmons BP. Guideline for isolation precautions in hospitals. *Infect. Control* 1983;4:245-325.
555. Van Bueren J, Simpson RA, Salman H, Farrelly HD, Cookson BD. Inactivation of HIV-1 by chemical disinfectants: sodium hypochlorite. *Epidemiol. Infect.* 1995;115:567-79.
556. Coates D. Disinfection of spills of body fluids: how effective is a level of 10,000 ppm available chlorine? *J. Hosp. Infect.* 1991;18:319-22.
557. Chitnis V, Chitnis S, Patil S, Chitnis D. Practical limitations of disinfection of body fluid spills with 10,000 ppm sodium hypochlorite (NaOCl). *Am. J. Infect. Control* 2004;32:306-8.
558. Anonymous. Recommendations for decontaminating manikins used in cardiopulmonary resuscitation training, 1983 update. *Infect. Control* 1984;5:399-401.
559. Centers for Disease Control. Use of bleach for disinfection of drug injection equipment. *MMWR* 1993;42:418-9.
560. Shapshak P, McCoy CB, Rivers JE, et al. Inactivation of human immunodeficiency virus-1 at short time intervals using undiluted bleach. *J. Acquir. Immune Defic. Syndr.* 1993;6:218-9.
561. Shapshak P, McCoy CB, Shah SM, et al. Preliminary laboratory studies of inactivation of HIV-1 in needles and syringes containing infected blood using undiluted household bleach. *J. Acquir. Immune Defic. Syndr.* 1994;7:754-9.
562. Brystrom A, Sundqvist G. Bacteriologic evaluation of the effect of 0.5 percent sodium hypochlorite in endodontic therapy. *Oral Surg. Oral Med. Oral Pathol.* 1983;55:307-12.
563. Favero MJ, Tokars JI, Arduino MJ, Alter MJ. Nosocomial infections associated with hemodialysis. In: Mayhall CG, ed. *Infect. Control and Hosp. Epidemiol.* Philadelphia: Lippincott Williams & Wilkins, 1999:897-917.
564. Helms CM, Massanari RM, Zeitler R, et al. Legionnaires' disease associated with a hospital water system: a cluster of 24 nosocomial cases. *Ann. Intern. Med.* 1983;99:172-8.
565. Heffelfinger JD, Kool JL, Fridkin S, et al. Risk of hospital-acquired legionnaires' disease in cities using monochloramine versus other water disinfectants. *Infect Control Hosp Epidemiol* 2003;24:569-74.
566. Moore MR, Pryor M, Fields B, Lucas C, Phelan M, Besser RE. Introduction of monochloramine into a municipal water system: Impact on colonization of buildings by *Legionella* spp. *Appl. Environ. Microbiol.* 2006;72:378-83.
567. Srinivasan A, Bova G, Ross T, et al. A 17-month evaluation of a chlorine dioxide water treatment system to control *Legionella* species in a hospital water supply. *Infect Control Hosp Epidemiol* 2003;24:575-9.
568. Steve L, Goodhart P, Alexander J. Hydrotherapy burn treatment: use of chloramine-T against resistant microorganisms. *Arch. Phys. Med. Rehabil.* 1979;60:301-3.

569. Coates D, Wilson M. Powders, composed of chlorine-releasing agent acrylic resin mixtures or based on peroxygen compounds, for spills of body fluids. *J. Hosp. Infect.* 1992;21:241-52.
570. Tulis JJ. Formaldehyde as a gas. In: Phillips GB, Miller WS, eds. *Industrial Sterilization*. Durham: Duke University Press, 1972:209-38.
571. Emmons CW. Fungicidal action of some common disinfectants on two dermatophytes. *Arch. Dermatol. Syphil.* 1933;28:15-21.
572. McCulloch EC, Costigan S. A comparison of the efficiency of phenol, liquor cresolis, formaldehyde, sodium hypochlorite and sodium hydroxide against *Eberthella typhi* at various temperatures. *J. Infect. Dis.* 1936;59:281-4.
573. Sagripanti JL, Eklund CA, Trost PA, et al. Comparative sensitivity of 13 species of pathogenic bacteria to seven chemical germicides. *Am. J. Infect. Control* 1997;25:335-9.
574. NIOSH. Formaldehyde: evidence of carcinogenicity. NIOSH Current Intelligence Bulletin 34. DHEW (NIOSH) Publication No. 81-111. 1981.
575. Occupational Safety and Health Administration. OSHA amends formaldehyde standard. *Occupational Safety and Health News* 1991:1.
576. Occupational Health and Safety Administration. OSHA Fact Sheet: Formaldehyde: Occupational Safety and Health Administration, U.S. Department of Labor, 2002.
577. Occupational Safety and Health Administration. Air Contaminants Final Rule. *Fed. Regist.* 1993;58:35338-51.
578. Occupational Safety and Health Administration. Formaldehyde: OSHA Fact Sheet: Occupational Safety and Health Administration, 2002.
579. Centers for Disease Control. Occupational exposures to formaldehyde in dialysis units. *MMWR* 1986;35:399-01.
580. Centers for Disease Control. Formaldehyde exposures in a gross anatomy laboratory - Colorado. *MMWR* 1983;52:698-700.
581. Tokars JI, Miller ER, Alter MJ, Arduino MJ. National surveillance of dialysis associated diseases in the United States, 1995. *ASAIO J.* 1998;44:98-107.
582. Favero MS, Alter MJ, Tokars JI, Bland LA. Dialysis-associated disease and their control. In: Bennett JV, Brachman PS, eds. *Hospital Infections*. Boston: Little, Brown and Company, 1998:357-80.
583. Bland LA, Favero MS. Microbial contamination control strategies for hemodialysis system. *Plant, Technology & Safety Management Series: infection control issues in PTSM 1990*. Oakbrook Terrace, Illinois.
584. Boucher RM. Potentiated acid 1,5 pentanedial solution--a new chemical sterilizing and disinfecting agent. *Am. J. Hosp. Pharm.* 1974;31:546-57.
585. Miner NA, McDowell JW, Willcockson GW, Bruckner NI, Stark RL, Whitmore EJ. Antimicrobial and other properties of a new stabilized alkaline glutaraldehyde disinfectant/sterilizer. *Am. J. Hosp. Pharm.* 1977;34:376-82.
586. Pepper RE. Comparison of the activities and stabilities of alkaline glutaraldehyde sterilizing solutions. *Infect. Control* 1980;1:90-2.
587. Leach ED. A new synergized glutaraldehyde-phenate sterilizing solution and concentrated disinfectant. *Infect. Control* 1981;2:26-30.
588. Miner NA, Ross C. Clinical evaluation of ColdSpor, a glutaraldehyde-phenolic disinfectant. *Respir. Care* 1991;36:104-9.
589. Collins FM, Montalbino V. Mycobactericidal activity of glutaraldehyde solutions. *J. Clin. Microbiol.* 1976;4:408-12.
590. Masferrer R, Marquez R. Comparison of two activated glutaraldehyde solutions: Cidex Solution and Sonacide. *Respir. Care* 1977;22:257-62.
591. Jette LP, Ringuette L, Ishak M, Miller M, Saint-Antoine P. Evaluation of three glutaraldehyde-based disinfectants used in endoscopy. *J. Hosp. Infect.* 1995;30:295-303.
592. Scott EM, Gorman SP. Glutaraldehyde. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001:361-81.
593. Scott EM, Gorman SP. Glutaraldehyde. In: Block SS, ed. *Disinfection, Sterilization, and Preservation*. Philadelphia: Lea & Febiger, 1991:596-616.
594. Stonehill AA, Krop S, Borick PM. Buffered glutaraldehyde - a new chemical sterilizing solution. *Am. J.*

- Hosp. Pharm. 1963;20:458-65.
595. Borick PM, Dondershine FH, Chandler VL. Alkalinized glutaraldehyde, a new antimicrobial agent. J. Pharm. Sci. 1964;53:1273-5.
596. Russell AD. Glutaraldehyde: current status and uses. Infect. Control Hosp. Epidemiol. 1994;15:724-33.
597. Hanson PJ, Bennett J, Jeffries DJ, Collins JV. Enteroviruses, endoscopy and infection control: an applied study. J. Hosp. Infect. 1994;27:61-7.
598. van Klingeren B, Pullen W. Glutaraldehyde resistant mycobacteria from endoscope washers. J. Hosp. Infect. 1993;25:147-9.
599. Griffiths PA, Babb JR, Bradley CR, Fraise AP. Glutaraldehyde-resistant *Mycobacterium chelonae* from endoscope washer disinfectors. J. Appl. Microbiol. 1997;82:519-26.
600. Dauendorffer JN, Laurain C, Weber M, Dailloux M. Evaluation of the bactericidal efficiency of a 2% alkaline glutaraldehyde solution on *Mycobacterium xenopi*. J. Hosp. Infect. 2000;46:73-6.
601. Nomura K, Ogawa M, Miyamoto H, Muratani T, Taniguchi H. Antibiotic susceptibility of glutaraldehyde-tolerant *Mycobacterium chelonae* from bronchoscope washing machines. J. Hosp. Infect. 2004;32:185-8.
602. Webster E, Ribner B, Streed LL, Hutton N. Microbial contamination of activated 2% glutaraldehyde used in high-level disinfection of endoscopes (abstract). Am. J. Infect. Control 1996;24:153.
603. Casemore DP, Blewett DA, Wright SE. Cleaning and disinfection of equipment for gastrointestinal flexible endoscopy: interim recommendations of a Working Party of the British Society of Gastroenterology. Gut 1989;30:1156-7.
604. Laskowski LF, Marr JJ, Spernoga JF, et al. Fastidious mycobacteria grown from porcine prosthetic-heart-valve cultures. N. Engl. J. Med. 1977;297:101-2.
605. Collins FM. Bactericidal activity of alkaline glutaraldehyde solution against a number of atypical mycobacterial species. J. Appl. Bacteriol. 1986;61:247-51.
606. Food and Drug Administration. Sterilants and high level disinfectants cleared by FDA in a 510(k) as of January 30, 2002 with general claims for processing reusable medical and dental devices, <http://www.fda.gov/cdrh/ode/germlab.html>, 2001.
607. Hernandez A, Martro E, Pizo C, et al. In-use evaluation of Perasafe compared with Cidex in fiberoptic bronchoscope disinfection. J. Hosp. Infect. 2003;54:46-52.
608. Leong D, Dorsey G, Klapp M. Dilution of glutaraldehyde by automatic endoscope machine washers: the need for a quality control program. Abstracts of the 14th Annual Educational Conference of Association for Practitioners in Infection Control, 1987:108, p.130.
609. Mbithi JN, Springthorpe VS, Sattar SA, Pacquette M. Bactericidal, virucidal, and mycobactericidal activities of reused alkaline glutaraldehyde in an endoscopy unit. J. Clin. Microbiol. 1993;31:2988-95.
610. Kleier DJ, Averbach RE. Glutaraldehyde nonbiologic monitors. Infect. Control Hosp. Epidemiol. 1990;11:439-41.
611. Kleier DJ, Tucker JE, Averbach RE. Clinical evaluation of glutaraldehyde nonbiologic monitors. Quintessence Int. 1989;20:271-7.
612. Overton D, Burgess JO, Beck B, Matis B. Glutaraldehyde test kits: evaluation for accuracy and range. Gen. Dent. 1989;37:126, 128.
613. Cooke RPD, Goddard SV, Chatterley R, Whyman-Morris A, Cheale J. Monitoring glutaraldehyde dilution in automated washer/disinfectors. J. Hosp. Infect. 2001;48:242-6.
614. Ayliffe GA, Babb JR, Bradley CR. Disinfection of endoscopes. J. Hosp. Infect. 1986;7:296-9.
615. Centers for Disease Control. Federal regulatory action against sporicidin cold sterilizing solution. MMWR 1991;40:880-1.
616. Husni L, Kale E, Climer C, Bostwick B, Parker TF, 3rd. Evaluation of a new disinfectant for dialyzer reuse. Am. J. Kidney Dis. 1989;14:110-8.
617. Townsend TR, Wee SB, Koblin B. An efficacy evaluation of a synergized glutaraldehyde-phenate solution in disinfecting respiratory therapy equipment contaminated during patient use. Infect. Control 1982;3:240-4.
618. Petersen NJ, Carson LA, Doto IL, Aguerro SM, Favero MS. Microbiologic evaluation of a new glutaraldehyde-based disinfectant for hemodialysis systems. Trans. Am. Soc. Artif. Intern. Organs 1982;28:287-90.
619. Gundogdu H, Ocal K, Caglikulekci M, Karabiber N, Bayramoglu E, Karahan M. High-level disinfection with 2% alkalinized glutaraldehyde solution for reuse of laparoscopic disposable plastic trocars. J.

- Laparoendosc. Adv. Surg. Techniques. Part A 1998;8:47-52.
620. Castelli M, Qizilbash A, Seaton T. Post-colonoscopy proctitis. *Am. J. Gastroenterol.* 1986;81:887.
621. Jonas G, Mahoney A, Murray J, Gertler S. Chemical colitis due to endoscope cleaning solutions: a mimic of pseudomembranous colitis. *Gastroenterology* 1988;95:1403-8.
622. Levine DS. Proctitis following colonoscopy. *Gastrointest. Endosc.* 1988;34:269-72.
623. Riney S, Grimes M, Khalife K, Warbasse L, Massanari M. Diarrhea associated with disinfection of sigmoidoscopes. [abstract]. *Am J Infect Control* 1991;19:109.
624. Durante L, Zulty JC, Israel E, et al. Investigation of an outbreak of bloody diarrhea: association with endoscopic cleaning solution and demonstration of lesions in an animal model. *Am. J. Med.* 1992;92:476-80.
625. Burtin P, Ruget O, Petit R, Boyer J. Glutaraldehyde-induced proctitis after endorectal ultrasound examination: a higher risk of incidence than expected? *Gastrointest. Endosc.* 1993;39:859-60.
626. Babb RR, Paaso BT. Glutaraldehyde proctitis. *West. J. Med.* 1995;163:477-8.
627. Ryan CK, Potter GD. Disinfectant colitis. Rinse as well as you wash. *J. Clin. Gastroenterol.* 1995;21:6-9.
628. Rozen P, Somjen GJ, Baratz M, Kimel R, Arber N, Gilat T. Endoscope-induced colitis: description, probable cause by glutaraldehyde, and prevention. *Gastrointest. Endosc.* 1994;40:547-53.
629. West AB, Kuan SF, Bennick M, Lagarde S. Glutaraldehyde colitis following endoscopy: clinical and pathological features and investigation of an outbreak. *Gastroenterology* 1995;108:1250-5.
630. Dolce P, Gourdeau M, April N, Bernard PM. Outbreak of glutaraldehyde-induced proctocolitis. *Am. J. Infect. Control* 1995;23:34-9.
631. Farina A, Fievet MH, Plassart F, Menet MC, Thuillier A. Residual glutaraldehyde levels in fiberoptic endoscopes: measurement and implications for patient toxicity. *J. Hosp. Infect.* 1999;43:293-7.
632. Dailey JR, Parnes RE, Aminlari A. Glutaraldehyde keratopathy. *Am. J. Ophthalmol.* 1993;115:256-8.
633. Courtright P, Lewallen S, Holland SP, Wendt TM. Corneal decompensation after cataract surgery. An outbreak investigation in Asia. *Ophthalmology* 1995;102:1461-5.
634. Leinster P, Baum JM, Baxter PJ. An assessment of exposure to glutaraldehyde in hospitals: typical exposure levels and recommended control measures. *Br. J. Ind. Med.* 1993;50:107-11.
635. Beauchamp RO, St Clair MB, Fennell TR, Clarke DO, Morgan KT. A critical review of the toxicology of glutaraldehyde. *Crit. Rev. Toxicol.* 1992;22:143-74.
636. Corrado OJ, Osman J, Davies RJ. Asthma and rhinitis after exposure to glutaraldehyde in endoscopy units. *Hum. Toxicol.* 1986;5:325-8.
637. Norback D. Skin and respiratory symptoms from exposure to alkaline glutaraldehyde in medical services. *Scand. J. Work, Environ. Health* 1988;14:366-71.
638. Mwaniki DL, Guthua SW. Occupational exposure to glutaraldehyde in tropical climates. *Lancet* 1992;340:1476-7.
639. Centers for Disease Control. Symptoms of irritation associated with exposure to glutaraldehyde. *MMWR* 1987;36:190-1.
640. Wiggins P, McCurdy SA, Zeidenberg W. Epistaxis due to glutaraldehyde exposure. *J. Occup. Med.* 1989;31:854-6.
641. Di Prima T, De Pasquale R, Nigro M. Contact dermatitis from glutaraldehyde. *Contact Dermatitis* 1988;19:219-20.
642. Fowler JF, Jr. Allergic contact dermatitis from glutaraldehyde exposure. *J. Occup. Med.* 1989;31:852-3.
643. Fisher AA. Allergic contact dermatitis of the hands from Sporicidin (glutaraldehyde-phenate) used to disinfect endoscopes. *Cutis* 1990;45:227-8.
644. Nethercott JR, Holness DL, Page E. Occupational contact dermatitis due to glutaraldehyde in health care workers. *Contact Dermatitis* 1988;18:193-6.
645. Gannon PF, Bright P, Campbell M, O'Hickey SP, Burge PS. Occupational asthma due to glutaraldehyde and formaldehyde in endoscopy and x ray departments. *Thorax* 1995;50:156-9.
646. Chan-Yeung M, McMurren T, Catonio-Begley F, Lam S. Occupational asthma in a technologist exposed to glutaraldehyde. *J. Allergy Clin. Immunol.* 1993;91:974-8.
647. Schnuch A, Uter W, Geier J, Frosch PJ, Rustemeyer T. Contact allergies in healthcare workers. Results from the IVDK. *Acta Derm. Venereol.* 1998;78:358-63.
648. Wellons SL, Trawick EG, Stowers MF, Jordan SL, Wass TL. Laboratory and hospital evaluation of four personal monitoring methods for glutaraldehyde in ambient air. *Am. Ind. Hyg. Assoc. J.* 1998;59:96-103.

649. Newman MA, Kachuba JB. Glutaraldehyde: a potential health risk to nurses. *Gastroenterol. Nurs.* 1992;14:296-300, discussion 300-1.
650. Association for the Advancement of Medical Instrumentation. Safe use and handling of glutaraldehyde-based products in healthcare facilities. Arlington, VA: AAMI, 1995.
651. Anonymous. Glutaraldehyde. New York: Occupational Health Services, Inc., 1992.
652. Rutala WA, Hamory BH. Expanding role of hospital epidemiology: employee health--chemical exposure in the health care setting. *Infect. Control Hosp. Epidemiol.* 1989;10:261-6.
653. Turner FJ. Hydrogen peroxide and other oxidant disinfectants. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lea & Febiger, 1983:240-50.
654. Block SS. Peroxygen compounds. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001:185-204.
655. Sattar SA, Springthorpe VS, Rochon M. A product based on accelerated and stabilized hydrogen peroxide: Evidence for broad-spectrum germicidal activity. *Canadian J Infect Control* 1998 (Winter):123-30.
656. Omidbakhsh N, Sattar SA. Broad-spectrum microbicidal activity, toxicologic assessment, and materials compatibility of a new generation of accelerated hydrogen peroxide-based environmental surface disinfectant. *Am. J. Infect. Control* 2006;34:251-7.
657. Schaeffer AJ, Jones JM, Amundsen SK. Bacterial effect of hydrogen peroxide on urinary tract pathogens. *Appl. Environ. Microbiol.* 1980;40:337-40.
658. Wardle MD, Renninger GM. Bactericidal effect of hydrogen peroxide on spacecraft isolates. *Appl. Microbiol.* 1975;30:710-1.
659. Sagripanti JL, Bonifacino A. Comparative sporicidal effect of liquid chemical germicides on three medical devices contaminated with spores of *Bacillus subtilis*. *Am. J. Infect. Control* 1996;24:364-71.
660. Sagripanti JL, Bonifacino A. Effects of salt and serum on the sporicidal activity of liquid disinfectants. *J. AOAC Int.* 1997;80:1198-207.
661. Saurina G, Landman D, Quale JM. Activity of disinfectants against vancomycin-resistant *Enterococcus faecium*. *Infect. Control Hosp. Epidemiol.* 1997;18:345-7.
662. Kilvington S. Moist-heat disinfection of *Acanthamoeba* cysts. *Rev. Infect. Dis.* 1991;13:S418.
663. Sattar SA, Adegbonrin O, Ramirez J. Combined application of simulated reuse and quantitative carrier test to assess high-level disinfection: Experiments with an accelerated hydrogen peroxide-based formulation. *Am. J. Infect. Control* 2002;30:449-57.
664. Leaper S. Influence of temperature on the synergistic sporicidal effect of peracetic acid plus hydrogen peroxide in *Bacillus subtilis* SA22(NCA 72-52). *Food Microbiol.* 1984;1:199-203.
665. Mentel R, Schmidt J. Investigations on rhinovirus inactivation by hydrogen peroxide. *Acta Virol.* 1973;17:351-4.
666. Sattar SA. Effect of liquid chemical germicides on mycobacteria including multi-drug resistant isolates of *Mycobacteria tuberculosis*. Abstracts of the 37th Interscience Conference on Antimicrobial Agents of Chemotherapy; September 28-October 1, 1997; Toronto, Ontario, Canada; E166., 1997.
667. Reckitt & Colman. Sporox sterilant and high-level disinfectant technical report. Montvale, NJ: Reckitt & Colman, 1997:1-12.
668. Sattar SA, Taylor YE, Paquette M, Rubino J. In-hospital evaluation of 7.5% hydrogen peroxide as a disinfectant for flexible endoscopes. *Can. J. Infect. Control* 1996;11:51-4.
669. Hobson DW, Seal LA. Evaluation of a novel, rapid-acting, sterilizing solution at room temperature. *Am. J. Infect. Control* 2000;28:370-5.
670. Anonymous. Hydrogen peroxide, ACS reagent. Vol. 2001: Sigma Product Information Sheet, <http://www.sigma-sial.com/sigma/proddata/h0904.htm>.
671. Silvany RE, Dougherty JM, McCulley JP, Wood TS, Bowman RW, Moore MB. The effect of currently available contact lens disinfection systems on *Acanthamoeba castellanii* and *Acanthamoeba polyphaga*. *Ophthalmology* 1990;97:286-90.
672. Moore MB. *Acanthamoeba keratitis* and contact lens wear: the patient is at fault. *Cornea* 1990;9:S33-5; discussion S39-40.
673. Judd PA, Tomlin PJ, Whitby JL, Inglis TC, Robinson JS. Disinfection of ventilators by ultrasonic nebulisation. *Lancet* 1968;2:1019-20.
674. Levenson JE. Corneal damage from improperly cleaned tonometer tips. *Arch. Ophthalmol.* 1989;107:1117.
675. Thompson RL, Haley CE, Searcy MA, et al. Catheter-associated bacteriuria. Failure to reduce attack rates

- using periodic instillations of a disinfectant into urinary drainage systems. JAMA 1984;251:747-51.
676. Bilotta JJ, Wayne JD. Hydrogen peroxide enteritis: the "snow white" sign. Gastrointest. Endosc. 1989;35:428-30.
677. Gottardi W. Iodine and iodine compounds. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lea & Febiger, 1991:152-66.
678. Gottardi W. Iodine and iodine compounds. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lippincott Williams & Wilkins, 2001:159-84.
679. Craven DE, Moody B, Connolly MG, Kollisch NR, Stottmeier KD, McCabe WR. Pseudobacteremia caused by povidone-iodine solution contaminated with *Pseudomonas cepacia*. N. Engl. J. Med. 1981;305:621-3.
680. Berkelman RL, Lewin S, Allen JR, et al. Pseudobacteremia attributed to contamination of povidone-iodine with *Pseudomonas cepacia*. Ann. Intern. Med. 1981;95:32-6.
681. Parrott PL, Terry PM, Whitworth EN, et al. *Pseudomonas aeruginosa* peritonitis associated with contaminated poloxamer-iodine solution. Lancet 1982;2:683-5.
682. Favero MS. Iodine--champagne in a tin cup. Infect. Control 1982;3:30-2.
683. Berkelman RL, Holland BW, Anderson RL. Increased bactericidal activity of dilute preparations of povidone-iodine solutions. J. Clin. Microbiol. 1982;15:635-9.
684. Chang SL. Modern concept of disinfection. J. Sanit. Eng. Div. Proc. Am. Soc. Civ. Eng. 1971:689-705.
685. Wallbank AM, Drulak M, Poffenroth L, Barnes C, Kay C, Lebttag I. Wescodyne: lack of activity against poliovirus in the presence of organic matter. Health Lab. Sci. 1978;15:133-7.
686. Carson JA, Favero MS. Comparative resistance of nontuberculous mycobacteria to iodophor germicides. Abstracts of the Annual Meeting of the American Society for Microbiology, 1984:Q101, p.221.
687. Medcom. Medcom Frequently Asked Questions. www.medcompnet.com/faq/faq/html, 2000.
688. Simons C, Walsh SE, Maillard JY, Russell AD. A note: ortho-phthalaldehyde: proposed mechanism of action of a new antimicrobial agent. Lett. Appl. Microbiol. 2000;31:299-302.
689. Walsh SE, Maillard JY, Simons C, Russell AD. Studies on the mechanisms of the antibacterial action of ortho-phthalaldehyde. J. Appl. Microbiol. 1999;87:702-10.
690. Fraud S, Hann AC, Maillard J-Y, Russell AD. Effects of ortho-phthalaldehyde, glutaraldehyde and chlorhexidine diacetate on *Mycobacterium chelonae* and *Mycobacterium abscessus* strains with modified permeability. J. Antimicrob. Chemother. 2003;51:575-84.
691. Cabrera-Martinez RM, Setlow B, Setlow P. Studies on the mechanisms of the sporicidal action of ortho-phthalaldehyde. J. Appl. Microbiol. 2002;92:675-80.
692. Gordon MD, Ezzell RJ, Bruckner NI, Ascenzi JM. Enhancement of mycobactericidal activity of glutaraldehyde with α,β -unsaturated and aromatic aldehydes. J. Indust. Microbiol. 1994;13:77-82.
693. Gregory AW, Schaalje GB, Smart JD, Robison RA. The mycobactericidal efficacy of ortho-phthalaldehyde and the comparative resistances of *Mycobacterium bovis*, *Mycobacterium terrae*, and *Mycobacterium chelonae*. Infect. Control Hosp. Epidemiol. 1999;20:324-30.
694. Walsh SE, Maillard JY, Russell AD. Ortho-phthalaldehyde: a possible alternative to glutaraldehyde for high level disinfection. J. Appl. Microbiol. 1999;86:1039-46.
695. Roberts CG, Chan Myers H. Mycobactericidal activity of dilute ortho-phthalaldehyde solutions. In: Abstracts in Environmental and General Applied Microbiology, Q-265, ASM 98th General Meeting, Atlanta, Georgia, USA, 1998:464-5.
696. Chan-Myers H. Sporicidal activity of ortho-phthalaldehyde as a function of temperature (abstract). Infect. Control Hosp. Epidemiol. 2000;21:101.
697. Chan-Myers H, Roberts C. Effect of temperature and organic soil concentration on biocidal activity of ortho-phthalaldehyde solution (abstract). 2000 Education Meeting of the Association for Professional in Infection Control and Epidemiology, Minneapolis, MN, 2000:31.
698. Bruckner NI, Gordon MD, Howell RG. Odorless aromatic dialdehyde disinfecting and sterilizing composition. US Patent 4,851,449. July, 1989.
699. McDonnell G, Pretzer D. New and developing chemical antimicrobials. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lippincott Williams & Wilkins, 2001:431-43.
700. Fraud S, Maillard J-Y, Russell AD. Comparison of the mycobactericidal activity of ortho-phthalaldehyde, glutaraldehyde, and other dialdehydes by a quantitative suspension test. J. Hosp. Infect. 2001;48:214-21.

701. Sattar SA, Springthorpe VS. New methods for efficacy testing of disinfectants and antiseptics. In: Rutala WA, ed. Disinfection, sterilization and antisepsis: principles and practices in healthcare facilities. Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:174-86.
702. Walsh SE, Maillard J-Y, Russell AD, Hann AC. Possible mechanisms for the relative efficiencies of ortho-phthalaldehyde and glutaraldehyde against glutaraldehyde-resistant *Mycobacterium chelonae*. J. Appl. Microbiol. 2001;91:80-92.
703. Herruzo-Cabrera R, Vizcaino-Alcaide MJ, Rodriguez J. Comparison of the microbicidal efficacy on germ carriers of several tertiary amine compounds with ortho-phthalaldehyde and Perasafe. J. Hosp. Infect. 2006;63:73-8.
704. Herruzo-Cabrera R, Vizcaino-Alcaide MJ, Fernandez-Acenero MJ. The influence of laboratory adaptation on test strains, such as *Pseudomonas aeruginosa*, in the evaluation of the antimicrobial efficacy of ortho-phthalaldehyde. J. Hosp. Infect. 2004;57:217-22.
705. Favero MS. Naturally occurring microorganisms and their resistance to physical and chemical agents. In: Rutala WA, ed. Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research. Washington, DC: Association for Professionals in Infection Control and Epidemiology, 2004:1-15.
706. Cooke RPD, Goddard SV, Whyman-Morris A, Sherwood J, Chatterly R. An evaluation of Cidex OPA (0.55% ortho-phthalaldehyde) as an alternative to 2% glutaraldehyde for high-level disinfection of endoscopes. J. Hosp. Infect. 2003;54:226-31.
707. Streckenbach SC, Alston TA. Perioral stains after ortho-phthalaldehyde disinfection of echo probes. Anesthesiol 2003;99:1032.
708. Wardle E, Jones D. Determination of rinsing volumes following manual endoscope disinfection with ortho-phthalaldehyde (OPA). J Gastroenterol Nurses College Australia 2003;January:7-9.
709. Sokol WN. Nine episodes of anaphylaxis following cystoscopy caused by Cidex OPA (ortho-phthalaldehyde) high-level disinfectant in 4 patients after cystoscopy. J. Allergy Clin. Immunol. 2004;114:392-7.
710. Hession SM. Endoscopic disinfection by ortho-phthalaldehyde in a clinical setting: An evaluation of reprocessing time and costs compared with glutaraldehyde. Gastroenterol. Nurs. 2003;26:110-4.
711. Tucker RC, Lestini BJ, Marchant RE. Surface analysis of clinically used expanded PTFE endoscopic tubing treated by the STERIS PROCESS. ASAIO J. 1996;42:306-13.
712. Hernandez A, Martro E, Matas L, Ausina V. In-vitro evaluation of Pearsafe compared with 2% alkaline glutaraldehyde against *Mycobacterium* spp. J. Hosp. Infect. 2003;54:52-6.
713. Vizcaino-Alcaide MJ, Herruzo-Cabrera R, Fernandez-Acenero MJ. Comparison of the disinfectant efficacy of Persafe and 2% glutaraldehyde in in vitro tests. J. Hosp. Infect. 2003;53:124-8.
714. Lensing HH, Oei HL. Investigations on the sporicidal and fungicidal activity of disinfectants. Zentralblatt für Bakteriologie, Mikrobiologie und Hygiene - 1 - Abt - Originale B, Hygiene 1985;181:487-95.
715. Sagripanti JL, Bonifacino A. Comparative sporicidal effects of liquid chemical agents. Appl. Environ. Microbiol. 1996;62:545-51.
716. Crow S. Peracetic acid sterilization: a timely development for a busy healthcare industry. Infect. Control Hosp. Epidemiol. 1992;13:111-3.
717. Malchesky PS. Medical applications of peracetic acid. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lippincott Williams & Wilkins, 2001:979-96.
718. Mannion PT. The use of peracetic acid for the reprocessing of flexible endoscopes and rigid cystoscopes and laparoscopes. J. Hosp. Infect. 1995;29:313-5.
719. Bradley CR, Babb JR, Ayliffe GA. Evaluation of the Steris System 1 Peracetic Acid Endoscope Processor. J. Hosp. Infect. 1995;29:143-51.
720. Duc DL, Ribiollet A, Dode X, Duceil G, Marchetti B, Calop J. Evaluation of the microbicidal efficacy of Steris System I for digestive endoscopes using GERMANDE and ASTM validation protocols. J. Hosp. Infect. 2001;48:135-41.
721. Alfa MJ, Olson N, Degagne P, Hizon R. New low temperature sterilization technologies: microbicidal activity and clinical efficacy. In: Rutala WA, ed. Disinfection, sterilization, and antisepsis in healthcare. Champlain, New York: Polyscience Publications, 1998:67-78.
722. Alfa MJ, DeGagne P, Olson N, Hizon R. Comparison of liquid chemical sterilization with peracetic acid and ethylene oxide sterilization for long narrow lumens. Am. J. Infect. Control 1998;26:469-77.

723. Seballos RJ, Walsh AL, Mehta AC. Clinical evaluation of a liquid chemical sterilization system for flexible bronchoscopes. *J. Bronch.* 1995;2:192-99.
724. Wallace CG, Agee PM, Demicco DD. Liquid chemical sterilization using peracetic acid. An alternative approach to endoscope processing. *ASAIO J.* 1995;41:151-4.
725. Centers for Disease Control and Prevention. Bronchoscopy-related infections and pseudoinfections - New York, 1996 and 1998. *MMWR* 1999;48:557-60.
726. Middleton AM, Chadwick MV, Gaya H. Disinfection of bronchoscopes, contaminated in vitro with *Mycobacterium tuberculosis*, *Mycobacterium avium-intracellulare* and *Mycobacterium chelonae* in sputum, using stabilized, buffered peracetic acid solution ('Nu-Cidex'). *J. Hosp. Infect.* 1997;37:137-43.
727. Holton J, Shetty N. In-use stability of Nu-Cidex. *J. Hosp. Infect.* 1997;35:245-8.
728. Alasri A, Roques C, Michel G, Cabassud C, Aptel P. Bactericidal properties of peracetic acid and hydrogen peroxide, alone and in combination, and chlorine and formaldehyde against bacterial water strains. *Can. J. Microbiol.* 1992;38:635-42.
729. Stanley P. Destruction of a glutaraldehyde-resistant mycobacterium by a per-oxygen disinfectant. (Abstract). *Am. J. Infect. Control* 1998;26:185.
730. Fleming SJ, Foreman K, Shanley K, Mihrshahi R, Siskind V. Dialyser reprocessing with Renalin. *Am. J. Nephrol.* 1991;11:27-31.
731. Kahn G. Depigmentation caused by phenolic detergent germicides. *Arch. Dermatol.* 1970;102:177-87.
732. Prindle RF. Phenolic compounds. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lea & Febiger, 1983:197-224.
733. Hegna IK. A comparative investigation of the bactericidal and fungicidal effects of three phenolic disinfectants. *J. Appl. Bacteriol.* 1977;43:177-81.
734. Hegna IK. An examination of the effect of three phenolic disinfectants on *Mycobacterium tuberculosis*. *J. Appl. Bacteriol.* 1977;43:183-7.
735. Bergan T, Lystad A. Antitubercular action of disinfectants. *J. Appl. Bacteriol.* 1971;34:751-6.
736. Narang HK, Codd AA. Action of commonly used disinfectants against enteroviruses. *J. Hosp. Infect.* 1983;4:209-12.
737. Cole EC, Rutala WA, Samsa GP. Disinfectant testing using a modified use-dilution method: collaborative study. *J. Assoc. Off. Anal. Chem.* 1988;71:1187-94.
738. Goddard PA, McCue KA. Phenolic compounds. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001:255-81.
739. Wysowski DK, Flynt JW, Jr., Goldfield M, Altman R, Davis AT. Epidemic neonatal hyperbilirubinemia and use of a phenolic disinfectant detergent. *Pediatrics* 1978;61:165-70.
740. Doan HM, Keith L, Shennan AT. Phenol and neonatal jaundice. *Pediatrics* 1979;64:324-5.
741. Shickman MD, Guze LB, Pearce ML. Bacteremia following cardiac catheterization. *N. Engl. J. Med.* 1959;260:1164-6.
742. Ehrenkranz NJ, Bolyard EA, Wiener M, Cleary TJ. Antibiotic-sensitive *Serratia marcescens* infections complicating cardiopulmonary operations: contaminated disinfectant as a reservoir. *Lancet* 1980;2:1289-92.
743. Shere L. Some comparisons of the disinfecting properties of hypochlorites and quaternary ammonium compounds. *Milk Plant Monthly* March 1948:66-9.
744. MacDougall KD, Morris C. Optimizing disinfectant application in healthcare facilities. *Infect Control Today* 2006;June:62-7.
745. Sykes G. *Disinfection and sterilization*. London: E & FN Spon Ltd, 1965.
746. Merianos JJ. Surface-active agents. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001:283-320.
747. Purohit A, Kopferschmitt-Kubler MC, Moreau C, Popin E, Blaumeiser M, Pauli G. Quaternary ammonium compounds and occupational asthma. *International Archives of Occupational & Environmental Health* 2000;73:423-7.
748. Petrocci AN. Surface active agents: quaternary ammonium compounds. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lea & Febiger, 1983:309-29.
749. Smith CR, Nishihara H, Golden F, Hoyt A, Guss CO, Kloetzel MC. The bactericidal effect of surface-active agents on tubercle bacilli. *Public Health Rep.* 1950;48:1588-1600.
750. Broadley SJ, Furr JR, Jenkins PA, Russell AD. Antimycobacterial activity of 'Virkon'. *J. Hosp. Infect.*

- 1993;23:189-97.
751. Angelillo IF, Bianco A, Nobile CG, Pavia M. Evaluation of the efficacy of glutaraldehyde and peroxygen for disinfection of dental instruments. *Lett. Appl. Microbiol.* 1998;27:292-6.
 752. Coates D. Disinfectants and spills of body fluids. *Nurs. RSA* 1992;7:25-7.
 753. Hamouda T, Hayes MM, Cao ZH, et al. A novel surfactant nanoemulsion with broad-spectrum sporicidal activity against *Bacillus* species. *J. Infect. Dis.* 1999;180:1939-49.
 754. Hamouda T, Myc A, Donovan B, Shih AY, Reuter JD, Baker JR. A novel surfactant nanoemulsion with a unique non-irritant topical antimicrobial activity against bacteria, enveloped viruses and fungi. *Microbiol. Res.* 2001;156:1-7.
 755. Hamouda T, Baker JR, Jr. Antimicrobial mechanism of action of surfactant lipid preparations in enteric Gram-negative bacilli. *J. Appl. Microbiol.* 2000;89:397-403.
 756. Widmer AF, Frei R. Antimicrobial activity of glucoprotamin: A clinical study of a new disinfectant for instruments. *Infect Control Hosp Epidemiol* 2003;24:762-4.
 757. Wilson M. Light-activated antimicrobial coating for the continuous disinfection of surfaces. *Infect Control Hosp Epidemiol* 2003;24:782-4.
 758. Schneider PM. New technologies for disinfection and sterilization. In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research.* Washington DC: Association for Professionals in Infection Control and Epidemiology, 2004:127-39.
 759. Rotter ML. Handwashing, hand disinfection, and skin disinfection. In: Wenzel RP, ed. *Prevention and control of nosocomial infections.* Baltimore: Williams and Wilkins, 1997:691-709.
 760. Mandel GL, Bennett JE, Dolin R. *Principles and practices of infectious diseases.* New York: Livingstone, 2000.
 761. Weber DJ, Rutala WA. Use of metals and microbicides in the prevention of nosocomial infections. In: Rutala W, ed. *Disinfection, sterilization, and antisepsis in healthcare.* Champlain, New York: Polyscience Publications, 1995:271-85.
 762. Weber DJ, Rutala WA. Use of metals as microbicides in preventing infections in healthcare. In: Block SS, ed. *Disinfection, sterilization, and preservation.* Philadelphia: Lippincott Williams & Wilkins, 2001:415-30.
 763. Brady MJ, Lisay CM, Yurkovetskiy AV, Sawan SP. Persistent silver disinfectant for the environmental control of pathogenic bacteria. *Am. J. Infect. Control* 2003;31:208-214.
 764. Rusin P, Bright K, Gerba C. Rapid reduction of *Legionella pneumophila* on stainless steel with zeolite coatings containing silver and zinc ions. *Lett. Appl. Microbiol.* 2003;36:69-72.
 765. Bright KR, Gerba CP, Rusin PA. Rapid reduction of *Staphylococcus aureus* populations on stainless steel surfaces by zeolite ceramic coatings containing silver and zinc ions. *J. Hosp. Infect.* 2002;52:307-9.
 766. Landeen LK, Yahya MT, Gerba CP. Efficacy of copper and silver ions and reduced levels of free chlorine in inactivation of *Legionella pneumophila*. *Appl. Environ. Microbiol.* 1989;55:3045-50.
 767. Pyle BH, Broadaway SC, McFeters GA. Efficacy of copper and silver ions with iodine in the inactivation of *Pseudomonas cepacia*. *J. Appl. Bacteriol.* 1992;72:71-9.
 768. Yahya MT, Landeen LK, Messina MC, Kutz SM, Schulze R, Gerba CP. Disinfection of bacteria in water systems by using electrolytically generated copper:silver and reduced levels of free chlorine. *Can. J. Microbiol.* 1990;36:109-16.
 769. Liu Z, Stout JE, Tedesco L, et al. Controlled evaluation of copper-silver ionization in eradicating *Legionella pneumophila* from a hospital water distribution system. *J. Infect. Dis.* 1994;169:919-22.
 770. Noyce JO, Michels H, Keevil CW. Potential use of copper surfaces to reduce survival of epidemic methicillin-resistant *Staphylococcus aureus* in the healthcare environment. *J. Hosp. Infect.* 2006;63:289-97.
 771. Goetz A, Yu VL. Copper-silver ionization: cautious optimism for *Legionella* disinfection and implications for environmental culturing. *Am. J. Infect. Control* 1997;25:449-51.
 772. Miuetzner S, Schwille RC, Farley A, et al. Efficacy of thermal treatment and copper-silver ionization for controlling *Legionella pneumophila* in high-volume hot water plumbing systems in hospitals. *Am. J. Infect. Control* 1997;25:452-7.
 773. Stout JE, Lin YS, Goetz AM, Muder RR. Controlling *Legionella* in hospital water systems: experience with the superheat-and-flush method and copper-silver ionization. *Infect. Control Hosp. Epidemiol.* 1998;19:911-4.

774. Stout JE, Yu VL. Experience of the first 16 hospitals using copper-silver ionization for *Legionella* control: Implications for the evaluation of other disinfection modalities. *Infect Control Hosp Epidemiol* 2003;24:563-8.
775. Russell AD. Ultraviolet radiation. In: Russell AD, Hugo WB, Ayliffe GAJ, eds. Principles and practices of disinfection, preservation and sterilization. Oxford: Blackwell Science, 1999:688-702.
776. Hall KK, Giannetta ET, Getchell-White SI, Durbin LJ, Farr BM. Ultraviolet light disinfection of hospital water for preventing nosocomial *Legionella* infection: A 13-year follow-up. *Infect Control Hosp Epidemiol* 2003;24:580-3.
777. Singh S, Schaaf NG. Dynamic sterilization of titanium implants with ultraviolet light. *Internat. J. Oral Maxillofac. Implants* 1989;4:139-46.
778. Dolman PJ, Dobrogowski MJ. Contact lens disinfection by ultraviolet light. *Am. J. Ophthalmol.* 1989;108:665-9.
779. Shechmeister IL. Sterilization by ultraviolet irradiation. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lea & Febiger, 1991:553-65.
780. National Research Council. Postoperative wound infections - the influence of ultraviolet irradiation of the operating room and of various other factors. *Ann. Surg.* 1964;160:1-125.
781. Sensakovic JW, Smith LG. Nosocomial ultraviolet keratoconjunctivitis. *Infect. Control* 1982;3:475-6.
782. Cefai C, Richards J, Gould FK, McPeake P. An outbreak of respiratory tract infection resulting from incomplete disinfection of ventilatory equipment. *J. Hosp. Infect.* 1990;15:177-82.
783. Gurevich I, Tafuro P, Ristuccia P, Herrmann J, Young AR, Cunha BA. Disinfection of respirator tubing: a comparison of chemical versus hot water machine-assisted processing. *J. Hosp. Infect.* 1983;4:199-208.
784. Rutala WA, Weber DJ, Gergen MF, Gratta AR. Efficacy of a washer-pasteurizer for disinfection of respiratory-care equipment. *Infect. Control Hosp. Epidemiol.* 2000;21:333-6.
785. Jette LP, Lambert NG. Evaluation of two hot water washer disinfectors for medical instruments. *Infect. Control Hosp. Epidemiol.* 1988;9:194-9.
786. Wang C-Y, Wu H-D, Lee L-N, et al. Pasteurization is effective against multidrug-resistant bacteria. *Am. J. Infect. Control* 2006;34:320-2.
787. Dempsey KM, Chiew RF, McKenzie JA, Mitchell DH. Evaluation of the cleaning and disinfection efficacy of the DEKO-190; award-based automated washer/disinfectant. *J. Hosp. Infect.* 2000;46:50-4.
788. Kearns AM, Freeman R, Lightfoot NF. Nosocomial enterococci: resistance to heat and sodium hypochlorite. *J. Hosp. Infect.* 1995;30:193-9.
789. Bradley CR, Fraise AP. Heat and chemical resistance of enterococci. *J. Hosp. Infect.* 1996;34:191-6.
790. Chadwick PR, Oppenheim BA. Vancomycin-resistant enterococci and bedpan washer machines. *Lancet* 1994;344:685.
791. Nystrom B. New technology for sterilization and disinfection. *Am. J. Med.* 1991;91:264S-266S.
792. Sanders FT, Morrow MS. The EPA's role in the regulation of antimicrobial pesticides in the United States. In: Rutala WA, ed. Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research. Washington, DC: Association for Professionals in Infection Control and Epidemiology, 2004:29-41.
793. Anonymous. Memorandum of understanding between the Food and Drug Administration, Public Health Service, and the Environmental Protection Agency, 1993.
794. Ulatowski TA. Current activities concerning the premarket evaluation of infection control devices at the Food and Drug Administration. In: Rutala WA, ed. Disinfection, sterilization, and antisepsis in healthcare. Champlain, New York: Polyscience Publications, 1998:1-7.
795. Cole EC, Rutala WA. Bacterial numbers on penicylinders used in disinfectant testing: use of 24 hour adjusted broth cultures. *J. Assoc. Off. Anal. Chem.* 1988;71:9-11.
796. Cole EC, Rutala WA, Alfano EM. Comparison of stainless steel penicylinders used in disinfectant testing. *J. Assoc. Off. Anal. Chem.* 1988;71:288-9.
797. Cole EC, Rutala WA, Carson JL. Evaluation of penicylinders used in disinfectant testing: bacterial attachment and surface texture. *J. Assoc. Off. Anal. Chem.* 1987;70:903-6.
798. Cole EC, Rutala WA, Samsa GP. Standardization of bacterial numbers of penicylinders used in disinfectant testing: interlaboratory study. *J. Assoc. Off. Anal. Chem.* 1987;70:635-7.
799. Alfano EM, Cole EC, Rutala WA. Quantitative evaluation of bacteria washed from stainless steel penicylinders during AOAC use-dilution method. *J. Assoc. Off. Anal. Chem.* 1988;71:868-71.

800. Favero MS, Groschel DHM. Chemical germicides in the health care field: current status and evaluation of efficacy and research needs. Washington, DC: American Society for Microbiology, 1987.
801. Sattar SA. Microbicidal testing of germicides: an update. In: Rutala WA, ed. Disinfection, sterilization, and antisepsis in healthcare. Champlain, New York: Polyscience Publications, 1998:227-40.
802. Best M. Development of a combined carrier test for disinfectant efficacy. Ottawa, Canada: University of Ottawa, 1994.
803. Sattar SA, Springthorpe VS. Recent developments in methods for testing the germicidal activity of disinfectants and antiseptics. In: Rutala WA, ed. Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research. Washington, DC: Association for Professionals in Infection Control and Epidemiology, 2004:180-8.
804. Sanders FT. Environmental protection agency's role in the regulation of antimicrobial pesticides in the United States. In: Rutala WA, ed. Disinfection, Sterilization and Antisepsis: principles and practices in healthcare facilities. Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:28-40.
805. Groschel DHM. Caveat emptor: do your disinfectants work? Infect. Control 1983;4:144.
806. United States General Accounting Office. Disinfectants: EPA lacks assurance they work., 1990.
807. Johnston MD, Lambert RJW, Hanlon GW, Denyer SP. A rapid method for assessing the suitability of quenching agents for individual biocides as well as combinations. J. Appl. Microbiol. 2002;92:784-9.
808. Russell AD. Neutralization procedures in the evaluation of bactericidal activity. In: Collins CH, Allwood MC, Bloomfield SF, Fox A, eds. Disinfectants: their use and evaluation of effectiveness. London: Academic Press, 1981:45-59.
809. Russell AD, Ahonkhai I, Rogers DT. Microbiological applications of the inactivation of antibiotics and other antimicrobial agents. J. Appl. Bacteriol. 1979;46:207-45.
810. Engley FB, Jr, Dey BP. A universal neutralizing medium for antimicrobial chemicals. Chem. Specialists Manuf. Assoc. Proc. 1970:100-6.
811. Association for the Advancement of Medical Instrumentation. Good hospital practice: Steam sterilization and sterility assurance. AAMI. Arlington, VA, 1993.
812. Association for the Advancement of Medical Instrumentation. Flash sterilization: Steam sterilization of patient care items for immediate use. AAMI. Arlington, VA, 1996.
813. Association for the Advancement of Medical Instrumentation. Steam sterilization and sterility assurance in health care facilities. ANSI/AAMI ST46. Arlington, VA, 2002:ANSI/AAMI ST46:2002.
814. Association for the Advancement of Medical Instrumentation. Ethylene oxide sterilization in health care facilities: Safety and effectiveness. AAMI. Arlington, VA, 1999.
815. Association of Operating Room Nurses. Recommended practices for sterilization in perioperative practice settings. 2000 Standards, Recommended Practices, and Guidelines. Denver, CO: AORN, 2000:347-58.
816. Association for peri-Operative Registered Nurses. Recommended practices for cleaning and caring for surgical instruments and powered equipment. AORN J. 2002;75:727-41.
817. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for prevention of surgical site infection, 1999. Hospital Infection Control Practices Advisory Committee. Infect. Control Hosp. Epidemiol. 1999;20:250-78.
818. Education Design. Best practices for the prevention of surgical site infection. Denver Colorado: Education Design, 1998.
819. Association for the Advancement of Medical Instrumentation. Comprehensive guide to steam sterilization and sterility assurance in health care facilities, ANSI/AAMI ST79. 2006.
820. Association for peri-Operative Registered Nurses. Recommended practice for sterilization in the perioperative practice setting. AORN J. 2006;83:700-22.
821. Singh J, Bhatia R, Gandhi JC, et al. Outbreak of viral hepatitis B in a rural community in India linked to inadequately sterilized needles and syringes. Bull. World Health Organ. 1998;76:93-8.
822. Eickhoff TC. An outbreak of surgical wound infections due to *Clostridium perfringens*. Surg. Gynecol. Obstet. 1962;114:102-8.
823. Favero MS. Sterility assurance: Concepts for patient safety. In: Rutala WA, ed. Disinfection, sterilization and antisepsis: principles and practices in healthcare facilities. Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:110-9.
824. Oxborrow GS, Berube R. Sterility testing-validation of sterilization processes, and sporicide testing. In:

- Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lea & Febiger, 1991:1047-57.
825. Rutala WA, Weber DJ. Clinical effectiveness of low-temperature sterilization technologies. *Infect. Control Hosp. Epidemiol.* 1998;19:798-804.
826. Adler S, Scherrer M, Daschner FD. Costs of low-temperature plasma sterilization compared with other sterilization methods. *J. Hosp. Infect.* 1998;40:125-34.
827. Joslyn L. Sterilization by heat. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lippincott Williams & Wilkins, 2001:695-728.
828. Bucx MJ, Veldman DJ, Beenhakker MM, Koster R. The effect of steam sterilization at 134 degrees C on light intensity provided by fibrelight Macintosh laryngoscopes. *Anaesthesia* 2000;55:185-6.
829. Gilbert JA, Phillips HO. The effect of steam sterilization on plaster casting material. *Clinical Orthopaed Rel Res* 1984:241-4.
830. Agaloco JP, Akers JE, Madsen RE. Moist heat sterilization--myths and realities. *PDA J. Pharmaceutical Sci. Technol.* 1998;52:346-50.
831. Rutala WA, Stiegel MM, Sarubbi FA, Jr. Decontamination of laboratory microbiological waste by steam sterilization. *Appl. Environ. Microbiol.* 1982;43:1311-6.
832. Lauer JL, Battles DR, Vesley D. Decontaminating infectious laboratory waste by autoclaving. *Appl. Environ. Microbiol.* 1982;44:690-4.
833. Rhodes P, Zelner L, Laufman H. A new disposable bowie-Dick-type test pack for prevacuum high-temperature sterilizers. *Med. Instrum.* 1982;16:117-20.
834. Association for the Advancement of Medical Instrumentation. Technical Information Report on process challenge devices/test packs for use in health care facilities, 2003.
835. Young M. Sterilization process monitoring. *Managing Infect Control* 2004;August:70-6.
836. American Society for Healthcare Central Service Professionals. Training Manual for Health Care Central Service Technicians. In: Association AH, ed. Chicago: The Jossey-Bass/American Hospital Association Press Series, 2001:1-271.
837. Crow S. Steam sterilizers: an evolution in design. *Infect. Control Hosp. Epidemiol.* 1993;14:488-90.
838. Gurevich I, Jacobsen E, Cunha BA. Pseudoautoclave failure caused by differences in spore test steam sensitivities. *Am. J. Infect. Control* 1996;24:402-4.
839. Bryce EA, Roberts FJ, Clements B, MacLean S. When the biological indicator is positive: investigating autoclave failures. *Infect. Control Hosp. Epidemiol.* 1997;18:654-6.
840. Barone MA, Faisal AJ, Andrews L, Ahmed J, Rashida B, Kristensen D. Adaptation and validation of a portable steam sterilizer for processing intrauterine device insertion instruments and supplies in low-resource settings. *Am. J. Infect. Control* 1997;25:350-6.
841. Young JH. Sterilization with steam under pressure. In: Morrissey RF, Phillips GB, eds. Sterilization technology: a practical guide for manufacturers and users of health care product. New York: Van Nostrand Reinhold, 1993:81-119.
842. Palenik CJ, Cumberlander ND. Effects of steam sterilization on the contents of sharps containers. *Am. J. Infect. Control* 1993;21:28-33.
843. Rutala WA. Disinfection and flash sterilization in the operating room. *J. Ophthal. Nurs. Technol.* 1991;10:106-15.
844. Maki DG, Hassemer CA. Flash sterilization: carefully measured haste. *Infect. Control* 1987;8:307-10.
845. Barrett T. Flash sterilization: What are the risks? In: Rutala WA, ed. Disinfection, sterilization and antisepsis: principles and practices in healthcare facilities. Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:70-6.
846. Vesley D, Langholz AC, Rohlfing SR, Foltz WE. Fluorimetric detection of a *Bacillus stearothermophilus* spore-bound enzyme, α -D-glucosidase, for rapid identification of flash sterilization failure. *Appl. Environ. Microbiol.* 1992;58:717-9.
847. Rutala WA, Gergen MF, Weber DJ. Evaluation of a rapid readout biological indicator for flash sterilization with three biological indicators and three chemical indicators. *Infect. Control Hosp. Epidemiol.* 1993;14:390-4.
848. Strzelecki LR, Nelson JH. Evaluation of closed container flash sterilization system. *Orthoped. Nurs.* 1989;8:21-4.
849. Hood E, Stout N, Catto B. Flash sterilization and neurosurgical site infections: Guilt by association. *Am. J.*

- Infect. Control 1997;25:156.
850. Rutala WA, Weber DJ, Chappell KJ. Patient injury from flash-sterilized instruments. *Infect. Control Hosp. Epidemiol.* 1999;20:458.
 851. Schneider PM. Low-temperature sterilization alternatives in the 1990s. *Tappi J.* 1994;77:115-9.
 852. Environmental Protection Agency. Protection of stratospheric ozone; Proposed Rule. 40 CFR Part 82. *Fed. Regist.* 1993.
 853. Schneider PM. Emerging low temperature sterilization technologies (non-FDA approved). In: Rutala WA, ed. *Disinfection, sterilization, and antisepsis in healthcare.* Champlain, New York: Polyscience Publications, 1998:79-92.
 854. Gross D. Ethylene oxide sterilization and alternative methods. *Surg. Serv. Management* 1995;1:16-7.
 855. Holler C, Martiny H, Christiansen B, Ruden H, Gundermann KO. The efficacy of low temperature plasma (LTP) sterilization, a new sterilization technique. *Zentralbl. Hyg. Umweltmed.* 1993;194:380-91.
 856. Rutala WA, Gergen MF, Weber DJ. Comparative evaluation of the sporicidal activity of new low-temperature sterilization technologies: ethylene oxide, 2 plasma sterilization systems, and liquid peracetic acid. *Am. J. Infect. Control* 1998;26:393-8.
 857. Ernst RR, Doyle JE. Sterilization with gaseous ethylene oxide: a review of chemical and physical factors. *Biotech. Bioeng.* 1968;10.
 858. Joslyn L. Gaseous chemical sterilization. In: Block SS, ed. *Disinfection, sterilization, and preservation.* Philadelphia: Lippincott Williams & Wilkins, 2001:337-60.
 859. Fisher AA. Ethylene oxide dermatitis. *Cutis* 1984;34:20, 22, 24.
 860. Jay WM, Swift TR, Hull DS. Possible relationship of ethylene oxide exposure to cataract formation. *Am. J. Ophthalmol.* 1982;93:727-32.
 861. Salinas E, Sasich L, Hall DH, Kennedy RM, Morriss H. Acute ethylene oxide intoxication. *Drug Intell. Clin. Pharm.* 1981;15:384-6.
 862. Marchand M, Delesvonn R, Claeys C. The toxicity of ethylene oxide and a report on three fatal cases of poisoning. *Am. Arch. Indust. Health* 1958;18:60.
 863. Finelli PF, Morgan TF, Yaar I, Granger CV. Ethylene oxide-induced polyneuropathy. A clinical and electrophysiologic study. *Arch. Neurol.* 1983;40:419-21.
 864. Estrin WJ, Becker CE. Evidence of neurologic dysfunction related to long-term ethylene oxide exposure. *Arch. Neurol.* 1987;44:1283-6.
 865. Estrin WJ, Bowler RM, Lash A, Becker CE. Neurotoxicological evaluation of hospital sterilizer workers exposed to ethylene oxide. *J. Toxicol. Clin. Toxicol.* 1990;28:1-20.
 866. Crystal HA, Schaumburg HH, Grober E, Fuld PA, Lipton RB. Cognitive impairment and sensory loss associated with chronic low-level ethylene oxide exposure. *Neurology* 1988;38:567-9.
 867. Shaham J, Levi Z, Gurvich R, Shain R, Ribak J. Hematological changes in hospital workers due to chronic exposure to low levels of ethylene oxide. *J. Occup. Environ. Med.* 2000;42:843-50.
 868. Lindbohm ML, Hemminki K, Bonhomme MG, et al. Effects of paternal occupational exposure on spontaneous abortions. *Am. J. Public Health* 1991;81:1029-33.
 869. Hemminki K, Mutanen P, Saloniemi I, Niemi M-L, Vainio H. Spontaneous abortions in hospital staff engaged in sterilising instruments with chemical agents. *Br. Med. J.* 1982;285:1461-3.
 870. Rowland AS, Baird DD, Shore DL, Darden B, Wilcox AJ. Ethylene oxide exposure may increase the risk of spontaneous abortion, preterm birth, and postterm birth. *Epidemiology* 1996;7:363-8.
 871. National Toxicology Program. <http://ntp-server.niehs.nih.gov/>.
 872. Anonymous. Ethylene oxide sterilization: How hospitals can adapt to the changes. *Health Devices* 1994;23:485-92.
 873. Occupational Safety and Health Administration. Ethylene Oxide: OSHA Fact Sheet: Occupational Safety and Health Administration, 2002.
 874. Cardenas-Camarena L. Ethylene oxide burns from improperly sterilized mammary implants. *Ann. Plast. Surg.* 1998;41:361-9.
 875. Windebank AJ, Blehrud MD. Residual ethylene oxide in hollow fiber hemodialysis units is neurotoxic in vitro. *Ann. Neurol.* 1989;26:63-8.
 876. Occupational Health and Safety Administration. Chemical sampling information-Ethylene chlorohydrin: Occupational Safety and Health Administration, 2002.
 877. Parisi AN, Young WE. Sterilization with ethylene oxide and other gases. In: Block SS, ed. *Disinfection,*

- sterilization, and preservation. Philadelphia: Lea & Febiger, 1991:580-95.
878. Ries MD, Weaver K, Beals N. Safety and efficacy of ethylene oxide sterilized polyethylene in total knee arthroplasty. *Clin. Orthop.* 1996;159-63.
879. Alfa MJ, DeGagne P, Olson N. Bacterial killing ability of 10% ethylene oxide plus 90% hydrochlorofluorocarbon sterilizing gas. *Infect. Control Hosp. Epidemiol.* 1997;18:641-5.
880. Parker HH, Johnson RB. Effectiveness of ethylene oxide for sterilization of dental handpieces. *J. Dent.* 1995;23:113-5.
881. Jacobs PT, Lin SM. Sterilization processes utilizing low-temperature plasma. In: Block SS, ed. *Disinfection, sterilization, and preservation.* Philadelphia: Lippincott Williams & Wilkins, 2001:747-63.
882. Rutala WA, Gergen MF, Weber DJ. Sporicidal activity of a new low-temperature sterilization technology: the Sterrad 50 sterilizer. *Infect. Control Hosp. Epidemiol.* 1999;20:514-6.
883. Kyi MS, Holton J, Ridgway GL. Assessment of the efficacy of a low temperature hydrogen peroxide gas plasma sterilization system. *J. Hosp. Infect.* 1995;31:275-84.
884. Jacobs PT, Smith D. The new Sterrad 100S sterilization system: Features and advantages. *Zentr. Steril.* 1998;6:86-94.
885. Rudolph H, Hilbert M. Practical testing of the new plasma sterilizer "Sterrad 100S" in the Diakonkrankenhaus Rotenburg. *Zentr. Steril.* 1997;5:207-15.
886. Bar W, Marquez de Bar G, Naumann A, Rusch-Gerdes S. Contamination of bronchoscopes with *Mycobacterium tuberculosis* and successful sterilization by low-temperature hydrogen peroxide plasma sterilization. *Am. J. Infect. Control* 2001;29:306-11.
887. Centers for Disease Control and Prevention. Corneal decompensation after intraocular ophthalmic surgery—Missouri, 1998. *MMWR* 1998;47:306-9.
888. Duffy RE, Brown SE, Caldwell KL, et al. An epidemic of corneal destruction caused by plasma gas sterilization. *Arch. Ophthalmol.* 2000;118:1167-76.
889. Jarvis WR. Hospital Infections Program, Centers for Disease Control and Prevention: On-site outbreak investigations, 1990-1999: How often are germicides or sterilants the source? In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: principles and practices in healthcare facilities.* Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:41-8.
890. Borneff M, Ruppert J, Okpara J, et al. Efficacy testing of low-temperature plasma sterilization (LTP) with test object models simulating practice conditions. *Zentr. Steril.* 1995;3:361-71.
891. Borneff-Lipp M, Okpara J, Bodendorf M, Sonntag HG. Validation of low-temperature-plasma (LPT) sterilization systems: Comparison of two technical versions, the Sterrad 100, 1.8 and the 100S. *Hygiene und Mikrobiologie* 1997;3:21-8.
892. Roberts C, Antonoplos P. Inactivation of human immunodeficiency virus type 1, hepatitis A virus, respiratory syncytial virus, vaccinia virus, herpes simplex virus type 1, and poliovirus type 2 by hydrogen peroxide gas plasma sterilization. *Am. J. Infect. Control* 1998;26:94-101.
893. Okpara-Hofmann J, Knoll M, Durr M, Schmitt B, Borneff-Lipp M. Comparison of low-temperature hydrogen peroxide gas plasma sterilization for endoscopes using various Sterrad models. *J. Hosp. Infect.* 2005;59:280-5.
894. Timm D, Gonzales D. Effect of sterilization on microstructure and function of microsurgical scissors. *Surg. Serv. Management* 1997;3:47-9.
895. Feldman LA, Hui HK. Compatibility of medical devices and materials with low-temperature hydrogen peroxide gas plasma. *Med. Dev. Diag. Indust.* 1997;19:57-62.
896. Muscarella LF. Leading a horse to water: Are crucial lessons in endoscopy and outbreak investigations being learned? *Infect. Control Hosp. Epidemiol.* 2002;23:358-60.
897. Gurevich I, Qadri SMH, Cunha BA. False-positive results of spore tests from improper clip use with the Steris chemical sterilant system. *Infect. Control Hosp. Epidemiol.* 1992;21:42-3.
898. Kralovic RC. Use of biological indicators designed for steam or ethylene oxide to monitor a liquid chemical sterilization process. *Infect. Control Hosp. Epidemiol.* 1993;14:313-9.
899. Bond WW. Biological indicators for a liquid chemical sterilizer. *Infect. Control Hosp. Epidemiol.* 1993;14:565.
900. Bond WW. Biological indicators for a liquid chemical sterilizer: a solution to the instrument reprocessing problem? *Infect. Control Hosp. Epidemiol.* 1993;14:309-12.
901. Malchesky PS. Biological indicators for a liquid chemical sterilizer. *Infect. Control Hosp. Epidemiol.*

- 1993;14:563-6.
902. Daschner F. STERIS SYSTEM 1 in Germany. *Infect. Control Hosp. Epidemiol.* 1994;15:294, 296.
 903. Sorin M, Segal-Maurer S, Urban C, Combest A, Rahal JJ. Nosocomial transmission of imipenem-resistant *Pseudomonas aeruginosa* following bronchoscopy associated with improper connection to the STERIS System 1 Processor. *Infect. Control Hosp. Epidemiol.* 2001;22:409-13.
 904. Food and Drug Administration, Division of General and Restorative Devices. Guidance on Premarket Notification [510(k)] Submissions for Sterilizers Intended for Use in Health Care Facilities. Rockville, MD. 1993.
 905. Vickery K, Deva AK, Zou J, Kumaradeva P, Bissett L, Cossart YE. Inactivation of duck hepatitis B virus by a hydrogen peroxide gas plasma sterilization system: laboratory and 'in use' testing. *J. Hosp. Infect.* 1999;41:317-22.
 906. Vassal S, Favennec L, Ballet JJ, Brasseur P. Hydrogen peroxide gas plasma sterilization is effective against *Cryptosporidium parvum* oocysts. *Am. J. Infect. Control* 1998;26:136-8.
 907. Penna TC, Ferraz CA, Cassola MA. The presterilization microbial load on used medical devices and the effectiveness of hydrogen peroxide gas plasma against *Bacillus subtilis* spores. *Infect. Control Hosp. Epidemiol.* 1999;20:465-72.
 908. Bryce EA, Chia E, Logelin G, Smith JA. An evaluation of the AbTox Plazlyte Sterilization System. *Infect. Control Hosp. Epidemiol.* 1997;18:646-53.
 909. Graham GS, Riley R. Sterilization manufacturers: Interactions with regulatory agencies. In: Rutala WA, ed. *Disinfection, sterilization, and antisepsis in healthcare.* Champlain, New York: Polyscience Publications, 1998:41-8.
 910. Royce A, Bowler C. Ethylene oxide sterilisation-some experiences and some practical limitations. *J. Pharm. Pharmacol.* 1961;13:87t-94t.
 911. Nystrom B. Disinfection of surgical instruments. *J. Hosp. Infect.* 1981;2:363-8.
 912. Rutala WA, Gergen MF, Jones JF, Weber DJ. Levels of microbial contamination on surgical instruments. *Am. J. Infect. Control* 1998;26:143-5.
 913. Alfa MJ, Nemes R. Inadequacy of manual cleaning for reprocessing single-use, triple-lumen sphinctertomes: Simulated-use testing comparing manual with automated cleaning methods. *Am. J. Infect. Control* 2003;31:193-207.
 914. Alfa MJ, Nemes R. Reprocessing of lumened instruments. In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research.* Washington DC: Association for Professionals in Infection Control and Epidemiology, 2004:189-99.
 915. Bargmann LS, Bargmann BC, Collier JP, Currier BH, Mayor MB. Current sterilization and packaging methods for polyethylene. *Clin. Orthop.* 1999:49-58.
 916. Williams IR, Mayor MB, Collier JP. The impact of sterilization method on wear in knee arthroplasty. *Clin. Orthop.* 1998:170-80.
 917. Russell AD. Ionizing radiation. In: Russell AD, Hugo WB, Ayliffe GAJ, eds. *Principles and practices of disinfection, preservation and sterilization.* Oxford: Blackwell Science, 1999:675-87.
 918. Hansen JM, Shaffer HL. Sterilization and preservation by radiation sterilization. In: Block SS, ed. *Disinfection, sterilization, and preservation.* Philadelphia: Lippincott Williams & Wilkins, 2001:729-46.
 919. Lagergren ER. Recent advances in sterilization. *J Infect Control (Asia)* 1998;1:11-3.
 920. Perkins JJ. *Principles and methods of sterilization in health sciences.* Springfield, IL: Charles C Thomas, 1969.
 921. Favero MS, Bond WW. The use of liquid chemical germicides. In: Morrissey RF, Phillips GB, eds. *Sterilization technology: A practical guide for manufacturers and users of health care products.* New York: Van Nostrand Reinhold, 1993:309-334.
 922. Muscarella LF. Are all sterilization processes alike? *AORN J.* 1998;67:966-70, 973-6.
 923. Levy RV. Sterile filtration of liquids and gases. In: Block SS, ed. *Disinfection, sterilization, and preservation.* Philadelphia: Lippincott Williams & Wilkins, 2001:795-822.
 924. Wallhausser KH. Is the removal of microorganisms by filtration really a sterilization method? *J. Parenter. Drug Assoc.* 1979;33:156-70.
 925. Webb BC, Thomas CJ, Harty DW, Willcox MD. Effectiveness of two methods of denture sterilization. *J. Oral Rehabil.* 1998;25:416-23.
 926. Rohrer MD, Bulard RA. Microwave sterilization. *J. Am. Dent. Assoc.* 1985;110:194-8.

927. Rohrer MD, Terry MA, Bulard RA, Graves DC, Taylor EM. Microwave sterilization of hydrophilic contact lenses. *Am. J. Ophthalmol.* 1986;101:49-57.
928. Douglas C, Burke B, Kessler DL, Cicmanec JF, Bracken RB. Microwave: practical cost-effective method for sterilizing urinary catheters in the home. *Urology* 1990;35:219-22.
929. Kindle G, Busse A, Kampa D, Meyer-Konig U, Daschner FD. Killing activity of microwaves in milk. *J. Hosp. Infect.* 1996;33:273-8.
930. Harris MG, Rechberger J, Grant T, Holden BA. In-office microwave disinfection of soft contact lenses. *Optom. Vis. Sci.* 1990;67:129-32.
931. Mervine J, Temple R. Using a microwave oven to disinfect intermittent-use catheters. *Rehabil. Nurs.* 1997;22:318-20.
932. Najdovski L, Dragas AZ, Kotnik V. The killing activity of microwaves on some non-sporogenic and sporogenic medically important bacterial strains. *J. Hosp. Infect.* 1991;19:239-47.
933. Rosaspina S, Salvatorelli G, Anzanel D, Bovolenta R. Effect of microwave radiation on *Candida albicans*. *Microbios* 1994;78:55-9.
934. Welt BA, Tong CH, Rossen JL, Lund DB. Effect of microwave radiation on inactivation of *Clostridium sporogenes* (PA 3679) spores. *Appl. Environ. Microbiol.* 1994;60:482-8.
935. Latimer JM, Matsen JM. Microwave oven irradiation as a method for bacterial decontamination in a clinical microbiology laboratory. *J. Clin. Microbiol.* 1977;6:340-2.
936. Sanborn MR, Wan SK, Bulard R. Microwave sterilization of plastic tissue culture vessels for reuse. *Appl. Environ. Microbiol.* 1982;44:960-4.
937. Rosaspina S, Salvatorelli G, Anzanel D. The bactericidal effect of microwaves on *Mycobacterium bovis* dried on scalpel blades. *J. Hosp. Infect.* 1994;26:45-50.
938. Engelhardt JP, Grun L, Dahl HJ. Factors affecting sterilization in glass bead sterilizers. *J. Endod.* 1984;10:465-70.
939. Smith GE. Glass bead sterilization of orthodontic bands. *Am. J. Orthod. Dentofacial Orthop.* 1986;90:243-9.
940. Sisco V, Winters LL, Zange LL, Brennan PC. Efficacy of various methods of sterilization of acupuncture needles. *J. Manip. Physiol. Therap.* 1988;11:94-7.
941. Klapes NA, Vesley D. Vapor-phase hydrogen peroxide as a surface decontaminant and sterilant. *Appl. Environ. Microbiol.* 1990;56:503-6.
942. French GL, Otter JA, Shannon KP, Adams NMT, Watling D, Parks MJ. Tackling contamination of the hospital environment by methicillin-resistant *Staphylococcus aureus* (MRSA): A comparison between conventional terminal cleaning and hydrogen peroxide vapour decontamination. *J. Hosp. Infect.* 2004;57:31-7.
943. Jeanes A, Rao G, Osman M, Merrick P. Eradication of persistent environmental MRSA. *J. Hosp. Infect.* 2005;61:85-6.
944. Bates CJ, Pearse R. Use of hydrogen peroxide vapour for environmental control during a *Serratia* outbreak in a neonatal intensive care unit. *J. Hosp. Infect.* 2005;61:364-6.
945. Boyce JM, Havill NL, Otter JA, et al. Impact of hydrogen peroxide vapor room bio-decontamination on environmental contamination and nosocomial transmission of *Clostridium difficile*. *The Society of Healthcare Epidemiology of America*, 2006;Abstract 155:109.
946. Berrington AW, Pedler SJ. Investigation of gaseous ozone for MRSA decontamination of hospital side-rooms. *J. Hosp. Infect.* 1998;40:61-5.
947. Gaspar MC, Pelaez B, Fernandez C, Fereres J. Microbiological efficacy of Sterrad 100S and LTSF sterilisation systems compared to ethylene oxide. *Zentr. Steril.* 2002;10:91-9.
948. Kanemitsu K, Kunishima H, Imasaka T, et al. Evaluation of a low-temperature steam and formaldehyde sterilizer. *J. Hosp. Infect.* 2003;55:47-52.
949. Kanemitsu K, Imasaka T, Ishikawa S, et al. A comparative study of ethylene oxide gas, hydrogen peroxide gas plasma, and low-temperature steam formaldehyde sterilization. *Infect Control Hosp Epidemiol* 2005;26:486-9.
950. Roncoroni AJ, Casewell MW, Phillips I. The disinfection of clinically contaminated Matburn suction pumps and baby incubators in an 'Aseptor' formalin cabinet. *J. Hosp. Infect.* 1980;1:251-9.
951. Cumberland NS, Botting FG. Formaldehyde vapour cabinets. *J. Hosp. Infect.* 1991;19:67-70.
952. Jeng DK, Woodworth AG. Chlorine dioxide gas sterilization of oxygenators in an industrial scale

- sterilizer: a successful model. *Artif. Organs* 1990;14:361-8.
953. Knapp JE, Battisti DL. Chloride dioxide. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001:215-27.
954. Kowalski JB. Sterilization of medical devices, pharmaceutical components, and barrier isolation systems with gaseous chlorine dioxide. In: Morrissey RF, Kowalski JB, eds. *Sterilization of medical products*. Champlain, NY: Polyscience Publications, 1998:313-23.
955. Portner DM, Hoffman RK. Sporicidal effect of peracetic acid vapor. *Appl. Microbiol.* 1968;16:1782-5.
956. Mata-Portuguez VH, Perez LS, Acosta-Gio E. Sterilization of heat-resistant instruments with infrared radiation. *Infect. Control Hosp. Epidemiol.* 2002;23.
957. Frey R. The structural and functional prerequisites for a central sterile supply department (CSSD). *Zentr. Steril.* 2000;8:128-40.
958. Reich RR, Fleming W, Burgess DJ. Sterilization validation: it's not just for industry. *Infect. Control Steril. Technol.* 1996;2.
959. American Institute of Architects. *Guidelines for design and construction of hospital and health care facilities*. Washington, DC: The American Institute of Architects Press, 2001.
960. DesCoteaux JG, Poulin EC, Julien M, Guidoin R. Residual organic debris on processed surgical instruments. *AORN J.* 1995;62:23-30.
961. Rutala WA, Weber DJ. A review of the use of gowns and drapes (single use and reusable) in healthcare. *Infect. Control Hosp. Epidemiol.* 2001;22:248-57.
962. Association of peri-Operative Registered Nurses. Recommended practices for sterilization in the perioperative practice setting. *AORN J.* 2006;83:700-22.
963. Taurasi R. Comfortable PPE? Maximum tray weight? *Healthcare Purchasing News* 2004;July:48.
964. Chobin N, Furr D, Nuyttens A. Wet packs and plastic accessory cases. *Infect Control Today* 2004;August:24, 28-30.
965. Dunkelberg H, Fleitmann-Glende F. Measurement of the microbial barrier effectiveness of sterilization containers in terms of the log reduction value for prevention of nosocomial infections. *Am. J. Infect. Control* 2006;34:285-9.
966. Rutala WA, Weber DJ. Choosing a sterilization wrap. *Infect. Control Today* 2000;4:64,70.
967. Maloney JM, Kohut RD. Infection control, barrier protection and the treatment environment. *Dent. Hyg. (Chic.)* 1987;61:310-3.
968. Mayworm D. Sterile shelf life and expiration dating. *J. Hosp. Supply, Process. Distri.* 1984;2:32-5.
969. Cardo DM, Sehulster LM. Central sterile supply. In: Mayhall CG, ed. *Infect. Control and Hosp. Epidemiol.* Philadelphia: Lippincott Williams & Wilkins, 1999:1023-30.
970. Klapes NA, Greene VW, Langholz AC. Microbial contamination associated with routine aseptic practice. *J. Hosp. Infect.* 1987;10:299-304.
971. Butt WE, Bradley DV, Jr., Mayhew RB, Schwartz RS. Evaluation of the shelf life of sterile instrument packs. *Oral Surg. Oral Med. Oral Pathol.* 1991;72:650-4.
972. Webster J, Lloyd W, Ho P, Burrige C, George N. Rethinking sterilization practices: Evidence for event-related outdated. *Infect. Control Hosp. Epidemiol.* 2003;24:622-4.
973. Widmer AF, Houston A, Bollinger E, Wenzel RP. A new standard for sterility testing for autoclaved surgical trays. *J. Hosp. Infect.* 1992;21:253-60.
974. Schneider PM, Reich RR, Kirckof SS, Foltz WG. Performance of various steam sterilization indicators under optimum and sub-optimum exposure conditions. In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research*. Washington DC: Association for Professionals in Infection Control and Epidemiology, 2004:200-23.
975. Greene VW. Control of sterilization process. In: Russell AD, Hugo WB, Ayliffe GAJ, eds. *Principles and practice of disinfection, preservation and sterilization*. Oxford, England: Blackwell Scientific Publications, 1992:605-24.
976. Vesley D, Nellis MA, Allwood PB. Evaluation of a rapid readout biological indicator for 121°C gravity and 132°C vacuum-assisted steam sterilization cycles. *Infect. Control Hosp. Epidemiol.* 1995;16:281-6.
977. Rutala WA, Jones SM, Weber DJ. Comparison of a rapid readout biological indicator for steam sterilization with four conventional biological indicators and five chemical indicators. *Infect. Control Hosp. Epidemiol.* 1996;17:423-8.
978. Alfa MJ, Olson N, DeGagne P, Jackson M. Evaluation of rapid readout biological indicators for 132°C

- gravity and 132°C vacuum-assisted steam sterilization cycles using a new automated fluorescent reader. *Infect. Control Hosp. Epidemiol.* 2002;23:388-92.
979. Koncur P, Janes JE, Ortiz PA. 20 second sterilization indicator tests equivalent to BIs. *Infect. Control Steril. Technol.* 1998;26-8, 30, 32-4.
980. Perkins RE, Bodman HA, Kundsins RB, Walter CW. Monitoring steam sterilization of surgical instruments: a dilemma. *Appl. Environ. Microbiol.* 1981;42:383-7.
981. Kotilainen HR, Gantz NM. An evaluation of three biological indicator systems in flash sterilization. *Infect. Control* 1987;8:311-6.
982. Kleinegger CL, Yeager DL, Huling JK, Drake DR. The effects of contamination on biological monitoring. *Infect. Control Hosp. Epidemiol.* 2001;22:391-2.
983. Centers for Disease Control. False-positive results of spore tests in ethylene oxide sterilizers - Wisconsin. *MMWR* 1981;30:238-40.
984. Association of Operating Room Nurses. AORN standards and recommended practices for perioperative nursing. 1987:Section III:14.1-III:14.11, AORN, Denver, CO.
985. Gurevich I, Holmes JE, Cunha BA. Presumed autoclave failure due to false-positive spore strip tests. *Infect. Control* 1982;3:388-92.
986. Epstein BJ, Lattimer JM, Matsen JM, Garibaldi RA. False positive spore strip sterility tests with steam sterilization. *Am. J. Infect. Control* 1983;11:71-3.
987. Association for the Advancement of Medical Instrumentation. Good hospital practice: steam sterilization and sterility assurance. Arlington, VA: AAMI, 1988.
988. Baird RM. Sterility assurance: Concepts, methods and problems. In: Russell AD, Hugo WB, Ayliffe GAJ, eds. *Principles and practice of disinfection, preservation and sterilization*. Oxford, England: Blackwell Scientific Publications, 1999:787-99.
989. Coulter WA, Chew-Graham CA, Cheung SW, Burke FJT. Autoclave performance and operator knowledge of autoclave use in primary care: a survey of UK practices. *J. Hosp. Infect.* 2001;48:180-5.
990. Greene VW. Reuse of disposable devices. In: Mayhall CG, ed. *Infect. Control and Hosp. Epidemiol.* Philadelphia: Lippincott Williams & Wilkins, 1999:1201-8.
991. Avital B, Khan M, Krum D, Jazayeri M, Hare J. Repeated use of ablation catheters: a prospective study. *J. Am. Coll. Cardiol.* 1993;22:1367-72.
992. Dunnigan A, Roberts C, McNamara M, Benson DW, Jr., Benditt DG. Success of re-use of cardiac electrode catheters. *Am. J. Cardiol.* 1987;60:807-10.
993. Aton EA, Murray P, Fraser V, Conaway L, Cain ME. Safety of reusing cardiac electrophysiology catheters. *Am. J. Cardiol.* 1994;74:1173-5.
994. Brown SA, Merritt K, Woods TO, McNamee SG, Hitchins VM. Effects of different disinfection and sterilization methods on tensile strength of materials used for single-use devices. *Biomed. Instrum. Technol.* 2002;January/February:23-7.
995. Food and Drug Administration. Enforcement Priorities for Single-Use Devices Reprocessed by Third Parties and Hospitals, Rockville, MD., 2000.
996. Ulatowski TA. FDA: Reuse of Single-Use Devices. In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research*. Washington, DC: Association for Professionals in Infection Control and Epidemiology, 2004:15-23.
997. Occupational Health and Safety Administration. Hazard Communication Standard. 29 CFR 1910.1200, OSHA, Washington, DC.
998. Edens AL. Occupational Safety and Health Administration: Regulations affecting healthcare facilities. In: Rutala WA, ed. *Disinfection, Sterilization and Antisepsis: Principles and practices in healthcare facilities*. Washington, D.C.: Association for Professionals in Infection Control and Epidemiology, Inc., 2001:49-58.
999. Schultz JK. Decontamination: recommended practices. In: Reichert M, Young JH, eds. *Sterilization technology for the health care facility*. Gaithersburg, MD: Aspen Publication, 1997:10-20.
1000. Occupational Health and Safety Administration. Ethylene Oxide Standard. Vol. 29 CFR 1910.1047, OSHA, Washington, DC.
1001. Occupational Safety and Health Administration. Formaldehyde Standard. Vol. 29 CFR 1910.1048, Washington, DC.
1002. Buxton AE, Anderson RL, Werdegar D, Atlas E. Nosocomial respiratory tract infection and colonization with *Acinetobacter calcoaceticus*. Epidemiologic characteristics. *Am. J. Med.* 1978;65:507-13.

1003. Snyderman DR. Hepatitis B infection from medical personnel. *JAMA* 1976;236:1009.
1004. Martiny H, Floss H. Residuals on medical devices following reprocessing. *J. Hosp. Infect.* 2001;48 (Supplement):S88-S92.
1005. Taylor DM. Inactivation of prions by physical and chemical means. *J. Hosp. Infect.* 1999;43 (supplement):S69-S76.
1006. Best M, Sattar SA, Springthorpe VS, Kennedy ME. Comparative mycobactericidal efficacy of chemical disinfectants in suspension and carrier tests. *Appl. Environ. Microbiol.* 1988;54:2856-8.
1007. Weber DJ, Rutala WA. Environmental issues and nosocomial infections. In: Wenzel RP, ed. *Prevention and control of nosocomial infections*. Baltimore: Williams and Wilkins, 1991:1042-64.
1008. Palmer PH, Yeoman DM. A study to assess the value of disinfectants when washing ward floors. *Med. J. Aust.* 1972;2:1237-9.
1009. Centers for Disease Control and Prevention. Preventing the spread of vancomycin resistance - report from the Hospital Infection Control Practices Advisory Committee. *Fed. Regist.* 1994;25758-63.
1010. Ayliffe GA, Collins BJ, Lowbury EJ. Cleaning and disinfection of hospital floors. *BMJ* 1966;5511:442-5.
1011. Neely AN. A survey of gram-negative bacteria survival on hospital fabrics and plastics. *J. Burn Care Rehabil.* 2000;21:523-7.
1012. Rutala WA. Disinfection and sterilization of patient-care items. *Infect. Control Hosp. Epidemiol.* 1996;17:377-84.
1013. Environmental Protection Agency. Pesticides: Regulating Pesticides. <http://www.epa.gov/oppad001/chemregindex.htm>, 2003.
1014. Hoffman PN, Layzell SK. Household bleach as disinfectant for use by injecting drug users. *Lancet* 1993;342:743.
1015. Chu NS, Chan-Myers H, Ghazanfari N, Antonoplos P. Levels of naturally occurring microorganisms on surgical instruments after clinical use and after washing. *Am. J. Infect. Control* 1999;27:315-9.
1016. Hoffmann KK, Weber DJ, Rutala WA. Pseudoepidemic of *Rhodotorula rubra* in patients undergoing fiberoptic bronchoscopy. *Infect. Control Hosp. Epidemiol.* 1989;10:511-4.
1017. Lowry PW, Jarvis WR. Use of tap water and disinfection practices in outpatient settings. A survey of otolaryngologists. *Arch. Otolaryngol. Head Neck Surg.* 1991;117:886-8.
1018. Fahey BJ, Koziol DE, Banks SM, Henderson DK. Frequency of nonparenteral occupational exposures to blood and body fluids before and after universal precautions training. *Am. J. Med.* 1991;90:145-53.
1019. Beekmann SE, Vlahov D, Koziol DE, McShalley ED, Schmitt JM, Henderson DK. Temporal association between implementation of universal precautions and a sustained, progressive decrease in percutaneous exposures to blood. *Clin. Infect. Dis.* 1994;18:562-9.
1020. Gerberding JL, Littell C, Tarkington A, Brown A, Schechter WP. Risk of exposure of surgical personnel to patients' blood during surgery at San Francisco General Hospital. *N. Engl. J. Med.* 1991;324:1788-93.
1021. Mast ST, Woolwine JD, Gerberding JL. Efficacy of gloves in reducing blood volumes transferred during simulated needlestick injury. *J. Infect. Dis.* 1993;168:1589-92.
1022. Wendt C, Herwaldt LA. Epidemics: Identification and Management. In: Wenzel RP, ed. *Prevention and Control of Nosocomial Infections*. Baltimore: Williams & Wilkins, 1997:175-214.
1023. Feigal DW, Gardner SN, McClellan M. Ensuring safe and effective medical devices. *N. Engl. J. Med.* 2003;348:191-2.
1024. Oie S, Kamiya A. Microbial contamination of antiseptics and disinfectants. *Am. J. Infect. Control* 1996;24:389-95.
1025. Strzelecki LR, Nelson JH. Evaluation of closed container flash sterilization system. *Orthop. Nurs.* 1989;8:21-4.
1026. Burgess DJ, Reich RR. Industrial ethylene oxide sterilization. In: Morrissey RF, Phillips GB, eds. *Sterilization technology: a practical guide for manufacturers and users of health care product*. New York: Van Nostrand Reinhold, 1993:120-51.
1027. Conviser CA, C W. Ethylene oxide sterilization: sterilant alternatives. In: Reichert M, Young JH, eds. *Sterilization technology for the health care facility*. Gaithersburg, MD: Aspen Publication, 1997:189-99.
1028. Young JH. Steam sterilization: scientific principles. In: Reichert M, Young JH, eds. *Sterilization technology for the health care facility*. Gaithersburg, MD: Aspen Publication, 1997:123-144.
1029. Alfa MJ. Importance of lumen flow in liquid chemical sterilization. *Am. J. Infect. Control* 1999;27:373-5.
1030. Mallison GF, Standard PG. Safe storage times for sterile packs. *Hospitals* 1974;48:77-8, 80.

1031. Klapes NA, Greene VW, Langholz AC, Hunstiger C. Effect of long-term storage on sterile status of devices in surgical packs. *Infect. Control* 1987;8:289-93.
1032. Japp NF. Packaging: Shelf life. In: Reichert M, Young JH, eds. *Sterilization Technology*. Gaithersburg, Maryland: Aspen, 1997:99-102.
1033. Joint Commission for the Accreditation of Healthcare Organizations. *Comprehensive accreditation manual for hospitals*, JCAHO, Chicago, IL. 2003.
1034. Block SS. Definition of terms. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001:19-28.
1035. Molinari JA, Gleason MJ, Cottone JA, Barrett ED. Comparison of dental surface disinfectants. *Gen. Dent.* 1987;35:171-5.

GUIDELINE FOR THE PREVENTION AND CONTROL OF NOROVIRUS GASTROENTERITIS OUTBREAKS IN HEALTHCARE SETTINGS

Taranisia MacCannell, PhD, MSc¹; Craig A. Umscheid, MD, MSCE²; Rajender K. Agarwal, MD, MPH²; Ingi Lee, MD, MSCE²; Gretchen Kuntz, MSW, MSLIS²; Kurt B. Stevenson, MD, MPH³ and the Healthcare Infection Control Practices Advisory Committee (HICPAC)⁴

¹ Division of Healthcare Quality Promotion
Centers for Disease Control and Prevention
Atlanta, GA

² Center for Evidence-based Practice
University of Pennsylvania Health System
Philadelphia, PA

³ Division of Infectious Diseases
The Ohio State University,
Columbus, OH



4 Healthcare Infection Control Practices Advisory Committee (HICPAC)

CHAIRMAN

BRENNAN, Patrick J., MD
Chief Medical Officer
Division of Infectious Diseases
University of Pennsylvania Health System
Philadelphia, PA

MEMBERS

BRATZLER, Dale, DO, MPH
President and CEO
Oklahoma Foundation for Medical Quality
Oklahoma City, OK

BURNS, Lillian A., MT, MPH
Director Infection Control/Epidemiology
Staten Island University Hospital
Staten Island, NY

ELWARD, Alexis, MD
Assistant Professor, Pediatrics Infectious
Diseases
Washington University School of Medicine
St. Louis, MO

HUANG, Susan, MD, MPH
UC Irvine School of Medicine
Division of Infectious Diseases
Orange, CA

LUNDSTROM, Tammy, MD, JD
Chief Medical Officer
Providence Hospital
Southfield, MI

MCCARTER, Yvette S., PhD
Director, Clinical Microbiology
Laboratory
Department of Pathology
University of Florida Health Science
Center
Jacksonville, FL

MURPHY, Denise M. MPH, RN, CIC
Vice President, Quality and Patient Safety
Main Line Health System
Bryn Mawr, PA

EXECUTIVE SECRETARY

BELL, Michael R., MD
Deputy Director
Division of Healthcare Quality Promotion
Centers for Disease Control and Prevention
Atlanta, GA

OLMSTED, Russell N. MPH
Epidemiologist
Infection Control Services
St. Joseph Mercy Health System
Ann Arbor, MI

OSTROFF, Stephen, M.D.
Director, Bureau of Epidemiology
Pennsylvania Department of Health
Harrisburg, PA

PEGUES, David Alexander, MD
Professor of Medicine, Hospital Epidemiologist
David Geffen School of Medicine at UCLA
Los Angeles, CA

PRONOVOST, Peter J. M.D., Ph.D., F.C.C.M.
Director, Johns Hopkins Quality and Safety
Research Group
Department of Anesthesiology & Critical Care
Medicine
Johns Hopkins University
Baltimore, Maryland

SCHECTER, William, P., MD
Professor of Surgery
Department of Surgery
San Francisco General Hospital
San Francisco, CA

SOULE, Barbara M., RN, MPA, CIC
Practice Leader
Infection Prevention and Control Services
Joint Commission Resources/Joint
Commission International
Olympia, Washington

EX-OFFICIO MEMBERS

Agency for Healthcare Research and Quality (AHRQ)

BAINE, William B., MD
Senior Medical Advisor
Center for Outcomes and Evidence
Rockville, MD

Center for Medicare & Medicaid Services (CMS)

MILLER, Jeannie, RN, MPH
Deputy Director, Clinical Standards Group
Baltimore, MD

Food and Drug Administration (FDA)

MURPHEY, Sheila A., MD
Infection Control Devices Branch
Division of Anesthesiology, General Hospital
Infection Control Dental Devices
Center for Devices and Radiology Health
Rockville, MD

Health Resources and Services Administration (HRSA)

MOORE, Paul D., Ph.D
Senior Health Policy Advisor
Health Resources and Services Administration
U.S. Department of Health and Human Services
Office of Rural Health Policy
Rockville, MD

National Institutes of Health (NIH)

HENDERSON, David, MD
Deputy Director for Clinical Care
Associate Director for Hospital Epidemiology
and Quality Improvement
NIH Clinical Center
Bethesda, MD

Department of Veterans Affairs (VA)

ROSELLE, Gary A., MD
National Program Director, Infectious Diseases
VA Central Office
Cincinnati VA Medical Center
Cincinnati, OH

LIAISONS

Advisory Council for the Elimination of Tuberculosis (ACET)

STRICOF, Rachel L., MPH
Albany, New York

American College of Occupational and Environmental Medicine

RUSSI, Mark, MD, MPH
Professor of Medicine
Yale University School of Medicine
Director, Occupational Health
Yale-New Haven Hospital
New Haven, CT

American Health Care Assn (AHCA)

FITZLER, Sandra L., RN
Senior Director of Clinical Services
Washington, DC

American Hospital Association (AHA)

SCHULMAN, Roslyne, MHA, MBA
Senior Associate Director, Policy Development
Washington, DC

Association of periOperative Registered Nurses (AORN)

BLANCHARD, Joan C., RN, BSN, MSS, CNOR, CIC
Denver, CO

Association of Professionals of Infection Control and Epidemiology, Inc. (APIC)

DeBAUN, Barbara, MSN, RN, CIC
San Francisco, CA

Council of State and Territorial Epidemiologists (CSTE)

KAINER, Marion MD, MPH
Director, Hospital Infections and Antimicrobial Resistance Program
Tennessee Department Health
Nashville, TN

Consumers Union

MCGIFFERT, Lisa
Senior Policy Analyst on Health Issues
Project Director, Stop Hospital Infections Organization
Yonkers, NY

Infectious Diseases Society of America (IDSA)

HUSKINS, W. Charles M.D., M.Sc.
Division of Pediatric Infectious Diseases
Assistant Professor of Pediatrics
College of Medicine
Rochester, MN

Public Health Agency of Canada

PATON, Shirley, RN, MN
Senior Technical Advisor Healthcare Acquired Infections
Center for Communicable Diseases and Infection Control
Ottawa, Ontario

Society for Healthcare Epidemiology of America (SHEA)

MARAGAKIS, Lisa, MD
Assistant Professor of Medicine
John Hopkins Medical Institutions
Baltimore, MD

Society of Critical Care Medicine

William A. Brock, MD, FCCM, FCCP, FACP
Mount Prospect, IL

Society of Hospital Medicine

SAINT, Sanjay, MD, MPH
Director, Ann Arbor VA Medical Center/University of Michigan Patient Safety Enhancement Program
Ann Arbor VA Medical Center
Ann Arbor, Michigan

The Joint Commission

WISE, Robert A., MD
Vice President
Division of Standards & Survey Methods
Oakbrook Terrace, IL

Acknowledgement

HICPAC thanks the following members who served on the HICPAC Norovirus Gastroenteritis Guideline writing group during the guideline development process: Alexis Elward and Keith M. Ramsey.

HICPAC thanks the following experts for reviewing a draft of this guideline: John Boyce, Hospital of St. Raphael, New Haven, CT; Aron Hall, NCIRD, CDC; L. Clifford McDonald, DHQP, CDC; and Umesh Parashar, NCIRD, CDC. The opinions of the reviewers might not be reflected in all of the recommendations contained in this document.

Disclosure of Financial Interests and Relationships

The authors T.M., C.A.U., R.K.A., I.L., G.K., and K.B.S. report no actual or potential conflicts of interest. C.A.U., R.K.A. and I.L. received funding from the CDC to support the guideline development process.

Table of Contents

Abbreviations	7
I. Executive Summary	10
II. Summary of Recommendations.....	11
III. Implementation and Audit	18
IV. Recommendations for Further Research	19
V. Background	20
VI. Scope and Purpose.....	22
VII. Methods	22
VIII. Evidence Review.....	27
References	43

Abbreviations

AIDS	Acquired immune deficiency syndrome
BAS	Basic science study
°C	Celsius
CaCV	Calicivirus
CCU	Cardiac/coronary care unit
CDC	Centers for Disease Control and Prevention
CI	Confidence interval
CICU	Cardiac/coronary intensive care unit
CSTE	Council of State and Territorial Epidemiologists
DES	Descriptive study
DHQP	Division of Healthcare Quality Promotion
DIAG	Diagnostic study
DNA	Deoxyribonucleic acid
ECL	Electrochemiluminescence
EFORS	Electronic Foodborne Outbreak Reporting System
EIA	Enzyme immunoassay
ELISA	Enzyme-linked immunoassay
EM	Electron microscopy
EPA	Environmental Protection Agency
FBDSS	Foodborne Disease Outbreak Surveillance System
FCV	Feline calicivirus
FDA	Food and Drug Administration
FN	False negative
FP	False positive
GRADE	Grading of Recommendations Assessment, Development, and Evaluation
HBGA	Histo-blood group antigen

HICPAC	Healthcare Infection Control Practices Advisory Committee
HIV	Human immunodeficiency virus
Km	Kilometer
LUX	Light-upon-extension
MI	Milliliter
MMWR	Morbidity and Mortality Weekly Report
MNV	Murine norovirus
N/A	Not applicable
NASBA	Nucleic acid sequence-based amplification
NCIRD	National Center for Immunization and Respiratory Diseases
NIH	National Institutes of Health
NLV	Norwalk-like virus
No	Number
NORS	National Outbreak Reporting System
NPV	Negative predictive value
OBS	Observational study
OR	Odds ratio
ORF	Open reading frame
P	P value
PCR	Polymerase chain reaction
PPE	Personal protective equipment
PPM	Part per million
PPV	Positive predictive value
RCT	Randomized controlled trial
RHD	Rapid humidifying device
RIA	Radioimmunoassay
RF	Reduction factor

RR	Relative risk
RT	Room temperature
RT-LAMP	Reverse transcription loop-mediated amplification assay
RT-PCR	Reverse transcriptase polymerase chain reaction
SD	Standard deviation
SPIEM	Solid-phase immune electron microscopy
SR	Systematic review
SRFV	Small round featureless virus
SRSV	Small round structured virus
TCID	Tissue culture infective dose
TE	Transcriptional enhancement
TEM	Transmission electron microscopy
TN	True negative
TP	True positive
UV	Ultraviolet
Vs	Versus

I. Executive Summary

Norovirus gastroenteritis infections and outbreaks have been increasingly described and reported in both non-healthcare and healthcare settings during the past several years. In response, several states have developed guidelines to assist both healthcare institutions and communities on preventing the transmission of norovirus infections and helped develop the themes and key questions to answer through an evidence-based review. This guideline addresses prevention and control of norovirus gastroenteritis outbreaks in healthcare settings. The guideline also includes specific recommendations for implementation, performance measurement, and surveillance. Recommendations for further research are provided to address knowledge gaps identified during the literature review in the prevention and control of norovirus gastroenteritis outbreaks. Guidance for norovirus outbreak management and disease prevention in non-healthcare settings can be found at <http://www.cdc.gov/mmwr/pdf/rr/rr6003.pdf>.

This document is intended for use by infection prevention staff, physicians, healthcare epidemiologists, healthcare administrators, nurses, other healthcare providers, and persons responsible for developing, implementing, and evaluating infection prevention and control programs for healthcare settings across the continuum of care. The guideline can also be used as a resource for societies or organizations that wish to develop more detailed implementation guidance for prevention and control of norovirus gastroenteritis outbreaks for specialized settings or populations.

To evaluate the evidence on preventing and controlling norovirus gastroenteritis outbreaks in healthcare settings, published material addressing three key questions were examined:

1. What host, viral, or environmental characteristics increase or decrease the risk of norovirus infection in healthcare settings?
2. What are the best methods to identify an outbreak of norovirus gastroenteritis in a healthcare setting?
3. What interventions best prevent or contain outbreaks of norovirus gastroenteritis in the healthcare setting?

Explicit links between the evidence and recommendations are available in the [Evidence Review](#) in the body of the guideline and [Evidence Tables](#) and [GRADE Tables](#) in the [Appendices](#). **It is important to note that the Category I recommendations are all considered strong and should be implemented;** it is only the *quality* of the evidence underlying the recommendation that distinguishes between levels A and B. Category IC recommendations are required by state or federal regulation and may have any level of supporting evidence. The categorization scheme used in this guideline is presented in Table 1: [Summary of Recommendations](#) and described further in the [Methods](#) section. The [Implementation and Audit](#) section includes a prioritization of recommendations (i.e., high-priority recommendations that are essential for every healthcare facility) in order to provide facilities more guidance on implementation of these guidelines. A list of recommended performance measures that can potentially be used for reporting purposes is also included.

Evidence-based recommendations were cross-checked with those from other guidelines identified in an initial systematic search. Recommendations from other guidelines on topics not directly addressed by this systematic review of the evidence were included in the [Summary of Recommendations](#) if they were deemed critical to the target users of this guideline. Unlike recommendations informed by the search of primary studies, these recommendations are stated independently of a key question.

The [Summary of Recommendations](#) includes recommendations organized into the following categories: 1) Patient Cohorting and Isolation Precautions, 2) Hand Hygiene, 3) Patient Transfer and Ward Closure, 4) Indirect Patient Care Staff - Food Handlers in Healthcare, 5) Diagnostics, 6) Personal Protective Equipment, 7) Environmental Cleaning, 8) Staff Leave and Policy, 9) Visitors, 10) Education, 11) Active Case-finding, and 12) Communication and Notification.

Areas for further research identified during the evidence review are outlined in the Recommendations for Further Research. This section includes gaps that were identified during the literature review where specific recommendations could not be supported because of the absence of available information that matched the inclusion criteria for GRADE. These recommendations provide guidance for new research or methodological approaches that should be prioritized for future studies

Readers who wish to examine the primary evidence underlying the recommendations are referred to the Evidence Review in the body of the guideline, and the Evidence and GRADE Tables in the Appendices. The Evidence Review includes narrative summaries of the data presented in the Evidence and GRADE Tables. The Evidence Tables include all study-level data used in the guideline, and the GRADE Tables assess the overall quality of evidence for each question. The Appendices also contain a defined search strategy that will be used for periodic reviews to ensure that the guideline is updated as new information becomes available.

II. Summary of Recommendations

Table 1. HICPAC Categorization Scheme for Recommendations	
Category IA	A strong recommendation supported by high to moderate quality evidence suggesting net clinical benefits or harms.
Category IB	A strong recommendation supported by low-quality evidence suggesting net clinical benefits or harms, or an accepted practice (e.g., aseptic technique) supported by low to very low-quality evidence.
Category IC	A strong recommendation required by state or federal regulation.
Category II	A weak recommendation supported by any quality evidence suggesting a tradeoff between clinical benefits and harms.
Recommendation for further research	An unresolved issue for which there is low to very low-quality evidence with uncertain tradeoffs between benefits and harms.

*Please refer to Methods Section (p.23) and Umscheid et al. Updating the Guideline Methodology of the Healthcare Infection Control Practices Advisory Committee (HICPAC) (<http://www.cdc.gov/hicpac/guidelineMethod/guidelineMethod.html>) for the process used to grade quality of evidence and implications of category designation

**Key questions are described within the Evidence Review Section (p.31)

PATIENT COHORTING AND ISOLATION PRECAUTIONS

1. Avoid exposure to vomitus or diarrhea. Place patients on Contact Precautions in a single occupancy room if they have symptoms consistent with norovirus gastroenteritis. **(Category IB)** (Key Question 1.A.1)
 - 1a. When patients with norovirus gastroenteritis cannot be accommodated in single occupancy rooms, efforts should be made to separate them from asymptomatic patients. Dependent upon facility characteristics, approaches for cohorting patients during outbreaks may include placing patients in multi-occupancy rooms, or designating patient care areas or contiguous sections within a facility for patient cohorts. **(Category IB)** (Key Question 3C.4.b)
2. During outbreaks, place patients with norovirus gastroenteritis on Contact Precautions for a minimum of 48 hours after the resolution of symptoms to prevent further exposure of susceptible patients **(Category IB)** (Key Question 3.C.4.a)
 - 2a. Consider longer periods of isolation or cohorting precautions for complex medical patients (e.g., those with cardiovascular, autoimmune, immunosuppressive, or renal disorders) as they can experience protracted episodes of diarrhea and prolonged viral shedding. Patients with these or other comorbidities have the potential to relapse, and facilities may choose longer periods of isolation based on clinical judgment. **(Category II)** (Key Question 1.A.2.a)
 - 2b. Consider extending the duration of isolation or cohorting precautions for outbreaks among infants and young children (e.g., under 2 years), even after resolution of symptoms, as there is a potential for prolonged viral shedding and environmental contamination. Among infants, there is evidence to consider extending contact precautions for up to 5 days after the resolution of symptoms. **(Category II)** (Key Question 3.A.1)
3. Further research is needed to understand the correlation between prolonged shedding of norovirus and the risk of infection to susceptible patients **(No recommendation/unresolved issue)** (Key Question 3.A.2)
4. Consider minimizing patient movements within a ward or unit during norovirus gastroenteritis outbreaks. **(Category II)** (Key Question 3.C.4.c)
 - 4a. Consider restricting symptomatic and recovering patients from leaving the patient-care area unless it is for essential care or treatment to reduce the likelihood of environmental contamination and transmission of norovirus in unaffected clinical areas. **(Category II)** (Key Question 3.C.4.c.1)
5. Consider suspending group activities (e.g., dining events) for the duration of a norovirus outbreak. **(Category II)** (Key Question 3.C.4.d)
6. Staff who have recovered from recent suspected norovirus infection associated with an outbreak may be best suited to care for symptomatic patients until the outbreak resolves. **(Category II)**(Key Question 3.C.5.b)

HAND HYGIENE

7. Actively promote adherence to hand hygiene among healthcare personnel, patients, and visitors in patient care areas affected by outbreaks of norovirus gastroenteritis. **(Category IB)** (Key Question 3.C.1.a)
8. During outbreaks, use soap and water for hand hygiene after providing care or having contact with patients suspected or confirmed with norovirus gastroenteritis. **(Category IB)** (Key Question 3.C.1.b)
 - 8a. For all other hand hygiene indications (e.g., before having contact with norovirus patients) refer to the 2002 HICPAC Guideline for Hand Hygiene in Health-Care Settings (<http://www.cdc.gov/mmwr/PDF/rr/rr51116.pdf>), which includes the indications for use of FDA-compliant alcohol-based hand sanitizer. **(Category IB)** (Key Question 3.C.1.b.1)
 - 8a.1 Consider ethanol-based hand sanitizers (60-95%) as the preferred active agent compared to other alcohol or non-alcohol based hand sanitizer products during outbreaks of norovirus gastroenteritis. **(Category II)** (Key Question 3.C.1.b.2)
 - 8b. Further research is required to directly evaluate the efficacy of alcohol-based hand sanitizers against human strains of norovirus, or against a surrogate virus with properties convergent with human strains of norovirus. **(No recommendation/unresolved issue)** (Key Question 3.C.1.b.3)
9. More research is required to evaluate the virucidal capabilities of alcohol-based as well as non-alcohol based hand sanitizers against norovirus. **(No recommendation/unresolved issue)** (Key Question 3.C.12.e.4)

PATIENT TRANSFER AND WARD CLOSURE

10. Consider the closure of wards to new admissions or transfers as a measure to attenuate the magnitude of an outbreak of norovirus gastroenteritis. The threshold for ward closure varies and depends on risk assessments by infection prevention personnel and facility leadership. **(Category II)** (Key Question 3.C.6)
11. Consider limiting transfers to those for which the receiving facility is able to maintain Contact Precautions; otherwise, it may be prudent to postpone transfers until patients no longer require Contact Precautions. During outbreaks, medically suitable individuals recovering from norovirus gastroenteritis can be discharged to their place of residence. **(Category II)** (Key Question 3.C.11)
12. Implement systems to designate patients with symptomatic norovirus and to notify receiving healthcare facilities or personnel prior to transfer of such patients within or between facilities. **(Category IC)**

INDIRECT PATIENT CARE STAFF – FOOD HANDLERS IN HEALTHCARE

13. To prevent food-related outbreaks of norovirus gastroenteritis in healthcare settings, food handlers must perform hand hygiene prior to contact with or the preparation of food items and beverages

(<http://www.fda.gov/Food/FoodSafety/RetailFoodProtection/FoodCode/default.htm>). **(Category IC)** (Key Question 1.C.3.a)

14. Personnel who work with, prepare or distribute food must be excluded from duty if they develop symptoms of acute gastroenteritis. Personnel should not return to these activities until a minimum of 48 hours after the resolution of symptoms or longer as required by local health regulations (<http://www.fda.gov/Food/FoodSafety/RetailFoodProtection/FoodCode/default.htm>). **(Category IC)** (Key Question 1.C.3.b)
15. Remove all shared or communal food items for patients or staff from clinical areas for the duration of the outbreak. **(Category IB)** (Key Question 3.B.2)

DIAGNOSTICS

16. Consider the development and adoption of facility policies to enable rapid clinical and virological confirmation of suspected cases of symptomatic norovirus infection while implementing prompt control measures to reduce the magnitude of a potential norovirus outbreak. **(Category II)** (Key Question 1.C.1)
17. In the absence of clinical laboratory diagnostics or in the case of delay in obtaining laboratory results, use Kaplan's clinical and epidemiologic criteria to identify a norovirus gastroenteritis outbreak (see Table 4 for Kaplan's criteria). **(Category IA)** (Key Question 2.A.1)
18. Further research is needed to compare the Kaplan criteria with other early detection criteria for outbreaks of norovirus gastroenteritis in healthcare settings, and to assess whether additional clinical or epidemiologic criteria can be applied to detect norovirus clusters or outbreaks in healthcare settings. **(No recommendation/unresolved issue)** (Key Question 2.A.1)
19. Consider submitting stool specimens as early as possible during a suspected norovirus gastroenteritis outbreak and ideally from individuals during the acute phase of illness (within 2-3 days of onset). It is suggested that healthcare facilities consult with state or local public health authorities regarding the types of and number of specimens to obtain for testing. **(Category II)** (Key Question 2.B)
20. Use effective laboratory diagnostic protocols for testing of suspected cases of viral gastroenteritis (e.g., refer to the Centers for Disease Control and Prevention (CDC)'s most current recommendations for norovirus diagnostic testing at <http://www.cdc.gov/mmwr/pdf/rr/rr6003.pdf>). **(Category IB)** (Key Question 2.C)
21. Routine collecting and processing of environmental swabs during a norovirus outbreak is not required. When supported by epidemiologic evidence, environmental sampling can be considered useful to confirm specific sources of contamination during investigations. **(Category II)**
22. Specimens obtained from vomitus can be submitted for laboratory identification of norovirus when fecal specimens are unavailable. Testing of vomitus as compared to fecal specimens can be less sensitive due to lower detectable viral concentrations. **(Category II)**

PERSONAL PROTECTIVE EQUIPMENT

23. If norovirus infection is suspected, adherence to PPE use according to Contact and Standard Precautions is recommended for individuals entering the patient care area (i.e., gowns and gloves upon entry) to reduce the likelihood of exposure to infectious vomitus or fecal material. **(Category IB)** (Key Question 1.C.4)
24. Use a surgical or procedure mask and eye protection or a full face shield if there is an anticipated risk of splashes to the face during the care of patients, particularly among those who are vomiting. **(Category IB)** (Key Question 3.C.2.a)
25. More research is needed to evaluate the utility of implementing Universal Gloving (e.g., routine use of gloves for all patient care) during norovirus outbreaks. **(No recommendation/unresolved issue)**

ENVIRONMENTAL CLEANING

26. Perform routine cleaning and disinfection of frequently touched environmental surfaces and equipment in isolation and cohorted areas, as well as high-traffic clinical areas. Frequently touched surfaces include, but are not limited to, commodes, toilets, faucets, hand/bedrailing, telephones, door handles, computer equipment, and kitchen preparation surfaces. **(Category IB)** (Key Question 3.B.1)
27. Clean and disinfect shared equipment between patients using EPA-registered products with label claims for use in healthcare. Follow the manufacturer's recommendations for application and contact times. The EPA lists products with activity against norovirus on their website (<http://www.epa.gov/oppad001/chemregindex.htm>). **(Category IC)** (Key Question 3.C.12.a)
28. Increase the frequency of cleaning and disinfection of patient care areas and frequently touched surfaces during outbreaks of norovirus gastroenteritis (e.g., increase ward/unit level cleaning to twice daily to maintain cleanliness, with frequently touched surfaces cleaned and disinfected three times daily using EPA-approved products for healthcare settings). **(Category IB)** (Key Question 3.C.12.b.1)
29. Clean and disinfect surfaces starting from the areas with a lower likelihood of norovirus contamination (e.g., tray tables, counter tops) to areas with highly contaminated surfaces (e.g., toilets, bathroom fixtures). Change mop heads when a new bucket of cleaning solution is prepared, or after cleaning large spills of emesis or fecal material. **(Category IB)** (Key Question 3.C.12.b.2)
30. Consider discarding all disposable patient-care items and laundering unused linens from patient rooms after patients on isolation for norovirus gastroenteritis are discharged or transferred. Facilities can minimize waste by limiting the number of disposable items brought into rooms/areas on Contact Precautions. **(Category II)** (Key Question 3.C.12.c.1)
31. No additional provisions for using disposable patient service items such as utensils or dishware are suggested for patients with symptoms of norovirus infection. Silverware and dishware may undergo normal processing and cleaning using standard procedures. **(Category II)** (Key Question 3.C.12.c.2)
32. Use Standard Precautions for handling soiled patient-service items or linens, including the use of appropriate PPE. **(Category IB)** (Key Question 3.C.12.c.3)

33. Consider avoiding the use of upholstered furniture and rugs or carpets in patient care areas, as these objects are difficult to clean and disinfect completely. If this option is not possible, immediately clean soilage, such as emesis or fecal material, from upholstery, using a manufacturer-approved cleaning agent or detergent. Opt for seating in patient-care areas that can withstand routine cleaning and disinfection. **(Category II)** (Key Question 3.C.12.d.1)
34. Consider steam cleaning of upholstered furniture in patient rooms upon discharge. Consult with manufacturer's recommendations for cleaning and disinfection of these items. Consider discarding items that cannot be appropriately cleaned/disinfected. **(Category II)**(Key Question 3.C.12.d.2)
35. During outbreaks, change privacy curtains when they are visibly soiled and upon patient discharge or transfer. **(Category IB)** (Key Question 3.C.12.d.3)
36. Handle soiled linens carefully, without agitating them, to avoid dispersal of virus. Use Standard Precautions, including the use of appropriate PPE (e.g., gloves and gowns), to minimize the likelihood of cross-contamination. **(Category IB)** (Key Question 3.C.12.d.4)
37. Double bagging, incineration, or modifications for laundering are not indicated for handling or processing soiled linen. **(Category II)** (Key Question 3.C.12.d.5)
38. Clean surfaces and patient equipment prior to the application of a disinfectant. Follow the manufacturer's recommendations for optimal disinfectant dilution, application, and surface contact time with an EPA-approved product with claims against norovirus. **(Category IC)** (Key Question 3.C.12.e.1)
39. More research is required to clarify the effectiveness of cleaning and disinfecting agents against norovirus, either through the use of surrogate viruses or the development of human norovirus culture system. **(No recommendation/unresolved issue)** (Key Question 3.C.12.e.2)
40. More research is required to clarify the effectiveness and reliability of fogging, UV irradiation, and ozone mists to reduce norovirus environmental contamination. **(No recommendation/unresolved issue)** (Key Question 3.C.12.e.3)
41. Further research is required to evaluate the utility of medications that might attenuate the duration and severity of norovirus illness. **(No recommendation/unresolved issue)** (Key Question 3.D)

STAFF LEAVE AND POLICY

42. Develop and adhere to sick leave policies for healthcare personnel who have symptoms consistent with norovirus infection. **(Category IB)** (Key Question 3.C.3)
 - 42a. Exclude ill personnel from work for a minimum of 48 hours after the resolution of symptoms. Once personnel return to work, the importance of performing frequent hand hygiene should be reinforced, especially before and after each patient contact. **(Category IB)** (Key Question 3.C.3.a)
43. Establish protocols for staff cohorting in the event of an outbreak of norovirus gastroenteritis. Ensure staff care for one patient cohort on their ward and do not move between patient cohorts (e.g., patient cohorts may include symptomatic, asymptomatic exposed, or asymptomatic unexposed patient groups). **(Category IB)**(Key Question 3.C.5.a)

44. Exclude non-essential staff, students, and volunteers from working in areas experiencing outbreaks of norovirus gastroenteritis. **(Category IB)** (Key Question 3.C.5.c)

VISITORS

45. Establish visitor policies for acute gastroenteritis (e.g., norovirus) outbreaks. **(Category IB)** (Key Question 3.C.7.a)
46. Restrict non-essential visitors from affected areas of the facility during outbreaks of norovirus gastroenteritis. **(Category IB)** (Key Question 3.C.7.b)
- 46a. For those affected areas where it is necessary to have continued visitor privileges during outbreaks, screen and exclude visitors with symptoms consistent with norovirus infection and ensure that they comply with hand hygiene and Contact Precautions. **(Category IB)** (Key Question 3.C.7.b.1)

EDUCATION

47. Provide education to staff, patients, and visitors, including recognition of norovirus symptoms, preventing infection, and modes of transmission upon the recognition and throughout the duration of a norovirus gastroenteritis outbreak. **(Category IB)** (Key Question 3.C.8.a)
48. Consider providing educational sessions and making resources available on the prevention and management of norovirus before outbreaks occur, as part of annual trainings, and when sporadic cases are detected. **(Category II)** (Key Question 3.C.8.b)

ACTIVE CASE-FINDING

49. Begin active case-finding when a cluster of acute gastroenteritis cases is detected in the healthcare facility. Use a specified case definition, and implement line lists to track both exposed and symptomatic patients and staff. Collect relevant epidemiological, clinical, and demographic data as well as information on patient location and outcomes. **(Category IB)** (Key Question 3.C.9.a)

COMMUNICATION AND NOTIFICATION

50. Develop written policies that specify the chains of communication needed to manage and report outbreaks of norovirus gastroenteritis. Key stakeholders such as clinical staff, environmental services, laboratory administration, healthcare facility administration and public affairs, as well as state or local public health authorities, should be included in the framework. **(Category IB)** (Key Question 3.C.10)
- 50a. Provide timely communication to personnel and visitors when an outbreak of norovirus gastroenteritis is suspected and outline what policies and provisions need to be followed to prevent further transmission **(Category IB)** (Key Question 3.C.10.a)
51. As with all outbreaks, notify appropriate local and state health departments, as required by state and local public health regulations, if an outbreak of norovirus gastroenteritis is suspected. **(Category IC)** (Key Question 3.C.9.b)

III. Implementation and Audit

Prioritization of Recommendations

Category I recommendations in this guideline are all considered strong recommendations and should be implemented. If it is not feasible to implement all of these recommendations concurrently, e.g., due to differences in facility characteristics such as nursing homes and other non-hospital settings, priority should be given to the recommendations below. A limited number of Category II recommendations are included, and while these currently are limited by the strength of the available evidence, they are considered key activities in preventing further transmission of norovirus in healthcare settings.

PATIENT COHORTING AND ISOLATION PRECAUTIONS

1. Avoid exposure to vomitus or diarrhea. Place patients on Contact Precautions in a single occupancy room if they present with symptoms consistent with norovirus gastroenteritis. **(Category IB)** (Key Question 1.A.1)

HAND HYGIENE

8. During outbreaks, use soap and water for hand hygiene after providing care or having contact with patients suspected or confirmed with norovirus gastroenteritis. **(Category IB)** (Key Question 3.C.1.b)

PATIENT TRANSFER AND WARD CLOSURE

11. Consider limiting transfers to those for which the receiving facility is able to maintain Contact Precautions; otherwise, it may be prudent to postpone transfers until patients no longer require Contact Precautions. During outbreaks, medically suitable individuals recovering from norovirus gastroenteritis can be discharged to their place of residence. **(Category II)** (Key Question 3.C.11)

DIAGNOSTICS

17. In the absence of clinical laboratory diagnostics or in the case of delay in obtaining laboratory results, use Kaplan's clinical and epidemiologic criteria to identify a norovirus gastroenteritis outbreak. **(Category IA)** (Key Question 2.A.1)

ENVIRONMENTAL CLEANING

28. Increase the frequency of cleaning and disinfection of patient care areas and frequently touched surfaces during outbreaks of norovirus gastroenteritis (e.g., consider increasing ward/unit level cleaning to twice daily to maintain cleanliness, with frequently touched surfaces cleaned and disinfected three times daily using EPA-approved products for healthcare settings). **(Category IB)** (Key Question 3.C.12.b.1)

STAFF LEAVE AND POLICY

42. Develop and adhere to sick leave policies for healthcare personnel who have symptoms consistent with norovirus infection. **(Category IB)** (Key Question 3.C.3)

42a. Exclude ill personnel from work for a minimum of 48 hours after the resolution of symptoms. Once personnel return to work, the importance of performing frequent hand hygiene should be reinforced, especially before and after each patient contact. **(Category IB)** (Key Question 3.C.3.a)

43. Establish protocols for staff cohorting in the event of an outbreak of norovirus gastroenteritis. Ensure staff care for one patient cohort on their ward and do not move between patient cohorts (e.g., patient cohorts may include symptomatic, asymptomatic exposed, or asymptomatic unexposed patient groups). **(Category IB)**(Key Question 3.C.5.a)

COMMUNICATION AND NOTIFICATION

51. As with all outbreaks, notify appropriate local and state health departments, as required by state and local public health regulations, if an outbreak of norovirus gastroenteritis is suspected. **(Category IC)** (Key Question 3.C.9.b)

Performance Measures for Health Departments

Use of performance measures may assist individual healthcare facilities, as well as local and state health departments to recognize increasing and peak activities of norovirus infection, and may allow for prevention and awareness efforts to be implemented rapidly or as disease incidence escalates. Evaluate fluctuations in the incidence of norovirus in healthcare settings using the National Outbreak Reporting System (NORS) (<http://www.cdc.gov/outbreaknet/nors/>). This system monitors the reporting of waterborne, foodborne, enteric person-to-person, and animal contact-associated disease outbreaks to CDC by state and territorial public health agencies. This surveillance program was previously used only for reporting foodborne disease outbreaks, but it has now expanded to include all enteric outbreaks, regardless of mode of transmission. Additionally, CDC is currently implementing a national surveillance system (CaliciNet) for genetic sequences of noroviruses; this system may also be used to measure changes in the epidemiology of healthcare-associated norovirus infections.

IV. Recommendations for Further Research

The literature review for this guideline revealed that many of the studies addressing strategies to prevent norovirus gastroenteritis outbreaks in healthcare facilities were not of sufficient quality to allow firm conclusions regarding the benefit of certain interventions. Future studies of norovirus gastroenteritis prevention in healthcare settings should include:

1. Analyses of the impact of specific or bundled infection control interventions,
2. Use of controls or comparison groups in both clinical and laboratory trials,
3. Comparisons of surrogate and human norovirus strains, focusing on the differences in their survival and persistence after cleaning and disinfection, and compare the natural history of disease in animal models to that in human norovirus infections,
4. Assessment of healthcare-focused risk factors (e.g the impact of isolation vs. cohorting practices, duration of isolation, hand hygiene policies during outbreaks of norovirus, etc.)
5. Statistically powerful studies able to detect small but significant effects of norovirus infection control strategies or interventions, and
6. Quantitative assessments of novel, and practical methods for effective cleaning and disinfection during norovirus outbreaks.

The following are specific areas in need of further research in order to make more precise prevention recommendations (see also recommendations under the category of No recommendation/unresolved issue in the Evidence Review):

Measurement and Case Detection

1. Assess the benefit of using the Kaplan criteria as an early detection tool for outbreaks of norovirus gastroenteritis in healthcare settings and examine whether the Kaplan criteria are differentially predictive of select strains of norovirus.

Host Contagiousness and Transmission

1. Determine correlations between prolonged shedding of norovirus after symptoms have subsided and the likelihood of secondary transmission of norovirus infection.
2. Assess the utility of medications that may attenuate the duration and severity of norovirus illness.
3. Determine the role of asymptomatic shedding (among recovered persons and carriers) in secondary transmission.
4. Evaluate the duration of protective immunity and other protective host factors, including histo-blood group antigens (HBGA) and secretor status.
5. Assess the contribution of water or food sources to outbreaks of norovirus gastroenteritis in healthcare settings.

Environmental Issues

1. Quantify the effectiveness of cleaning and disinfecting agents against norovirus or appropriate surrogates.
2. Evaluate effectiveness and reliability of novel environmental disinfection strategies such as fogging, UV irradiation, vapor-phase hydrogen peroxides, and ozone mists to reduce norovirus contamination.
3. Develop methods to evaluate norovirus persistence in the environment, with a focus on persistent infectivity.
4. Identify a satisfactory animal model for surrogate testing of norovirus properties and pathogenesis. Translate laboratory findings into practical infection prevention strategies.

Hygiene and Infection Control

1. Evaluate the effectiveness of FDA-approved hand sanitizers against norovirus or appropriate surrogates, including viral persistence after treatment with non-alcohol based products.
2. Assess the benefits and impact of implementing Universal Gloving practices during outbreaks of norovirus gastroenteritis

V. Background

Norovirus is the most common etiological agent of acute gastroenteritis and is often responsible for outbreaks in a wide spectrum of community and healthcare settings. These single-stranded RNA viruses belong to the family *Caliciviridae*, which also includes the genera Sapovirus, Lagovirus, and Vesivirus.¹ Illness is typically self-limiting, with acute symptoms of fever, nausea, vomiting, cramping, malaise, and diarrhea persisting for 2 to 5 days.^{2,3} Noteworthy sequelae of norovirus infection include hypovolemia and electrolyte imbalance, as well as more severe medical presentations such as hypokalemia and renal insufficiency. As most healthy children and adults experience relatively mild symptoms, sporadic cases and outbreaks may be undetected or underreported. However, it is estimated that norovirus may be the causative agent in over 23 million gastroenteritis cases every year in the United States, representing approximately 60% of all acute gastroenteritis cases.⁴ Based on pooled analysis, it is estimated that norovirus may lead to over 91,000 emergency room visits and 23,000 hospitalizations for severe diarrhea among children under the age of five each year in the United States.^{5,6}

Noroviruses are classified into five genogroups, with most human infections resulting from genogroups GI and GII.⁶ Over 80% of confirmed human norovirus infections are associated with genotype GII.4.^{7,8} Since 2002, multiple new variants of the GII.4 genotype have emerged and quickly become the predominant cause of human norovirus disease.⁹ As recently as late 2006, two new GII.4 variants were detected across the United States and resulted in a 254% increase in acute gastroenteritis outbreaks in 2006 compared to 2005.¹⁰ The increase in incidence was likely associated with potential increases in pathogenicity and transmissibility of, and depressed population immunity to these new strains.¹⁰ CDC conducts surveillance for foodborne outbreaks, including norovirus or norovirus-like outbreaks, through voluntary state and local health reports using the Foodborne Disease Outbreak Surveillance System (FBDSS). CDC summary data for 2001-2005 indicate that caliciviruses (CaCV), primarily norovirus, were responsible for 29% of all reported foodborne outbreaks, while in 2006, 40% of foodborne outbreaks were attributed to norovirus.¹¹ In 2009, the National Outbreak Reporting System (NORS) was launched by the CDC after the Council of State

and Territorial Epidemiologists (CSTE) passed a resolution to commit states to reporting all acute gastroenteritis outbreaks, including those that involve person-to-person or waterborne transmission.

Norovirus infections are seen in all age groups, although severe outcomes and longer durations of illness are most likely to be reported among the elderly.² Among hospitalized persons who may be immunocompromised or have significant medical comorbidities, norovirus infection can directly result in a prolonged hospital stay, additional medical complications, and, rarely, death.¹⁰ Immunity after infection is strain-specific and appears to be limited in duration to a period of several weeks, despite the fact that seroprevalence of antibody to this virus reaches 80-90% as populations transition from childhood to adulthood.² There is currently no vaccine available for norovirus and, generally, no medical treatment is offered for norovirus infection apart from oral or intravenous repletion of volume.²

Food or water can be easily contaminated by norovirus, and numerous point-source outbreaks are attributed to improper handling of food by infected food-handlers, or through contaminated water sources where food is grown or cultivated (e.g., shellfish and produce) (<http://www.cdc.gov/mmwr/pdf/rr/rr6003.pdf>) The ease of its transmission, with a very low infectious dose of <10 -100 virions, primarily by the fecal-oral route, along with a short incubation period (24-48 hours)^{12,13}, environmental persistence, and lack of durable immunity following infection, enables norovirus to spread rapidly through confined populations.⁶

Institutional settings such as hospitals and long-term care facilities commonly report outbreaks of norovirus gastroenteritis, which may make up over 50% of reported outbreaks.¹¹ However, cases and outbreaks are also reported in a wide breadth of community settings such as cruise ships, schools, day-care centers, and food services, such as hotels and restaurants. In healthcare settings, norovirus may be introduced into a facility through ill patients, visitors, or staff. Typically, transmission occurs through exposure to direct or indirect fecal contamination found on fomites, by ingestion of fecally-contaminated food or water, or by exposure to aerosols of norovirus from vomiting persons.^{2,6} Healthcare facilities managing outbreaks of norovirus gastroenteritis may experience significant costs relating to isolation precautions and PPE, ward closures, supplemental environmental cleaning, staff cohorting or replacement, and sick time.

The pathogenesis of human norovirus infection

The P2 subdomain of the viral capsid is the likely binding site of norovirus, and is the most variable region on the norovirus genome.¹⁴ The P2 ligand is the natural binding site with human HBGA, which may be the point of initial viral attachment.¹⁴ HBGA is found on the surfaces of red blood cells and is also expressed in saliva, in the gut, and in respiratory epithelia. The strength of the virus binding may be dependent on the human host HBGA receptor sites, as well as on the infecting strain of norovirus. Infection appears to involve the lamina propria of the proximal portion of the small intestine,¹⁵ yet the cascade of changes to the local environment is unknown.

Clinical diagnosis of norovirus gastroenteritis is common, and, under outbreak conditions, the Kaplan Criteria are often used to determine whether gastroenteritis clusters or outbreaks of unknown etiology are likely to be attributable to norovirus.¹⁶ These criteria are:

1. Submitted fecal specimens negative for bacterial and if tested, parasitic pathogens,
2. Greater than 50% of cases reporting vomiting as a symptom of illness,
3. Mean or median duration of illness ranging between 12 and 60 hours, and
4. Mean or median incubation period ranging between 24 and 48 hours.

The current standard for norovirus diagnostics is reverse transcriptase polymerase chain reaction (RT-PCR), but clinical laboratories may use commercial enzyme immunoassays (EIA), or electron microscopy (EM).⁶ ELISA and transmission electron microscopy (TEM) demonstrate high sensitivity but lower specificities against the RT-PCR gold standard. The use of enzyme-linked immunosorbent assays (ELISA) and EM together can improve the overall test characteristics—particularly test specificity.¹⁷ Improvements in PCR have included the development of multiple nucleotide probes to detect a spectrum of genotypes as

well as methods to improve detection of norovirus from dilute samples or low viral loads and those containing PCR-inhibitors.¹⁸ While the currently available diagnostic methods are capable, with differing degrees of sensitivity and specificity, of detecting the physical presence of human norovirus from a sample, its detection does not directly translate into information about residual infectivity.

A significant challenge to controlling the environmental spread of norovirus in healthcare and other settings is the paucity of data available on the ability of human strains of norovirus to persist and remain infective in environments after cleaning and disinfection.¹⁹ Identifying the physical and chemical properties of norovirus is limited by the fact that human strains are presently uncultivable *in vitro*. The majority of research evaluating the efficacy of both environmental and hand disinfectants against human norovirus over the past two decades has primarily utilized feline calicivirus (FCV) as a surrogate. It is still unclear whether FCV is an appropriate surrogate for human norovirus, with some research suggesting that human norovirus may exhibit more resistance to disinfectants than does FCV.²⁰ Newer research has identified and utilized a murine norovirus (MNV) surrogate, which exhibits physical properties and pathophysiology more similar to those of human norovirus.²⁰ Currently, the Environmental Protection Agency (EPA) offers a list of approved disinfectants demonstrating efficacy against FCV, and the Federal Drug Administration (FDA) is responsible for evaluating hand disinfectants with label-claims against FCV as a surrogate for human norovirus (among other epidemiologically significant pathogens). It is unknown whether there are variations of physical and chemical tolerances to disinfectants and other virucidal agents among the various human norovirus genotypes. Other research pathways are evaluating the efficacy of fumigants, such as vapor phase hydrogen peroxides, as well as fogging methods as virucidal mechanisms to eliminate norovirus from environmental surfaces.

VI. Scope and Purpose

This guideline provides recommendations for the prevention and control of norovirus gastroenteritis outbreaks in healthcare settings. All patient populations and healthcare settings have been included in the review of the evidence. The guideline also includes specific recommendations for implementation, performance measurement, and surveillance strategies. Recommendations for further research are also included to address the knowledge gaps relating to norovirus gastroenteritis outbreak prevention and management that were identified during the literature review.

To evaluate the evidence on preventing and managing norovirus gastroenteritis outbreaks, three key questions were examined and addressed:

1. What host, viral, or environmental characteristics increase or decrease the risk of norovirus infection in healthcare settings?
2. What are the best methods to identify an outbreak of norovirus gastroenteritis in a healthcare setting?
3. What interventions best prevent or contain outbreaks of norovirus gastroenteritis in the healthcare setting?

This document is intended for use by infection prevention staff, healthcare epidemiologists, healthcare administrators, nurses, other healthcare providers, and persons responsible for developing, implementing, and evaluating infection prevention and control programs for healthcare settings across the continuum of care. The guideline can also be used as a resource for professional societies or organizations that wish to develop guidance on prevention or management of outbreaks of norovirus gastroenteritis for specialized settings or populations.

VII. Methods

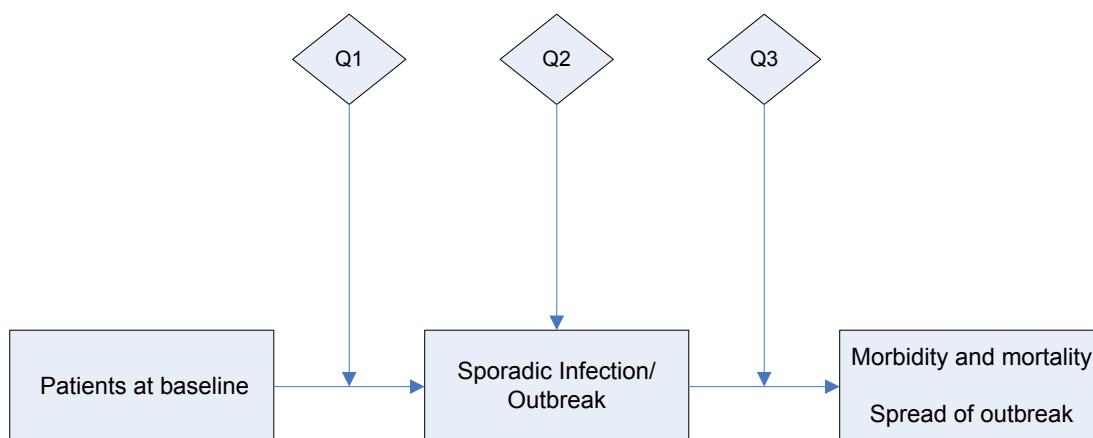
This guideline was based on a targeted systematic review of the best available evidence on the prevention and control of norovirus gastroenteritis outbreaks in healthcare settings. The Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach was used²¹⁻²⁴ to provide explicit links

between the available evidence and the resulting recommendations. Methods and/or details that were unique to this guideline are included below.

Development of Key Questions

First, an electronic search of the National Guideline Clearinghouse, MEDLINE, EMBASE, the Cochrane Health Technology Assessment Database, the NIH Consensus Development Program, and the National Institute for Health and Clinical Excellence, the Scottish Intercollegiate Guidelines Network and the United States Preventive Services Task Force databases was conducted for existing national and international guidelines relevant to norovirus. The strategy used for the guideline search and the search results can be found in *Appendix 1A*. A preliminary list of key questions was developed from a review of the relevant guidelines identified in the search.²⁵⁻⁴⁹ Key questions were put in final form after vetting them with a panel of content experts and HICPAC members. An analytic framework depicting the relationship among the key questions is included in *Figure 2*.

Figure 2. Norovirus Analytic Framework



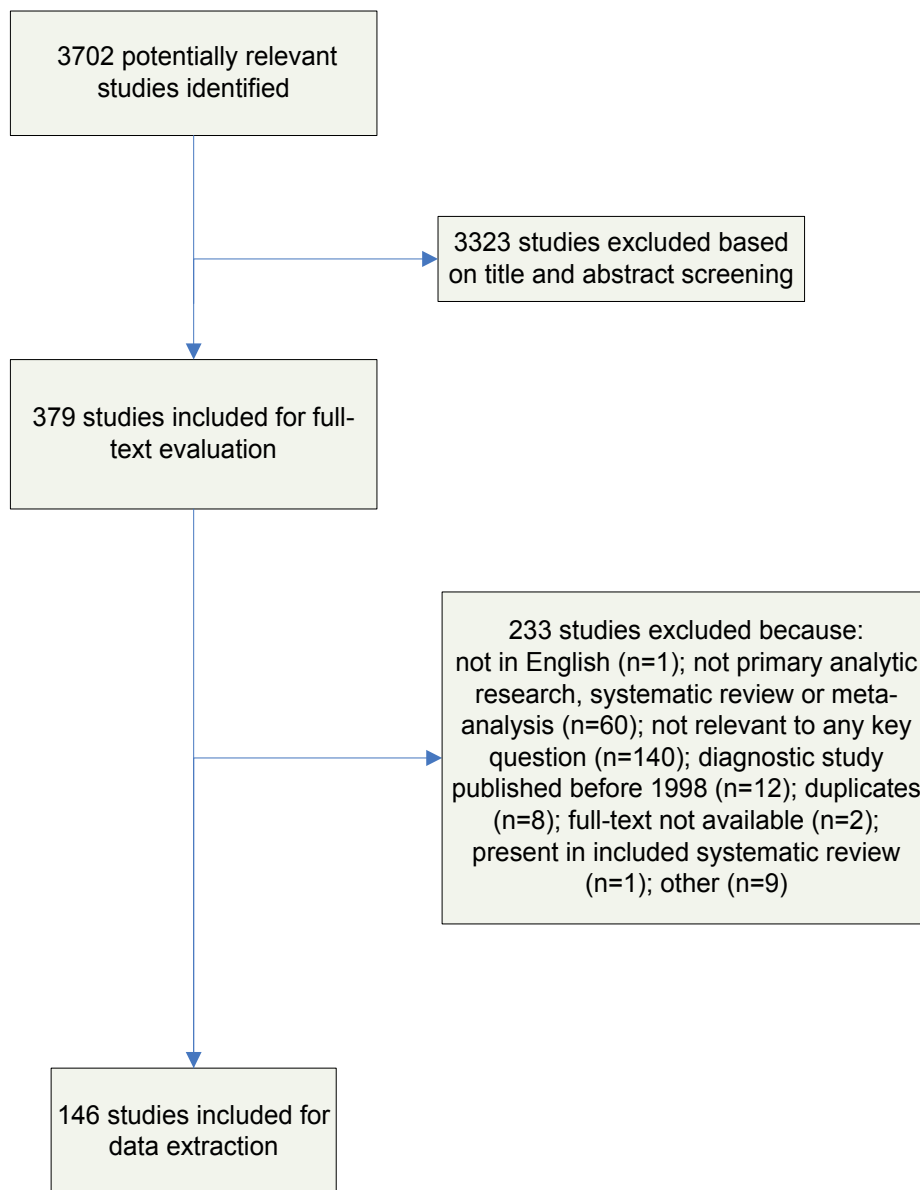
Literature Search

Following the development of the key questions, search terms were developed for identifying literature most relevant to those questions. For the purposes of quality assurance, these terms were compared to those used in relevant seminal studies and guidelines. These search terms were then incorporated into search strategies for the relevant electronic databases. Searches were performed in MEDLINE, EMBASE, CINAHL, the Cochrane Library, Global Health and ISI Web of Science (all databases were searched to the end of February 2008), and the resulting references were imported into a reference manager, where duplicates were resolved. The detailed search strategy used for identifying primary literature and the results of the search can be found in *Appendix 1B*.

Study Selection

Titles and abstracts from references were screened by a single reviewer (T.M. or K.B.S.). Full text articles were retrieved if they were 1) relevant to one or more key questions, 2) primary research, systematic reviews or meta-analyses, and 3) written in English. To be included, studies had to measure ≥ 1 clinically relevant outcome. For Key Questions 1 and 3, this included symptoms of norovirus infection, or stool antigen, virus, or EM results. For Key Question 2, this included any study published after 1997 that reported test characteristics (e.g., sensitivity, specificity, predictive values, likelihood ratios). Outbreak descriptions were included if: 1) norovirus was confirmed as the cause by EM, PCR, or antigen tests AND 2) the outbreak occurred in a healthcare setting and included a list of interventions or practices used to prevent or contain the outbreak OR 3) the outbreak occurred in any setting, but the report included statistical analyses. Full-text articles were screened by two independent reviewers (T.M., and I.L., or K.B.S.) and disagreements were resolved by discussion. The results of this process are depicted in *Figure 3*.

Figure 3. Results of the Study Selection Process



Data Extraction and Synthesis

For those studies meeting inclusion criteria, data on the study author, year, design, objective, population, setting, sample size, power, follow-up, and definitions and results of clinically relevant outcomes were extracted into standardized data extraction forms (*Appendix 3*). From these, three evidence tables were developed, each of which represented one of the key questions (*Appendix 2*). Studies were extracted into the most relevant evidence table. Then, studies were organized by the common themes that emerged within each evidence table. Data were extracted by a single author (R.K.A or I.L.) and cross-checked by another author (R.K.A or I.L.). Disagreements were resolved by the remaining authors. Data and analyses were extracted as originally presented in the included studies. Meta-analyses were performed only where

their use was deemed critical to a recommendation and only in circumstances in which multiple studies with sufficiently homogenous populations, interventions, and outcomes could be analyzed. Systematic reviews were included in this review. To avoid duplication of data, primary studies were excluded if they were also included in a systematic review captured through the broader search strategy. The only exception to this was if the primary study also addressed a relevant question that was outside the scope of the included systematic review. Before exclusion, data from primary studies that were originally captured were abstracted into the evidence tables and reviewed. Systematic reviews that analyzed primary studies that were fully captured in a more recent systematic review were excluded. The only exception to this was if the older systematic review also addressed a relevant question that was outside the scope of the newer systematic review. To ensure that all relevant studies were captured in the search, the bibliography was vetted by a panel of content experts. For the purposes of the review, statistical significance was defined as $p \leq 0.05$.

For all other methods (i.e., Grading of Evidence, Formulation of Recommendations, and Finalizing of the Guideline) please refer to the [Guideline Methods supplement](#).

Updating the Guideline

Future revisions to this guideline will be dictated by new research and technological advancements for preventing and managing norovirus gastroenteritis outbreaks.

VIII. Evidence Review

Question 1: What host, viral or environmental characteristics increase or decrease the risk of norovirus infection in healthcare settings?

To answer this question, the quality of evidence was evaluated among risk factors identified in 57 studies. In areas for which the outcome of symptomatic norovirus infection was available, this was considered the critical outcome in decision-making. The evidence for this question consisted of one systematic review,⁵⁶ 51 observational,^{57-62,62-64,64-77,77-107} and 4 descriptive studies,¹⁰⁸⁻¹¹¹ as well as one basic science study.¹¹² The paucity of randomized controlled trials (RCT) and the large number of observational studies greatly influenced the quality of evidence supporting the conclusions in the evidence review. Based on the available evidence, the risk factors were categorized as host, viral or environmental characteristics. Host characteristics were further categorized into demographics, clinical characteristics, and laboratory characteristics. Environmental characteristics were further categorized into institution, pets, diet, and exposure. The findings of the evidence review and the grades for all clinically relevant outcomes are shown in Evidence and Grade Table 1.

Q1.A Person characteristics

Q1.A.1 Demographic characteristics

Low-quality evidence was available to support age as a risk factor for norovirus infection,^{57-60,62-64} and very low-quality evidence to support black race as a protective factor.⁶⁴ Three studies indicated that persons over the age of 65 may be at greater risk than younger patients for prolonged duration and recovery from diarrhea in healthcare settings.⁵⁷⁻⁵⁹ Studies including children under the age of five showed an increased risk of household transmission as well as asymptomatic infection compared with older children and adults.^{60,62}

A single but large-scale observational study among military personnel found blacks to be at lower risk of infection than whites.⁶⁴ Very low-quality evidence failed to demonstrate meaningful differences in the risk of infection corresponding to strata on the basis of educational background (in the community setting).⁶¹ Based upon very low-quality evidence, outbreaks originating from patients were more likely to affect a large proportion of patients than were outbreaks originating from staff.⁵⁶ Exposure to vomitus and patients with diarrhea increased the likelihood that long-term care facility staff would develop norovirus infection.⁶⁶

The search did not identify studies that established a clear association between sex and symptomatic norovirus infection or complications of norovirus infection.^{57,59, 79, 98} Low-quality evidence from one prospective controlled trial did not identify sex as a significant predictor of symptomatic norovirus in univariate analyses.⁵⁷ There is low-quality evidence suggesting that sex is not a risk factor for protracted illness or complications of norovirus infection including acute renal failure and hypokalemia.⁵⁷

Q1.A.2 Clinical characteristics

Review of the available studies revealed very low-quality evidence identifying clinical characteristics as risk factors for norovirus infection.^{57,60,65,68} One small study found hospitalized children with human immunodeficiency virus (HIV) and chronic diarrhea were more likely to have symptomatic infection with small round structured virus (SRSV) than those without HIV and affected with chronic diarrhea.^{65,68} Adult patients with symptomatic norovirus receiving immunosuppressive therapy or admitted with underlying trauma were at risk for a greater than 10% rise in their serum creatinine.⁵⁷ Norovirus-infected patients with cardiovascular disease or having had a renal transplant were at greater risk for a decrease in their potassium levels by greater than 20%.⁵⁷ Observational, univariate study data also supported an increased duration of diarrhea (longer than two days) among hospitalized patients of advanced age and those with

malignancies.⁵⁷ This search did not reveal data on the risk of norovirus acquisition among those co-infected with other acute gastrointestinal infections, such as *C. difficile*.

Q1.A.3 Laboratory characteristics

Q1.A.3.a Antibody levels

There was very low-quality evidence to support limited protective effects of serum antibody levels against subsequent norovirus infection.⁷⁴⁻⁷⁶ In two challenge studies, adult and pediatric subjects with prior exposure to norovirus showed higher antibody titers than found in previously unexposed subjects after initial infection and after challenge.^{74,76} The detection of preexisting serum antibody does not appear to correlate with protection against subsequent norovirus challenge, nor did increasing detectable pre-existing antibody titres correlate with attenuations in the clinical severity of disease.^{74,75} In one study, symptoms such as vomiting, nausea, headaches, and arthralgia were correlated with increasing antibody titres.⁷⁴ In a serial challenge study, 50% of participants (n=6) developed infection, and upon subsequent challenge 27-42 months later, only those same participants developed symptoms. A third challenge 4-8 weeks after the second series resulted in symptoms in just a single volunteer.⁷⁶ Pre-existing antibody may offer protection to susceptible persons only for a limited window of time, on the order of a few weeks. The search strategy did not reveal data on the persistence of immunity to norovirus nor elevations in antibody titers that were consistently suggestive of immunity.

Q1.A.3.b Secretor genotype

Review of the outlined studies demonstrated high-quality evidence to support the protective effects of human host non-secretor genotypes against norovirus infection.^{70-72,113} Two observational studies and one intervention study examined volunteers with and without the expression of the secretor (FUT2) genotype after norovirus challenge.⁷⁰⁻⁷² Statistically significant differences were reported with secretor-negative persons demonstrating a greater likelihood of protection against, or innate resistance to symptomatic and asymptomatic norovirus infection than seen in persons with secretor-positive genotypes. This search did not reveal data on the dose-response effects of norovirus in persons with homozygous and heterozygous secretor genotypes. Because the FUT2-mediated secretor positive phenotype appears to confer susceptibility to subsequent norovirus infection following challenge, there is an association between this phenotype and measurable circulating antibody (suggesting prior infection) in the population. One study estimated that 80% of the population is secretor-positive (or susceptible to norovirus) and 20% is secretor-negative (resistant to norovirus challenge independent of inoculum dose). Among susceptible persons, approximately 35% are protected from infection. This protection is potentially linked to a memory-mediated rapid mucosal IgA response to norovirus exposure that is not seen in the other 45% of susceptibles, who demonstrate delayed mucosal IgA and serum IgG responses.⁷² Although elevated antibody levels following infection appear to confer some protective immunity to subsequent challenge, paradoxically, measurable antibody titers in the population may be a marker of *increased* susceptibility to norovirus because of the association between such antibodies and FUT2-positive status.

Q1.A.3.c ABO phenotype

There was low-quality evidence suggesting any association of ABO blood type with the risk of norovirus infection.^{69,72,73,77,78,114,115} An RCT suggested that persons with histo-blood group type O was associated with an increased risk of symptomatic or asymptomatic norovirus infection among secretor-positive patients.⁷² Binding of norovirus to the mucosal epithelium may be facilitated by ligands associated with type-O blood. The other blood types—A, B, and AB—were not associated with norovirus infection after controlling for secretor status. Three studies showed no protective effect of any of the blood types against norovirus.^{69,77,78} The search strategy did not reveal prospective cohort data to correlate the role of ABO blood types with risk of norovirus infection.

Q1.B Viral characteristics

There was very low-quality evidence to suggest an association of virus characteristics with norovirus infection.^{57,108-110} Very low-quality descriptive evidence suggested that increases in overall norovirus activity may result from the emergence of new variants among circulating norovirus strains, and strains may differ in pathogenicity, particularly among GII.3 and GII.4 variants.¹⁰⁸⁻¹¹⁰ In recent years, GII.4 strains are increasingly reported in the context of healthcare-associated outbreaks, but further epidemiologic and laboratory studies are required to expand on this body of information. This search did not identify studies examining genotypic characteristics of viruses associated with healthcare-acquired norovirus infection.

Q1.C Environmental characteristics

Q1.C.1 Institutional characteristics

Very low-quality evidence was available to support the association of institutional characteristics with symptomatic norovirus infection.^{82,99} Among two observational studies, the number of beds within a ward, nurse understaffing, admission to an acute care hospital (compared to smaller community-based facilities), and having experienced a prior outbreak of norovirus gastroenteritis within the past 30 days were all possible risk factors for new infections.^{82,99} These increased institutional risks were identified from univariate analyses in pediatric and adult hospital populations. There were statistically significant, increased risks of infection among those admitted to geriatric, mental health, orthopedic, and general medicine wards. The review process did not reveal data on the comparative risks of infection among those admitted to private and shared patient rooms.

Q1.C.2 Pets

Review of the outlined studies demonstrated very low-quality evidence to support exposure to pets (e.g., cats and dogs) as a risk factor for norovirus infection.⁶¹ One case-control study examined pet exposure among households in the community and concluded that the effect of cats was negligible.⁶¹ The single study did not demonstrate any evidence of transmission between pets and humans of norovirus infection. This search strategy did not reveal studies that evaluated the impact of therapy pets in healthcare settings during outbreaks of norovirus gastroenteritis or data examining domestic animals as reservoirs for human infection.

Q1.C.3 Diet

There was low-quality evidence to suggest that extrinsically contaminated food items are commonly implicated as vehicles of norovirus exposure in healthcare settings.^{61,77,80,84,86,87,89-97,100-102,104-107,111} Nineteen observational studies itemized statistically significant food sources implicated in community outbreaks.^{80,81,84,86,87,89-97,100,101,104-106} Common to most of these food sources was a symptomatic or asymptomatic food-handler. Sauces, sandwiches, fruits and vegetables, salads, and other moisture-containing foods were most often cited as extrinsically contaminated sources of outbreaks of norovirus gastroenteritis. Importantly, these data reflected the breadth of foods that can become contaminated. Tap water and ice were also associated with norovirus contamination during an outbreak with an ill food-handler. This literature review did not identify studies that examined the introduction of intrinsically contaminated produce or meats as a nidus for norovirus infection and dissemination within healthcare facilities.

Q1.C.4 Proximity to infected persons

This review demonstrated high-quality evidence to suggest that proximity to infected persons with norovirus is associated with increased risk of symptomatic infection.^{61,62,64,79,83,88,98,103,111} Eight observational studies found statistically significant factors such as proximate exposure to an infected source within households or in crowded quarters increased infection risk, as did exposures to any or frequent vomiting episodes.^{61,62,64,79,83,88,98,103} These data suggest person-to-person transmission is dependent on close or direct contact as well as short-range aerosol exposures. One observational study established a linear relationship

between a point source exposure and attack rate based on proximity to an infected and vomiting source.⁸⁸ This search process did not identify studies that quantified the spatial radius necessary for transmission to successfully occur.

Q1 Recommendations

1.A.1 Avoid exposure to vomitus or diarrhea. Place patients on Contact Precautions in a single occupancy room if they have symptoms consistent with norovirus gastroenteritis. **(Category IB)** (Key Question 1A)

1.A.2.a Consider longer periods of isolation or cohorting precautions for complex medical patients (e.g., those with cardiovascular, autoimmune, immunosuppressive, or renal disorders) as they can experience protracted episodes of diarrhea and prolonged viral shedding. Patients with these or other comorbidities have the potential to relapse and facilities may choose longer periods of isolation based on clinical judgment. **(Category II)** (Key Question 1A)

1.C.1 Consider the development and adoption of facility policies to enable rapid clinical and virological confirmation of suspected cases of symptomatic norovirus infection while implementing prompt control measures to reduce the magnitude of a potential norovirus outbreak. **(Category II)** (Key Question 1C)

1.C.3.a To prevent food-related outbreaks of norovirus gastroenteritis in healthcare settings, food handlers must perform hand hygiene prior to contact with or the preparation of food items and beverages (<http://www.fda.gov/Food/FoodSafety/RetailFoodProtection/FoodCode/default.htm>). **(Category IC)** (Key Question 1C)

1.C.3.b Personnel who work with, prepare or distribute food must be excluded from duty if they develop symptoms of acute gastroenteritis. Personnel should not return to these activities until a minimum of 48 hours after the resolution of symptoms or longer as required by local health regulations (<http://www.fda.gov/Food/FoodSafety/RetailFoodProtection/FoodCode/default.htm>). **(Category IC)** (Key Question 1C)

1.C.4 If norovirus infection is suspected, adherence to PPE use according to Contact and Standard Precautions is recommended for individuals entering the patient care area (i.e., gowns and gloves upon entry) to reduce the likelihood of exposure to infectious vomitus or fecal material. **(Category IB)** (Key Question 1C)

Question 2: What are the best methods to identify an outbreak of norovirus gastroenteritis in a healthcare setting?

To address this question, studies that provided test characteristics for the diagnosis of norovirus or outbreaks of norovirus gastroenteritis were critically reviewed. The available data examined the use of clinical criteria for the diagnosis of an outbreak of norovirus, methods of specimen collection for the diagnosis of a norovirus outbreak, and characteristics of tests used to diagnose norovirus. The evidence consisted of 33 diagnostic studies.^{17,18,116-146} The findings from the evidence review and the grades of evidence for clinically relevant outcomes are shown in Evidence and Grade Table 2.

Q2.A Clinical Criteria

There was moderate quality evidence from a single diagnostic study supporting the use of the Kaplan criteria to detect outbreaks of norovirus gastroenteritis.^{16,116} Of 362 confirmed gastroenteritis outbreaks with complete clinical or laboratory data, the sensitivity of the Kaplan Criteria to detect an outbreak of norovirus gastroenteritis without an identified bacterial pathogen was 68.2%, with a specificity of 98.6%. The positive predictive value (PPV) was 97.1% and the negative predictive value was 81.8%. Individual criteria, such as vomiting among >50% of a patient cohort, brief duration of illness (12-60 hours), or mean incubation time of 24-48 hours, demonstrated high sensitivities (85.8-89.2%), but specificities were low (60.7-69.6%). The use of additional criteria, such as the ratios of fever-to-vomiting and diarrhea-to-vomiting, provided sensitivities of 90.1% and 96.6%, and specificities of 46.6% and 44.5%, respectively. Applied to the 1141 outbreaks of unconfirmed etiology, suspected norovirus or bacterial sources with complete data, the Kaplan criteria estimated that 28% of all 1998-2000 CDC-reported *foodborne* outbreaks might be attributable to norovirus. The search strategy did not identify studies that have assessed the utility of the Kaplan criteria in healthcare-associated outbreaks of norovirus gastroenteritis.

Q2.B Specimen Collection

There was low-quality evidence from three diagnostic studies outlining the minimum number of stool samples from symptomatic patients required to confirm an outbreak of norovirus gastroenteritis.^{117,119,120,122,123} In modeling analyses using a hypothetical test demonstrating 100% sensitivity and 100% specificity, obtaining a positive EIA result from two or more submitted samples demonstrated a sensitivity of 52.2-57%, with a peak in sensitivity when at least one from a total of six submitted samples was positive for norovirus (71.4-92%). Specificity was 100% when at least one positive EIA was obtained from a minimum of two submitted stool samples.

Using a reverse transcriptase polymerase chain reaction (RT-PCR) method, if at least one positive test was identified among 2 to 4 submitted stool specimens from symptomatic persons, the test sensitivity was greater than 84%. When 5-11 stool samples were submitted and at least 2 were confirmed as positive, the sensitivity of PCR was greater than 92%. When at least one stool specimen was submitted for identification, PCR confirmed norovirus as the causative agent in a larger proportion of outbreaks than those using EM or ELISA methods, and is currently the Gold Standard. This evaluation was unable to determine how diagnostic test characteristics are affected by the timing of specimen collection relative to the disease process.

Q2.C Diagnostic Methods

28 diagnostic studies^{17,18,118-120,122,124-139,141-145,147} and 1 descriptive study¹²¹ that evaluated the test characteristics of EIA such as ELISA, EM, reverse transcriptase PCR, and nucleic acid sequence-based amplification (NASBA) in the detection of norovirus in human fecal specimens were summarized. Test characteristics for the most common or commercially-available norovirus diagnostics are summarized in the following Table.

Q2 Recommendations

2.A.1 In the absence of clinical laboratory diagnostics or in the case of delay in obtaining laboratory results, use Kaplan's clinical and epidemiologic criteria to identify a norovirus gastroenteritis outbreak (see Table 4 for Kaplan's criteria). **(Category IA)** (Key Question 2A)

2.A.2 Further research is needed to compare the Kaplan criteria with other early detection criteria for outbreaks of norovirus gastroenteritis in healthcare settings, and to assess whether additional clinical or epidemiologic criteria can be applied to detect norovirus clusters or outbreaks in healthcare settings. **(No recommendation/unresolved issue)** (Key Question 2A)

2.B Consider submitting stool specimens as early as possible during a suspected norovirus gastroenteritis outbreak and ideally from individuals during the acute phase of illness (within 2-3 days of onset). It is suggested that healthcare facilities consult with state or local public health regarding the types of and number of specimens to obtain for testing. **(Category II)** (Key Question 2B)

2.C Use effective laboratory diagnostic protocols for testing of suspected cases of viral gastroenteritis (e.g., refer to the Centers for Disease Control and Prevention (CDC)'s most current recommendations for norovirus diagnostic testing at <http://www.cdc.gov/mmwr/pdf/rr/rr6003.pdf>. **(Category IB)** (Key Question 2C)

Table 3. Test Characteristics for Norovirus in Fecal Specimens

Diagnostic method	Reference standard	Quantity and type of evidence	Findings*			
			Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Kaplan criteria	PCR	1 DIAG ¹¹⁶	68	99	97	82
EIA/ELISA	PCR	10 DIAG ^{17,118-120,123-128,139}	31 – 90	65 – 100	52 – 100	56-97
EM	PCR	2 DIAG ^{17,119}	24 – 58	98-99	88-94	71-91
NASBA	PCR	1 DIAG ¹⁴⁴	100	50	-	-

* Range from studies that reported test characteristics
Negative predictive Value, NPV; Positive predictive value, PPV

Table 4. Kaplan Criteria¹⁶

- 1) Vomiting in more than half of symptomatic cases
- 2) Mean (or median) incubation period of 24 to 48 hours
- 3) Mean (or median) duration of illness of 12 to 60 hours
- 4) No bacterial pathogen isolated in stool culture

Question 3: What interventions best prevent or contain outbreaks of norovirus gastroenteritis in the healthcare setting?

To address this question, 69 studies^{58,63,66,79,83-85,87,89,92,102,103,112,148-203} were critically reviewed for evidence of interventions that might prevent or attenuate an outbreak of norovirus. The available data dealt with viral shedding, recovery of norovirus, and components of an outbreak prevention or containment program, including the use of medications. The evidence consisted of 1 randomized controlled trial,²⁰² 1 systematic review,¹⁵³ 20 basic science studies,^{112,162,163,185-201} 43 descriptive studies,^{58,63,79,83-85,87,89,92,102,103,149-152,154-161,165-184} and 4 observational studies.^{66,148,164,203} The findings from the evidence review and the grades of evidence for clinically relevant outcomes are shown in Evidence and Grade Table 3.

Q3.A Viral Shedding

This review did not identify studies demonstrating direct associations between viral shedding and infectivity. However, there was low-quality evidence to support an association between age and duration of viral shedding.^{149,150} One observational study suggested that children under the age of six months may be at an increased risk of prolonged viral shedding (greater than two weeks), even after the resolution of symptoms.¹⁴⁸ Other findings suggest that infants can shed higher titers of virus than levels reported in other

age groups.¹⁴⁹ High-quality evidence was available to demonstrate the presence of viral shedding in asymptomatic subjects, and low-quality evidence demonstrating that shedding can persist for up to 22 days following infection and 5 days after the resolution of symptoms.¹⁵⁰⁻¹⁵² The search strategy employed did not identify studies that correlated other clinical factors to duration of viral shedding.

Q3.B Recovery of Norovirus

Q3.B.1 Fomites

There was low-quality evidence positively associating fomite contamination with norovirus infection.^{153-159,161,163,194} Similarly, there was low-quality evidence demonstrating transfer of norovirus from fomites to hands.¹⁹⁴ One basic science study demonstrated that norovirus on surfaces can be readily transferred to other fomites (telephones, taps, door handles) via fingertips in 30-50% of opportunities even when virus has been left to dry for 15 minutes.¹⁹⁴ There was moderate quality evidence examining the norovirus contamination of the environment.^{153-159,161,163} A single systematic review evaluated 5 outbreaks with environmental sampling data.¹⁵³ Three of those outbreaks confirmed environmental contamination with norovirus. Of the over 200 swabs examined from the 5 outbreaks in this review, 36% identified norovirus contamination on various fomites such as curtains, carpets, cushions, commodes and toilets, furnishings and equipment within 3-4 feet of the patient, handrails, faucets, telephones, and door handles. However, in two outbreaks from which 47 environmental samples were collected, norovirus was not detected. Additional studies detected norovirus on kitchen surfaces, elevator buttons, and other patient equipment.^{154-157, 194}

There was low-quality evidence regarding the duration of norovirus persistence.^{154,155,157-159,161} Norovirus can persist in a dried state at room temperature for up to 21-28 days and, in a single observational study, was undetectable in areas of previously known contamination after 5 months had elapsed.¹⁵⁹ Laboratory studies comparing FCV and MNV-1 also demonstrated persistence of virus in both dried and in fecal suspensions for a minimum of seven days on stainless steel preparations at 4°C and at room temperature.²⁰ Within a systematic review, it was observed that norovirus may remain viable in carpets up to 12 days, despite regular vacuuming.¹⁵³ Similarly, a cultivable surrogate for human strains of norovirus (FCV) was detected on computer keyboards and mice, as well as telephone components up to 72 hrs from its initial inoculation.¹⁵⁶ This search strategy did not find studies in which the recovery of norovirus from fomites, food, and water sources was directly associated with transmission of infection in healthcare settings; however transmission from these sources has been well documented in other settings.

Q3.B.2 Foods and Food Preparation Surfaces

There was low-quality evidence suggesting that foods and food-preparation surfaces are significant sources of norovirus transmission in healthcare settings.^{112,162,163} There was moderate quality evidence among three basic science studies to suggest that norovirus can be recovered from foods such as meats and produce as well as from utensils and non-porous surfaces (e.g., stainless steel, laminate, ceramics) upon which foods are prepared.^{112,162,163} Two of these studies, comprised of low-quality evidence, suggested that the transfer of diluted aliquots of norovirus from stainless steel surfaces to wet and dry food, and through contaminated gloves was detectable using PCR methods. Norovirus transfer was statistically more efficient when it was inoculated onto moist surfaces compared to dry ones.^{162,163}

There was low-quality evidence to suggest that norovirus persists for longer periods in meats compared to other foods and non-porous surfaces, both at 4°C and at room temperature.¹¹² There was moderate quality evidence demonstrating that over a period of 7 days after application, both human norovirus genogroup I and a surrogate (FCV) could be detected among all surfaces tested.^{112,162} Within the first hour, the log₁₀ of FCV titers declined by 2-3, with an additional drop of 2-4 after 48 hours elapsed.¹⁶² Food and food-preparation areas can serve as a common source of contamination with norovirus in the absence of cleaning and disinfection.

Q3.B.3 Water

This search strategy did not identify studies that measured the contribution of norovirus-contaminated water to outbreaks in the healthcare setting. However, there was moderate quality evidence to suggest that norovirus could be recovered from water.^{155,158,160} Among three outbreaks that examined water as a source, one identified norovirus in 3 of 7 water samples.¹⁶⁰ In outbreaks in the community, which were outside the scope of this review, contaminated surface water sources, well water, and recreational water venues have been associated with outbreaks of norovirus gastroenteritis.²⁰⁴

Q3.C Components of an Outbreak Prevention/Containment Program

As with most infection-prevention and control activities, multiple strategies are instituted simultaneously during outbreaks in healthcare settings. Thus, it is difficult to single out particular interventions that may be more influential than others, as it is normally a combination of prudent interventions that reduce disease transmission. Numerous studies cite the early recognition of cases and the rapid implementation of infection control measures as key to controlling disease transmission. The following interventions represent a summary of key components in light of published primary literature and addressed in seminal guidelines on outbreaks of norovirus gastroenteritis.

Q3.C.1 Hand Hygiene

Q3.C.1.a Handwashing with soap and water

Very low-quality evidence was available to confirm that handwashing with soap and water prevents symptomatic norovirus infections.^{63,66,79,85,89,102,103,165,166,168-171,173-177,183} Several descriptive studies emphasized hand hygiene as a primary prevention behavior and promoted it simultaneously with other practical interventions. Several outbreaks centered in healthcare augmented or reinforced hand hygiene behavior as an early intervention and considered it an effective measure aimed at outbreak control.^{103,165,168,170,174,176,177,183} The protocols for hand hygiene that were reviewed included switching to the exclusive use of handwashing with soap and water, and a blend of handwashing with the adjunct use of alcohol-based hand sanitizers. Additional guidance is available in the 2002 HICPAC Guideline for Hand Hygiene in Health-Care Settings (<http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf>).

Q3.C.1.b Alcohol-based hand sanitizers

Very low-quality evidence was available to suggest that hand hygiene using alcohol-based hand sanitizers may reduce the likelihood of symptomatic norovirus infection.^{66,87,169,171,205} Several studies used FDA-compliant alcohol-based hand antiseptics during periods of norovirus activity as an adjunct measure of hand hygiene.^{66,87,168,169,171,205,206} Two studies used a commercially available 95% ethanol-based hand sanitizer along with handwashing with soap and water; but without a control group and with hand hygiene comprising one of several interventions, the relative contribution of hand hygiene to attenuating transmission was difficult to evaluate.^{169,171} In the laboratory, even with 95% ethanol products, the maximum mean reduction in log₁₀ titer reduction was 2.17.¹⁹³ Evidence to evaluate the efficacy of alcohol-based hand disinfectants consisted of basic science studies using FCV as a surrogate for norovirus. Moderate quality evidence supported ethanol as a superior active ingredient in alcohol-based hand disinfectants compared to 1-propanol, particularly when simulated organic loads (e.g. fecal material) were used in conjunction with exposure to norovirus.^{189,191,193,196} The use of hand sanitizers with mixtures of ethanol and propanol have shown effectiveness against FCV compared to products with single active ingredients (70% ethanol or propanol) under controlled conditions.¹⁸⁹ There were no studies available to evaluate the effect of non-alcohol based hand sanitizers on norovirus persistence on skin surfaces.

Q3.C.1.c Role of artificial nails

Very low-quality evidence suggested that the magnitude in reduction of a norovirus surrogate (FCV) using a spectrum of soaps and hand disinfectants was significantly greater among volunteers with natural nails compared to those with artificial nails.¹⁹⁷ A subanalysis showed that longer fingernails were associated with consistently greater hand contamination. Further evidence summarizing the impact of artificial and long fingernails in healthcare settings can be found in the HICPAC Guidelines for Hand Hygiene in Healthcare Settings (<http://www.cdc.gov/Handhygiene/>).

Q3.C.2 Personal Protective Equipment

Very low-quality evidence among 1 observational⁶⁶ and 13 descriptive studies^{167-173,176-179,181,183} support the use of personal protective equipment (PPE) as a prevention measure against symptomatic norovirus infection. A single retrospective study failed to support the use of gowns as a significantly protective measure against norovirus infection during the outbreak among staff but did not consider the role of wearing gowns in avoiding patient-to-patient transmission.⁶⁶ Mask or glove use was not evaluated in the self-administered questionnaire used in the study. Several observational and descriptive studies emphasized the use of gloves and isolation gowns for routine care of symptomatic patients, with the use of masks recommended when staff anticipated exposure to emesis or circumstances where virus may be aerosolized.^{167-173,176-179,181,183} The use of PPE was advocated for both staff and visitors in two outbreak studies.^{169,179}

Q3.C.3 Leave Policies for Staff

There was very low-quality evidence among several studies to support the implementation of staff exclusion policies to prevent symptomatic norovirus infections in healthcare settings.^{84,85,92,165,167-169,172,174,176,177,179-181,183,184} Fifteen descriptive studies emphasized granting staff sick time from the time of symptom onset to a minimum of 24 hours after symptom resolution.^{84,85,92,167-169,172,176,177,179,180,183,184} The majority of studies opted for 48 hours after symptom resolution before staff could return to the workplace.^{84,92,167,169,172,176,177,179,180,183,184} One study instituted a policy to exclude symptomatic staff from work until they had remained symptom-free for 72 hours.¹⁶⁸ While selected studies have identified the ability of persons to shed virus for protracted periods post-infection, it is not well understood whether virus detection translates to norovirus infectivity. The literature search was unable to determine whether return to work policies were effective in reducing secondary transmission of norovirus in healthcare facilities.

Q3.C.4 Isolation/Cohorting of Symptomatic Patients

There was very low-quality evidence among several descriptive studies to support patient cohorting or placing patients on Contact Precautions as an intervention to prevent symptomatic norovirus infections in healthcare settings.^{87,166-171,173,176,177,179-182,184} No evidence was available to encourage the use of Contact Precautions for sporadic cases, and the standard of care in these circumstances is to manage such cases with Standard Precautions (<http://www.cdc.gov/ncidod/dhqp/pdf/guidelines/Isolation2007.pdf>). Fifteen descriptive studies used isolation precautions or cohorting practices as a primary means of outbreak management.^{87,166-171,173,176,177,179-182,184} Patients were cared for in single occupancy (e.g., private) rooms, physically grouped into cohorts of symptomatic, exposed but asymptomatic, or unexposed within a ward, or alternatively, with entire wards placed under Contact Precautions. Exposure status typically was based on a person's symptoms and/or physical and temporal proximity to norovirus activity. A few studies cited restricting patient movements within the ward, suspending group activities, and special considerations for therapy or other medical appointments during outbreak periods as adjunct measures to control the spread of norovirus.^{63,169,182,183}

Q3.C.5 Staff Cohorting

Very low-quality evidence supported the implementation of staff cohorting and the exclusion of non-essential staff and volunteers to prevent symptomatic norovirus infections.^{87,103,165,168-170,172,173,177,179,180,182,183} All studies addressing this topic were descriptive. Staff was designated to care for one cohort of patients

(symptomatic, exposed but asymptomatic, or unexposed). Exposed staff was discouraged from working in unaffected clinical areas and from returning to care for unexposed patients before, at a minimum, allowing 48 hours from their last putative exposure to elapse.¹⁷⁷ The search strategy did not identify healthcare personnel other than nursing, medical, environmental services, and paramedical staff who were assigned to staff cohorting. There were no identified studies that evaluated the infectious risk of assigning recovered staff as caregivers for asymptomatic patients.

Q3.C.6 Ward Closure

Low-quality evidence was available to support ward closure as an intervention to prevent symptomatic norovirus infections.^{85,164-166,168,173,176-179,183,184} Ward closure focused on temporarily suspending transfers in or out of the ward, and discouraged or disallowed staff from working in clinical areas outside of the closed ward. One prospective controlled study evaluating 227 ward-level outbreaks between 2002 and 2003 demonstrated that outbreaks were significantly shorter (7.9 vs. 15.4 days, $p < 0.01$) when wards were closed to new admissions.¹⁶⁴ The mean duration of ward closure was 9.65 days, with a loss of 3.57 bed-days for each day the ward was closed. The duration of ward closure in the descriptive studies examined was dependent on facility resources and magnitude of the outbreaks. Allowing at least 48 hours from the resolution of the last case, followed by thorough environmental cleaning and disinfection was common before re-opening a ward. Other community-based studies have used closures as an opportunity to perform thorough environmental cleaning and disinfection before re-opening. Two studies moved all patients with symptoms of norovirus infection to a closed infectious disease ward and then performed thorough terminal cleaning of the vacated area.^{170,172} In most instances, studies defended that it was preferable to minimize patient movements and transfers in an effort to contain environmental contamination.

Q3.C.7 Visitor Policies

There was very low-quality evidence demonstrating the impact of restriction and/or screening of visitors for symptoms consistent with norovirus infection.^{168,170,173,182,183} In two studies, visitors were screened for symptoms of gastroenteritis using a standard questionnaire or evaluated by nursing staff prior to ward entry as part of multi-faceted outbreak control measures.^{168,170} Other studies restricted visitors to immediate family, suspended all visitor privileges, or curtailed visitors from accessing multiple clinical areas.^{182,183} The reviewed literature failed to identify research that considered the impact of different levels of visitor restrictions on outbreak containment.

Q3.C.8 Education

There was very low-quality evidence on the impact of staff and/or patient education on symptomatic norovirus infections.^{166,168,169,172,173,182} Six studies simply described education promoted during outbreaks.^{166,168,169,172,173,182} Content for education included recognizing symptoms of norovirus, understanding basic principles of disease transmission, understanding the components of transmission-based precautions, patient discharges and transfer policies, as well as cleaning and disinfection procedures. While many options are available, the studies that were reviewed used posters to emphasize hand hygiene and conducted one-on-one teaching with patients and visitors, as well as holding departmental seminars for staff. The literature reviewed failed to identify research that examined the impact of educational measures on the magnitude and duration of outbreaks of norovirus gastroenteritis, or what modes of education were most effective in promoting adherence to outbreak measures.

Q3.C.9 Surveillance

There was very low-quality evidence to suggest that surveillance for norovirus activity was an important measure in preventing symptomatic infection.^{58,84,166,170} Four descriptive studies identified surveillance as a component of outbreak measurement and containment. Establishing a working case definition and performing active surveillance through contact tracing, admission screening, and patient chart review were suggested as actionable items during outbreaks. There was no available literature to determine whether

active case-finding and tracking of new norovirus cases were directly associated with shorter outbreaks or more efficient outbreak containment.

Q3.C.10 Policy Development and Communication

Very low-quality evidence was available to support the benefits of having established written policies and a pre-arranged communication framework in facilitating the prevention and management of symptomatic norovirus infections.^{63,84,172,182-184} Six descriptive studies outlined the need for mechanisms to disseminate outbreak information and updates to staff, laboratory liaisons, healthcare facility administration, and public health departments.^{63,84,172,182-184} The search of the literature did not yield any studies to demonstrate that facilities with written norovirus policies already in place had fewer or shorter outbreaks of norovirus gastroenteritis.

Q3.C.11 Patient Transfers and Discharges

There was very low-quality evidence examining the benefit of delayed discharge or transfer for patients with symptomatic norovirus infection.^{172,179,183,184} Transfer of patients after symptom resolution was supported in one study but discouraged unless medically necessary in three others. Discharge home was supported once a minimum of 48 hours had elapsed since the patient's symptoms had resolved. For transfers to long-term care or assisted living, patients were held for five days after symptom resolution before transfer occurred. The literature search was unable to identify studies that compared the impact of conservative patient discharge policies for recovered, asymptomatic patients.

Q3.C.12 Environmental Disinfection

Q3.C.12.a Targeted surface disinfection

Very low-quality evidence was available to support cleaning and disinfection of frequently touched surfaces to prevent symptomatic norovirus infection.^{79,153,168,183} One systematic review¹⁵³ and three descriptive studies^{79,168,183} highlighted the need to routinely clean and disinfect frequently touched surfaces (e.g., patient and staff bathrooms and clean and dirty utility rooms, tables, chairs, commodes, computer keyboards and mice, and items in close proximity to symptomatic patients). One systematic review¹⁵³ and two descriptive studies^{102,177,183,184} supported-steam cleaning carpets once an outbreak was declared over. Within the review, a single case report suggested that contaminated carpets may contain viable virus for a minimum of twelve days even after routine dry vacuuming.¹⁵³ Routine cleaning and disinfection of non-porous flooring were supported by several studies, with particular attention to prompt cleaning of visible soiling from emesis or fecal material.^{153,168} There were no studies directly addressing the impact of surface disinfection of frequently touched areas on outbreak prevention or containment.

Q3.C.12.b Process of environmental disinfection

There was very low-quality evidence supportive of enhanced cleaning during an outbreak of norovirus gastroenteritis.^{168,170,177,179} Several studies cited increasing the frequency of cleaning and disinfection during outbreaks of norovirus gastroenteritis.^{168,170,177,179} Ward-level cleaning was performed once to twice per day, with frequently touched surfaces and bathrooms cleaned and disinfected more frequently (e.g., hourly, once per shift, or three times daily). Studies also described enhancements to the process of environmental cleaning. Environmental services staff wore PPE while cleaning patient-care areas during outbreaks of norovirus gastroenteritis.^{176,177,179,205} Personnel first cleaned the rooms of unaffected patients and then moved to the symptomatic patient areas¹⁵⁹. Adjunct measures to minimize environmental contamination from two descriptive studies included labeling patient commodes and expanding the cleaning radius for enhanced cleaning within the immediate patient area to include other proximal fixtures and equipment.^{170,177} In another study, mop heads were changed at an interval of once every three rooms.¹⁶⁸ This literature search was not able to identify whether there was an association with enhanced cleaning regimens during outbreaks of norovirus gastroenteritis and the attenuation in outbreak magnitude or duration.

Q3.C.12.c Patient-service items

There was very low-quality evidence to support the cleaning of patient equipment or service items to reduce symptomatic norovirus infections.^{168,172,177} Three descriptive studies suggested that patient equipment/service items be cleaned and disinfected after use, with disposable patient care items discarded from patient rooms upon discharge.^{168,172,177} A single descriptive study used disposable dishware and cutlery for symptomatic patients.¹⁷² There were no identified studies that directly examined the impact of disinfection of patient equipment on outbreaks of norovirus gastroenteritis.

Q3.C.12.d Fabrics

Very low-quality evidence was available to examine the impact of fabric disinfection on norovirus infections.^{153,168,177,183} One systematic review¹⁵³ and three descriptive studies^{168,177,183} suggested changing patient privacy curtains if they are visibly soiled or upon patient discharge. One descriptive study suggested that soiled, upholstered patient equipment should be steam cleaned^{135, 159}. If this was not possible, those items were discarded. Two descriptive studies emphasized careful handling of soiled linens to minimize re-aerosolization of virus.^{177,183} Wheeling hampers to the bedside or using hot soluble hamper bags (e.g., disposable) were suggested mechanisms to reduce self-contamination. This literature search did not identify studies that examined the direct impact of disinfection of fabrics on outbreaks of norovirus gastroenteritis or whether self-contamination with norovirus was associated with new infection.

Q.3.C.12.e Cleaning and disinfection agents

The overall quality of evidence on cleaning and disinfection agents was very low.^{63,83,87,89,153,167,168,170,174,176-179,182,184} The outcomes examined were symptomatic norovirus infection, inactivation of human norovirus, and inactivation of FCV. Evidence for efficacy against norovirus was usually based on studies using FCV as a surrogate. However, FCV and norovirus exhibit different physiochemical properties and it is unclear whether inactivation of FCV reflects efficacy against human strains of norovirus. One systematic review¹⁵³ and 14 descriptive studies^{63,83,87,89,167,168,170,174,176-179,182,184} outlined strategies for containing environmental bioburden. The majority of outbreaks were managed with sodium hypochlorite in various concentrations as the primary disinfectant. The concentrations for environmental cleaning among these studies ranged from 0.1% to 6.15% sodium hypochlorite.

There was found moderate quality evidence to examine the impact of disinfection agents on human norovirus inactivation.^{187,194,201} Three basic science studies evaluated the virucidal effects of select disinfectants against norovirus.^{187,194,201} A decline of 3 in the log₁₀ of human norovirus exposed to disinfectants in the presence of fecal material, a fetal bovine serum protein load, or both was achieved with 5% organic acid after 60 minutes of contact time, 6000 ppm free chlorine with 15 minutes of contact time, or a 1 or 2% peroxide solution for 60 minutes.¹⁸⁷ This study also demonstrated that the range of disinfectants more readily inactivated FCV than human norovirus samples, suggesting that FCV may not have equivalent physical properties to those of human norovirus. One basic science study demonstrated a procedure to eliminate norovirus (genogroup II) from a melamine substrate using a two step process - a cleaning step to remove gross fecal material, followed by a 5000-ppm hypochlorite product with a one minute contact time.¹⁹⁴ Cleaning with a detergent, or using a disinfectant alone failed to eliminate the virus.

Moderate quality evidence was available on the impact of disinfection agents on the human norovirus surrogate, FCV.^{185,187,188,190-192,198-200} Nine basic science studies evaluated the activity of several disinfectants agents against FCV.^{185,187,188,190-192,198-200} Only a single study showed equivalent efficacy between a quaternary ammonium compound and 1000 ppm hypochlorite on non-porous surfaces.¹⁸⁸ In contrast, selected quaternary ammonium based-products, ethanol, and a 1% anionic detergent were all unable to inactivate FCV beyond a reduction of 1.25 in the log₁₀ of virus, compared to 1000 ppm and 5000 ppm hypochlorite, 0.8% iodine, and 0.5% glutaraldehyde products.²⁰⁰ 4% organic acid, 1% peroxide, and >2% aldehyde products showed inactivation of FCV but only with impractical contact times exceeding 1 hour.¹⁸⁷

Studies of disinfecting non-porous surfaces and hands evaluated the efficacy of varying dilutions of ethanol and isopropanol and determined that 70-90% ethanol was more efficacious at inactivating FCV compared to isopropanol, but unable to achieve a reduction of 3 in the log₁₀ of the viral titer (99.9%), even after 10 minutes of contact.¹⁹¹ Other studies have shown that combinations of phenolic and quaternary ammonium compounds and peroxyacetic acid were only effective against FCV if they exceeded the manufacturers' recommended concentrations by a factor of 2 to 4.¹⁹⁹ The included basic science studies agents demonstrating complete inactivation of FCV were those containing hypochlorite, glutaraldehyde, hydrogen peroxide, iodine, or >5% sodium bicarbonate active ingredients. Not all of these products are feasible for use in healthcare settings.

In applications to various fabrics (100% cotton, 100% polyester, and cotton blends), FCV was inactivated completely by 2.6% glutaraldehyde, and showed >90% reductions of FCV titers when phenolics, 2.5% or 10% sodium bicarbonate, or 70% isopropanol were evaluated.¹⁹⁰ In carpets consisting of olefin, polyester, nylon, or blends, 2.6% glutaraldehyde demonstrated >99.7% inactivation of FCV, with other disinfectants showing moderate to modest reductions in FCV titers.¹⁹⁰ The experimental use of monochloramine as an alternative disinfectant to free chlorine in water treatment systems only demonstrated modest reductions in viral titer after 3 hours of contact time. The literature search did not evaluate publications using newer methods for environmental disinfection, such as ozone mist from a humidifying device, fumigation, UV irradiation, and fogging.

This search strategy was unable to find well-designed studies that compared virucidal efficacy of products on human norovirus, FCV, or other surrogate models among commonly used hospital disinfectants agents to establish practical standards, conditions, concentrations, and contact times. Ongoing laboratory studies are now exploring murine models as a surrogate that may exhibit greater similarity to human norovirus than FCV. Forthcoming research using this animal model may provide clearer direction regarding which disinfectants reduce norovirus environmental contamination from healthcare environments, while balancing occupational safety issues with the practicality of efficient and ready-to-use products.

Q3.D Medications

There was very low-quality evidence suggesting that select medications may reduce the risk of illness or attenuate symptoms of norovirus.^{202,203} Among elderly psychiatric patients, those on antipsychotic drugs plus trihexyphenidyl or benztropine were less likely to become symptomatic, as were those taking psyllium hydrophilic mucilloid.²⁰³ The pharmacodynamics to explain this outcome are unknown, and it is likely that these medications may either be a surrogate marker for another biologically plausible protective factor, or may impact norovirus through central or local effects on gastrointestinal motility. Those who received nitazoxanide, an anti-protozoal drug, were more likely to exhibit longer periods of norovirus illness than those patients who received placebo.²⁰² The search strategy used in this review did not identify research that considered the effect of anti-peristaltics on the duration or outcomes of norovirus infection.

Q3 Recommendations

3.A.1 Consider extending the duration of isolation or cohorting precautions for outbreaks among infants and young children (e.g., under 2 years), even after resolution of symptoms, as there is a potential for prolonged viral shedding and environmental contamination. Among infants, there is evidence to consider extending contact precautions for up to 5 days after the resolution of symptoms. **(Category II)** (Key Question 3A)

3.A.2 Further research is needed to understand the correlation between prolonged shedding of norovirus and the risk of infection to susceptible patients **(No recommendation/unresolved issue)** (Key Question 3A)

3.B.1 Perform routine cleaning and disinfection of frequently touched environmental surfaces and equipment in isolation and cohorted areas, as well as high-traffic clinical areas. Frequently touched surfaces include, but are not limited to, commodes, toilets, faucets, hand/bedrailing, telephones, door handles, computer equipment, and kitchen preparation surfaces. **(Category IB)** (Key Question 3B)

3.B.2 Remove all shared or communal food items for patients or staff from clinical areas for the duration of the outbreak. **(Category IB)** (Key Question 3B)

3.C.1.a. Actively promote adherence to hand hygiene among healthcare personnel, patients, and visitors in patient care areas affected by outbreaks of norovirus gastroenteritis. **(Category IB)** (Key Question 3C)

3.C.1.b. During outbreaks, use soap and water for hand hygiene after providing care or having contact with patients suspected or confirmed with norovirus gastroenteritis. **(Category IB)** (Key Question 3C)

3.C.1.b.1. For all other hand hygiene indications (e.g., when hands are not visibly soiled and have not been in contact with diarrheal patients, contaminated surfaces, or other body fluids) refer to the 2002 HICPAC Guideline for Hand Hygiene in Health-Care Settings (<http://www.cdc.gov/mmwr/PDF/rr/rr51116.pdf>), which includes the indications for use of FDA-compliant alcohol based hand sanitizer. **(Category IB)** (Key Question 3C)

3.C.1.b.2. Consider ethanol-based hand sanitizers (60-95%) as the preferred active agent compared to other alcohol or non-alcohol based hand sanitizer products during outbreaks of norovirus gastroenteritis. **(Category II)** (Key Question 3C)

3.C.1.b.3. Further research is required to directly evaluate the efficacy of alcohol-based hand sanitizers against human strains of norovirus, or against a surrogate virus with properties convergent with human strains of norovirus. **(No recommendation/unresolved issue)** (Key Question 3C)

3.C.2.a Use a surgical or procedure mask and eye protection or a full face shield if there is an anticipated risk of splashes to the face during the care of patients, particularly among those who are vomiting. **(Category IB)** (Key Question 3C)

3.C.3 Develop and adhere to sick leave policies for healthcare personnel who have symptoms consistent with norovirus infection. **(Category IB)** (Key Question 3C)

3.C.3.a Exclude ill personnel from work for a minimum of 48 hours after the resolution of symptoms. Once personnel return to work, the importance of performing frequent hand hygiene should be reinforced, especially before and after each patient contact. **(Category IB)** (Key Question 3C)

3.C.4.a During outbreaks, place patients with norovirus gastroenteritis on Contact Precautions for a minimum of 48 hours after the resolution of symptoms to prevent further transmission. **(Category IB)** (Key Question 3C)

3.C.4.b When patients with norovirus gastroenteritis cannot be accommodated in single occupancy rooms, efforts should be made to separate them from asymptomatic patients. Dependent upon facility characteristics, approaches for cohorting patients during outbreaks may include placing patients in multi-occupancy rooms, or designating patient care areas or contiguous sections within a facility for patient cohorts. **(Category IB)** (Key Question 3C)

3.C.4.c Consider minimizing patient movements within a ward or unit during norovirus gastroenteritis outbreaks. **(Category II)** (Key Question 3C)

3.C.4.c.1 Consider restricting symptomatic and recovering patients from leaving the patient-care area

unless it is for essential care or treatment to reduce the likelihood of environmental contamination and transmission of norovirus in unaffected clinical areas. **(Category II)** (Key Question 3C)

3.C.4.d Consider suspending group activities (e.g., dining events) for the duration of a norovirus outbreak. **(Category II)** (Key Question 3C)

3.C.5.a Establish protocols for staff cohorting in the event of an outbreak of norovirus gastroenteritis. Ensure staff care for one patient cohort on their ward and do not move between patient cohorts (e.g., patient cohorts may include symptomatic, asymptomatic exposed, or asymptomatic unexposed patient groups). **(Category IB)** (Key Question 3C)

3.C.5.b Staff who have recovered from recent suspected norovirus infection associated with this outbreak may be best suited to care for symptomatic patients until the outbreak resolves. **(Category II)** (Key Question 3C)

3.C.5.c Exclude non-essential staff, students, and volunteers from working in areas experiencing outbreaks of norovirus gastroenteritis. **(Category IB)** (Key Question 3C)

3.C.6 Consider the closure of wards to new admissions or transfers as a measure to attenuate the magnitude of an outbreak of norovirus gastroenteritis. The threshold for ward closure varies and depends on risk assessments by infection prevention personnel and facility leadership. **(Category II)** (Key Question 3C)

3.C.7.a Establish visitor policies for acute gastroenteritis (e.g., norovirus) outbreaks. **(Category IB)** (Key Question 3C)

3.C.7.b Restrict non-essential visitors from affected areas of the facility during outbreaks of norovirus gastroenteritis. **(Category IB)** (Key Question 3C)

3.C.7.b.1 For those affected areas where it is necessary to have continued visitor privileges during outbreaks, screen and exclude visitors with symptoms consistent with norovirus infection and ensure that they comply with hand hygiene and Contact Precautions. **(Category IB)** (Key Question 3C)

3.C.8.a Provide education to staff, patients, and visitors, including recognition of norovirus symptoms, preventing infection, and modes of transmission upon the recognition and throughout the duration of a norovirus gastroenteritis outbreak. **(Category IB)** (Key Question 3C)

3.C.8.b Consider providing educational sessions and making resources available on the prevention and management of norovirus before outbreaks occur, as part of annual trainings, and when sporadic cases are detected. **(Category II)** (Key Question 3C)

3.C.9.a Begin active case-finding when a cluster of acute gastroenteritis cases is detected in the healthcare facility. Use a specified case definition, and implement line lists to track both exposed and symptomatic patients and staff. Collect relevant epidemiological, clinical, and demographic data as well as information on patient location and outcomes. **(Category IB)** (Key Question 3C)

3.C.9.b As with all outbreaks, notify appropriate local and state health departments, as required by state and local public health regulations, if an outbreak of norovirus gastroenteritis is suspected. **(Category IC)** (Key Question 3C)

3.C.10 Develop written policies that specify the chains of communication needed to manage and report outbreaks of norovirus gastroenteritis. Key stakeholders such as clinical staff, environmental services, laboratory administration, healthcare facility administration and public affairs, as well as state or local public

health authorities, should be included in the framework. **(Category IB)** (Key Question 3C)

3.C.10.a Provide timely communication to personnel and visitors when an outbreak of norovirus gastroenteritis is identified and outline what policies and provisions need to be followed to prevent further transmission **(Category IB)** (Key Question 3C)

3.C.11 Consider limiting transfers to those for which the receiving facility is able to maintain Contact Precautions; otherwise, it may be prudent to postpone transfers until patients no longer require Contact Precautions. During outbreaks, medically suitable individuals recovering from norovirus gastroenteritis can be discharged to their place of residence. **(Category II)** (Key Question 3C)

3.C.12.a Clean and disinfect shared equipment between patients using EPA-registered products with label claims for use in healthcare. Follow the manufacturer's recommendations for application and contact times. The EPA lists products with activity against norovirus on their website (<http://www.epa.gov/oppad001/chemregindex.htm>). **(Category IC)** (Key Question 3C)

3.C.12.b.1 Increase the frequency of cleaning and disinfection of patient care areas and frequently touched surfaces during outbreaks of norovirus gastroenteritis (e.g., consider increasing ward/unit level cleaning to twice daily to maintain cleanliness, with frequently touched surfaces cleaned and disinfected three times daily using EPA-approved products for healthcare settings). **(Category IB)** (Key Question 3C)

3.C.12.b.2 Clean and disinfect surfaces starting from the areas with a lower likelihood of norovirus contamination (e.g., tray tables, counter tops) to areas with highly contaminated surfaces (e.g., toilets, bathroom fixtures). Change mop heads when a new bucket of cleaning solution is prepared, or after cleaning large spills of emesis or fecal material. **(Category IB)** (Key Question 3C)

3.C.12.c.1 Consider discarding all disposable patient-care items and laundering unused linens from patient rooms after patients on isolation for norovirus gastroenteritis are discharged or transferred. Facilities can minimize waste by limiting the number of disposable items brought into rooms/areas on Contact Precautions. **(Category II)** (Key Question 3C)

3.C.12.c.2 No additional provisions for using disposable patient service items such as utensils or dishware are suggested for patients with symptoms of norovirus infection. Silverware and dishware may undergo normal processing and cleaning using standard procedures. **(Category II)** (Key Question 3C)

3.C.12.c.3 Use Standard Precautions for handling soiled patient-service items or linens, including the use of appropriate PPE. **(Category IB)** (Key Question 3C)

3.C.12.d.1 Consider avoiding the use of upholstered furniture and rugs or carpets in patient care areas, as these objects are difficult to clean and disinfect completely. If this option is not possible, immediately clean soilage, such as emesis or fecal material, from upholstery, using a manufacturer-approved cleaning agent or detergent. Opt for seating in patient-care areas that can withstand routine cleaning and disinfection. **(Category II)** (Key Question 3C)

3.C.12.d.2 Consider steam cleaning of upholstered furniture in patient rooms upon discharge. Consult with manufacturer's recommendations for cleaning and disinfection of these items. Consider discarding items that cannot be appropriately cleaned/disinfected. **(Category II)**(Key Question 3C)

3.C.12.d.3 During outbreaks, change privacy curtains when they are visibly soiled and upon patient discharge or transfer. **(Category IB)** (Key Question 3C)

3.C.12.d.4 Handle soiled linens carefully, without agitating them, to avoid dispersal of virus. Use Standard

Precautions, including the use of appropriate PPE (e.g., gloves and gowns), to minimize the likelihood of cross-contamination. **(Category IB)** (Key Question 3C)

3.C.12.d.5 Double bagging, incineration, or modifications for laundering are not indicated for handling or processing soiled linen. **(Category II)** (Key Question 3C)

3.C.12.e.1 Clean surfaces and patient equipment prior to the application of a disinfectant. Follow the manufacturer's recommendations for optimal disinfectant dilution, application, and surface contact time with an EPA-approved product with claims against norovirus. **(Category IC)** (Key Question 3C)

3.C.12.e.2 More research is required to clarify the effectiveness of cleaning and disinfecting agents against norovirus, either through the use of surrogate viruses or the development of human norovirus culture system. **(No recommendation/unresolved issue)** (Key Question 3C)

3.C.12.e.3 More research is required to clarify the effectiveness and reliability of fogging, UV irradiation, and ozone mists to reduce norovirus environmental contamination. **(No recommendation/unresolved issue)** (Key Question 3C)

3.C.12.e.4 More research is required to evaluate the virucidal capabilities of alcohol-based as well as non-alcohol based hand sanitizers against norovirus. **(No recommendation/unresolved issue)** (Key Question 3C)

3.D Further research is required to evaluate the utility of medications that may attenuate the duration and severity of norovirus illness. **(No recommendation/unresolved issue)** (Key Question 3D)

References

1. Green KY, Ando T, Balayan, et al. Taxonomy of the caliciviruses. *J Infect Dis.* 2000;181 (Supplement 2):S322-30.
2. Atmar RL, Estes MK. The epidemiologic and clinical importance of norovirus infection. *Gastroenterol Clin North Am.* 2006;35(2):275-290.
3. Kaplan JE, Schonberger LB, Varano G, Jackman N, Bied J, Gary GW. An outbreak of acute nonbacterial gastroenteritis in a nursing home. Demonstration of person-to-person transmission by temporal clustering of cases. *Am J Epidemiol.* 1982;116(6):940-948.
4. Mead PS, Slutsker L, Dietz V, et al. Food-related illness and death in the United States. *Emerg Infect Dis.* 1999;5(5):607-625.
5. Widdowson MA, Meltzer MI, Zhang X, Bresee JS, Parashar UD, Glass RI. Cost-effectiveness and potential impact of rotavirus vaccination in the United States. *Pediatrics.* 2007;119(4):684-697.
6. Patel MM, Widdowson MA, Glass RI, Akazawa K, Vinje J, Parashar UD. Systematic literature review of role of noroviruses in sporadic gastroenteritis. *Emerg Infect Dis.* 2008;14(8):1224-1231.
7. Fankhauser RL, Monroe SS, Noel JS, et al. Epidemiologic and molecular trends of "Norwalk-like viruses" associated with outbreaks of gastroenteritis in the United States. *J Infect Dis.* 2002;186(1):1-7.
8. Widdowson MA, Cramer EH, Hadley L, et al. Outbreaks of acute gastroenteritis on cruise ships and on land: identification of a predominant circulating strain of norovirus--United States, 2002. *J Infect Dis.* 2004;190(1):27-36.
9. Siebenga JJ, Vennema H, Zheng DP, et al. Norovirus illness is a global problem: emergence and spread of norovirus GII.4 variants, 2001-2007. *J Infect Dis.* 2009;200(5):802-812.
10. Centers for Disease Control and Prevention (CDC). Norovirus activity--United States, 2006-2007. *MMWR.* 2007;56(33):842-846.
11. Centers for Disease Control and Prevention (CDC). Surveillance for foodborne disease outbreaks - United States, 2006. *MMWR.* 2009;58(22):609-615.

12. Caul EO. Small round structured viruses: airborne transmission and hospital control. *Lancet*. 1994;343(8908):1240-1242.
13. Hutson AM, Atmar RL, Estes MK. Norovirus disease: changing epidemiology and host susceptibility factors. *Trends Microbiol*. 2004;12(6):279-287.
14. Donaldson EF, Lindesmith LC, Lobue AD, Baric RS. Norovirus pathogenesis: mechanisms of persistence and immune evasion in human populations. *Immunol Rev*. 2008;225:190-211.
15. Widerlite L, Trier JS, Blacklow NR, Schreiber DS. Structure of the gastric mucosa in acute infectious bacterial gastroenteritis. *Gastroenterology*. 1975;68(3):425-430.
16. Kaplan JE, Feldman R, Campbell DS, Lookabaugh C, Gary GW. The frequency of a Norwalk-like pattern of illness in outbreaks of acute gastroenteritis. *Am J Public Health*. 1982;72(12):1329-1332.
17. Rabenau HF, Sturmer M, Buxbaum S, Walczok A, Preiser W, Doerr HW. Laboratory diagnosis of norovirus: which method is the best?. *Intervirology*. 2003;46(4):232-238.
18. Trujillo A, McCaustland K, Zheng D, et al. Use of TaqMan real-time reverse transcription-PCR for rapid detection, quantification, and typing of norovirus. *Journal of Clinical Microbiology*. American Society for Microbiology (ASM), Washington, USA. 2006;44(4):1405-1412. <http://dx.doi.org/10.1128/JCM.44.4.1405-1412.2006>.
19. Said MA, Perl TM, Sears CL. Healthcare epidemiology: gastrointestinal flu: norovirus in health care and long-term care facilities. *Clin Infect Dis*. 2008;47(9):1202-1208.
20. Cannon JL, Papafragkou E, Park GW, Osborne J, Jaykus LA, Vinje J. Surrogates for the study of norovirus stability and inactivation in the environment: a comparison of murine norovirus and feline calicivirus. *J Food Prot*. 2006;69(11):2761-2765.
21. Guyatt GH, Oxman AD, Vist GE, et al. GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. *BMJ*. 2008;336(7650):924-926.
22. Guyatt GH, Oxman AD, Kunz R, et al. What is "quality of evidence" and why is it important to clinicians? *BMJ*. 2008;336(7651):995-998.
23. Guyatt GH, Oxman AD, Kunz R, et al. Going from evidence to recommendations. *BMJ*. 2008;336(7652):1049-1051.
24. Schunemann HJ, Oxman AD, Brozek J, et al. Grading quality of evidence and strength of recommendations for diagnostic tests and strategies. *BMJ*. 2008;336(7653):1106-1110.
25. Vessel Sanitation Program Construction Guideline, July 2005. Available at: <http://www.cdc.gov/nceh/vsp/ConstructionGuidelines/ConstructionGuidelines2005.pdf>. Accessed September 24, 2007.
26. Vessel Sanitation Program Operations Manual, August 2005. Available at: <http://www.cdc.gov/nceh/vsp/operationsmanual/OPSMannual2005.pdf>. Accessed September 24, 2007.
27. Guidance for the Management of Norovirus Infection in Cruise Ships. July 2007. Available at: http://www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1206520183347. Accessed September 24, 2007.
28. Viral Gastroenteritis: Leeds Teaching Hospitals Trust Infection Control Policies. 2008. Available at: http://www.leedsteachinghospitals.com/sites/infection_control/documents/VGRevisedJan08.pdf. Accessed September 24, 2007.
29. National Guidelines on the Management of Outbreaks of Norovirus Infection in Healthcare Settings. 2003. Available at: <http://www.hpsc.ie/hpsc/A-Z/Gastroenteric/ViralGastroenteritis/Publications/File,1194,en.pdf>. Accessed September 24, 2007.
30. Netherlands: Norovirus Guidelines for Cruiseships and Hotels. 2007. Accessed September 24, 2007.
31. Gastroenteritis in an Institution Response Protocol for New South Wales Public Health Units, NSW Health 2005. (updated 2010). Available at: <http://www.health.nsw.gov.au/factsheets/guideline/gastro.html>. Accessed September 24, 2007.
32. Guidelines for the Management of Norovirus Outbreaks in Hospitals and Elderly Care Institutions, July 2007, Auckland Regional Public Health Service, New Zealand. (updated 2008). Available at: http://www.arphs.govt.nz/notifiable/downloads/Norovirus_Guidelines_2008.pdf. Accessed September 24, 2007.
33. Viral Gastroenteritis Outbreaks: Information for Supervisors in the Child Care and Hospitality Industries. Queensland Health, Australia. (updated 2010). Available at: <http://www.health.qld.gov.au/ph/documents/cdb/26888.pdf>. Accessed September 24, 2007.

34. Aide-Memoire for Managing Norovirus Outbreaks in Healthcare Settings Scottish Centre for Infection and Environmental Health, National Services Scotland. 2004. Available at: <http://www.hps.scot.nhs.uk/giz/publicationsdetail.aspx?id=22582>. Accessed September 24, 2007.
35. The Identification and Management of Outbreaks of Norovirus Infection in Tourists and Leisure Industry Settings. 2005. Available at: http://www.sefton.gov.uk/pdf/epd_norovirusguide.pdf. Accessed September 24, 2007.
36. Guidelines for the Management of Infectious Gastroenteritis in Aged Care Facilities in South Australia. 2005. Available at: <http://www.publications.health.sa.gov.au/cgi/viewcontent.cgi?article=1014&context=cdc>. Accessed September 24, 2007.
37. General Guidelines for the Management of Viral Gastroenteritis, Community Infection Prevention and Control Policy and Procedure, Gloucestershire Primary Care Trust, 2008. Available at: <http://www.glospct.nhs.uk/pdf/policies/infectioncontrol/gpct%20viral%20gastroenteritis.pdf>. Accessed September 24, 2007.
38. Recommendations for the Prevention and Control of Viral Gastroenteritis Outbreaks in California Long-Term Care Facilities. 2006 Available at: <http://www.cdph.ca.gov/pubsforms/Guidelines/Documents/PCofViralGastroenteritisOutbreaks.pdf>. Accessed September 24, 2007.
39. Control of Viral Gastroenteritis Outbreaks in Illinois Long-Term Care Facilities. Illinois Department of Public Health. 2006. Available at: <http://www.co.mchenry.il.us/departments/health/pdfDocs/PHS/CD/NoroLongFacGuide.pdf>. Accessed September 24, 2007.
40. Norovirus Prevention Guidance for Institutions/Facilities. Virginia Department of Health, January 2007. Available at: http://www.flpic.com/Norovirus_Prev_Guidance_Institutions_2007.pdf. Accessed September 24, 2007.
41. Viral Gastroenteritis Outbreak Guidelines for Child Care Facilities. Washoe County District Health Department, Reno, Nevada. http://www.co.washoe.nv.us/repository/files//4/Childcare_guidelines_for_norovirus.pdf. Accessed September 24, 2007
42. Viral Gastroenteritis Outbreak Guidelines for Community Living Facilities. Washoe County District Health Department, Reno, Nevada. 2005. Available at: <http://www.co.washoe.nv.us/repository/files/4/Guidelines%20for%20Norovirus%20in%20ECFs.pdf>. Accessed September 24, 2007.
43. Guidelines for the Epidemiological Investigation of Gastroenteritis Outbreaks in Long Term Care Facilities, Maryland Department of Health and Mental Hygiene. Revised 2001. Available at: <http://ideha.dhmm.maryland.gov/pdf/guidelines/gastroenteritis.aspx>. Accessed September 24, 2007.
44. Recommendations for the Control of Viral Gastroenteritis Outbreaks in Long-Term Care Facilities and Viral Gastroenteritis Outbreaks in Nursing Homes or Long-Term Care Facilities Guidelines for Environmental Decontamination. Georgia Department of Community Health. Available at: http://health.state.ga.us/pdfs/epi/notifiable/outbreaks/Norovirus%20cleaning%20guidelines_LTC.pdf. Accessed September 24, 2007.
45. Recommendations for the Prevention and Control of Viral Gastroenteritis Outbreaks in Wisconsin Long-Term Care Facilities. Wisconsin Division of Public Health. (updated 2009). Available at: http://www.dhs.wisconsin.gov/rl_dsl/Providers/norovirusRecoLTCF09.pdf. Accessed September 24, 2007.
46. Norovirus (Viral Gastroenteritis) Control Measures for Skilled Nursing Facilities. Los Angeles County Public Health. 2006. Available at: http://www.publichealth.lacounty.gov/acd/docs/Norovirus/NorovirusControlMeasures_12_1_06.pdf. Accessed September 24, 2007.
47. Norovirus (Viral Gastroenteritis): Information Packet for Nursing Facilities. Washington State Department of Health, May 2005. Available at: <http://www.co.thurston.wa.us/health/personalhealth/communicabledisease/DrYuUpdates/PDF/2011noropacket.pdf>. Accessed September 24, 2007.
48. Viral Gastroenteritis Guidance. Royal Devon and Exeter. NHS, August 2007. (revised July 2009). Available at: http://www.rdehospital.nhs.uk/docs/patients/services/infection_control/Viral%20gastroenteritis-Aug2009.pdf. Accessed September 24, 2007.
49. Norovirus Infections. Public Health Importance and Outbreak Management. Public Health-Seattle and King County. Updated March 2010. Available at:

<http://www.kingcounty.gov/healthservices/health/communicable/diseases/NorovirusControl.aspx>. Accessed September 24, 2007.

50. von Elm E, Altman DG, Egger M, et al. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *Ann Intern Med*. 2007;147(8):573-577.
51. Shea BJ, Grimshaw JM, Wells GA, et al. Development of AMSTAR: a measurement tool to assess the methodological quality of systematic reviews. *BMC Med Res Methodol*. 2007;7:10.
52. Sanderson S, Tatt ID, Higgins JP. Tools for assessing quality and susceptibility to bias in observational studies in epidemiology: a systematic review and annotated bibliography. *Int J Epidemiol*. 2007;36(3):666-676.
53. Moher D, Schulz KF, Altman D, CONSORT Group (Consolidated Standards of Reporting Trials). The CONSORT statement: revised recommendations for improving the quality of reports of parallel-group randomized trials. *JAMA*. 2001;285(15):1987-1991.
54. Jadad AR, Moore RA, Carroll D, et al. Assessing the quality of reports of randomized clinical trials: is blinding necessary? *Control Clin Trials*. 1996;17(1):1-12.
55. Yale Center for Medical Informatics. 2009. Available at: <http://ycmi.med.yale.edu/>. Accessed April 27, 2011.
56. Mattner F, Mattner L, Borck HU, Gastmeier P. Evaluation of the impact of the source (patient versus staff) on nosocomial norovirus outbreak severity. *Infect Control Hosp Epidemiol*. 2005;26(3):268-272.
57. Mattner F, Sohr D, Heim A, Gastmeier P, Vennema H, Koopmans M. Risk groups for clinical complications of norovirus infections: an outbreak investigation. *Clin Microbiol Infect*. 2006;12(1):69-74.
58. Lopman BA, Reacher MH, Vipond IB, Sarangi J, Brown DW. Clinical manifestation of norovirus gastroenteritis in health care settings. *Clin Infect Dis*. 2004;39(3):318-324.
59. Lee N, Chan MCW, Wong B, et al. Fecal viral concentration and diarrhea in norovirus gastroenteritis. *Emerg Infect Dis*. 2007;13(9):1399-1401. <http://www.cdc.gov/eid/content/13/9/pdfs/1399.pdf>.
60. Rodriguez-Guillen L, Vizzi E, Alcalá AC, Pujol FH, Liprandi F, Ludert JE. Calicivirus infection in human immunodeficiency virus seropositive children and adults. *J Clin Virol*. 2005;33(2):104-109.
61. de Wit MA, Koopmans MP, van Duynhoven YT. Risk factors for norovirus, Sapporo-like virus, and group A rotavirus gastroenteritis. *Emerg Infect Dis*. 2003;9(12):1563-1570.
62. Gotz H, Ekdahl K, Lindback J, de Jong B, Hedlund KO, Giesecke J. Clinical spectrum and transmission characteristics of infection with Norwalk-like virus: findings from a large community outbreak in Sweden. *Clin Infect Dis*. 2001;33(5):622-628.
63. Oppermann H, Mueller B, Takkinen J, Klauditz W, Schreier E, Ammon A. An outbreak of viral gastroenteritis in a mother-and-child health clinic. *Int J Hyg Environ Health*. 2001;203(4):369-373.
64. Sharp TW, Hyams KC, Watts D, et al. Epidemiology of Norwalk virus during an outbreak of acute gastroenteritis aboard a US aircraft carrier. *J Med Virol*. 1995;45(1):61-67.
65. Thea DM, Glass R, Grohmann GS, et al. Prevalence of enteric viruses among hospital patients with AIDS in Kinshasa, Zaire. *Trans R Soc Trop Med Hyg*. 1993;87(3):263-266.
66. Marx A, Shay DK, Noel JS, et al. An outbreak of acute gastroenteritis in a geriatric long-term-care facility: combined application of epidemiological and molecular diagnostic methods. *Infect Control Hosp Epidemiol*. 1999;20(5):306-311.
67. Caceres VM, Kim DK, Bresee JS, et al. A viral gastroenteritis outbreak associated with person-to-person spread among hospital staff. *Infect Control Hosp Epidemiol*. 1998;19(3):162-167.
68. Cegielski JP, Msengi AE, Miller SE. Enteric viruses associated with HIV infection in Tanzanian children with chronic diarrhea. *Pediatr AIDS HIV Infect*. 1994;5(5):296-299.
69. Halperin T, Vennema H, Koopmans M, et al. No association between histo-blood group antigens and susceptibility to clinical infections with genogroup II norovirus. *J Infect Dis*. 2008;197(1):63-65.
70. Hutson AM, Airaud F, LePendou J, Estes MK, Atmar RL. Norwalk virus infection associates with secretor status genotyped from sera. *J Med Virol*. 2005;77(1):116-120.
71. Thorven M, Grahn A, Hedlund KO, et al. A homozygous nonsense mutation (428G-->A) in the human secretor (FUT2) gene provides resistance to symptomatic norovirus (GGII) infections. *J Virol*. 2005;79(24):15351-15355.
72. Lindesmith L, Moe C, Marionneau S, et al. Human susceptibility and resistance to Norwalk virus infection. *Nat Med*. 2003;9(5):548-553.
73. Hutson AM, Atmar RL, Graham DY, Estes MK. Norwalk virus infection and disease is associated with ABO histo-blood group type. *J Infect Dis*. 2002;185(9):1335-1337.

74. Graham DY, Jiang X, Tanaka T, Opekun AR, Madore HP, Estes MK. Norwalk virus infection of volunteers: new insights based on improved assays. *J Infect Dis.* 1994;170(1):34-43.
75. Nakata S, Chiba S, Terashima H, Yokoyama T, Nakao T. Humoral immunity in infants with gastroenteritis caused by human calicivirus. *J Infect Dis.* 1985;152(2):274-279.
76. Parrino TA, Schreiber DS, Trier JS, Kapikian AZ, Blacklow NR. Clinical immunity in acute gastroenteritis caused by Norwalk agent. *N Engl J Med.* 1977;297(2):86-89.
77. Fretz R, Svoboda P, Schorr D, Tanner M, Baumgartner A. Risk factors for infections with Norovirus gastrointestinal illness in Switzerland. *Eur J Clin Microbiol Infect Dis.* 2005;24(4):256-261.
78. Meyer E, Ebner W, Scholz R, Dettkenkofer M, Daschner FD. Nosocomial outbreak of norovirus gastroenteritis and investigation of ABO histo-blood group type in infected staff and patients. *J Hosp Infect.* 2004;56(1):64-66.
79. Centers for Disease Control and Prevention (CDC). Norovirus outbreak in an elementary school--District of Columbia, February 2007. *MMWR.* 2008;56(51-52):1340-1343.
80. Centers for Disease Control and Prevention (CDC). Multistate outbreak on norovirus gastroenteritis among attendees at a family reunion -- Grant County, West Virginia, October 2006. *MMWR.* 2007;56(27):673-678.
81. Costas L, Vilella A, Llupia A, Bosch J, Jimenez de Anta MT, Trilla A. Outbreak of norovirus gastroenteritis among staff at a hospital in Barcelona, Spain, September 2007. *Euro Surveill.* 2007;12(11):E071122.5.
82. Lopman BA, Andrews N, Sarangi J, Vipond IB, Brown DW, Reacher MH. Institutional risk factors for outbreaks of nosocomial gastroenteritis: survival analysis of a cohort of hospital units in South-west England, 2002-2003. *J Hosp Infect.* 2005;60(2):135-143.
83. Evans MR, Meldrum R, Lane W, et al. An outbreak of viral gastroenteritis following environmental contamination at a concert hall. *Epidemiol Infect.* 2002;129(2):355-360.
84. Lachlan M, Licence K, Oates K, Vaughan S, Hill R. Practical lessons from the management of an outbreak of small round structured virus (Norwalk-like virus) gastroenteritis. *Commun Dis Public Health.* 2002;5(1):43-47.
85. Love SS, Jiang X, Barrett E, Farkas T, Kelly S. A large hotel outbreak of Norwalk-like virus gastroenteritis among three groups of guests and hotel employees in Virginia. *Epidemiol Infect.* 2002;129(1):127-132.
86. Anderson AD, Garrett VD, Sobel J, et al. Multistate outbreak of Norwalk-like virus gastroenteritis associated with a common caterer. *Am J Epidemiol.* 2001;154(11):1013-1019.
87. Cunney RJ, Costigan P, McNamara EB, et al. Investigation of an outbreak of gastroenteritis caused by Norwalk-like virus, using solid phase immune electron microscopy. *J Hosp Infect.* 2000;44(2):113-118.
88. Marks PJ, Vipond IB, Carlisle D, Deakin D, Fey RE, Caul EO. Evidence for airborne transmission of Norwalk-like virus (NLV) in a hotel restaurant. *Epidemiol Infect.* 2000;124(3):481-487.
89. Lo SV, Connolly AM, Palmer SR, Wright D, Thomas PD, Joynson D. The role of the pre-symptomatic food handler in a common source outbreak of food-borne SRSV gastroenteritis in a group of hospitals. *Epidemiol Infect.* 1994;113(3):513-521.
90. Patterson T, Hutchings P, Palmer S. Outbreak of SRSV gastroenteritis at an international conference traced to food handled by a post-symptomatic caterer. *Epidemiol Infect.* 1993;111(1):157-162.
91. Alexander WJ, Holmes JR, Shaw JF, Riley WE, Roper WL. Norwalk virus outbreak at a college campus. *South Med J.* 1986;79(1):33-36.
92. Wit MA, Widdowson V, H., Bruin Ed, Fernandes T, Koopmans M. Large outbreak of norovirus: the baker who should have known better. *Journal of Infection.* Elsevier, Amsterdam, Netherlands. 2007;55(2):188-193.
93. Centers for Disease Control and Prevention (CDC). Norovirus outbreak associated with ill food-service workers-- Michigan, January-February 2006. *MMWR.* 2007;56(46):1212-1216.
94. Rizzo C, Di Bartolo I, Santantonio M, et al. Epidemiological and virological investigation of a Norovirus outbreak in a resort in Puglia, Italy. *BMC Infect Dis.* 2007;7:135.
95. Schmid D, Stuger HP, Lederer I, et al. A foodborne norovirus outbreak due to manually prepared salad, Austria 2006. *Infection.* 2007;35(4):232-239.
96. Payne J, Hall M, Lutzke M, Armstrong C, King J. Multisite outbreak of norovirus associated with a franchise restaurant - Kent County, Michigan, May 2005. *MMWRt.* 2006;55(14):395-397.
97. Grotto I, Huerta M, Balicer RD, et al. An outbreak of norovirus gastroenteritis on an Israeli military base. *Infection.* 2004;32(6):339-343.
98. Marks PJ, Vipond IB, Regan FM, Wedgwood K, Fey RE, Caul EO. A school outbreak of Norwalk-like virus: evidence for airborne transmission. *Epidemiol Infect.* 2003;131(1):727-736.

99. Stegenga J, Bell E, Matlow A. The role of nurse understaffing in nosocomial viral gastrointestinal infections on a general pediatrics ward. *Infect Control Hosp Epidemiol.* 2002;23(3):133-136.
100. Becker KM, Moe CL, Southwick KL, MacCormack JN. Transmission of Norwalk virus during football game. *N Engl J Med.* 2000;343(17):1223-1227.
101. Parashar UD, Dow L, Fankhauser RL, et al. An outbreak of viral gastroenteritis associated with consumption of sandwiches: implications for the control of transmission by food handlers. *Epidemiol Infect.* 1998;121(3):615-621.
102. McEvoy M, Blake W, Brown D, Green J, Cartwright R. An outbreak of viral gastroenteritis on a cruise ship. *Commun Dis Rep CDR Rev.* 1996;6(13):R188-92.
103. Chadwick PR, McCann R. Transmission of a small round structured virus by vomiting during a hospital outbreak of gastroenteritis. *J Hosp Infect.* 1994;26(4):251-259.
104. Reid JA, Caul EO, White DG, Palmer SR. Role of infected food handler in hotel outbreak of Norwalk-like viral gastroenteritis: implications for control. *Lancet.* 1988;2(8606):321-323.
105. Iversen AM, Gill M, Bartlett CL, Cubitt WD, McSwiggan DA. Two outbreaks of foodborne gastroenteritis caused by a small round structured virus: evidence of prolonged infectivity in a food handler. *Lancet.* 1987;2(8558):556-558.
106. White KE, Osterholm MT, Mariotti JA, et al. A foodborne outbreak of Norwalk virus gastroenteritis. Evidence for post-recovery transmission. *Am J Epidemiol.* 1986;124(1):120-126.
107. Kaplan JE, Gary GW, Baron RC, et al. Epidemiology of Norwalk gastroenteritis and the role of Norwalk virus in outbreaks of acute nonbacterial gastroenteritis. *Ann Intern Med.* 1982;96(6 Pt 1):756-761.
108. Tu ETV, Bull RA, Greening GE, et al. Epidemics of gastroenteritis during 2006 were associated with the spread of norovirus GII.4 variants 2006a and 2006b. *Clinical Infectious Diseases.* 2008;46(3):413-420.
109. Adamson WE, Gunson RN, Maclean A, Carman WF. Emergence of a new norovirus variant in Scotland in 2006. *J Clin Microbiol.* 2007;45(12):4058-4060.
110. Gallimore CI, Cubitt D, du Plessis N, Gray JJ. Asymptomatic and symptomatic excretion of noroviruses during a hospital outbreak of gastroenteritis. *J Clin Microbiol.* 2004;42(5):2271-2274.
111. Blanton LH, Adams SM, Beard RS, et al. Molecular and epidemiologic trends of caliciviruses associated with outbreaks of acute gastroenteritis in the United States, 2000-2004. *J Infect Dis.* 2006;193(3):413-421.
112. Mattison K, Karthikeyan K, Abebe M, et al. Survival of calicivirus in foods and on surfaces: experiments with feline calicivirus as a surrogate for norovirus. *J Food Prot.* 2007;70(2):500-503.
113. Lindesmith L, Moe C, Lependu J, Frelinger JA, Treanor J, Baric RS. Cellular and humoral immunity following Snow Mountain virus challenge. *J Virol.* 2005;79(5):2900-2909.
114. Fretz R, Herrmann L, Christen A, et al. Frequency of Norovirus in stool samples from patients with gastrointestinal symptoms in Switzerland. *Eur J Clin Microbiol Infect Dis.* 2005;24(3):214-216.
115. Fretz R, Svoboda P, Luthi TM, Tanner M, Baumgartner A. Outbreaks of gastroenteritis due to infections with Norovirus in Switzerland, 2001-2003. *Epidemiol Infect.* 2005;133(3):429-437.
116. Turcios RM, Widdowson MA, Sulka AC, Mead PS, Glass RI. Reevaluation of epidemiological criteria for identifying outbreaks of acute gastroenteritis due to norovirus: United States, 1998-2000. *Clin Infect Dis.* 2006;42(7):964-969.
117. Duizer E, Pielaat A, Vennema H, Kroneman A, Koopmans M. Probabilities in norovirus outbreak diagnosis. *J Clin Virol.* 2007;40(1):38-42.
118. Gray JJ, Kohli E, Ruggeri FM, et al. European multicenter evaluation of commercial enzyme immunoassays for detecting norovirus antigen in fecal samples. *Clin Vaccine Immunol.* 2007;14(10):1349-1355.
119. Richards AF, Lopman B, Gunn A, et al. Evaluation of a commercial ELISA for detecting Norwalk-like virus antigen in faeces. *J Clin Virol.* 2003;26(1):109-115.
120. Khamrin P, Nguyen TA, Phan TG, et al. Evaluation of immunochromatography and commercial enzyme-linked immunosorbent assay for rapid detection of norovirus antigen in stool samples. *J Virol Methods.* 2008;147(2):360-363.
121. Wiechers C, Bissinger AL, Hamprecht K, Kimmig P, Jahn G, Poets CF. Apparently non-specific results found using a norovirus antigen immunoassay for fecal specimens from neonates. *Journal of Perinatology.* 2008;28(1):79-81.
122. Castriciano S, Luinstra K, Petrich A, et al. Comparison of the RIDASCREEN norovirus enzyme immunoassay to IDEIA NLV GI/GII by testing stools also assayed by RT-PCR and electron microscopy. *J Virol Methods.* 2007;141(2):216-219.

123. Wilhelmi de Cal I, Revilla A, del Alamo JM, Roman E, Moreno S, Sanchez-Fauquier A. Evaluation of two commercial enzyme immunoassays for the detection of norovirus in faecal samples from hospitalised children with sporadic acute gastroenteritis. *Clin Microbiol Infect.* 2007;13(3):341-343.
124. de Bruin E, Duizer E, Vennema H, Koopmans MP. Diagnosis of Norovirus outbreaks by commercial ELISA or RT-PCR. *J Virol Methods.* 2006;137(2):259-264.
125. Okitsu-Negishi S, Okame M, Shimizu Y, et al. Detection of norovirus antigens from recombinant virus-like particles and stool samples by a commercial norovirus enzyme-linked immunosorbent assay kit. *J Clin Microbiol.* 2006;44(10):3784-3786.
126. Burton-MacLeod JA, Kane EM, Beard RS, Hadley LA, Glass RI, Ando T. Evaluation and comparison of two commercial enzyme-linked immunosorbent assay kits for detection of antigenically diverse human noroviruses in stool samples. *J Clin Microbiol.* 2004;42(6):2587-2595.
127. Christen A, Fretz R, Tanner M, Svoboda P. Evaluation of a commercial ELISA kit for the detection of Norovirus antigens in human stool specimens. *Mitteilungen aus Lebensmitteluntersuchung und Hygiene. Bundesamt für Gesundheit.* 2003;94(6):594-602.
128. Gunson RN, Miller J, Carman WF. Comparison of real-time PCR and EIA for the detection of outbreaks of acute gastroenteritis caused by norovirus. *Commun Dis Public Health.* 2003;6(4):297-299.
129. Nordgren J, Bucardo F, Dienus O, Svensson L, Lindgren PE. Novel light-upon-extension real-time PCR assays for detection and quantification of genogroup I and II noroviruses in clinical specimens. *J Clin Microbiol.* 2008;46(1):164-170.
130. De Medici D, Suffredini E, Crudeli S, Ruggeri FM. Effectiveness of an RT-booster-PCR method for detection of noroviruses in stools collected after an outbreak of gastroenteritis. *J Virol Methods.* 2007;144(1-2):161-164.
131. Hymas W, Atkinson A, Stevenson J, Hillyard D. Use of modified oligonucleotides to compensate for sequence polymorphisms in the real-time detection of norovirus. *J Virol Methods.* 2007;142(1-2):10-14.
132. Logan C, O'Leary JJ, O'Sullivan N. Real-time reverse transcription PCR detection of norovirus, sapovirus and astrovirus as causative agents of acute viral gastroenteritis. *J Virol Methods.* 2007;146(1-2):36-44.
133. Menton JF, Kearney K, Morgan JG. Development of a real-time RT-PCR and Reverse Line probe Hybridisation assay for the routine detection and genotyping of Noroviruses in Ireland. *Virol J.* 2007;4:86.
134. Wolf S, Williamson WM, Hewitt J, et al. Sensitive multiplex real-time reverse transcription-PCR assay for the detection of human and animal noroviruses in clinical and environmental samples. *Appl Environ Microbiol.* 2007;73(17):5464-5470.
135. Yoda T, Suzuki Y, Yamazaki K, et al. Evaluation and application of reverse transcription loop-mediated isothermal amplification for detection of noroviruses. *J Med Virol.* 2007;79(3):326-334.
136. Antonishyn NA, Crozier NA, McDonald RR, Levett PN, Horsman GB. Rapid detection of Norovirus based on an automated extraction protocol and a real-time multiplexed single-step RT-PCR. *J Clin Virol.* 2006;37(3):156-161.
137. Hohne M, Schreier E. Detection and Characterization of Norovirus Outbreaks in Germany: Application of a One-Tube RT-PCR Using a Fluorogenic Real-Time Detection System. *J Med Virol.* 2004;72(2):312-319.
138. Rohayem J, Berger S, Juretzek T, et al. A simple and rapid single-step multiplex RT-PCR to detect Norovirus, Astrovirus and Adenovirus in clinical stool samples. *J Virol Methods.* 2004;118(1):49-59.
139. Schmid M, Oehme R, Schalasta G, Brockmann S, Kimmig P, Enders G. Fast detection of Noroviruses using a real-time PCR assay and automated sample preparation. *BMC Infect Dis.* 2004;4:15.
140. Vinje J, Vennema H, Maunula L, et al. International collaborative study to compare reverse transcriptase PCR assays for detection and genotyping of noroviruses. *J Clin Microbiol.* 2003;41(4):1423-1433.
141. Tatsumi M, Nakata S, Sakai Y, Honma S, Numata-Kinoshita K, Chiba S. Detection and differentiation of Norwalk virus by reverse transcription-polymerase chain reaction and enzyme-linked immunosorbent assay. *J Med Virol.* 2002;68(2):285-290.
142. O'Neill HJ, McCaughey C, Wyatt DE, Mitchell F, Coyle PV. Gastroenteritis outbreaks associated with Norwalk-like viruses and their investigation by nested RT-PCR. *BMC Microbiol.* 2001;1:14.
143. Jean J, D'Souza D, Jaykus LA. Transcriptional enhancement of RT-PCR for rapid and sensitive detection of Noroviruses. *FEMS Microbiol Lett.* 2003;226(2):339-345.
144. Greene SR, Moe CL, Jaykus LA, Cronin M, Grosso L, Aarle P. Evaluation of the NucliSens Basic Kit assay for detection of Norwalk virus RNA in stool specimens. *J Virol Methods.* 2003;108(1):123-131.

145. Tian P, Mandrell R. Detection of norovirus capsid proteins in faecal and food samples by a real time immuno-PCR method. *Journal of Applied Microbiology*. 2006;100(3):564-574.
146. Beuret C. A simple method for isolation of enteric viruses (noroviruses and enteroviruses) in water. *J Virol Methods*. 2003;107(1):1-8.
147. Vinje J, Hamidjaja RA, Sobsey MD. Development and application of a capsid VP1 (region D) based reverse transcription PCR assay for genotyping of genogroup I and II noroviruses. *J Virol Methods*. 2004;116(2):109-117.
148. Murata T, Katsushima N, Mizuta K, Muraki Y, Hongo S, Matsuzaki Y. Prolonged norovirus shedding in infants <or=6 months of age with gastroenteritis. *Pediatr Infect Dis J*. 2007;26(1):46-49.
149. Rockx B, De Wit M, Vennema H, et al. Natural history of human calicivirus infection: a prospective cohort study. *Clin Infect Dis*. 2002;35(3):246-253.
150. Marshall JA, Salamone S, Yuen L, Catton MG, Wright JP. High level excretion of Norwalk-like virus following resolution of clinical illness. *Pathology*. 2001;33(1):50-52.
151. Hedlund K-, Bennet R, Eriksson M, Ehrnst A. Norwalk-like virus as a cause of diarrhea in a pediatric hospital. *Clinical Microbiology and Infection*. 1998;4(8):417-421.
152. Chiba S, Sakuma Y, Kogasaka R, et al. Fecal shedding of virus in relation to the days of illness in infantile gastroenteritis due to calicivirus. *J Infect Dis*. 1980;142(2):247-249.
153. Dalling J. A review of environmental contamination during outbreaks of Norwalk-like virus. *Br J Infect Control*. 2004;5(2):9-13.
154. Wu HM, Fornek M, Schwab KJ, et al. A norovirus outbreak at a long-term-care facility: the role of environmental surface contamination. *Infect Control Hosp Epidemiol*. 2005;26(10):802-810.
155. Jones EL, Kramer A, Gaither M, Gerba CP. Role of fomite contamination during an outbreak of norovirus on houseboats. *Int J Environ Health Res*. 2007;17(2):123-131.
156. Clay S, Maherchandani S, Malik YS, Goyal SM. Survival on uncommon fomites of feline calicivirus, a surrogate of noroviruses. *Am J Infect Control*. 2006;34(1):41-43.
157. Gallimore CI, Taylor C, Gennery AR, et al. Environmental monitoring for gastroenteric viruses in a pediatric primary immunodeficiency unit. *J Clin Microbiol*. 2006;44(2):395-399.
158. Kuusi M, Nuorti JP, Maunula L, et al. A prolonged outbreak of Norwalk-like calicivirus (NLV) gastroenteritis in a rehabilitation centre due to environmental contamination. *Epidemiol Infect*. 2002;129(1):133-138.
159. Cheesbrough JS, Green J, Gallimore CI, Wright PA, Brown DW. Widespread environmental contamination with Norwalk-like viruses (NLV) detected in a prolonged hotel outbreak of gastroenteritis. *Epidemiol Infect*. 2000;125(1):93-98.
160. Schvoerer E, Bonnet F, Dubois V, et al. A hospital outbreak of gastroenteritis possibly related to the contamination of tap water by a small round structured virus. *J Hosp Infect*. 1999;43(2):149-154.
161. Green J, Wright PA, Gallimore CI, Mitchell O, Morgan-Capner P, Brown DW. The role of environmental contamination with small round structured viruses in a hospital outbreak investigated by reverse-transcriptase polymerase chain reaction assay. *J Hosp Infect*. 1998;39(1):39-45.
162. D'Souza DH, Sair A, Williams K, et al. Persistence of caliciviruses on environmental surfaces and their transfer to food. *Int J Food Microbiol*. 2006;108(1):84-91.
163. Paulson DS. The transmission of surrogate Norwalk virus - from inanimate surfaces to gloved hands: is it a threat? *Food Protection Trends*. International Association for Food Protection. 2005;25(6):450-454.
164. Lopman BA, Reacher MH, Vipond IB, et al. Epidemiology and cost of nosocomial gastroenteritis, Avon, England, 2002-2003. *Emerg Infect Dis*. 2004;10(10):1827-1834.
165. Billgren M, Christenson B, Hedlund KO, Vinje J. Epidemiology of Norwalk-like human caliciviruses in hospital outbreaks of acute gastroenteritis in the Stockholm area in 1996. *J Infect*. 2002;44(1):26-32.
166. Hansen S, Stamm-Balderjahn S, Zuschneid I, et al. Closure of medical departments during nosocomial outbreaks: data from a systematic analysis of the literature. *J Hosp Infect*. 2007;65(4):348-353.
167. Zingg W, Colombo C, Jucker T, Bossart W, Rued C. Impact of an outbreak of norovirus infection on hospital resources. *Infect Control Hosp Epidemiol*. 2005;26(3):263-267.
168. Johnston CP, Qiu H, Ticehurst JR, et al. Outbreak management and implications of a nosocomial norovirus outbreak. *Clin Infect Dis*. 2007;45(5):534-540.
169. Leuenberger S, Widdowson MA, Feilchenfeldt J, Egger R, Streuli RA. Norovirus outbreak in a district general hospital--new strain identified. *Swiss Med Wkly*. 2007;137(3-4):57-81.

170. Cheng FW, Leung TF, Lai RW, Chan PK, Hon EK, Ng PC. Rapid control of norovirus gastroenteritis outbreak in an acute paediatric ward. *Acta Paediatr.* 2006;95(5):581-586.
171. Simon A, Schildgen O, Maria Eis-Hubinger A, et al. Norovirus outbreak in a pediatric oncology unit. *Scand J Gastroenterol.* 2006;41(6):693-699.
172. Conway R, Bunt S, Mathias E, Said H. The Norovirus experience: an exercise in outbreak management at a tertiary referral hospital. *Aust Infect Control.* 2005;10(3):95, 97-102.
173. Cooper E, Blamey S. A norovirus gastroenteritis epidemic in a long-term-care facility. *Infection Control and Hospital Epidemiology.* 2005;26(3):256-258.
174. Navarro G, Sala RM, Segura F, et al. An outbreak of norovirus infection in a long-term-care unit in Spain. *Infect Control Hosp Epidemiol.* 2005;26(3):259-262.
175. Schmid D, Lederer I, Pichler AM, Berghold C, Schreier E, Allerberger F. An outbreak of Norovirus infection affecting an Austrian nursing home and a hospital. *Wien Klin Wochenschr.* 2005;117(23-24):802-808.
176. Weber DJ, Sickbert-Bennett EE, Vinje J, et al. Lessons learned from a norovirus outbreak in a locked pediatric inpatient psychiatric unit. *Infect Control Hosp Epidemiol.* 2005;26(10):841-843.
177. Lynn S, Toop J, Hanger C, Millar N. Norovirus outbreaks in a hospital setting: the role of infection control. *N Z Med J.* 2004;117(1189):U771.
178. Khanna N, Goldenberger D, Graber P, Battegay M, Widmer AF. Gastroenteritis outbreak with norovirus in a Swiss university hospital with a newly identified virus strain. *J Hosp Infect.* 2003;55(2):131-136.
179. McCall J, Smithson R. Rapid response and strict control measures can contain a hospital outbreak of Norwalk-like virus. *Commun Dis Public Health.* 2002;5(3):243-246.
180. Milazzo A, Tribe IG, Ratcliff R, Doherty C, Higgins G, Givney R. A large, prolonged outbreak of human calicivirus infection linked to an aged-care facility. *Commun Dis Intell.* 2002;26(2):261-264.
181. Miller M, Carter L, Scott K, Millard G, Lynch B, Guest C. Norwalk-like virus outbreak in Canberra: implications for infection control in aged care facilities. *Commun Dis Intell.* 2002;26(4):555-561.
182. Hoyle J. Managing the challenge of an acute gastroenteritis outbreak caused by a Norwalk-like virus in a 239 bed long-term care facility. *Aust Infect Control.* 2001;6(4):128-133.
183. Russo PL, Spelman DW, Harrington GA, et al. Concise communications. Hospital outbreak of Norwalk-like virus. *Infect Control Hosp Epidemiol.* 1997;18(8):576-579.
184. Stevenson P, McCann R, Duthie R, Glew E, Ganguli L. A hospital outbreak due to Norwalk virus. *J Hosp Infect.* 1994;26(4):261-272.
185. Hudson JB, Sharma M, Petric M. Inactivation of Norovirus by ozone gas in conditions relevant to healthcare. *J Hosp Infect.* 2007;66(1):40-45.
186. Park GW, Boston DM, Kase JA, Sampson MN, Sobsey MD. Evaluation of liquid- and fog-based application of Sterilox hypochlorous acid solution for surface inactivation of human norovirus. *Appl Environ Microbiol.* 2007;73(14):4463-4468.
187. Poschetto LF, Ike A, Papp T, Mohn U, Bohm R, Marschang RE. Comparison of the sensitivities of noroviruses and feline calicivirus to chemical disinfection under field-like conditions. *Appl Environ Microbiol.* 2007;73(17):5494-5500.
188. Jimenez L, Chiang M. Virucidal activity of a quaternary ammonium compound disinfectant against feline calicivirus: a surrogate for norovirus. *Am J Infect Control.* 2006;34(5):269-273.
189. Kramer A, Galabov AS, Sattar SA, et al. Virucidal activity of a new hand disinfectant with reduced ethanol content: comparison with other alcohol-based formulations. *J Hosp Infect.* 2006;62(1):98-106.
190. Malik YS, Allwood PB, Hedberg CW, Goyal SM. Disinfection of fabrics and carpets artificially contaminated with calicivirus: relevance in institutional and healthcare centres. *J Hosp Infect.* 2006;63(2):205-210.
191. Malik YS, Maherchandani S, Goyal SM. Comparative efficacy of ethanol and isopropanol against feline calicivirus, a norovirus surrogate. *Am J Infect Control.* 2006;34(1):31-35.
192. Malik YS, Goyal SM. Virucidal efficacy of sodium bicarbonate on a food contact surface against feline calicivirus, a norovirus surrogate. *International Journal of Food Microbiology.* 2006;109(1/2):160-163.
193. Kampf G, Grothier D, Steinmann J. Efficacy of three ethanol-based hand rubs against feline calicivirus, a surrogate virus for norovirus. *J Hosp Infect.* 2005;60(2):144-149.
194. Barker J, Vipond IB, Bloomfield SF. Effects of cleaning and disinfection in reducing the spread of Norovirus contamination via environmental surfaces. *J Hosp Infect.* 2004;58(1):42-49.

195. Duizer E, Bijkerk P, Rockx B, De Groot A, Twisk F, Koopmans M. Inactivation of caliciviruses. *Appl Environ Microbiol.* 2004;70(8):4538-4543.
196. Gehrke C, Steinmann J, Goroncy-Bermes P. Inactivation of feline calicivirus, a surrogate of norovirus (formerly Norwalk-like viruses), by different types of alcohol in vitro and in vivo. *J Hosp Infect.* 2004;56(1):49-55.
197. Lin CM, Wu FM, Kim HK, Doyle MP, Michael BS, Williams LK. A comparison of hand washing techniques to remove *Escherichia coli* and caliciviruses under natural or artificial fingernails. *J Food Prot.* 2003;66(12):2296-2301.
198. Nuanualsuwan S, Mariam T, Himathongkham S, Cliver DO. Ultraviolet inactivation of feline calicivirus, human enteric viruses and coliphages. *Photochemistry and Photobiology.* 2002;76(4):406-410.
199. Gulati BR, Allwood PB, Hedberg CW, Goyal SM. Efficacy of commonly used disinfectants for the inactivation of calicivirus on strawberry, lettuce, and a food-contact surface. *J Food Prot.* 2001;64(9):1430-1434.
200. Doultree JC, Druce JD, Birch CJ, Bowden DS, Marshall JA. Inactivation of feline calicivirus, a Norwalk virus surrogate. *J Hosp Infect.* 1999;41(1):51-57.
201. Shin GA, Sobsey MD. Reduction of norwalk virus, poliovirus 1 and coliphage MS2 by monochloramine disinfection of water. *Water Science and Technology.* 1998;38(12):151-154.
202. Rossignol JF, El-Gohary YM. Nitazoxanide in the treatment of viral gastroenteritis: a randomized double-blind placebo-controlled clinical trial. *Aliment Pharmacol Ther.* 2006;24(10):1423-1430.
203. Gustafson TL, Kobylik B, Hutcheson RH, Schaffner W. Protective effect of anticholinergic drugs and psyllium in a nosocomial outbreak of Norwalk gastroenteritis. *J Hosp Infect.* 1983;4(4):367-374.
204. Yoder J. Roberts V. Craun GF. Hill V. Hicks LA. Alexander NT. Radke V. Calderon RL. Hlavsa MC. Beach MJ. Roy SL. Centers for Disease Control and Prevention (CDC). Surveillance for waterborne disease and outbreaks associated with drinking water and water not intended for drinking--United States, 2005-2006. *Morb Mortal Wkly Rep Surveill Summ.* 2008;57(9):39-62.
205. Cooper E, Blamey S. A norovirus gastroenteritis epidemic in a long-term-care facility. *Infect Control Hosp Epidemiol.* 2005;26(3):256-258.
206. Russo PL, Spelman DW, Harrington GA, et al. Hospital outbreak of Norwalk-like virus. *Infect Control Hosp Epidemiol.* 1997;18(8):576-579.
207. Centers for Disease Control and Prevention (CDC). Multisite outbreak of norovirus associated with a franchise restaurant--Kent County, Michigan, May 2005. *MMWR.* 2006;55(14):395-397.

The recommendations in this guideline for Ebola Virus Disease has been superseded by [CDC's Infection Prevention and Control Recommendations for Hospitalized Patients with Known or Suspected Ebola Virus Disease in U.S. Hospitals](#) and by [CDC's Interim Guidance for Environmental Infection Control in Hospitals for Ebola Virus](#) issued on August 1, 2014.

This information is on pages [12](#), [13](#), [113](#) and [124](#).

[Click here](#) for current information on how Ebola virus is transmitted.

Guidelines for Environmental Infection Control in Health-Care Facilities

**Recommendations of CDC and the Healthcare Infection Control
Practices Advisory Committee (HICPAC)**

**U.S. Department of Health and Human Services
Centers for Disease Control and Prevention (CDC)
Atlanta, GA 30333**

2003

Suggested Citations:

Available from the CDC Internet Site:

The full-text version of the guidelines appears as a web-based document at the CDC's Division of Healthcare Quality Promotion's Internet site at:
www.cdc.gov/ncidod/hip/enviro/guide.htm

The full-text version of the guidelines should be cited when reference is made primarily to material in Parts I and IV. The print version of the guidelines appears as:

Sehulster LM, Chinn RYW, Arduino MJ, Carpenter J, Donlan R, Ashford D, Besser R, Fields B, McNeil MM, Whitney C, Wong S, Juranek D, Cleveland J. Guidelines for environmental infection control in health-care facilities. Recommendations from CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). Chicago IL; American Society for Healthcare Engineering/American Hospital Association; 2004.

Part II of these guidelines appeared in the CDC's "Morbidity and Mortality Weekly Report:"

Centers for Disease Control and Prevention. Guidelines for environmental infection control in health-care facilities: recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). MMWR 2003; 52 (No. RR-10): 1–48.

Updates to the Part II recommendations also appeared in the MMWR in 2003 as "Errata: Vol. 52 (No. RR-10)" (MMWR Vol. 52 [42]: 1025–6) on October 24, 2003 and as a "Notice to Readers" scheduled to appear in February 2004. The full-text version of these guidelines (this document) incorporates these updates.

Centers for Disease Control and Prevention Healthcare Infection Control Practices Advisory Committee (HICPAC)

Guidelines for Environmental Infection Control in Health-Care Facilities

Abstract

Background:

Although the environment serves as a reservoir for a variety of microorganisms, it is rarely implicated in disease transmission except in the immunocompromised population. Inadvertent exposures to environmental opportunistic pathogens (e.g., *Aspergillus* spp. and *Legionella* spp.) or airborne pathogens (e.g., *Mycobacterium tuberculosis* and varicella-zoster virus) may result in infections with significant morbidity and/or mortality. Lack of adherence to established standards and guidance (e.g., water quality in dialysis, proper ventilation for specialized care areas such as operating rooms, and proper use of disinfectants) can result in adverse patient outcomes in health-care facilities.

Objective:

The objective is to develop an environmental infection-control guideline that reviews and reaffirms strategies for the prevention of environmentally-mediated infections, particularly among health-care workers and immunocompromised patients. The recommendations are evidence-based whenever possible.

Search Strategies:

The contributors to this guideline reviewed predominantly English-language articles identified from MEDLINE literature searches, bibliographies from published articles, and infection-control textbooks.

Criteria for Selecting Citations and Studies for This Review:

Articles dealing with outbreaks of infection due to environmental opportunistic microorganisms and epidemiological- or laboratory experimental studies were reviewed. Current editions of guidelines and standards from organizations (i.e., American Institute of Architects [AIA], Association for the Advancement of Medical Instrumentation [AAMI], and American Society of Heating, Refrigeration, and Air-Conditioning Engineers [ASHRAE]) were consulted. Relevant regulations from federal agencies (i.e., U.S. Food and Drug Administration [FDA]; U.S. Department of Labor, Occupational Safety and Health Administration [OSHA]; U.S. Environmental Protection Agency [EPA]; and U.S. Department of Justice) were reviewed. Some topics did not have well-designed, prospective studies nor reports of outbreak investigations. Expert opinions and experience were consulted in these instances.

Types of Studies:

Reports of outbreak investigations, epidemiological assessment of outbreak investigations with control strategies, and *in vitro* environmental studies were assessed. Many of the recommendations are derived

from empiric engineering concepts and reflect industry standards. A few of the infection-control measures proposed cannot be rigorously studied for ethical or logistical reasons.

Outcome Measures:

Infections caused by the microorganisms described in this guideline are rare events, and the effect of these recommendations on infection rates in a facility may not be readily measurable. Therefore, the following steps to measure performance are suggested to evaluate these recommendations:

1. Document whether infection-control personnel are actively involved in all phases of a health-care facility's demolition, construction, and renovation. Activities should include performing a risk assessment of the necessary types of construction barriers, and daily monitoring and documenting of the presence of negative airflow within the construction zone or renovation area.
2. Monitor and document daily the negative airflow in airborne infection isolation rooms (AII) and positive airflow in protective environment rooms (PE), especially when patients are in these rooms.
3. Perform assays at least once a month by using standard quantitative methods for endotoxin in water used to reprocess hemodialyzers, and for heterotrophic, mesophilic bacteria in water used to prepare dialysate and for hemodialyzer reprocessing.
4. Evaluate possible environmental sources (e.g., water, laboratory solutions, or reagents) of specimen contamination when nontuberculous mycobacteria (NTM) of unlikely clinical importance are isolated from clinical cultures. If environmental contamination is found, eliminate the probable mechanisms.
5. Document policies to identify and respond to water damage. Such policies should result in either repair and drying of wet structural materials within 72 hours, or removal of the wet material if drying is unlikely within 72 hours.

Main Results:

Infection-control strategies and engineering controls, when consistently implemented, are effective in preventing opportunistic, environmentally-related infections in immunocompromised populations. Adherence to proper use of disinfectants, proper maintenance of medical equipment that uses water (e.g., automated endoscope reprocessors and hydrotherapy equipment), water-quality standards for hemodialysis, and proper ventilation standards for specialized care environments (i.e., airborne infection isolation [AII], protective environment [PE], and operating rooms [ORs]), and prompt management of water intrusion into facility structural elements will minimize health-care-associated infection risks and reduce the frequency of pseudo-outbreaks. Routine environmental sampling is not advised except in the few situations where sampling is directed by epidemiologic principles and results can be applied directly to infection control decisions, and for water quality determinations in hemodialysis.

Reviewers' Conclusions:

Continued compliance with existing environmental infection control measures will decrease the risk of health-care-associated infections among patients, especially the immunocompromised, and health-care workers.

**Centers for Disease Control and Prevention
Healthcare Infection Control Practices Advisory Committee (HICPAC)**

***Guidelines for Environmental Infection Control in
Health-Care Facilities***

Table of Contents

Executive Summary	1
Part I. Background Information: Environmental Infection Control in Health-Care Facilities	3
A. Introduction	3
B. Key Terms Used in this Guideline	5
C. Air	6
1. Modes of Transmission of Airborne Diseases	6
2. Airborne Infectious Diseases in Health-Care Facilities	7
3. Heating, Ventilation, and Air Conditioning Systems in Health-Care Facilities	13
4. Construction, Renovation, Remediation, Repair, and Demolition	21
5. Environmental Infection-Control Measures for Special Health-Care Settings.....	34
6. Other Aerosol Hazards in Health-Care Facilities	40
D. Water	40
1. Modes of Transmission of Waterborne Diseases	40
2. Waterborne Infectious Diseases in Health-Care Facilities	41
3. Water Systems in Health-Care Facilities	46
4. Strategies for Controlling Waterborne Microbial Contamination	53
5. Cooling Towers and Evaporative Condensers.....	57
6. Dialysis Water Quality and Dialysate.....	59
7. Ice Machines and Ice	65
8. Hydrotherapy Tanks and Pools.....	67
9. Miscellaneous Medical/Dental Equipment Connected to Main Water Systems	69
E. Environmental Services	71
1. Principles of Cleaning and Disinfecting Environmental Surfaces.....	71
2. General Cleaning Strategies for Patient-Care Areas.....	74
3. Cleaning Strategies for Spills of Blood and Body Substances	77
4. Carpeting and Cloth Furnishings	78
5. Flowers and Plants in Patient-Care Areas	80
6. Pest Control	81
7. Special Pathogen Concerns.....	82
F. Environmental Sampling	88
1. General Principles: Microbiologic Sampling of the Environment	88
2. Air Sampling.....	89
3. Water Sampling	94
4. Environmental Surface Sampling.....	95
G. Laundry and Bedding	98
1. General Information	98
2. Epidemiology and General Aspects of Infection Control.....	98
3. Collecting, Transporting, and Sorting Contaminated Textiles and Fabrics.....	99

4. Parameters of the Laundry Process	100
5. Special Laundry Situations.....	102
6. Surgical Gowns, Drapes, and Disposable Fabrics.....	103
7. Antimicrobial-Impregnated Articles and Consumer Items Bearing Antimicrobial Labeling ..	103
8. Standard Mattresses, Pillows, and Air-Fluidized Beds	104
H. Animals in Health-Care Facilities	105
1. General Information	105
2. Animal-Assisted Activities, Animal-Assisted Therapy, and Resident Animals	106
3. Service Animals	108
4. Animals as Patients in Human Health-Care Facilities	110
5. Research Animals in Health-Care Facilities	111
I. Regulated Medical Waste	112
1. Epidemiology	112
2. Categories of Medical Waste	112
3. Management of Regulated Medical Waste in Health-Care Facilities	113
4. Treatment of Regulated Medical Waste.....	113
5. Discharging Blood, Fluids to Sanitary Sewers or Septic Tanks.....	116
6. Medical Waste and CJD	116
Part II. Recommendations for Environmental Infection Control in Health-Care Facilities	117
A. Rationale for Recommendations	117
B. Rating Categories.....	117
C. Recommendations—Air	118
D. Recommendations—Water	125
E. Recommendations—Environmental Services	133
F. Recommendations—Environmental Sampling	138
G. Recommendations—Laundry and Bedding	138
H. Recommendations—Animals in Health-Care Facilities.....	141
I. Recommendations—Regulated Medical Waste.....	143
Part III. References.....	145
Part IV. Appendices.....	201
Appendix A. Glossary of Terms	201
Appendix B. Air	210
1. Airborne Contaminant Removal	210
2. Air Sampling for Aerosols Containing Legionellae.....	210
3. Calculation of Air Sampling Results.....	211
4. Ventilation Specifications for Health-Care Facilities.....	212
Appendix C. Water	220
1. Biofilms	220
2. Water and Dialysate Sampling Strategies in Dialysis.....	222
3. Water Sampling Strategies and Culture Techniques for Detecting Legionellae	223
4. Procedure for Cleaning Cooling Towers and Related Equipment	225
5. Maintenance Procedures Used to Decrease Survival and Multiplications of <i>Legionella</i> spp. in Potable-Water Distribution Systems	227
Appendix D. Insects and Microorganisms.....	228
Appendix E. Information Resources.....	229
Appendix F. Areas of Future Research	230
Index—Parts I and IV	231

List of Figures, Boxes, and Tables

Figures

Figure 1. Diagram of a ventilation system	14
Figure 2. Example of positive-pressure room control for protection from airborne environmental microbes (PE)	35
Figure 3. Example of negative-pressure room control for airborne infection isolation (AII)	36
Figure 4. Example of airborne infection isolation (AII) room with anteroom and neutral anteroom	37
Figure 5. Diagram of a typical air conditioning (induced draft) cooling tower	58
Figure 6. Dialysis water treatment system	60

Boxes

Box 1. Environmental infection control: performance measures	2
Box 2. Eight criteria for evaluating the strength of evidence for environmental sources of infection	4
Box 3. Chain of infection components	4
Box 4. Suggested members and functions of a multi-disciplinary coordination team for construction, renovation, repair, and demolition projects	24
Box 5. Construction design and function considerations for environmental infection control	25
Box 6. Unresolved issues associated with microbiologic air sampling	28
Box 7. Construction/repair projects that require barrier structures	32
Box 8. Strategy for managing TB patients and preventing airborne transmission in operating rooms	39
Box 9. Recovery and remediation measures for water-related emergencies	52
Box 10. Contingency planning for flooding	53
Box 11. Steps in an epidemiologic investigation for legionellosis	56
Box 12. General steps for cleaning and maintaining ice machines, dispensers, and storage chests	66
Box 13. Preliminary concerns for conducting air sampling	90
Box 14. Selecting an air sampling device	93
Box 15. Undertaking environmental-surface sampling	95
Box C.1. Potential sampling sites for <i>Legionella</i> spp. in health-care facilities	224
Box C.2. Procedures for collecting and processing environmental specimens for <i>Legionella</i> spp.	225

Tables

Table 1. Clinical and epidemiologic characteristics of aspergillosis	7
Table 2. Environmental fungal pathogens: entry into and contamination of the health-care facility	8
Table 3. Clinical and epidemiologic characteristics of tuberculosis (TB)	10
Table 4. Microorganisms associated with airborne transmission	13
Table 5. Filtration methods	15
Table 6. Engineered specifications for positive- and negative pressure rooms	19
Table 7. Ventilation hazards in health-care facilities that may be associated with increased potential of airborne disease transmission	22
Table 8. Strategies to reduce dust and moisture intrusion during external demolition and construction	30

Table 9. Infection-control measures for internal construction and repair projects	32
Table 10. Summary of ventilation specifications in selected areas of health-care facilities.....	39
Table 11. Clinical and epidemiologic characteristics of legionellosis/Legionnaires disease.....	41
Table 12. <i>Pseudomonas aeruginosa</i> infections in health-care facilities	42
Table 13. Other gram-negative bacteria associated with water and moist environments	43
Table 14. Nontuberculous mycobacteria—environmental vehicles	45
Table 15. Water and point-of-use fixtures as sources and reservoirs of waterborne pathogens	47
Table 16. Water demand in health-care facilities during water disruption emergencies	50
Table 17. Additional infection-control measures to prevent exposure of high-risk patients to waterborne pathogens.....	57
Table 18. Microbiologic limits for hemodialysis fluids.....	62
Table 19. Factors influencing microbial contamination in hemodialysis systems.....	63
Table 20. Microorganisms and their sources in ice and ice machines	65
Table 21. Infections associated with use of hydrotherapy equipment	67
Table 22. Levels of disinfection by type of microorganism	72
Table 23. Air sampling methods and examples of equipment	91
Table 24. Examples of eluents and diluents for environmental-surface sampling.....	96
Table 25. Methods of environmental-surface sampling.....	97
Table 26. Examples of diseases associated with zoonotic transmission.....	105
Table 27. Microorganisms and biologicals identified as select agents	115
Table B.1. Air changes/hour (ACH) and time required for airborne-contaminant removal efficiencies of 99% and 99.9%	210
Table B.2. Ventilation requirements for areas affecting patient care in hospitals and outpatient facilities	212
Table B.3. Pressure relationships and ventilation of certain areas of nursing facilities.....	217
Table B.4. Filter efficiencies for central ventilation and air conditioning systems in general hospitals	219
Table B.5. Filter efficiencies for central ventilation and air conditioning systems in outpatient facilities	219
Table B.6. Filter efficiencies for central ventilation and air conditioning systems in nursing facilities	220
Table B.7. Filter efficiencies for central ventilation and air conditioning systems in psychiatric hospitals	220
Table D.1. Microorganisms isolated from arthropods in health-care settings	228

List of Abbreviations Used in This Publication

AAA	animal-assisted activity
AAMI	Association for the Advancement of Medical Instrumentation
AAT	animal-assisted therapy
ACGIH	American Council of Governmental Industrial Hygienists
ACH	air changes per hour
ADA	Americans with Disabilities Act
AER	automated endoscope reprocessor
AFB	acid-fast bacilli
AHA	American Hospital Association
AHJ	authorities having jurisdiction
AIA	American Institute of Architects
AII	airborne infection isolation
AmB	amphotericin B
ANC	absolute neutrophil count
ANSI	American National Standards Institute
AORN	Association of periOperative Registered Nurses
ASHE	American Society for Healthcare Engineering
ASHRAE	American Society of Heating, Refrigeration, and Air-Conditioning Engineers
BCG	Bacille Calmette-Guérin
BCYE	buffered charcoal yeast extract medium
BHI	brain-heart infusion
BMBL	CDC/NIH publication “Biosafety in Microbiological and Biomedical Laboratories”
BOD	biological oxygen demand
BSE	bovine spongiform encephalopathy
BSL	biosafety level
C	Centigrade
CAPD	continuous ambulatory peritoneal dialysis
CCPD	continual cycling peritoneal dialysis
CMAD	count median aerodynamic diameter
CDC	U.S. Centers for Disease Control and Prevention
CFR	Code of Federal Regulations
CFU	colony-forming unit
CJD	Creutzfeldt-Jakob disease
cm	centimeter
CMS	U.S. Centers for Medicare and Medicaid Services
CPL	compliance document (OSHA)
CT/EC	cooling tower/evaporative condenser
DFA	direct fluorescence assay; direct fluorescent antibody
DHHS	U.S. Department of Health and Human Services
DHBV	duck hepatitis B virus
DNA	deoxyribonucleic acid
DOP	dioctylphthalate
DOT	U.S. Department of Transportation
EC	environment of care (JCAHO)
ELISA	enzyme-linked immunosorbent assay
EPA	U.S. Environmental Protection Agency
ESRD	end-stage renal disease

EU	endotoxin unit
F	Fahrenheit
FDA	U.S. Food and Drug Administration
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FRC	free residual chlorine
ft	foot (feet)
FTC	U.S. Federal Trade Commission
GISA	glycopeptide intermediate resistant <i>Staphylococcus aureus</i>
HBV	hepatitis B virus
HCV	hepatitis C virus
HEPA	high efficiency particulate air
HICPAC	Healthcare Infection Control Practices Advisory Committee
HIV	human immunodeficiency virus
HPV	human papilloma virus
HSCT	hematopoietic stem cell transplant
HVAC	heating, ventilation, air conditioning
ICRA	infection control risk assessment
ICU	intensive care unit
ID₅₀	50% median infectious dose
IPD	intermittent peritoneal dialysis
JCAHO	Joint Commission on Accreditation of Healthcare Organizations
kg	kilogram
L	liter
MAC	<i>Mycobacterium avium</i> complex; also used to denote MacConkey agar
MDRO	multiple-drug resistant organism
MIC	minimum inhibitory concentration
µm	micrometer; micron
mL	milliliter
min	minute
mg	milligram
MMAD	mass median aerodynamic diameter
MMWR	“Morbidity and Mortality Weekly Report”
MRSA	methicillin-resistant <i>Staphylococcus aureus</i>
MSDS	material safety data sheet
N	Normal
NaCl	sodium chloride
NaOH	sodium hydroxide
NCID	National Center for Infectious Diseases
NCCDPHP	National Center for Chronic Disease Prevention and Health Promotion
NCCLS	National Committee for Clinical Laboratory Standards
ng	nanogram
NICU	neonatal intensive care unit
NIH	U.S. National Institutes of Health
NIOSH	National Institute for Occupational Safety and Health
nm	nanometer
NNIS	National Nosocomial Infection Surveillance
NTM	nontuberculous mycobacteria
OPL	on-premises laundry
OSHA	U.S. Occupational Safety and Health Administration
Pa	Pascal
PCP	<i>Pneumocystis carinii</i> pneumonia

PCR	polymerase chain reaction
PD	peritoneal dialysis
PE	protective environment
PEL	permissible exposure limit
PPE	personal protective equipment
ppm	parts per million
PVC	polyvinylchloride
RAPD	randomly amplified polymorphic DNA
RODAC	replicate organism direct agar contact
RSV	respiratory syncytial virus
RO	reverse osmosis
SARS	severe acute respiratory syndrome
SARS-CoV	SARS coronavirus
sec	second
spp	species
SSI	surgical site infection
TB	tuberculosis
TLV®-TWA	threshold limit value-time weighted average
TSA	tryptic soy agar
TSB	tryptic soy broth
TSE	transmissible spongiform encephalopathy
U.S.	United States
USC	United States Code
USDA	U.S. Department of Agriculture
USPS	U.S. Postal Service
UV	ultraviolet
UVGI	ultraviolet germicidal irradiation
VAV	variable air ventilation
vCJD	variant Creutzfeldt-Jakob disease
VISA	vancomycin intermediate resistant <i>Staphylococcus aureus</i>
VRE	vancomycin-resistant <i>Enterococcus</i>
VRSA	vancomycin-resistant <i>Staphylococcus aureus</i>
v/v	volume/volume
VZV	varicella-zoster virus

Note: Use of trade names and commercial sources is for identification only and does not imply endorsement by the U.S. Department of Health and Human Services. References to non-CDC sites on the Internet are provided as a service to the reader and does not constitute or imply endorsement of these organization s or their programs by CDC or the U.S. Department of Health and Human Services. CDC is not responsible for the content of pages found at these sites.

The following CDC staff member and HICPAC member prepared this report:

Lynne Schulster, PhD
Division of Healthcare Quality Promotion
National Center for Infectious Diseases

Raymond Y.W. Chinn, MD
HICPAC Advisor
Sharp Memorial Hospital
San Diego, California

Disclosure of Relationship: Raymond Y.W. Chinn is a private-practice physician and salaried employee of Sharp Memorial Hospital in San Diego, California. Dr. Chinn received no research funds from commercial sources either directly, or indirectly through awards made to the hospital, before or during the development of these guidelines.

Contributions were made by the following CDC staff members:

Matthew Arduino, DrPH
Joe Carpenter, PE
Rodney Donlan, PhD
Lynne Schulster, PhD
Division of Healthcare Quality Promotion
National Center for Infectious Diseases

David Ashford, DVM, Dsc, MPH
Richard Besser, MD
Barry Fields, PhD
Michael M. McNeil, MBBS, MPH
Cynthia Whitney, MD, MPH
Stephanie Wong, DMV, MPH
Division of Bacterial and Mycotic Diseases
National Center for Infectious Diseases

Dennis Juranek, DVM, MSC
Division of Parasitic Diseases
National Center for Infectious Diseases

Jennifer Cleveland, DDS, MPH
Division of Oral Health
National Center for Chronic Disease Prevention and Health Promotion

***In collaboration with the Healthcare Infection Control Practices Advisory Committee
(HICPAC)***

HICPAC Members, February 2002

Robert A. Weinstein, MD
Chair
Cook County Hospital
Chicago, IL

Jane D. Siegel, MD
Co-Chair
University of Texas Southwestern Medical Center
Dallas, TX

Michele L. Pearson, MD
Executive Secretary
Centers for Disease Control and Prevention
Atlanta, GA

Raymond Y.W. Chinn, MD
Sharp Memorial Hospital
San Diego, CA

Alfred DeMaria, Jr., MD
Massachusetts Department of Public Health
Jamaica Plain, MA

Elaine L. Larson, RN, PhD
Columbia University School of Nursing
New York, NY

James T. Lee, MD, PhD
University of Minnesota
VA Medical Center
St. Paul, MN

Ramon E. Moncada, MD
Coronado Physician's Medical Center
Coronado, CA

William A. Rutala, PhD, MPH, CIC
University of North Carolina School of
Medicine
Chapel Hill, NC

William E. Scheckler, MD
University of Wisconsin Medical School
Madison, WI

Beth H. Stover, RN, CIC
Kosair Children's Hospital
Louisville, KY

Marjorie A. Underwood, RN, BSN, CIC
Mt. Diablo Medical Center
Concord, CA

Liaison Members

Loretta L. Fauerbach, MS, CIC
Association for Professionals in Infection
Control and Epidemiology (APIC)
Washington, DC

Sandra L. Fitzler, RN
American Health Care Association
Washington, DC

Dorothy M. Fogg, RN, BSN, MA
Association of periOperative Registered
Nurses (AORN)
Denver, CO

Stephen F. Jencks, MD, MPH
U.S. Centers for Medicare and Medicaid
Services
Baltimore, MD

Chiu S. Lin, PhD
U.S. Food and Drug Administration
Rockville, MD

James P. Steinberg, MD
Society for Healthcare Epidemiology of America,
Inc. (SHEA)
Atlanta, GA

Liaison Members (continued)

Michael L. Tapper, MD
Advisory Committee for the Elimination of
Tuberculosis (ACET)
New York, NY

Expert Reviewers

Trisha Barrett, RN, MBA, CIC
Alta Bates Medical Center
Berkeley, CA

Michael Berry
University of North Carolina
Chapel Hill, NC

Walter W. Bond, MS
RCSA, Inc.
Lawrenceville, GA

Douglas Erickson, FASHE
American Society for Healthcare
Engineering (ASHE)
Park Ridge, IL

Richard Miller, MD
University of Louisville School of Medicine
Louisville, KY

Gina Pugliese, RN, MS
Premier Safety Institute
Oak Brook, IL

James D. Scott, PE
Michigan Department of Consumer and
Industry Services
Lansing, MI

Dale Woodin
American Society for Healthcare Engineering
(ASHE)
Chicago, IL

Judene Bartley, MS, MPH, CIC
Epidemiology Consulting Services, Inc.
Beverly Hills, MI

Col. Nancy Bjerke, BSN, MA, MEd, MPH, CIC
(USAF, Retired)
Infection Control Associates (ICA)
San Antonio, TX

Cheryl Carter, RN
University of Iowa Health Center
Iowa City, IA

Martin S. Favero, PhD
Advanced Sterilization Products, Johnson and
Johnson
Irvine, CA

Col. Shannon E. Mills, DDS
HQ USAF / Surgeon General Detail
Bolin AFB, DC

Craig E. Rubens, MD, PhD
Children's Hospital and Medical Center
Seattle, WA

Andrew J. Streifel, MPH, REHS
University of Minnesota
Minneapolis, MN

Executive Summary

The *Guidelines for Environmental Infection Control in Health-Care Facilities* is a compilation of recommendations for the prevention and control of infectious diseases that are associated with health-care environments. This document a) revises multiple sections from previous editions of the Centers for Disease Control and Prevention [CDC] document titled *Guideline for Handwashing and Hospital Environmental Control*;^{1,2} b) incorporates discussions of air and water environmental concerns from CDC's *Guideline for the Prevention of Nosocomial Pneumonia*;³ c) consolidates relevant environmental infection-control measures from other CDC guidelines;⁴⁻⁹ and d) includes two topics not addressed in previous CDC guidelines — infection-control concerns related to animals in health-care facilities and water quality in hemodialysis settings.

Part I of this report, *Background Information: Environmental Infection Control in Health-Care Facilities*, provides a comprehensive review of the scientific literature. Attention is given to engineering and infection-control concerns during construction, demolition, renovation, and repairs of health-care facilities. Use of an infection-control risk assessment is strongly supported before the start of these or any other activities expected to generate dust or water aerosols. Also reviewed in Part I are infection-control measures used to recover from catastrophic events (e.g., flooding, sewage spills, loss of electricity and ventilation, and disruption of the water supply) and the limited effects of environmental surfaces, laundry, plants, animals, medical wastes, cloth furnishings, and carpeting on disease transmission in healthcare facilities.

Part II of this guideline, *Recommendations for Environmental Infection Control in Health-Care Facilities*, outlines environmental infection control in health-care facilities, describing measures for preventing infections associated with air, water, and other elements of the environment. These recommendations represent the views of different divisions within CDC's National Center for Infectious Diseases (NCID) (e.g., the Division of Healthcare Quality Promotion [DHQP] and the Division of Bacterial and Mycotic Diseases [DBMD]) and the consensus of the Healthcare Infection Control Practices Advisory Committee (HICPAC), a 12-member group that advises CDC on concerns related to the surveillance, prevention, and control of health-care-associated infections, primarily in U.S. health-care facilities.¹⁰ In 1999, HICPAC's infection-control focus was expanded from acute-care hospitals to all venues where health care is provided (e.g., outpatient surgical centers, urgent care centers, clinics, outpatient dialysis centers, physicians' offices, and skilled nursing facilities). The topics addressed in this guideline are applicable to the majority of health-care venues in the United States. This document is intended for use primarily by infection-control professionals (ICPs), epidemiologists, employee health and safety personnel, information system specialists, administrators, engineers, facility managers, environmental service professionals, and architects for health-care facilities.

Key recommendations include a) infection-control impact of ventilation system and water system performance; b) establishment of a multidisciplinary team to conduct infection-control risk assessment; c) use of dust-control procedures and barriers during construction, repair, renovation, or demolition; d) environmental infection-control measures for special care areas with patients at high risk; e) use of airborne particle sampling to monitor the effectiveness of air filtration and dust-control measures; f) procedures to prevent airborne contamination in operating rooms when infectious tuberculosis [TB] patients require surgery; g) guidance regarding appropriate indications for routine culturing of water as part of a comprehensive control program for legionellae; h) guidance for recovering from water system disruptions, water leaks, and natural disasters [e.g., flooding]; i) infection-control concepts for equipment that uses water from main lines [e.g., water systems for hemodialysis, ice machines, hydrotherapy equipment, dental unit water lines, and automated endoscope reprocessors]; j) environmental surface cleaning and disinfection strategies with respect to antibiotic-resistant

microorganisms; k) infection-control procedures for health-care laundry; l) use of animals in health care for activities and therapy; m) managing the presence of service animals in health-care facilities; n) infection-control strategies for when animals receive treatment in human health-care facilities; and o) a call to reinstate the practice of inactivating amplified cultures and stocks of microorganisms on-site during medical waste treatment.

Whenever possible, the recommendations in Part II are based on data from well-designed scientific studies. However, certain of these studies were conducted by using narrowly defined patient populations or for specific health-care settings (e.g., hospitals versus long-term care facilities), making generalization of findings potentially problematic. Construction standards for hospitals or other health-care facilities may not apply to residential home-care units. Similarly, infection-control measures indicated for immunosuppressed patient care are usually not necessary in those facilities where such patients are not present. Other recommendations were derived from knowledge gained during infectious disease investigations in health-care facilities, where successful termination of the outbreak was often the result of multiple interventions, the majority of which cannot be independently and rigorously evaluated. This is especially true for construction situations involving air or water.

Other recommendations are derived from empiric engineering concepts and may reflect an industry standard rather than an evidence-based conclusion. Where recommendations refer to guidance from the American Institute of Architects (AIA), the statements reflect standards intended for new construction or renovation. Existing structures and engineered systems are expected to be in continued compliance with the standards in effect at the time of construction or renovation. Also, in the absence of scientific confirmation, certain infection-control recommendations that cannot be rigorously evaluated are based on a strong theoretical rationale and suggestive evidence. Finally, certain recommendations are derived from existing federal regulations. The references and the appendices comprise Parts III and IV of this document, respectively.

Infections caused by the microorganisms described in these guidelines are rare events, and the effect of these recommendations on infection rates in a facility may not be readily measurable. Therefore, the following steps to measure performance are suggested to evaluate these recommendations (Box 1):

Box 1. Environmental infection control: performance measures

-
1. **Document whether infection-control personnel are actively involved in all phases of a health-care facility's demolition, construction, and renovation. Activities should include performing a risk assessment of the necessary types of construction barriers, and daily monitoring and documenting of the presence of negative airflow within the construction zone or renovation area.**
 2. **Monitor and document daily the negative airflow in airborne infection isolation (AII) rooms and positive airflow in protective environment (PE) rooms, especially when patients are in these rooms.**
 3. **Perform assays at least once a month by using standard quantitative methods for endotoxin in water used to reprocess hemodialyzers, and for heterotrophic and mesophilic bacteria in water used to prepare dialysate and for hemodialyzer reprocessing.**
 4. **Evaluate possible environmental sources (e.g., water, laboratory solutions, or reagents) of specimen contamination when nontuberculous mycobacteria (NTM) of unlikely clinical importance are isolated from clinical cultures. If environmental contamination is found, eliminate the probable mechanisms.**
 5. **Document policies to identify and respond to water damage. Such policies should result in either repair and drying of wet structural or porous materials within 72 hours, or removal of the wet material if drying is unlikely with 72 hours.**
-

Topics outside the scope of this document include a) noninfectious adverse events (e.g., sick building syndrome); b) environmental concerns in the home; c) home health care; d) bioterrorism; and e) health-care–associated foodborne illness. This document includes only limited discussion of a) handwashing/hand hygiene; b) standard precautions; and c) infection-control measures used to prevent instrument or equipment contamination during patient care (e.g., preventing waterborne contamination of nebulizers or ventilator humidifiers). These topics are mentioned only if they are important in minimizing the transfer of pathogens to and from persons or equipment and the environment. Although the document discusses principles of cleaning and disinfection as they are applied to maintenance of environmental surfaces, the full discussion of sterilization and disinfection of medical instruments and direct patient-care devices is deferred for inclusion in the *Guideline for Disinfection and Sterilization in Health-Care Facilities*, a document currently under development. Similarly, the full discussion of hand hygiene is available as the *Guideline for Hand Hygiene in Health-Care Settings: Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force*. Where applicable, the *Guidelines for Environmental Infection Control in Health-Care Facilities* are consistent in content to the drafts available as of October 2002 of both the revised *Guideline for Prevention of Health-Care–Associated Pneumonia* and *Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health-Care Facilities*.

This guideline was prepared by CDC staff members from NCID and the National Center for Chronic Disease Prevention and Health Promotion (NCCDPHP) and the designated HICPAC advisor. Contributors to this document reviewed predominantly English-language manuscripts identified from reference searches using the National Library of Medicine’s MEDLINE, bibliographies of published articles, and infection-control textbooks. Working drafts of the guideline were reviewed by CDC scientists, HICPAC committee members, and experts in infection control, engineering, internal medicine, infectious diseases, epidemiology, and microbiology. All recommendations in this guideline may not reflect the opinions of all reviewers.

Part I. Background Information: Environmental Infection Control in Health-Care Facilities

A. Introduction

The health-care environment contains a diverse population of microorganisms, but only a few are significant pathogens for susceptible humans. Microorganisms are present in great numbers in moist, organic environments, but some also can persist under dry conditions. Although pathogenic microorganisms can be detected in air and water and on fomites, assessing their role in causing infection and disease is difficult.¹¹ Only a few reports clearly delineate a “cause and effect” with respect to the environment and in particular, housekeeping surfaces.

Eight criteria are used to evaluate the strength of evidence for an environmental source or means of transmission of infectious agents (Box 2).^{11,12} Applying these criteria to disease investigations allows scientists to assess the contribution of the environment to disease transmission. An example of this application is the identification of a pathogen (e.g., vancomycin-resistant enterococci [VRE]) on an environmental surface during an outbreak. The presence of the pathogen does not establish its causal role; its transmission from source to host could be through indirect means (e.g., via hand transferral).¹¹ The surface, therefore, would be considered one of a number of potential reservoirs for the pathogen, but not the “de facto” source of exposure. An understanding of how infection occurs after exposure,

based on the principles of the “chain of infection,” is also important in evaluating the contribution of the environment to health-care–associated disease.¹³ All of the components of the “chain” must be operational for infection to occur (Box 3).

Box 2. Eight criteria for evaluating the strength of evidence for environmental sources of infection* +

1. **The organism can survive after inoculation onto the fomite.**
 2. **The organism can be cultured from in-use fomites.**
 3. **The organism can proliferate in or on the fomite.**
 4. **Some measure of acquisition of infection cannot be explained by other recognized modes of transmission.**
 5. **Retrospective case-control studies show an association between exposure to the fomite and infection.**
 6. **Prospective case-control studies may be possible when more than one similar type of fomite is in use.**
 7. **Prospective studies allocating exposure to the fomite to a subset of patients show an association between exposure and infection.**
 8. **Decontamination of the fomite results in the elimination of infection transmission.**
-

* These criteria are listed in order of strength of evidence.

+ Adapted from references 11 and 12.

Box 3. Chain of infection components*

1. **Adequate number of pathogenic organisms (dose)**
 2. **Pathogenic organisms of sufficient virulence**
 3. **A susceptible host**
 4. **An appropriate mode of transmission or transferal of the organism in sufficient number from source to host**
 5. **The correct portal of entry into the host**
-

* Adapted from reference 13.

The presence of the susceptible host is one of these components that underscores the importance of the health-care environment and opportunistic pathogens on fomites and in air and water. As a result of advances in medical technology and therapies (e.g., cytotoxic chemotherapy and transplantation medicine), more patients are becoming immunocompromised in the course of treatment and are therefore at increased risk for acquiring health-care–associated opportunistic infections. Trends in health-care delivery (e.g., early discharge of patients from acute care facilities) also are changing the distribution of patient populations and increasing the number of immunocompromised persons in non-acute-care hospitals. According to the American Hospital Association (AHA), in 1998, the number of hospitals in the United States totaled 6,021; these hospitals had a total of 1,013,000 beds,¹⁴ representing a 5.5% decrease in the number of acute-care facilities and a 10.2% decrease in the number of beds over the 5-year period 1994–1998.¹⁴ In addition, the total average daily number of patients receiving care in U.S. acute-care hospitals in 1998 was 662,000 (65.4%) – 36.5% less than the 1978 average of 1,042,000.¹⁴ As the number of acute-care hospitals declines, the length of stay in these facilities is concurrently decreasing, particularly for immunocompetent patients. Those patients remaining in acute-care facilities are likely to be those requiring extensive medical interventions who therefore are at high risk for opportunistic infection.

The growing population of severely immunocompromised patients is at odds with demands on the health-care industry to remain viable in the marketplace; to incorporate modern equipment, new diagnostic procedures, and new treatments; and to construct new facilities. Increasing numbers of health-care facilities are likely to be faced with construction in the near future as hospitals consolidate to reduce costs, defer care to ambulatory centers and satellite clinics, and try to create more “home-like” acute-care settings. In 1998, approximately 75% of health-care–associated construction projects focused on renovation of existing outpatient facilities or the building of such facilities;¹⁵ the number of projects associated with outpatient health care rose by 17% from 1998 through 1999.¹⁶ An aging population is also creating increasing demand for assisted-living facilities and skilled nursing centers. Construction of assisted-living facilities in 1998 increased 49% from the previous year, with 138 projects completed at a cost of \$703 million.¹⁶ Overall, from 1998 to 1999, health-care–associated construction costs increased by 28.5%, from \$11.56 billion to \$14.86 billion.¹⁶

Environmental disturbances associated with construction activities near health-care facilities pose airborne and waterborne disease threats risks for the substantial number of patients who are at risk for health-care–associated opportunistic infections. The increasing age of hospitals and other health-care facilities is also generating ongoing need for repair and remediation work (e.g., installing wiring for new information systems, removing old sinks, and repairing elevator shafts) that can introduce or increase contamination of the air and water in patient-care environments. Aging equipment, deferred maintenance, and natural disasters provide additional mechanisms for the entry of environmental pathogens into high-risk patient-care areas.

Architects, engineers, construction contractors, environmental health scientists, and industrial hygienists historically have directed the design and function of hospitals’ physical plants. Increasingly, however, because of the growth in the number of susceptible patients and the increase in construction projects, the involvement of hospital epidemiologists and infection-control professionals is required. These experts help make plans for building, maintaining, and renovating health-care facilities to ensure that the adverse impact of the environment on the incidence of health-care–associated infections is minimal. The following are examples of adverse outcomes that could have been prevented had such experts been involved in the planning process: a) transmission of infections caused by *Mycobacterium tuberculosis*, varicella-zoster virus (VZV), and measles (i.e., rubeola) facilitated by inappropriate air-handling systems in health-care facilities;⁶ b) disease outbreaks caused by *Aspergillus* spp.,^{17–19} *Mucoraceae*,²⁰ and *Penicillium* spp. associated with the absence of environmental controls during periods of health-care facility-associated construction;²¹ c) infections and/or colonizations of patients and staff with vancomycin-resistant *Enterococcus faecium* [VRE] and *Clostridium difficile* acquired indirectly from contact with organisms present on environmental surfaces in health-care facilities,^{22–25} and d) outbreaks and pseudoepidemics of legionellae,^{26, 27} *Pseudomonas aeruginosa*,^{28–30} and the nontuberculous mycobacteria (NTM)^{31, 32} linked to water and aqueous solutions used in health-care facilities. The purpose of this guideline is to provide useful information for both health-care professionals and engineers in efforts to provide a safe environment in which quality health care may be provided to patients. The recommendations herein provide guidance to minimize the risk for and prevent transmission of pathogens in the indoor environment.

B. Key Terms Used in this Guideline

Although Appendix A provides definitions for terms discussed in Part I, several terms that pertain to specific patient-care areas and patients who are at risk for health-care–associated opportunistic infections are presented here. Specific engineering parameters for these care areas are discussed more

fully in the text. **Airborne Infection Isolation (AII)** refers to the isolation of patients infected with organisms spread via airborne droplet nuclei $<5\ \mu\text{m}$ in diameter. This isolation area receives numerous air changes per hour (ACH) (≥ 12 ACH for new construction as of 2001; ≥ 6 ACH for construction before 2001), and is under negative pressure, such that the direction of the airflow is from the outside adjacent space (e.g., corridor) into the room. The air in an AII room is preferably exhausted to the outside, but may be recirculated provided that the return air is filtered through a high efficiency particulate air (HEPA) filter. The use of personal respiratory protection is also indicated for persons entering these rooms.

Protective Environment (PE) is a specialized patient-care area, usually in a hospital, with a positive airflow relative to the corridor (i.e., air flows from the room to the outside adjacent space). The combination of HEPA filtration, high numbers of air changes per hour (≥ 12 ACH), and minimal leakage of air into the room creates an environment that can safely accommodate patients who have undergone allogeneic hematopoietic stem cell transplant (HSCT).

Immunocompromised patients are those patients whose immune mechanisms are deficient because of immunologic disorders (e.g., human immunodeficiency virus [HIV] infection, congenital immune deficiency syndrome, chronic diseases [such as diabetes, cancer, emphysema, and cardiac failure]) or immunosuppressive therapy (e.g., radiation, cytotoxic chemotherapy, anti-rejection medication, and steroids). Immunocompromised patients who are identified as **high-risk patients** have the greatest risk of infection caused by airborne or waterborne microorganisms. Patients in this subset include those who are severely neutropenic for prolonged periods of time (i.e., an absolute neutrophil count [ANC] of ≤ 500 cells/mL), allogeneic HSCT patients, and those who have received intensive chemotherapy (e.g., childhood acute myelogenous leukemia patients).

C. Air

1. Modes of Transmission of Airborne Diseases

A variety of airborne infections in susceptible hosts can result from exposures to clinically significant microorganisms released into the air when environmental reservoirs (i.e., soil, water, dust, and decaying organic matter) are disturbed. Once these materials are brought indoors into a health-care facility by any of a number of vehicles (e.g., people, air currents, water, construction materials, and equipment), the attendant microorganisms can proliferate in various indoor ecological niches and, if subsequently disbursed into the air, serve as a source for airborne health-care-associated infections.

Respiratory infections can be acquired from exposure to pathogens contained either in droplets or droplet nuclei. Exposure to microorganisms in droplets (e.g., through aerosolized oral and nasal secretions from infected patients³³) constitutes a form of direct contact transmission. When droplets are produced during a sneeze or cough, a cloud of infectious particles $>5\ \mu\text{m}$ in size is expelled, resulting in the potential exposure of susceptible persons within 3 feet of the source person.⁶ Examples of pathogens spread in this manner are influenza virus, rhinoviruses, adenoviruses, and respiratory syncytial virus (RSV). Because these agents primarily are transmitted directly and because the droplets tend to fall out of the air quickly, measures to control air flow in a health-care facility (e.g., use of negative pressure rooms) generally are not indicated for preventing the spread of diseases caused by these agents. Strategies to control the spread of these diseases are outlined in another guideline.³

The spread of airborne infectious diseases via droplet nuclei is a form of indirect transmission.³⁴ Droplet nuclei are the residuals of droplets that, when suspended in air, subsequently dry and produce

particles ranging in size from 1–5 μm . These particles can a) contain potentially viable microorganisms, b) be protected by a coat of dry secretions, c) remain suspended indefinitely in air, and d) be transported over long distances. The microorganisms in droplet nuclei persist in favorable conditions (e.g., a dry, cool atmosphere with little or no direct exposure to sunlight or other sources of radiation). Pathogenic microorganisms that can be spread via droplet nuclei include *Mycobacterium tuberculosis*, VZV, measles virus (i.e., rubeola), and smallpox virus (i.e., variola major).⁶ Several environmental pathogens have life-cycle forms that are similar in size to droplet nuclei and may exhibit similar behavior in the air. The spores of *Aspergillus fumigatus* have a diameter of 2–3.5 μm , with a settling velocity estimated at 0.03 cm/second (or about 1 meter/hour) in still air. With this enhanced buoyancy, the spores, which resist desiccation, can remain airborne indefinitely in air currents and travel far from their source.³⁵

2. Airborne Infectious Diseases in Health-Care Facilities

a. Aspergillosis and Other Fungal Diseases

Aspergillosis is caused by molds belonging to the genus *Aspergillus*. *Aspergillus* spp. are prototype health-care-acquired pathogens associated with dusty or moist environmental conditions. Clinical and epidemiologic aspects of aspergillosis (Table 1) are discussed extensively in another guideline.³

Table 1. Clinical and epidemiologic characteristics of aspergillosis

		References
Causative agents	<i>Aspergillus fumigatus</i> (90%–95% of <i>Aspergillus</i> infections among hematopoietic stem cell transplant (HSCT) patients; <i>A. flavus</i> , <i>A. niger</i> , <i>A. terreus</i> , <i>A. nidulans</i>)	36–43
Modes of transmission	Airborne transmission of fungal spores; direct inhalation; direct inoculation from environmental sources (rare)	37
Activities associated with infection	Construction, renovation, remodeling, repairs, building demolition; rare episodes associated with fomites	44–51
Clinical syndromes and diseases	Acute invasive: pneumonia; ulcerative tracheobronchitis; osteomyelitis; abscesses (aspergillomas) of the lungs, brain, liver, spleen, and kidneys; thrombosis of deep blood vessels; necrotizing skin ulcers; endophthalmitis; and sinusitis Chronic invasive: chronic pneumonitis Hypersensitivity: allergic bronchopulmonary aspergillosis Cutaneous: primary skin and burn-wound infections	44, 45, 52–58
Patient populations at greatest risk	Hematopoietic stem cell transplant patients (HSCT): immunocompromised patients (i.e., those with underlying disease), patients undergoing chemotherapy, organ transplant recipients, preterm neonates, hemodialysis patients, patients with identifiable immune system deficiencies who receive care in general intensive care units (ICUs), and cystic fibrosis patients (may be colonized, occasionally become infected)	36, 59–78
Factors affecting severity and outcomes	The immune status of the patient and the duration of severe neutropenia	79, 80
Occurrence	Rare and sporadic, but increasing as proportion of immunocompromised patients increases; 5% of HSCT patients infected, <5% of solid organ transplant recipients infected	36, 37, 81–88
Mortality rate	Rate can be as high as 100% if severe neutropenia persists; 13%–80% mortality among leukemia patients	58, 83, 89, 90

Aspergillus spp. are ubiquitous, aerobic fungi that occur in soil, water, and decaying vegetation; the organism also survives well in air, dust, and moisture present in health-care facilities.^{91–93} The presence of aspergilli in the health-care facility environment is a substantial extrinsic risk factor for opportunistic invasive aspergillosis (invasive aspergillosis being the most serious form of the disease).^{69, 94} Site renovation and construction can disturb *Aspergillus*-contaminated dust and produce bursts of airborne

fungal spores. Increased levels of atmospheric dust and fungal spores have been associated with clusters of health-care–acquired infections in immunocompromised patients.^{17, 20, 44, 47, 49, 50, 95–98}

Absorbent building materials (e.g., wallboard) serve as an ideal substrate for the proliferation of this organism if they become and remain wet, thereby increasing the numbers of fungal spores in the area. Patient-care items, devices, and equipment can become contaminated with *Aspergillus* spp. spores and serve as sources of infection if stored in such areas.⁵⁷

Most cases of aspergillosis are caused by *Aspergillus fumigatus*, a thermotolerant/thermophilic fungus capable of growing over a temperature range from 53.6°F–127.4°F (12°C–53°C); optimal growth occurs at approximately 104°F (40°C), a temperature inhibitory to most other saprophytic fungi.⁹⁹ It can use cellulose or sugars as carbon sources; because its respiratory process requires an ample supply of carbon, decomposing organic matter is an ideal substrate.

Other opportunistic fungi that have been occasionally linked with health-care–associated infections are members of the order *Mucorales* (e.g., *Rhizopus* spp.) and miscellaneous moniliaceous molds (e.g., *Fusarium* spp. and *Penicillium* spp.) (Table 2). Many of these fungi can proliferate in moist environments (e.g., water-damaged wood and building materials). Some fungi (e.g., *Fusarium* spp. and *Pseudoallescheria* spp.) also can be airborne pathogens.¹⁰⁰ As with aspergillosis, a major risk factor for disease caused by any of these pathogens is the host’s severe immunosuppression from either underlying disease or immunosuppressive therapy.^{101, 102}

Table 2. Environmental fungal pathogens: entry into and contamination of the health-care facility

Implicated environmental vehicle	References
<i>Aspergillus</i> spp.	
Improperly functioning ventilation systems	20, 46, 47, 97, 98, 103, 104
Air filters ^{*,†}	17, 18, 105–107
Air filter frames	17, 18
Window air conditioners	96
Backflow of contaminated air	107
Air exhaust contamination [†]	104
False ceilings	48, 57, 97, 108
Fibrous insulation and perforated metal ceilings	66
Acoustic ceiling tiles, plasterboard	18, 109
Fireproofing material	48, 49
Damp wood building materials	49
Opening doors to construction site	110
Construction	69
Open windows	20, 108, 111
Disposal conduit door	68
Hospital vacuum cleaner	68
Elevator	112
Arm boards	57
Walls	113
Unit kitchen	114
Food	21
Ornamental plants	21
<i>Mucorales</i> / <i>Rhizopus</i> spp.	
Air filter	20, 115
False ceilings	97
Heliport	115
<i>Scedosporium</i> spp.	
Construction	116

(Table 2. continued)

Implicated environmental vehicles	References
<i>Penicillium</i> spp.	
Rotting cabinet wood, pipe leak	21
Ventilation duct fiberglass insulation	112
Air filters	105
Topical anesthetic	117
<i>Acromonium</i> spp.	
Air filters	105
<i>Cladosporium</i> spp.	
Air filters	105
<i>Sporothrix</i>	
Construction (pseudoepidemic)	118

- *. Pigeons, their droppings and roosts are associated with spread of *Aspergillus*, *Cryptococcus*, and *Histoplasma* spp. There have been at least three outbreaks linked to contamination of the filtering systems from bird droppings^{98, 103, 104} Pigeon mites may gain access into a health-care facility through the ventilation system.¹¹⁹
- +. The American Institute of Architects (AIA) standards stipulate that for new or renovated construction a) exhaust outlets are to be placed >25 feet from air intake systems, b) the bottom of outdoor air intakes for HVAC systems should be 6 feet above ground or 3 feet above roof level, and c) exhaust outlets from contaminated areas are situated above the roof level and arranged to minimize the recirculation of exhausted air back into the building.¹²⁰

Infections due *Cryptococcus neoformans*, *Histoplasma capsulatum*, or *Coccidioides immitis* can occur in health-care settings if nearby ground is disturbed and a malfunction of the facility's air-intake components allows these pathogens to enter the ventilation system. *C. neoformans* is a yeast usually 4–8 μm in size. However, viable particles of <2 μm diameter (and thus permissive to alveolar deposition) have been found in soil contaminated with bird droppings, particularly from pigeons.^{98, 103, 104, 121} *H. capsulatum*, with the infectious microconidia ranging in size from 2–5 μm , is endemic in the soil of the central river valleys of the United States. Substantial numbers of these infectious particles have been associated with chicken coops and the roosts of blackbirds.^{98, 103, 104, 122} Several outbreaks of histoplasmosis have been associated with disruption of the environment; construction activities in an endemic area may be a potential risk factor for health-care-acquired airborne infection.^{123, 124} *C. immitis*, with arthrospores of 3–5 μm diameter, has similar potential, especially in the endemic southwestern United States and during seasons of drought followed by heavy rainfall. After the 1994 earthquake centered near Northridge, California, the incidence of coccidioidomycosis in the surrounding area exceeded the historical norm.¹²⁵

Emerging evidence suggests that *Pneumocystis carinii*, now classified as a fungus, may be spread via airborne, person-to-person transmission.¹²⁶ Controlled studies in animals first demonstrated that *P. carinii* could be spread through the air.¹²⁷ More recent studies in health-care settings have detected nucleic acids of *P. carinii* in air samples from areas frequented or occupied by *P. carinii*-infected patients but not in control areas that are not occupied by these patients.^{128, 129} Clusters of cases have been identified among immunocompromised patients who had contact with a source patient and with each other. Recent studies have examined the presence of *P. carinii* DNA in oropharyngeal washings and the nares of infected patients, their direct contacts, and persons with no direct contact.^{130, 131} Molecular analysis of the DNA by polymerase chain reaction (PCR) provides evidence for airborne transmission of *P. carinii* from infected patients to direct contacts, but immunocompetent contacts tend to become transiently colonized rather than infected.¹³¹ The role of colonized persons in the spread of *P. carinii* pneumonia (PCP) remains to be determined. At present, specific modifications to ventilation systems to control spread of PCP in a health-care facility are not indicated. Current recommendations

outline isolation procedures to minimize or eliminate contact of immunocompromised patients not on PCP prophylaxis with PCP-infected patients.^{6, 132}

b. Tuberculosis and Other Bacterial Diseases

The bacterium most commonly associated with airborne transmission is *Mycobacterium tuberculosis*. A comprehensive review of the microbiology and epidemiology of *M. tuberculosis* and guidelines for tuberculosis (TB) infection control have been published.^{4, 133, 134} A summary of the clinical and epidemiologic information from these materials is provided in this guideline (Table 3).

Table 3. Clinical and epidemiologic characteristics of tuberculosis (TB)*

Causative agents	<i>Mycobacterium tuberculosis</i> , <i>M. bovis</i> , <i>M. africanum</i>
Mode of transmission	Airborne transmission via droplet nuclei 1–5 µm in diameter
Patient factors associated with infectivity and transmission	<ul style="list-style-type: none"> ▪ Disease of the lungs, airways, or larynx; presence of cough or other forceful expiratory measures ▪ Presence of acid-fast bacilli (AFB) in the sputum ▪ Failure of the patient to cover the mouth and nose when coughing or sneezing ▪ Presence of cavitation on chest radiograph ▪ Inappropriate or shortened duration of chemotherapy
Activities associated with infections	<ul style="list-style-type: none"> ▪ Exposures in relatively small, enclosed spaces ▪ Inadequate ventilation resulting in insufficient removal of droplet nuclei ▪ Cough-producing procedures done in areas without proper environmental controls ▪ Recirculation of air containing infectious droplet nuclei ▪ Failure to use respiratory protection when managing open lesions for patients with suspected extrapulmonary TB¹³⁵
Clinical syndromes and disease	Pulmonary TB ; extrapulmonary TB can affect any organ system or tissue; laryngeal TB is highly contagious
Populations at greatest risk	<ul style="list-style-type: none"> ▪ Immunocompromised persons (e.g., HIV-infected persons) ▪ Medically underserved persons, urban poor, homeless persons, elderly persons, migrant farm workers, close contacts of known patients ▪ Substance abusers, present and former prison inmates ▪ Foreign-born persons from areas with high prevalence of TB ▪ Health-care workers
Factors affecting severity and outcomes	<ul style="list-style-type: none"> ▪ Concentration of droplet nuclei in air, duration of exposure ▪ Age at infection ▪ Immunosuppression due to therapy or disease, underlying chronic medical conditions, history of malignancies or lesions of the lungs
Occurrence	Worldwide; incidence in the United States is 5.6 cases/100,000 population (2001) ¹³⁶
Mortality	930 deaths in the United States (1999) ¹³⁶
Chemoprophylaxis / treatment	Treatment of latent infection includes isoniazid (INH) or rifampin (RIF). ^{4, 134, 137–139} Directly observed therapy (DOT) for active cases as indicated: INH, RIF, pyrazinamide (PZA), ethambutol (EMB), streptomycin (SM) in various combinations determined by prevalent levels of specific resistance. ^{4, 134, 137–139} Consult therapy guidelines for specific treatment indications. ¹³⁹

* Material in this table is compiled from references 4, 133–141.

M. tuberculosis is carried by droplet nuclei generated when persons (primarily adults and adolescents) who have pulmonary or laryngeal TB sneeze, cough, speak, or sing;¹³⁹ normal air currents can keep these particles airborne for prolonged periods and spread them throughout a room or building.¹⁴² However, transmission of TB has occurred from mycobacteria aerosolized during provision of care (e.g., wound/lesion care or during handling of infectious peritoneal dialysis fluid) for extrapulmonary TB patients.^{135, 140}

Gram-positive cocci (i.e., *Staphylococcus aureus*, group A beta-hemolytic streptococci), also important health-care-associated pathogens, are resistant to inactivation by drying and can persist in the

environment and on environmental surfaces for extended periods. These organisms can be shed from heavily colonized persons and discharged into the air. Airborne dispersal of *S. aureus* is directly associated with the concentration of the bacterium in the anterior nares.¹⁴³ Approximately 10% of healthy carriers will disseminate *S. aureus* into the air, and some persons become more effective disseminators of *S. aureus* than others.^{144–148} The dispersal of *S. aureus* into air can be exacerbated by concurrent viral upper respiratory infection, thereby turning a carrier into a “cloud shedder.”¹⁴⁹ Outbreaks of surgical site infections (SSIs) caused by group A beta-hemolytic streptococci have been traced to airborne transmission from colonized operating-room personnel to patients.^{150–153} In these situations, the strain causing the outbreak was recovered from the air in the operating room^{150, 151, 154} or on settle plates in a room in which the carrier exercised.^{151–153} *S. aureus* and group A streptococci have not been linked to airborne transmission outside of operating rooms, burn units, and neonatal nurseries.^{155, 156} Transmission of these agents occurs primarily via contact and droplets.

Other gram-positive bacteria linked to airborne transmission include *Bacillus* spp. which are capable of sporulation as environmental conditions become less favorable to support their growth. Outbreaks and pseudo-outbreaks have been attributed to *Bacillus cereus* in maternity, pediatric, intensive care, and bronchoscopy units; many of these episodes were secondary to environmental contamination.^{157–160}

Gram-negative bacteria rarely are associated with episodes of airborne transmission because they generally require moist environments for persistence and growth. The main exception is *Acinetobacter* spp., which can withstand the inactivating effects of drying. In one epidemiologic investigation of bloodstream infections among pediatric patients, identical *Acinetobacter* spp. were cultured from the patients, air, and room air conditioners in a nursery.¹⁶¹

Aerosols generated from showers and faucets may potentially contain legionellae and other gram-negative waterborne bacteria (e.g., *Pseudomonas aeruginosa*). Exposure to these organisms is through direct inhalation. However, because water is the source of the organisms and exposure occurs in the vicinity of the aerosol, the discussion of the diseases associated with such aerosols and the prevention measures used to curtail their spread is discussed in another section of the Guideline (see Part I: Water).

c. Airborne Viral Diseases

Some human viruses are transmitted from person to person via droplet aerosols, but very few viruses are consistently airborne in transmission (i.e., are routinely suspended in an infective state in air and capable of spreading great distances), and health-care–associated outbreaks of airborne viral disease are limited to a few agents. Consequently, infection-control measures used to prevent spread of these viral diseases in health-care facilities primarily involve patient isolation, vaccination of susceptible persons, and antiviral therapy as appropriate rather than measures to control air flow or quality.⁶ Infections caused by VZV frequently are described in health-care facilities. Health-care–associated airborne outbreaks of VZV infections from patients with primary infection and disseminated zoster have been documented; patients with localized zoster have, on rare occasions, also served as source patients for outbreaks in health-care facilities.^{162–166} VZV infection can be prevented by vaccination, although patients who develop a rash within 6 weeks of receiving varicella vaccine or who develop breakthrough varicella following exposure should be considered contagious.¹⁶⁷

Viruses whose major mode of transmission is via droplet contact rarely have caused clusters of infections in group settings through airborne routes. The factors facilitating airborne distribution of these viruses in an infective state are unknown, but a presumed requirement is a source patient in the early stage of infection who is shedding large numbers of viral particles into the air. Airborne transmission of measles has been documented in health-care facilities.^{168–171} In addition, institutional outbreaks of influenza virus infections have occurred predominantly in nursing homes,^{172–176} and less frequently in medical and neonatal intensive care units, chronic-care areas, HSCT units, and pediatric

12 [Click here for current information on how Ebola virus is transmitted.](#)

wards.^{177–180} Some evidence supports airborne transmission of influenza viruses by droplet nuclei,^{181, 182} and case clusters in pediatric wards suggest that droplet nuclei may play a role in transmitting certain respiratory pathogens (e.g., adenoviruses and respiratory syncytial virus [RSV]).^{177, 183, 184} Some evidence also supports airborne transmission of enteric viruses. An outbreak of a Norwalk-like virus infection involving more than 600 staff personnel over a 3-week period was investigated in a Toronto, Ontario hospital in 1985; common sources (e.g., food and water) were ruled out during the investigation, leaving airborne spread as the most likely mode of transmission.¹⁸⁵

Smallpox virus, a potential agent of bioterrorism, is spread predominantly via direct contact with infectious droplets, but it also can be associated with airborne transmission.^{186, 187} A German hospital study from 1970 documented the ability of this virus to spread over considerable distances and cause infection at low doses in a well-vaccinated population; factors potentially facilitating transmission in this situation included a patient with cough and an extensive rash, indoor air with low relative humidity, and faulty ventilation patterns resulting from hospital design (e.g., open windows).¹⁸⁸ Smallpox patients with extensive rash are more likely to have lesions present on mucous membranes and therefore have greater potential to disseminate virus into the air.¹⁸⁸ In addition to the smallpox transmission in Germany, two cases of laboratory-acquired smallpox virus infection in the United Kingdom in 1978 also were thought to be caused by airborne transmission.¹⁸⁹

Airborne transmission may play a role in the natural spread of hantaviruses and certain hemorrhagic fever viruses (e.g., Ebola, Marburg, and Lassa), but evidence for airborne spread of these agents in health-care facilities is inconclusive.¹⁹⁰ Although hantaviruses can be transmitted when aerosolized from rodent excreta,^{191, 192} person-to-person spread of hantavirus infection from source patients has not occurred in health-care facilities.^{193–195} Nevertheless, health-care workers are advised to contain potentially infectious aerosols and wear National Institute of Occupational Safety and Health (NIOSH) approved respiratory protection when working with this agent in laboratories or autopsy suites.¹⁹⁶ Lassa virus transmission via aerosols has been demonstrated in the laboratory and incriminated in health-care-associated infections in Africa,^{197–199} but airborne spread of this agent in hospitals in developed nations likely is inefficient.^{200, 201} Yellow fever is considered to be a viral hemorrhagic fever agent with high aerosol infectivity potential, but health-care-associated transmission of this virus has not been described.²⁰² Viral hemorrhagic fever diseases primarily occur after direct exposure to infected blood and body fluids, and the use of standard and droplet precautions prevents transmission early in the course of these illnesses.^{203, 204} However, whether these viruses can persist in droplet nuclei that might remain after droplet production from coughs or vomiting in the latter stages of illness is unknown.²⁰⁵ Although the use of a negative-pressure room is not required during the early stages of illness, its use might be prudent at the time of hospitalization to avoid the need for subsequent patient transfer. Current CDC guidelines recommend negative-pressure rooms with anterooms for patients with hemorrhagic fever and use of HEPA respirators by persons entering these rooms when the patient has prominent cough, vomiting, diarrhea, or hemorrhage.^{6, 203} Face shields or goggles will help to prevent mucous-membrane exposure to potentially-aerosolized infectious material in these situations. If an anteroom is not available, portable, industrial-grade high efficiency particulate air (HEPA) filter units can be used to provide the equivalent of additional air changes per hour (ACH).

Table 4. Microorganisms associated with airborne transmission*

	Fungi	Bacteria	Viruses
Numerous reports in health-care facilities	<i>Aspergillus</i> spp.+ <i>Mucorales (Rhizopus</i> spp.) ^{97, 115}	<i>Mycobacterium tuberculosis</i> +	Measles (rubeola) virus ¹⁶⁸⁻¹⁷⁰ Varicella-zoster virus ¹⁶²⁻¹⁶⁶
Atypical, occasional reports	<i>Acremonium</i> spp. ^{105, 206} <i>Fusarium</i> spp. ¹⁰² <i>Pseudoallescheria boydii</i> ¹⁰⁰ <i>Scedosporium</i> spp. ¹¹⁶ <i>Sporothrix cyanescens</i> ¶ ¹¹⁸	<i>Acinetobacter</i> spp. ¹⁶¹ <i>Bacillus</i> spp.¶ ^{160, 207} <i>Brucella</i> spp. ** ²⁰⁸⁻²¹¹ <i>Staphylococcus aureus</i> ^{148, 156} Group A <i>Streptococcus</i> ¹⁵¹	Smallpox virus (variola)§ ^{188, 189} Influenza viruses ^{181, 182} Respiratory syncytial virus ¹⁸³ Adenoviruses ¹⁸⁴ Norwalk-like virus ¹⁸⁵
Airborne in nature; airborne transmission in health care settings not described	<i>Coccidioides immitis</i> ¹²⁵ <i>Cryptococcus</i> spp. ¹²¹ <i>Histoplasma capsulatum</i> ¹²⁴	<i>Coxiella burnetii</i> (Q fever) ²¹²	Hantaviruses ^{193, 195} Lassa virus ²⁰⁵ Marburg virus ²⁰⁵ Ebola virus ²⁰⁵ Crimean-Congo virus ²⁰⁵
Under investigation	<i>Pneumocystis carinii</i> ¹³¹	—	—

* This list excludes microorganisms transmitted from aerosols derived from water.

+ Refer to the text for references for these disease agents.

§ Airborne transmission of smallpox is infrequent. Potential for airborne transmission increases with patients who are effective disseminators present in facilities with low relative humidity in the air and faulty ventilation.

¶ Documentation of pseudoepidemic during construction.

** Airborne transmission documented in the laboratory but not in patient-care areas

3. Heating, Ventilation, and Air Conditioning Systems in Health-Care Facilities

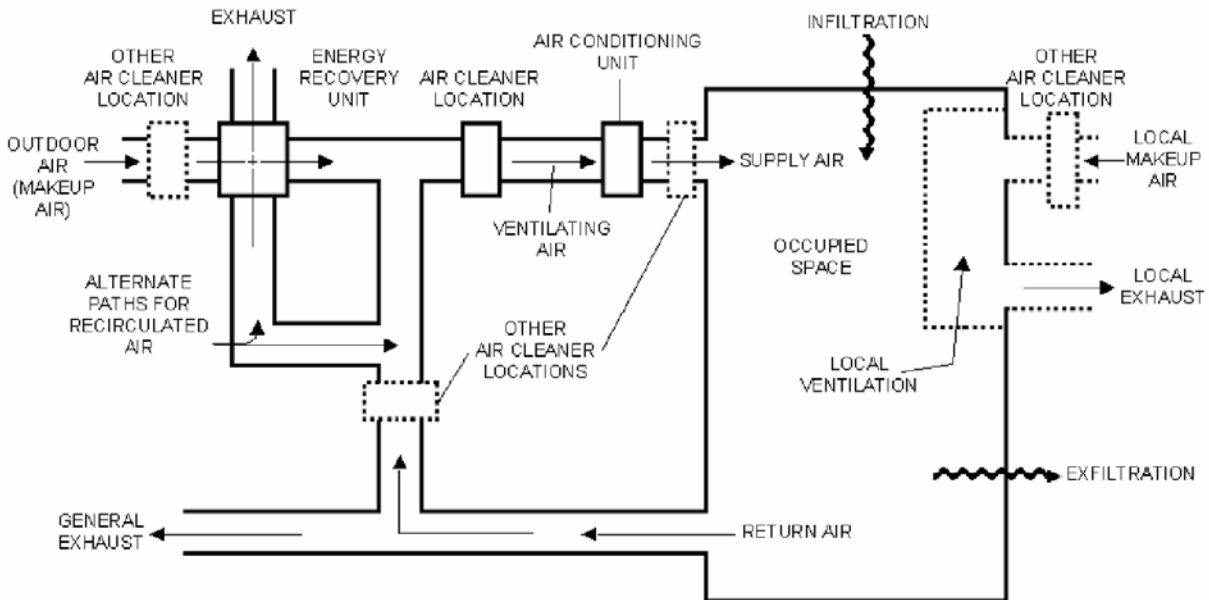
a. Basic Components and Operations

Heating, ventilation, and air conditioning (HVAC) systems in health-care facilities are designed to a) maintain the indoor air temperature and humidity at comfortable levels for staff, patients, and visitors; b) control odors; c) remove contaminated air; d) facilitate air-handling requirements to protect susceptible staff and patients from airborne health-care-associated pathogens; and e) minimize the risk for transmission of airborne pathogens from infected patients.^{35, 120} An HVAC system includes an outside air inlet or intake; filters; humidity modification mechanisms (i.e., humidity control in summer, humidification in winter); heating and cooling equipment; fans; ductwork; air exhaust or out-takes; and registers, diffusers, or grilles for proper distribution of the air (Figure 1).^{213, 214} Decreased performance of healthcare facility HVAC systems, filter inefficiencies, improper installation, and poor maintenance can contribute to the spread of health-care-associated airborne infections.

The American Institute of Architects (AIA) has published guidelines for the design and construction of new health-care facilities and for renovation of existing facilities. These AIA guidelines address indoor air-quality standards (e.g., ventilation rates, temperature levels, humidity levels, pressure relationships, and minimum air changes per hour [ACH]) specific to each zone or area in health-care facilities (e.g., operating rooms, laboratories, diagnostic areas, patient-care areas, and support departments).¹²⁰ These guidelines represent a consensus document among authorities having jurisdiction (AHJ), governmental regulatory agencies (i.e., Department of Health and Human Services [DHHS]; Department of Labor, Occupational Safety and Health Administration [OSHA]), health-care professionals, professional organizations (e.g., American Society of Heating, Refrigeration, and Air-Conditioning Engineers [ASHRAE], American Society for Healthcare Engineering [ASHE]), and accrediting organizations (i.e., Joint Commission on Accreditation of Healthcare Organizations [JCAHO]). More than 40 state agencies that license health-care facilities have either incorporated or adopted by reference these

guidelines into their state standards. JCAHO, through its surveys, ensures that facilities are in compliance with the ventilation guidelines of this standard for new construction and renovation.

Figure 1. Diagram of a ventilation system*



Outdoor air and recirculated air pass through air cleaners (e.g., filter banks) designed to reduce the concentration of airborne contaminants. Air is conditioned for temperature and humidity before it enters the occupied space as supply air. Infiltration is air leakage inward through cracks and interstitial spaces of walls, floors, and ceilings. Exfiltration is air leakage outward through these same cracks and spaces. Return air is largely exhausted from the system, but a portion is recirculated with fresh, incoming air.

* Used with permission of the publisher of reference 214 (ASHRAE)

Engineering controls to contain or prevent the spread of airborne contaminants center on a) local exhaust ventilation [i.e., source control], b) general ventilation, and c) air cleaning.⁴ General ventilation encompasses a) dilution and removal of contaminants via well-mixed air distribution of filtered air, b) directing contaminants toward exhaust registers and grilles via uniform, non-mixed airflow patterns, c) pressurization of individual spaces relative to all other spaces, and d) pressurization of buildings relative to the outdoors and other attached buildings.

A centralized HVAC system operates as follows. Outdoor air enters the system, where low-efficiency or “roughing” filters remove large particulate matter and many microorganisms. The air enters the distribution system for conditioning to appropriate temperature and humidity levels, passes through an additional bank of filters for further cleaning, and is delivered to each zone of the building. After the conditioned air is distributed to the designated space, it is withdrawn through a return duct system and delivered back to the HVAC unit. A portion of this “return air” is exhausted to the outside while the remainder is mixed with outdoor air for dilution and filtered for removal of contaminants.²¹⁵ Air from toilet rooms or other soiled areas is usually exhausted directly to the atmosphere through a separate duct exhaust system. Air from rooms housing tuberculosis patients is exhausted to the outside if possible, or passed through a HEPA filter before recirculation. Ultraviolet germicidal irradiation (UVGI) can be used as an adjunct air-cleaning measure, but it cannot replace HEPA filtration.

b. Filtration

i. Filter Types and Methods of Filtration

Filtration, the physical removal of particulates from air, is the first step in achieving acceptable indoor air quality. Filtration is the primary means of cleaning the air. Five methods of filtration can be used (Table 5). During filtration, outdoor air passes through two filter beds or banks (with efficiencies of 20%–40% and $\geq 90\%$, respectively) for effective removal of particles 1–5 μm in diameter.^{35, 120} The low-to-medium efficiency filters in the first bank have low resistance to airflow, but this feature allows some small particulates to pass onto heating and air conditioning coils and into the indoor environment.³⁵ Incoming air is mixed with recirculated air and reconditioned for temperature and humidity before being filtered by the second bank of filters. The performance of filters with $\leq 90\%$ efficiency is measured using either the dust-spot test or the weight-arrestance test.^{35, 216}

Table 5. Filtration methods*

Basic method	Principle of performance	Filtering efficiency
Straining	Particles in the air are larger than the openings between the filter fibers, resulting in gross removal of large particles.	Low
Impingement	Particles collide with filter fibers and remain attached to the filter. Fibers may be coated with adhesive.	Low
Interception	Particles enter into the filter and become entrapped and attached to the filter fibers.	Medium
Diffusion	Small particles, moving in erratic motion, collide with filter fibers and remain attached.	High
Electrostatic	Particles bearing negative electrostatic charge are attracted to the filter with positively charged fibers.	High

* Material in this table was compiled from information in reference 217.

The second filter bank usually consists of high-efficiency filters. This filtration system is adequate for most patient-care areas in ambulatory-care facilities and hospitals, including the operating room environment and areas providing central services.¹²⁰ Nursing facilities use 90% dust-spot efficient filters as the second bank of filters,¹²⁰ whereas a HEPA filter bank may be indicated for special-care areas of hospitals. HEPA filters are at least 99.97% efficient for removing particles $\geq 0.3 \mu\text{m}$ in diameter. (As a reference, *Aspergillus* spores are 2.5–3.0 μm in diameter.) Examples of care areas where HEPA filters are used include PE rooms and those operating rooms designated for orthopedic implant procedures.³⁵

Maintenance costs associated with HEPA filters are high compared with other types of filters, but use of in-line disposable prefilters can increase the life of a HEPA filter by approximately 25%. Alternatively, if a disposable prefilter is followed by a filter that is 90% efficient, the life of the HEPA filter can be extended ninefold. This concept, called progressive filtration, allows HEPA filters in special care areas to be used for 10 years.²¹³ Although progressive filtering will extend the mechanical ability of the HEPA filter, these filters may absorb chemicals in the environment and later desorb those chemicals, thereby necessitating a more frequent replacement program. HEPA filter efficiency is monitored with the dioctylphthalate (DOP) particle test using particles that are 0.3 μm in diameter.²¹⁸

HEPA filters are usually framed with metal, although some older versions have wood frames. A metal frame has no advantage over a properly fitted wood frame with respect to performance, but wood can compromise the air quality if it becomes and remains wet, allowing the growth of fungi and bacteria. Hospitals are therefore advised to phase out water-damaged or spent wood-framed filter units and replace them with metal-framed HEPA filters.

HEPA filters are usually fixed into the HVAC system; however, portable, industrial grade HEPA units are available that can filter air at the rate of 300–800 ft³/min. Portable HEPA filters are used to a) temporarily recirculate air in rooms with no general ventilation, b) augment systems that cannot provide adequate airflow, and c) provide increased effectiveness in airflow.⁴ Portable HEPA units are useful engineering controls that help clean the air when the central HVAC system is undergoing repairs,²¹⁹ but these units do not satisfy fresh-air requirements.²¹⁴ The effectiveness of the portable unit for particle removal is dependent on a) the configuration of the room, b) the furniture and persons in the room, c) the placement of the units relative to the contents and layout of the room, and d) the location of the supply and exhaust registers or grilles. If portable, industrial-grade units are used, they should be capable of recirculating all or nearly all of the room air through the HEPA filter, and the unit should be designed to achieve the equivalent of ≥ 12 ACH.⁴ (An average room has approximately 1,600 ft³ of airspace.) The hospital engineering department should be contacted to provide ACH information in the event that a portable HEPA filter unit is necessary to augment the existing fixed HVAC system for air cleaning.

ii. Filter Maintenance

Efficiency of the filtration system is dependent on the density of the filters, which can create a drop in pressure unless compensated by stronger and more efficient fans, thus maintaining air flow. For optimal performance, filters require monitoring and replacement in accordance with the manufacturer's recommendations and standard preventive maintenance practices.²²⁰ Upon removal, spent filters can be bagged and discarded with the routine solid waste, regardless of their patient-care area location.²²¹ Excess accumulation of dust and particulates increases filter efficiency, requiring more pressure to push the air through. The pressure differential across filters is measured by use of manometers or other gauges. A pressure reading that exceeds specifications indicates the need to change the filter. Filters also require regular inspection for other potential causes of decreased performance. Gaps in and around filter banks and heavy soil and debris upstream of poorly maintained filters have been implicated in health-care-associated outbreaks of aspergillosis, especially when accompanied by construction activities at the facility.^{17, 18, 106, 222}

c. Ultraviolet Germicidal Irradiation (UVGI)

As a supplemental air-cleaning measure, UVGI is effective in reducing the transmission of airborne bacterial and viral infections in hospitals, military housing, and classrooms, but it has only a minimal inactivating effect on fungal spores.^{223–228} UVGI is also used in air handling units to prevent or limit the growth of vegetative bacteria and fungi. Most commercially available UV lamps used for germicidal purposes are low-pressure mercury vapor lamps that emit radiant energy predominantly at a wave-length of 253.7 nm.^{229, 230} Two systems of UVGI have been used in health-care settings – duct irradiation and upper-room air irradiation. In duct irradiation systems, UV lamps are placed inside ducts that remove air from rooms to disinfect the air before it is recirculated. When properly designed, installed, and maintained, high levels of UVGI can be attained in the ducts with little or no exposure of persons in the rooms.^{231, 232} In upper-room air irradiation, UV lamps are either suspended from the ceiling or mounted on the wall.⁴ Upper air UVGI units have two basic designs: a) a “pan” fixture with UVGI unshielded above the unit to direct the irradiation upward and b) a fixture with a series of parallel plates to columnize the irradiation outward while preventing the light from getting to the eyes of the room's occupants. The germicidal effect is dependent on air mixing via convection between the room's irradiated upper zone and the lower patient-care zones.^{233, 234}

Bacterial inactivation studies using BCG mycobacteria and *Serratia marcescens* have estimated the effect of UVGI as equivalent to 10 ACH–39 ACH.^{235, 236} Another study, however, suggests that UVGI may result in fewer equivalent ACH in the patient-care zone, especially if the mixing of air between zones is insufficient.²³⁴ The use of fans or HVAC systems to generate air movement may increase the effectiveness of UVGI if airborne microorganisms are exposed to the light energy for a sufficient length

of time.^{233, 235, 237–239} The optimal relationship between ventilation and UVGI is not known.

Because the clinical effectiveness of UV systems may vary, UVGI is not recommended for air management prior to air recirculation from airborne isolation rooms. It is also not recommended as a substitute for HEPA filtration, local exhaust of air to the outside, or negative pressure.⁴ The use of UV lamps and HEPA filtration in a single unit offers only minimal infection-control benefits over those provided by the use of a HEPA filter alone.²⁴⁰ Duct systems with UVGI are not recommended as a substitute for HEPA filters if the air from isolation rooms must be recirculated to other areas of the facility.⁴ Regular maintenance of UVGI systems is crucial and usually consists of keeping the bulbs free of dust and replacing old bulbs as necessary. Safety issues associated with the use of UVGI systems are described in other guidelines.⁴

d. Conditioned Air in Occupied Spaces

Temperature and humidity are two essential components of conditioned air. After outside air passes through a low- or medium-efficiency filter, the air undergoes conditioning for temperature and humidity control before it passes through high-efficiency or HEPA filtration.

i. Temperature

HVAC systems in health-care facilities are often single-duct or dual-duct systems.^{35, 241} A single-duct system distributes cooled air (55°F [12.8°C]) throughout the building and uses thermostatically controlled reheat boxes located in the terminal ductwork to warm the air for individual or multiple rooms. The dual-duct system consists of parallel ducts, one with a cold air stream and the other with a hot air stream. A mixing box in each room or group of rooms mixes the two air streams to achieve the desired temperature. Temperature standards are given as either a single temperature or a range, depending on the specific health-care zone. Cool temperature standards (68°F–73°F [20°C–23°C]) usually are associated with operating rooms, clean workrooms, and endoscopy suites.¹²⁰ A warmer temperature (75°F [24°C]) is needed in areas requiring greater degrees of patient comfort. Most other zones use a temperature range of 70°F–75°F (21°C–24°C).¹²⁰ Temperatures outside of these ranges may be needed occasionally in limited areas depending on individual circumstances during patient care (e.g., cooler temperatures in operating rooms during specialized operations).

ii. Humidity

Four measures of humidity are used to quantify different physical properties of the mixture of water vapor and air. The most common of these is relative humidity, which is the ratio of the amount of water vapor in the air to the amount of water vapor air can hold at that temperature.²⁴² The other measures of humidity are specific humidity, dew point, and vapor pressure.²⁴²

Relative humidity measures the percentage of saturation. At 100% relative humidity, the air is saturated. For most areas within health-care facilities, the designated comfort range is 30%–60% relative humidity.^{120, 214} Relative humidity levels >60%, in addition to being perceived as uncomfortable, promote fungal growth.²⁴³ Humidity levels can be manipulated by either of two mechanisms.²⁴⁴ In a water-wash unit, water is sprayed and drops are taken up by the filtered air; additional heating or cooling of this air sets the humidity levels. The second mechanism is by means of water vapor created from steam and added to filtered air in humidifying boxes. Reservoir-type humidifiers are not allowed in health-care facilities as per AIA guidelines and many state codes.¹²⁰ Cool-mist humidifiers should be avoided, because they can disseminate aerosols containing allergens and microorganisms.²⁴⁵ Additionally, the small, personal-use versions of this equipment can be difficult to clean.

iii. Ventilation

The control of air pollutants (e.g., microorganisms, dust, chemicals, and smoke) at the source is the most effective way to maintain clean air. The second most effective means of controlling indoor air pollution is through ventilation. Ventilation rates are voluntary unless a state or local government specifies a standard in health-care licensing or health department requirements. These standards typically apply to only the design of a facility, rather than its operation.^{220, 246} Health-care facilities without specific ventilation standards should follow the AIA guideline specific to the year in which the building was built or the ANSI/ASHRAE Standard 62, *Ventilation for Acceptable Indoor Air Quality*.^{120, 214, 241}

Ventilation guidelines are defined in terms of air volume per minute per occupant and are based on the assumption that occupants and their activities are responsible for most of the contaminants in the conditioned space.²¹⁵ Most ventilation rates for health-care facilities are expressed as room ACH. Peak efficiency for particle removal in the air space occurs between 12 ACH–15 ACH.^{35, 247, 248} Ventilation rates vary among the different patient-care areas of a health-care facility (Appendix B).¹²⁰

Health-care facilities generally use recirculated air.^{35, 120, 241, 249, 250} Fans create sufficient positive pressure to force air through the building duct work and adequate negative pressure to evacuate air from the conditioned space into the return duct work and/or exhaust, thereby completing the circuit in a sealed system (Figure 1). However, because gaseous contaminants tend to accumulate as the air recirculates, a percentage of the recirculated air is exhausted to the outside and replaced by fresh outdoor air. In hospitals, the delivery of filtered air to an occupied space is an engineered system design issue, the full discussion of which is beyond the scope of this document.

Hospitals with areas not served by central HVAC systems often use through-the-wall or fan coil air conditioning units as the sole source of room ventilation. AIA guidelines for newly installed systems stipulate that through-the-wall fan-coil units be equipped with permanent (i.e., cleanable) or replaceable filters with a minimum efficiency of 68% weight arrestance.¹²⁰ These units may be used only as recirculating units; all outdoor air requirements must be met by a separate central air handling system with proper filtration, with a minimum of two outside air changes in general patient rooms (D. Erickson, ASHE, 2000).¹²⁰ If a patient room is equipped with an individual through-the-wall fan coil unit, the room should not be used as either AII or as PE.¹²⁰ These requirements, although directed to new HVAC installations also are appropriate for existing settings. Non-central air-handling systems are prone to problems associated with excess condensation accumulating in drip pans and improper filter maintenance; health-care facilities should clean or replace the filters in these units on a regular basis while the patient is out of the room.

Laminar airflow ventilation systems are designed to move air in a single pass, usually through a bank of HEPA filters either along a wall or in the ceiling, in a one-way direction through a clean zone with parallel streamlines. Laminar airflow can be directed vertically or horizontally; the unidirectional system optimizes airflow and minimizes air turbulence.^{63, 241} Delivery of air at a rate of 0.5 meters per second (90 ± 20 ft/min) helps to minimize opportunities for microorganism proliferation.^{63, 251, 252} Laminar airflow systems have been used in PE to help reduce the risk for health-care-associated airborne infections (e.g., aspergillosis) in high-risk patients.^{63, 93, 253, 254} However, data that demonstrate a survival benefit for patients in PE with laminar airflow are lacking. Given the high cost of installation and apparent lack of benefit, the value of laminar airflow in this setting is questionable.^{9, 37} Few data support the use of laminar airflow systems elsewhere in a hospital.²⁵⁵

iv. Pressurization

Positive and negative pressures refer to a pressure differential between two adjacent air spaces (e.g., rooms and hallways). Air flows away from areas or rooms with positive pressure (pressurized), while

air flows into areas with negative pressure (depressurized). All rooms are set at negative pressure to prevent airborne microorganisms in the room from entering hallways and corridors. PE rooms housing severely neutropenic patients are set at positive pressure to keep airborne pathogens in adjacent spaces or corridors from coming into and contaminating the airspace occupied by such high-risk patients. Self-closing doors are mandatory for both of these areas to help maintain the correct pressure differential.^{4,6,120} Older health-care facilities may have variable pressure rooms (i.e., rooms in which the ventilation can be manually switched between positive and negative pressure). These rooms are no longer permitted in the construction of new facilities or in renovated areas of the facility,¹²⁰ and their use in existing facilities has been discouraged because of difficulties in assuring the proper pressure differential, especially for the negative pressure setting, and because of the potential for error associated with switching the pressure differentials for the room. Continued use of existing variable pressure rooms depends on a partnership between engineering and infection control. Both positive- and negative-pressure rooms should be maintained according to specific engineering specifications (Table 6).

Table 6. Engineered specifications for positive- and negative pressure rooms*

	Positive pressure areas (e.g., protective environments [PE])	Negative pressure areas (e.g., airborne infection isolation [AII])
Pressure differentials	> +2.5 Pa§ (0.01" water gauge)	> -2.5 Pa (0.01" water gauge)
Air changes per hour (ACH)	>12	≥12 (for renovation or new construction)
Filtration efficiency	Supply: 99.97% @ 0.3 μm DOP¶ Return: none required**	Supply: 90% (dust spot test) Return: 99.97% @ 0.3 μm DOP¶ †
Room airflow direction	Out to the adjacent area	In to the room
Clean-to-dirty airflow in room	Away from the patient (high-risk patient, immunosuppressed patient)	Towards the patient (airborne disease patient)
Ideal pressure differential	> + 8 Pa	> - 2.5 Pa

* Material in this table was compiled from references 35 and 120. Table adapted from and used with permission of the publisher of reference 35 (Lippincott Williams and Wilkins).

§ Pa is the abbreviation for Pascal, a metric unit of measurement for pressure based on air velocity; 250 Pa equals 1.0 inch water gauge.

¶ DOP is the abbreviation for dioctylphthalate particles of 0.3 μm diameter.

** If the patient requires both PE and AII, return air should be HEPA-filtered or otherwise exhausted to the outside.

† HEPA filtration of exhaust air from AII rooms should not be required, providing that the exhaust is properly located to prevent re-entry into the building.

Health-care professionals (e.g., infection control, hospital epidemiologists) must perform a risk assessment to determine the appropriate number of AII rooms (negative pressure) and/or PE rooms (positive pressure) to serve the patient population. The AIA guidelines require a certain number of AII rooms as a minimum, and it is important to refer to the edition under which the building was built for appropriate guidance.¹²⁰

In large health-care facilities with central HVAC systems, sealed windows help to ensure the efficient operation of the system, especially with respect to creating and maintaining pressure differentials. Sealing the windows in PE areas helps minimize the risk of airborne contamination from the outside. One outbreak of aspergillosis among immunosuppressed patients in a hospital was attributed in part to an open window in the unit during a time when both construction and a fire happened nearby; sealing the window prevented further entry of fungal spores into the unit from the outside air.¹¹¹ Additionally, all emergency exits (e.g., fire escapes and emergency doors) in PE wards should be kept closed (except during emergencies) and equipped with alarms.

e. Infection Control Impact of HVAC System Maintenance and Repair

A failure or malfunction of any component of the HVAC system may subject patients and staff to discomfort and exposure to airborne contaminants. Only limited information is available from formal

studies on the infection-control implications of a complete air-handling system failure or shutdown for maintenance. Most experience has been derived from infectious disease outbreaks and adverse outcomes among high-risk patients when HVAC systems are poorly maintained. (See Table 7 for potential ventilation hazards, consequences, and correction measures.)

AIA guidelines prohibit U.S. hospitals and surgical centers from shutting down their HVAC systems for purposes other than required maintenance, filter changes, and construction.¹²⁰ Airflow can be reduced; however, sufficient supply, return, and exhaust must be provided to maintain required pressure relationships when the space is not occupied. Maintaining these relationships can be accomplished with special drives on the air-handling units (i.e., a variable air ventilation [VAV] system).

Microorganisms proliferate in environments wherever air, dust, and water are present, and air-handling systems can be ideal environments for microbial growth.³⁵ Properly engineered HVAC systems require routine maintenance and monitoring to provide acceptable indoor air quality efficiently and to minimize conditions that favor the proliferation of health-care-associated pathogens.^{35, 249} Performance monitoring of the system includes determining pressure differentials across filters, regular inspection of system filters, DOP testing of HEPA filters, testing of low- or medium efficiency filters, and manometer tests for positive- and negative-pressure areas in accordance with nationally recognized standards, guidelines, and manufacturers' recommendations. The use of hand-held, calibrated equipment that can provide a numerical reading on a daily basis is preferred for engineering purposes (A. Streifel, University of Minnesota, 2000).²⁵⁶ Several methods that provide a visual, qualitative measure of pressure differentials (i.e., airflow direction) include smoke-tube tests or placing flutter strips, ping-pong balls, or tissue in the air stream.

Preventive filter and duct maintenance (e.g., cleaning ductwork vents, replacing filters as needed, and properly disposing spent filters into plastic bags immediately upon removal) is important to prevent potential exposures of patients and staff during HVAC system shut-down. The frequency of filter inspection and the parameters of this inspection are established by each facility to meet their unique needs. Ductwork in older health-care facilities may have insulation on the interior surfaces that can trap contaminants. This insulation material tends to break down over time to be discharged from the HVAC system. Additionally, a malfunction of the air-intake system can overburden the filtering system and permit aerosolization of fungal pathogens. Keeping the intakes free from bird droppings, especially those from pigeons, helps to minimize the concentration of fungal spores entering from the outside.⁹⁸

Accumulation of dust and moisture within HVAC systems increases the risk for spread of health-care-associated environmental fungi and bacteria. Clusters of infections caused by *Aspergillus* spp., *P. aeruginosa*, *S. aureus*, and *Acinetobacter* spp. have been linked to poorly maintained and/or malfunctioning air conditioning systems.^{68, 161, 257, 258} Efforts to limit excess humidity and moisture in the infrastructure and on air-stream surfaces in the HVAC system can minimize the proliferation and dispersion of fungal spores and waterborne bacteria throughout indoor air.²⁵⁹⁻²⁶² Within the HVAC system, water is present in water-wash units, humidifying boxes, or cooling units. The dual-duct system may also create conditions of high humidity and excess moisture that favor fungal growth in drain pans as well as in fibrous insulation material that becomes damp as a result of the humid air passing over the hot stream and condensing.

If moisture is present in the HVAC system, periods of stagnation should be avoided. Bursts of organisms can be released upon system start-up, increasing the risk of airborne infection.²⁰⁶ Proper engineering of the HVAC system is critical to preventing dispersal of airborne organisms. In one hospital, endophthalmitis caused by *Acremonium kiliense* infection following cataract extraction in an ambulatory surgical center was traced to aerosols derived from the humidifier water in the ventilation system.²⁰⁶ The organism proliferated because the ventilation system was turned off routinely when the

center was not in operation; the air was filtered before humidification, but not afterwards.

Most health-care facilities have contingency plans in case of disruption of HVAC services. These plans include back-up power generators that maintain the ventilation system in high-risk areas (e.g., operating rooms, intensive-care units, negative- and positive-pressure rooms, transplantation units, and oncology units). Alternative generators are required to engage within 10 seconds of a loss of main power. If the ventilation system is out of service, rendering indoor air stagnant, sufficient time must be allowed to clean the air and re-establish the appropriate number of ACH once the HVAC system begins to function again. Air filters may also need to be changed, because reactivation of the system can dislodge substantial amounts of dust and create a transient burst of fungal spores.

Duct cleaning in health-care facilities has benefits in terms of system performance, but its usefulness for infection control has not been conclusively determined. Duct cleaning typically involves using specialized tools to dislodge dirt and a high-powered vacuum cleaner to clean out debris.²⁶³ Some duct-cleaning services also apply chemical biocides or sealants to the inside surfaces of ducts to minimize fungal growth and prevent the release of particulate matter. The U.S. Environmental Protection Agency (EPA), however, has concerns with the use of sanitizers and/or disinfectants to treat the surfaces of ductwork, because the label indications for most of these products may not specifically include the use of the product in HVAC systems.²⁶⁴ Further, EPA has not evaluated the potency of disinfectants in such applications, nor has the agency examined the potential attendant health and safety risks. The EPA recommends that companies use only those chemical biocides that are registered for use in HVAC systems.²⁶⁴ Although infrequent cleaning of the exhaust ducts in AII areas has been documented as a cause of diminishing negative pressure and a decrease in the air exchange rates,²¹⁴ no data indicate that duct cleaning, beyond what is recommended for optimal performance, improves indoor air quality or reduces the risk of infection. Exhaust return systems should be cleaned as part of routine system maintenance. Duct cleaning has not been shown to prevent any health problems,²⁶⁵ and EPA studies indicate that airborne particulate levels do not increase as a result of dirty air ducts, nor do they diminish after cleaning, presumably because much of the dirt inside air ducts adheres to duct surfaces and does not enter the conditioned space.²⁶⁵ Additional research is needed to determine if air-duct contamination can significantly increase the airborne infection risk in general areas of health-care facilities.

4. Construction, Renovation, Remediation, Repair, and Demolition

a. General Information

Environmental disturbances caused by construction and/or renovation and repair activities (e.g., disruption of the above-ceiling area, running cables through the ceiling, and structural repairs) in and near health-care facilities markedly increase the airborne *Aspergillus* spp. spore counts in the indoor air of such facilities, thereby increasing the risk for health-care-associated aspergillosis among high-risk patients. Although one case of health-care-associated aspergillosis is often difficult to link to a specific environmental exposure, the occurrence of temporarily clustered cases increase the likelihood that an environmental source within the facility may be identified and corrected.

Table 7. Ventilation hazards in health-care facilities that may be associated with increased potential of airborne disease transmission*

Problem§	Consequences	Possible solutions
Water-damaged building materials (18, 266)	Water leaks can soak wood, wall board, insulation, wall coverings, ceiling tiles, and carpeting. All of these materials can provide microbial habitat when wet. This is especially true for fungi growing on gypsum board.	<ol style="list-style-type: none"> 1. Replace water-damaged materials. 2. Incorporate fungistatic compounds into building materials in areas at risk for moisture problems. 3. Test for all moisture and dry in less than 72 hours. Replace if the material cannot dry within 72 hours.
Filter bypasses (17)	Rigorous air filtration requires air flow resistance. Air stream will elude filtration if openings are present because of filter damage or poor fit.	<ol style="list-style-type: none"> 1. Use pressure gauges to ensure that filters are performing at proper static pressure. 2. Make ease of installation and maintenance criteria for filter selection. 3. Properly train maintenance personnel in HVAC concerns. 4. Design system with filters downstream from fans. 5. Avoid water on filters or insulation.
Improper fan setting (267)	Air must be delivered at design volume to maintain pressure balances. Air flow in special vent rooms reverses.	<ol style="list-style-type: none"> 1. Routinely monitor air flow and pressure balances throughout critical parts of HVAC system. 2. Minimize or avoid using rooms that switch between positive and negative pressure.
Ductwork disconnections (268)	Dislodged or leaky supply duct runs can spill into and leaky returns may draw from hidden areas. Pressure balance will be interrupted, and infectious material may be disturbed and entrained into hospital air supply.	<ol style="list-style-type: none"> 1. Design a ductwork system that is easy to access, maintain, and repair. 2. Train maintenance personnel to regularly monitor air flow volumes and pressure balances throughout the system. 3. Test critical areas for appropriate air flow
Air flow impedance (213)	Debris, structural failure, or improperly adjusted dampers can block duct work and prevent designed air flow.	<ol style="list-style-type: none"> 1. Design and budget for a duct system that is easy to inspect, maintain, and repair. 2. Alert contractors to use caution when working around HVAC systems during the construction phase. 3. Regularly clean exhaust grilles. 4. Provide monitoring for special ventilation areas.
Open windows (96, 247)	Open windows can alter fan-induced pressure balance and allow dirty-to-clean air flow.	<ol style="list-style-type: none"> 1. Use sealed windows. 2. Design HVAC systems to deliver sufficient outdoor dilution ventilation. 3. Ensure that OSHA indoor air quality standards are met.
Dirty window air conditioners (96, 269)	Dirt, moisture, and bird droppings can contaminate window air conditioners, which can then introduce infectious material into hospital rooms.	<ol style="list-style-type: none"> 1. Eliminate such devices in plans for new construction. 2. Where they must be used, make sure that they are routinely cleaned and inspected.

Problem§	Consequences	Possible solutions
Inadequate filtration (270)	Infectious particles may pass through filters into vulnerable patient areas.	<ol style="list-style-type: none"> 1. Specify appropriate filters during new construction design phase. 2. Make sure that HVAC fans are sized to overcome pressure demands of filter system. 3. Inspect and test filters for proper installation.
Maintenance disruptions (271)	Fan shut-offs, dislodged filter cake material contaminates downstream air supply and drain pans. This may compromise air flow in special ventilation areas.	<ol style="list-style-type: none"> 1. Budget for a rigorous maintenance schedule when designing a facility. 2. Design system for easy maintenance. 3. Ensure communication between engineering and maintenance personnel. 4. Institute an ongoing training program for all involved staff members.
Excessive moisture in the HVAC system (120)	Chronically damp internal lining of the HVAC system, excessive condensate, and drip pans with stagnant water may result from this problem.	<ol style="list-style-type: none"> 1. Locate duct humidifiers upstream of the final filters. 2. Identify a means to remove water from the system. 3. Monitor humidity; all duct take-offs should be downstream of the humidifiers so that moisture is absorbed completely. 4. Use steam humidifiers in the HVAC system.
Duct contamination (18, 272)	Debris is released during maintenance or cleaning.	<ol style="list-style-type: none"> 1. Provide point-of-use filtration in the critical areas. 2. Design air-handling systems with insulation of the exterior of the ducts. 3. Do not use fibrous sound attenuators. 4. Decontaminate or encapsulate contamination.

* Reprinted with permission of the publisher of reference 35 (Lippincott Williams and Wilkins).

§ Numbers in parentheses are reference citations.

Construction, renovation, repair, and demolition activities in health-care facilities require substantial planning and coordination to minimize the risk for airborne infection both during projects and after their completion. Several organizations and experts have endorsed a multi-disciplinary team approach (Box 4) to coordinate the various stages of construction activities (e.g., project inception, project implementation, final walk-through, and completion).^{120, 249, 250, 273–276} Environmental services, employee health, engineering, and infection control must be represented in construction planning and design meetings should be convened with architects and design engineers. The number of members and disciplines represented is a function of the complexity of a project. Smaller, less complex projects and maintenance may require a minimal number of members beyond the core representation from engineering, infection control, environmental services, and the directors of the specialized departments.

Box 4. Suggested members and functions of a multi-disciplinary coordination team for construction, renovation, repair, and demolition projects

Members

Infection-control personnel, including hospital epidemiologists
Laboratory personnel
Facility administrators or their designated representatives, facility managers
Director of engineering
Risk-management personnel
Directors of specialized programs (e.g., transplantation, oncology and ICU* programs)
Employee safety personnel, industrial hygienists, and regulatory affairs personnel
Environmental services personnel
Information systems personnel
Construction administrators or their designated representatives
Architects, design engineers, project managers, and contractors

Functions and responsibilities

Coordinate members' input in developing a comprehensive project management plan.
Conduct a risk assessment of the project to determine potential hazards to susceptible patients.
Prevent unnecessary exposures of patients, visitors, and staff to infectious agents.
Oversee all infection-control aspects of construction activities.
Establish site-specific infection-control protocols for specialized areas.
Provide education about the infection-control impact of construction to staff and construction workers.
Ensure compliance with technical standards, contract provisions, and regulations.
Establish a mechanism to address and correct problems quickly.
Develop contingency plans for emergency response to power failures, water supply disruptions, and fires.
Provide a water-damage management plan (including drying protocols) for handling water intrusion from floods, leaks, and condensation.
Develop a plan for structural maintenance.

* ICU is intensive care unit.

Education of maintenance and construction workers, health-care staff caring for high-risk patients, and persons responsible for controlling indoor air quality heightens awareness that minimizing dust and moisture intrusion from construction sites into high-risk patient-care areas helps to maintain a safe environment.^{120, 250, 271, 275–278} Visual and printed educational materials should be provided in the language spoken by the workers. Staff and construction workers also need to be aware of the potentially catastrophic consequences of dust and moisture intrusion when an HVAC system or water system fails during construction or repair; action plans to deal quickly with these emergencies should be developed in advance and kept on file. Incorporation of specific standards into construction contracts may help to prevent departures from recommended practices as projects progress. Establishing specific lines of communication is important to address problems (e.g., dust control, indoor air quality, noise levels, and vibrations), resolve complaints, and keep projects moving toward completion. Health-care facility staff should develop a mechanism to monitor worker adherence to infection-control guidelines on a daily basis in and around the construction site for the duration of the project.

b. Preliminary Considerations

The three major topics to consider before initiating any construction or repair activity are as follows: a) design and function of the new structure or area, b) assessment of environmental risks for airborne disease and opportunities for prevention, and c) measures to contain dust and moisture during construction or repairs. A checklist of design and function considerations can help to ensure that a planned structure or area can be easily serviced and maintained for environmental infection control (Box 5).^{17, 250, 273, 275–277} Specifications for the construction, renovation, remodeling, and maintenance of health-care facilities are outlined in the AIA document, *Guidelines for Design and Construction of Hospitals and Health Care Facilities*.^{120, 275}

Box 5. Construction design and function considerations for environmental infection control

Location of sinks and dispensers for handwashing products and hand hygiene products
Types of faucets (e.g., aerated vs. non-aerated)
Air-handling systems engineered for optimal performance, easy maintenance, and repair
ACH and pressure differentials to accommodate special patient-care areas
Location of fixed sharps containers
Types of surface finishes (e.g., porous vs. non-porous)
Well-caulked walls with minimal seams
Location of adequate storage and supply areas
Appropriate location of medicine preparations areas (e.g., ≥ 3 ft. from a sink)
Appropriate location and type of ice machines (e.g., preferably ice dispensers rather than ice bins)
Appropriate materials for sinks and wall coverings
Appropriate traffic flow (e.g., no “dirty” movement through “clean” areas)
Isolation rooms with anterooms as appropriate
Appropriate flooring (e.g., seamless floors in dialysis units)
Sensible use carpeting (e.g., avoiding use of carpeting in special care areas or areas likely to become wet)*
Convenient location of soiled utility areas
Properly engineered areas for linen services and solid waste management
Location of main generator to minimize the risk of system failure from flooding or other emergency
Installation guidelines for sheetrock

* Use of carpet cleaning methods (e.g., “bonneting”) that disperse microorganisms into the air may increase the risk of airborne infection among at-risk patients, especially if they are in the vicinity of the cleaning activity.¹¹¹

Proactive strategies can help prevent environmentally mediated airborne infections in health-care facilities during demolition, construction, and renovation. The potential presence of dust and moisture and their contribution to health-care-associated infections must be critically evaluated early in the planning of any demolition, construction, renovation, and repairs.^{120, 250, 251, 273, 274, 276–279} Consideration must extend beyond dust generated by major projects to include dust that can become airborne if disturbed during routine maintenance and minor renovation activities (e.g., exposure of ceiling spaces for inspection; installation of conduits, cable, or sprinkler systems; rewiring; and structural repairs or replacement).^{273, 276, 277} Other projects that can compromise indoor air quality include construction and repair jobs that inadvertently allow substantial amounts of raw, unfiltered outdoor air to enter the facility (e.g., repair of elevators and elevator shafts) and activities that dampen any structure, area, or item made of porous materials or characterized by cracks and crevices (e.g., sink cabinets in need of repair, carpets, ceilings, floors, walls, vinyl wall coverings, upholstery, drapes, and countertops).^{18, 273, 277} Molds grow and proliferate on these surfaces when they become and remain wet.^{21, 120, 250, 266, 270, 272, 280} Scrubbable

materials are preferred for use in patient-care areas.

Containment measures for dust and/or moisture control are dictated by the location of the construction site. Outdoor demolition and construction require actions to keep dust and moisture out of the facility (e.g., sealing windows and vents and keeping doors closed or sealed). Containment of dust and moisture generated from construction inside a facility requires barrier structures (either pre-fabricated or constructed of more durable materials as needed) and engineering controls to clean the air in and around the construction or repair site.

c. Infection-Control Risk Assessment

An infection-control risk assessment (ICRA) conducted before initiating repairs, demolition, construction, or renovation activities can identify potential exposures of susceptible patients to dust and moisture and determine the need for dust and moisture containment measures. This assessment centers on the type and extent of the construction or repairs in the work area but may also need to include adjacent patient-care areas, supply storage, and areas on levels above and below the proposed project. An example of designing an ICRA as a matrix, the policy for performing an ICRA and implementing its results, and a sample permit form that streamlines the communication process are available.²⁸¹ Knowledge of the air flow patterns and pressure differentials helps minimize or eliminate the inadvertent dispersion of dust that could contaminate air space, patient-care items, and surfaces.^{57, 282, 283} A recent aspergillosis outbreak among oncology patients was attributed to depressurization of the building housing the HSCT unit while construction was underway in an adjacent building. Pressure readings in the affected building (including 12 of 25 HSCT-patient rooms) ranged from 0.1 Pa–5.8 Pa. Unfiltered outdoor air flowed into the building through doors and windows, exposing patients in the HSCT unit to fungal spores.²⁸³ During long-term projects, providing temporary essential services (e.g., toilet facilities) and conveniences (e.g., vending machines) to construction workers within the site will help to minimize traffic in and out of the area. The type of barrier systems necessary for the scope of the project must be defined.^{12, 120, 250, 279, 284}

Depending on the location and extent of the construction, patients may need to be relocated to other areas in the facility not affected by construction dust.^{51, 285} Such relocation might be especially prudent when construction takes place within units housing immunocompromised patients (e.g., severely neutropenic patients and patients on corticosteroid therapy). Advance assessment of high-risk locations and planning for the possible transport of patients to other departments can minimize delays and waiting time in hallways.⁵¹ Although hospitals have provided immunocompromised patients with some form of respiratory protection for use outside their rooms, the issue is complex and remains unresolved until more research can be done. Previous guidance on this issue has been inconsistent.⁹ Protective respirators (i.e., N95) were well tolerated by patients when used to prevent further cases of construction-related aspergillosis in a recent outbreak.²⁸³ The routine use of the N95 respirator by patients, however, has not been evaluated for preventing exposure to fungal spores during periods of non-construction. Although health-care workers who would be using the N95 respirator for personal respiratory protection must be fit-tested, there is no indication that either patients or visitors should undergo fit-testing.

Surveillance activities should augment preventive strategies during construction projects.^{3, 4, 20, 110, 286, 287} By determining baseline levels of health-care-acquired airborne and waterborne infections, infection-control staff can monitor changes in infection rates and patterns during and immediately after construction, renovations, or repairs.³

d. Air Sampling

Air sampling in health-care facilities may be conducted both during periods of construction and on a periodic basis to determine indoor air quality, efficacy of dust-control measures, or air-handling system performance via parametric monitoring. Parametric monitoring consists of measuring the physical

performance of the HVAC system in accordance with the system manufacturer's specifications. A periodic assessment of the system (e.g., air flow direction and pressure, ACH, and filter efficiency) can give assurance of proper ventilation, especially for special care areas and operating rooms.²⁸⁸

Air sampling is used to detect aerosols (i.e., particles or microorganisms). Particulate sampling (i.e., total numbers and size range of particulates) is a practical method for evaluating the infection-control performance of the HVAC system, with an emphasis on filter efficiency in removing respirable particles (<5 µm in diameter) or larger particles from the air. Particle size is reported in terms of the mass median aerodynamic diameter (MMAD), whereas count median aerodynamic diameter (CMAD) is useful with respect to particle concentrations.

Particle counts in a given air space within the health-care facility should be evaluated against counts obtained in a comparison area. Particle counts indoors are commonly compared with the particulate levels of the outdoor air. This approach determines the "rank order" air quality from "dirty" (i.e., the outdoor air) to "clean" (i.e., air filtered through high-efficiency filters [90%–95% filtration]) to "cleanest" (i.e., HEPA-filtered air).²⁸⁸ Comparisons from one indoor area to another may also provide useful information about the magnitude of an indoor air-quality problem. Making rank-order comparisons between clean, highly-filtered areas and dirty areas and/or outdoors is one way to interpret sampling results in the absence of air quality and action level standards.^{35, 289}

In addition to verifying filter performance, particle counts can help determine if barriers and efforts to control dust dispersion from construction are effective. This type of monitoring is helpful when performed at various times and barrier perimeter locations during the project. Gaps or breaks in the barriers' joints or seals can then be identified and repaired. The American Conference of Governmental Industrial Hygienists (ACGIH) has set a threshold limit value-time weighted average (TLV®-TWA) of 10 mg/m³ for nuisance dust that contains no asbestos and <1% crystalline silica.²⁹⁰ Alternatively, OSHA has set permissible exposure limits (PELs) for inert or nuisance dust as follows: respirable fraction at 5 mg/m³ and total dust at 15 mg/m³.²⁹¹ Although these standards are not measures of a bioaerosol, they are used for indoor air quality assessment in occupational settings and may be useful criteria in construction areas. Application of ACGIH guidance to health-care settings has not been standardized, but particulate counts in health-care facilities are likely to be well below this threshold value and approaching clean-room standards in certain care areas (e.g., operating rooms).¹⁰⁰

Particle counters and anemometers are used in particulate evaluation. The anemometer measures air flow velocity, which can be used to determine sample volumes. Particulate sampling usually does not require microbiology laboratory services for the reporting of results.

Microbiologic sampling of air in health-care facilities remains controversial because of currently unresolved technical limitations and the need for substantial laboratory support (Box 6). Infection-control professionals, laboratorians, and engineers should determine if microbiologic and/or particle sampling is warranted and assess proposed methods for sampling. The most significant technical limitation of air sampling for airborne fungal agents is the lack of standards linking fungal spore levels with infection rates. Despite this limitation, several health-care institutions have opted to use microbiologic sampling when construction projects are anticipated and/or underway in efforts to assess the safety of the environment for immunocompromised patients.^{35, 289} Microbiologic air sampling should be limited to assays for airborne fungi; of those, the thermotolerant fungi (i.e., those capable of growing at 95°F–98.6°F [35°C–37°C]) are of particular concern because of their pathogenicity in immunocompromised hosts.³⁵ Use of selective media (e.g., Sabouraud dextrose agar and inhibitory mold agar) helps with the initial identification of recovered organisms.

Microbiologic sampling for fungal spores performed as part of various airborne disease outbreak

investigations has also been problematic.^{18, 49, 106, 111, 112, 289} The precise source of a fungus is often difficult to trace with certainty, and sampling conducted after exposure may neither reflect the circumstances that were linked to infection nor distinguish between health-care–acquired and community-acquired infections. Because fungal strains may fluctuate rapidly in the environment, health-care–acquired *Aspergillus* spp. infection cannot be confirmed or excluded if the infecting strain is not found in the health-care setting.²⁸⁷ Sensitive molecular typing methods (e.g., randomly amplified polymorphic DNA (RAPD) techniques and a more recent DNA fingerprinting technique that detects restriction fragment length polymorphisms in fungal genomic DNA) to identify strain differences among *Aspergillus* spp., however, are becoming increasingly used in epidemiologic investigations of health-care–acquired fungal infection (A. Streifel, University of Minnesota, 2000).^{68, 110, 286, 287, 292–296} During case cluster evaluation, microbiologic sampling may provide an isolate from the environment for molecular typing and comparison with patient isolates. Therefore, it may be prudent for the clinical laboratory to save *Aspergillus* spp. isolated from colonizations and invasive disease cases among patients in PE, oncology, and transplant services for these purposes.

Box 6. Unresolved issues associated with microbiologic air sampling*

Lack of standards linking fungal spore levels with infection rates (i.e., no safe level of exposure)
Lack of standard protocols for testing (e.g., sampling intervals, number of samples, sampling locations)
Need for substantial laboratory support
Culture issues (e.g., false negatives, insensitivity, lag time between sampling and recording the results)
New, complex polymerase chain reaction (PCR) analytical methods
Unknown incubation period for *Aspergillus* spp. infection
Variability of sampler readings
Sensitivity of the sampler used (i.e., the volumes of air sampled)
Lack of details in the literature about describing sampling circumstances (e.g., unoccupied rooms vs. ongoing activities in rooms, expected fungal concentrations, and rate of outdoor air penetration)
Lack of correlation between fungal species and strains from the environment and clinical specimens
Confounding variables with high-risk patients (e.g., visitors and time spent outside of protective environment [PE] without respiratory protection)
Need for determination of ideal temperature for incubating fungal cultures (95°F [35°C] is the most commonly used temperature)

* Material in this box is compiled from references 35, 100, 222, 289, and 297.

Sedimentation methods using settle plates and volumetric sampling methods using solid impactors are commonly employed when sampling air for bacteria and fungi. Settle plates have been used by numerous investigators to detect airborne bacteria or to measure air quality during medical procedures (e.g., surgery).^{17, 60, 97, 151, 161, 287} Settle plates, because they rely on gravity during sampling, tend to select for larger particles and lack sensitivity for respirable particles (e.g., individual fungal spores), especially in highly-filtered environments. Therefore, they are considered impractical for general use.^{35, 289, 298–301} Settle plates, however, may detect fungi aerosolized during medical procedures (e.g., during wound dressing changes), as described in a recent outbreak of aspergillosis among liver transplant patients.³⁰²

The use of slit or sieve impactor samplers capable of collecting large volumes of air in short periods of time are needed to detect low numbers of fungal spores in highly filtered areas.^{35, 289} In some

outbreaks, aspergillosis cases have occurred when fungal spore concentrations in PE ambient air ranged as low as 0.9–2.2 colony-forming units per cubic meter (CFU/m³) of air.^{18, 94} On the basis of the expected spore counts in the ambient air and the performance parameters of various types of volumetric air samplers, investigators of a recent aspergillosis outbreak have suggested that an air volume of at least 1000 L (1 m³) should be considered when sampling highly filtered areas.²⁸³ Investigators have also suggested limits of 15 CFU/m³ for gross colony counts of fungal organisms and <0.1 CFU/m³ for *Aspergillus fumigatus* and other potentially opportunistic fungi in heavily filtered areas (≥12 ACH and filtration of ≥99.97% efficiency).¹²⁰ No correlation of these values with the incidence of health-care-associated fungal infection rates has been reported.

Air sampling in health-care facilities, whether used to monitor air quality during construction, to verify filter efficiency, or to commission new space prior to occupancy, requires careful notation of the circumstances of sampling. Most air sampling is performed under undisturbed conditions. However, when the air is sampled during or after human activity (e.g., walking and vacuuming), a higher number of airborne microorganisms likely is detected.²⁹⁷ The contribution of human activity to the significance of air sampling and its impact on health-care-associated infection rates remain to be defined. Comparing microbiologic sampling results from a target area (e.g., an area of construction) to those from an unaffected location in the facility can provide information about distribution and concentration of potential airborne pathogens. A comparison of microbial species densities in outdoor air versus indoor air has been used to help pinpoint fungal spore bursts. Fungal spore densities in outdoor air are variable, although the degree of variation with the seasons appears to be more dramatic in the United States than in Europe.^{92, 287, 303}

Particulate and microbiologic air sampling have been used when commissioning new HVAC system installations; however, such sampling is particularly important for newly constructed or renovated PE or operating rooms. Particulate sampling is used as part of a battery of tests to determine if a new HVAC system is performing to specifications for filtration and the proper number of ACH.^{268, 288, 304} Microbiologic air sampling, however, remains controversial in this application, because no standards for comparison purposes have been determined. If performed, sampling should be limited to determining the density of fungal spores per unit volume of air space. High numbers of spores may indicate contamination of air-handling system components prior to installation or a system deficiency when culture results are compared with known filter efficiencies and rates of air exchange.

e. External Demolition and Construction

External demolition, planned building implosions, and dirt excavation generate considerable dust and debris that can contain airborne microorganisms. In one study, peak concentrations in outdoor air at grade level and HVAC intakes during site excavation averaged 20,000 CFU/m³ for all fungi and 500 CFU/m³ for *Aspergillus fumigatus*, compared with 19 CFU/m³ and 4 CFU/m³, respectively, in the absence of construction.²⁸⁰ Many health-care institutions are located in large, urban areas; building implosions are becoming a more frequent concern. Infection-control risk assessment teams, particularly those in facilities located in urban renewal areas, would benefit by developing risk management strategies for external demolition and construction as a standing policy. In light of the events of 11 September 2001, it may be necessary for the team to identify those dust exclusion measures that can be implemented rapidly in response to emergency situations (Table 8). Issues to be reviewed prior to demolition include a) proximity of the air intake system to the work site, b) adequacy of window seals and door seals, c) proximity of areas frequented by immunocompromised patients, and d) location of the underground utilities (D. Erickson, ASHE, 2000).^{120, 250, 273, 276, 277, 280, 305}

Table 8. Strategies to reduce dust and moisture intrusion during external demolition and construction

<i>Item</i>	<i>Recommendation</i>
Demolition site	<ul style="list-style-type: none"> ● Shroud the site if possible to reduce environmental contamination.
Dust-generating equipment	<ul style="list-style-type: none"> ● Prior to placing dust-generating equipment, evaluate the location to ensure that dust produced by the equipment will not enter the building through open doorways or windows, or through ventilation air intakes.
Construction materials storage	<ul style="list-style-type: none"> ● Locate this storage away from the facility and ventilation air intakes.
Adjacent air intakes HVAC system	<ul style="list-style-type: none"> ● Seal off affected intakes, if possible, or move if funds permit. ● Consult with the facility engineer about pressure differentials and air recirculation options; keep facility air pressure positive to outside air.
Filters	<ul style="list-style-type: none"> ● Ensure that filters are properly installed; change roughing filters frequently to prevent dust build-up on high-efficiency filters.
Windows	<ul style="list-style-type: none"> ● Seal and caulk to prevent entry of airborne fungal spores.
Doors	<ul style="list-style-type: none"> ● Keep closed as much as possible; do not prop open; seal and caulk unused doors (i.e., those that are not designated as emergency exits); use mats with tacky surfaces at outside entrances.
Water utilities	<ul style="list-style-type: none"> ● Note location relative to construction area to prevent intrusion of dust into water systems.*
Medical gas piping	<ul style="list-style-type: none"> ● Ensure that these lines/pipes are insulated during periods of vibration.
Rooftops	<ul style="list-style-type: none"> ● Temporarily close off during active demolition/construction those rooftop areas that are normally open to the public (e.g., rooftop atrium).
Dust generation	<ul style="list-style-type: none"> ● Provide methods (e.g., misting the area with water) to minimize dust.
Immunocompromised patients	<ul style="list-style-type: none"> ● Use walk-ways protected from demolition/construction sites; avoid outside areas close to these sites; avoid rooftops.
Pedestrian traffic	<ul style="list-style-type: none"> ● Close off entry ways as needed to minimize dust intrusion.
Truck traffic	<ul style="list-style-type: none"> ● Reroute if possible, or arrange for frequent street cleaning.
Education and awareness+	<ul style="list-style-type: none"> ● Encourage reporting of hazardous or unsafe incidents associated with construction.

* Contamination of water pipes during demolition activities has been associated with health-care-associated transmission of *Legionella* spp.³⁰⁵

+ When health-care facilities have immunosuppressed patients in their census, telephoning the city building department each month to find out if buildings are scheduled for demolition is prudent.

Minimizing the entry of outside dust into the HVAC system is crucial in reducing the risk for airborne contaminants. Facility engineers should be consulted about the potential impact of shutting down the system or increasing the filtration. Selected air handlers, especially those located close to excavation sites, may have to be shut off temporarily to keep from overloading the system with dust and debris. Care is needed to avoid significant facility-wide reductions in pressure differentials that may cause the building to become negatively pressured relative to the outside. To prevent excessive particulate overload and subsequent reductions in effectiveness of intake air systems that cannot be shut off temporarily, air filters must be inspected frequently for proper installation and function. Excessive dust

penetration can be avoided if recirculated air is maximally utilized while outdoor air intakes are shut down. Scheduling demolition and excavation during the winter, when *Aspergillus* spp. spores may be present in lower numbers, can help, although seasonal variations in spore density differ around the world.^{92, 287, 303} Dust control can be managed by misting the dirt and debris during heavy dust-generating activities. To decrease the amount of aerosols from excavation and demolition projects, nearby windows, especially in areas housing immunocompromised patients, can be sealed and window and door frames caulked or weather-stripped to prevent dust intrusion.^{50, 301, 306} Monitoring for adherence to these control measures throughout demolition or excavation is crucial. Diverting pedestrian traffic away from the construction sites decreases the amount of dust tracked back into the health-care facility and minimizes exposure of high-risk patients to environmental pathogens. Additionally, closing entrances near construction or demolition sites might be beneficial; if this is not practical, creating an air lock (i.e., pressurizing the entry way) is another option.

f. Internal Demolition, Construction, Renovations, and Repairs

The focus of a properly implemented infection-control program during interior construction and repairs is containment of dust and moisture. This objective is achieved by a) educating construction workers about the importance of control measures; b) preparing the site; c) notifying and issuing advisories for staff, patients, and visitors; d) moving staff and patients and relocating patients as needed; e) issuing standards of practice and precautions during activities and maintenance; f) monitoring for adherence to control measures during construction and providing prompt feedback about lapses in control; g) monitoring HVAC performance; h) implementing daily clean-up, terminal cleaning and removal of debris upon completion; and i) ensuring the integrity of the water system during and after construction. These activities should be coordinated with engineering staff and infection-control professionals.

Physical barriers capable of containing smoke and dust will confine dispersed fungal spores to the construction zone.^{279, 284, 307, 308} The specific type of physical barrier required depends on the project's scope and duration and on local fire codes. Short-term projects that result in minimal dust dispersion (e.g., installation of new cables or wiring above ceiling tiles) require only portable plastic enclosures with negative pressure and HEPA filtration of the exhaust air from the enclosed work area. The placement of a portable industrial-grade HEPA filter device capable of filtration rate of 300–800 ft³/min. adjacent to the work area will help to remove fungal spores, but its efficacy is dependent on the supplied ACH and size of the area. If the project is extensive but short-term, dust-abatement, fire-resistant plastic curtains (e.g., Visqueen®) may be adequate. These should be completely airtight and sealed from ceiling to floor with overlapping curtains;^{276, 277, 309} holes, tears, or other perforations should be repaired promptly with tape. A portable, industrial-grade HEPA filter unit on continuous operation is needed within the contained area, with the filtered air exhausted to the outside of the work zone. Patients should not remain in the room when dust-generating activities are performed. Tools to assist the decision-making process regarding selection of barriers based on an ICRA approach are available.²⁸¹

More elaborate barriers are indicated for long-term projects that generate moderate to large amounts of dust. These barrier structures typically consist of rigid, noncombustible walls constructed from sheet rock, drywall, plywood, or plaster board and covered with sheet plastic (e.g., Visqueen®). Barrier requirements to prevent the intrusion of dust into patient-care areas include a) installing a plastic dust abatement curtain before construction of the rigid barrier; b) sealing and taping all joint edges including the top and bottom; c) extending the barrier from floor to floor, which takes into account the space [approximately 2–8 ft.] above the finished, lay-down ceiling; and d) fitting or sealing any temporary doors connecting the construction zone to the adjacent area. (See Box 7 for a list of the various construction and repair activities that require the use of some type of barrier.)

Box 7. Construction/repair projects that require barrier structures*

Demolition of walls, wallboard, plaster, ceramic tiles, ceiling tiles, and ceilings
Removal of flooring and carpeting, windows and doors, and casework
Working with sinks and plumbing that could result in aerosolization of water in high-risk areas
Exposure of ceiling spaces for demolition and for installation or rerouting of utility services (e.g., rewiring, electrical conduction installation, HVAC ductwork, and piping)
Crawling into ceiling spaces for inspection in a manner that may dislodge dust
Demolition, repair, or construction of elevator shafts
Repairing water damage

* Material for this box was compiled from references 120, 250, 273, 276, and 277.

Dust and moisture abatement and control rely primarily on the impermeable barrier containment approach; as construction continues, numerous opportunities can lead to dispersion of dust to other areas of the health-care facility. Infection-control measures that augment the use of barrier containment should be undertaken (Table 9).

Dust-control measures for clinical laboratories are an essential part of the infection-control strategy during hospital construction or renovation. Use of plastic or solid barriers may be needed if the ICRA determines that air flow from construction areas may introduce airborne contaminants into the laboratory space. In one facility, pseudofungemia clusters attributed to *Aspergillus* spp. and *Penicillium* spp. were linked to improper air flow patterns and construction projects adjacent to the laboratory; intrusion of dust and spores into a biological safety cabinet from construction activity immediately next to the cabinet resulted in a cluster of cultures contaminated with *Aspergillus niger*.^{310, 311} Reportedly, no barrier containment was used and the HEPA filtration system was overloaded with dust. In addition, an outbreak of pseudobacteremia caused by *Bacillus* spp. occurred in another hospital during construction above a storage area for blood culture bottles.²⁰⁷ Airborne spread of *Bacillus* spp. spores resulted in contamination of the bottles' plastic lids, which were not disinfected or handled with proper aseptic technique prior to collection of blood samples.

Table 9. Infection-control measures for internal construction and repair projects*+

Infection-control measure	Steps for implementation
Prepare for the project.	<ol style="list-style-type: none"> 1. Use a multi-disciplinary team approach to incorporate infection control into the project. 2. Conduct the risk assessment and a preliminary walk-through with project managers and staff.
Educate staff and construction workers.	<ol style="list-style-type: none"> 1. Educate staff and construction workers about the importance of adhering to infection-control measures during the project. 2. Provide educational materials in the language of the workers. 3. Include language in the construction contract requiring construction workers and subcontractors to participate in infection-control training.
Issue hazard and warning notices.	<ol style="list-style-type: none"> 1. Post signs to identify construction areas and potential hazards. 2. Mark detours requiring pedestrians to avoid the work area.
Relocate high-risk patients as needed, especially if the construction is in or adjacent to a PE area.	<ol style="list-style-type: none"> 1. Identify target patient populations for relocation based on the risk assessment. 2. Arrange for the transfer in advance to avoid delays. 3. At-risk patients should wear protective respiratory equipment (e.g., a high-efficiency mask) when outside their PE rooms.
Establish alternative traffic patterns for staff, patients, visitors, and construction workers.	<ol style="list-style-type: none"> 1. Determine appropriate alternate routes from the risk assessment. 2. Designate areas (e.g., hallways, elevators, and entrances/exits) for construction-worker use. 3. Do not transport patients on the same elevator with construction materials and debris.

Infection-control measure	Steps for implementation
Erect appropriate barrier containment.	<ol style="list-style-type: none"> 1. Use prefabricated plastic units or plastic sheeting for short-term projects that will generate minimal dust. 2. Use durable rigid barriers for ongoing, long-term projects.
Establish proper ventilation.	<ol style="list-style-type: none"> 1. Shut off return air vents in the construction zone, if possible, and seal around grilles. 2. Exhaust air and dust to the outside, if possible. 3. If recirculated air from the construction zone is unavoidable, use a pre-filter and a HEPA filter before the air returns to the HVAC system. 4. When vibration-related work is being done that may dislodge dust in the ventilation system or when modifications are made to ductwork serving occupied spaces, install filters on the supply air grilles temporarily. 5. Set pressure differentials so that the contained work area is under negative pressure. 6. Use air flow monitoring devices to verify the direction of the air pattern. 7. Exhaust air and dust to the outside, if possible. 8. Monitor temperature, air changes per hour (ACH), and humidity levels (humidity levels should be <65%). 9. Use portable, industrial grade HEPA filters in the adjacent area and/or the construction zone for additional ACH. 10. Keep windows closed, if possible.
Control solid debris.	<ol style="list-style-type: none"> 1. When replacing filters, place the old filter in a bag prior to transport and dispose as a routine solid waste. 2. Clean the construction zone daily or more often as needed. 3. Designate a removal route for small quantities of solid debris. 4. Mist debris and cover disposal carts before transport (i.e., leaving the construction zone). 5. Designate an elevator for construction crew use. 6. Use window chutes and negative pressure equipment for removal of larger pieces of debris while maintaining pressure differentials in the construction zone. 7. Schedule debris removal to periods when patient exposures to dust is minimal.
Control water damage.	<ol style="list-style-type: none"> 1. Make provisions for dry storage of building materials. 2. Do not install wet, porous building materials (i.e., sheet rock). 3. Replace water-damaged porous building materials if they cannot be completely dried out within 72 hours.
Control dust in air and on surfaces.	<ol style="list-style-type: none"> 1. Monitor the construction area daily for compliance with the infection-control plan. 2. Protective outer clothing for construction workers should be removed before entering clean areas. 3. Use mats with tacky surfaces within the construction zone at the entry; cover sufficient area so that both feet make contact with the mat while walking through the entry. 4. Construct an anteroom as needed where coveralls can be donned and removed. 5. Clean the construction zone and all areas used by construction workers with a wet mop. 6. If the area is carpeted, vacuum daily with a HEPA-filtered–equipped vacuum. 7. Provide temporary essential services (e.g., toilets) and worker conveniences (e.g., vending machines) in the construction zone as appropriate. 8. Damp-wipe tools if removed from the construction zone or left in the area. 9. Ensure that construction barriers remain well sealed; use particle sampling as needed. 10. Ensure that the clinical laboratory is free from dust contamination.

Infection-control measure	Steps for implementation
Complete the project.	<ol style="list-style-type: none"> 1. Flush the main water system to clear dust-contaminated lines. 2. Terminally clean the construction zone before the construction barriers are removed. 3. Check for visible mold and mildew and eliminate (i.e., decontaminate and remove), if present. 4. Verify appropriate ventilation parameters for the new area as needed. 5. Do not accept ventilation deficiencies, especially in special care areas. 6. Clean or replace HVAC filters using proper dust-containment procedures. 7. Remove the barriers and clean the area of any dust generated during this work. 8. Ensure that the designated air balances in the operating rooms (OR) and protective environments (PE) are achieved before occupancy. 9. Commission the space as indicated, especially in the OR and PE, ensuring that the room's required engineering specifications are met.

* Material in this table includes information from D. Erickson, ASHE, 2000.

+ Material in this table was compiled from references 19, 51, 67, 80, 106, 120, 250, 266, 273, 276–278, 280, 285, and 309, 312–315.

5. Environmental Infection-Control Measures for Special Health-Care Settings

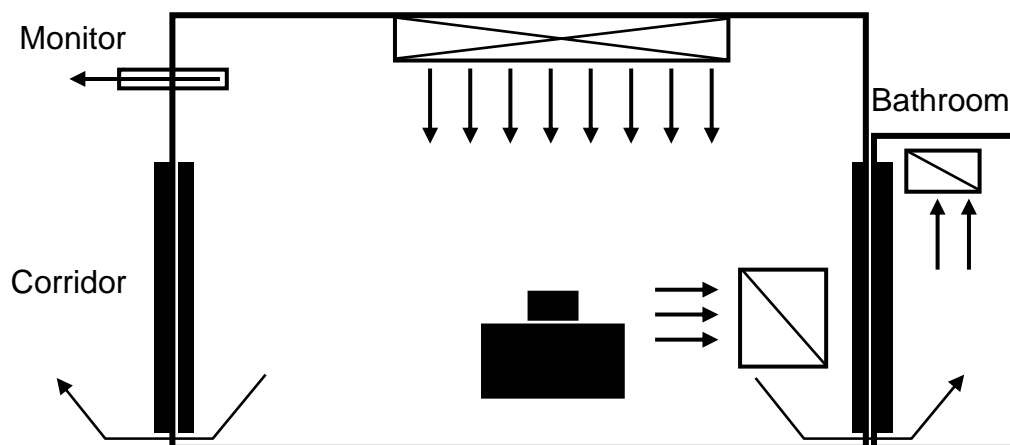
Areas in health-care facilities that require special ventilation include a) operating rooms; b) PE rooms used by high-risk, immunocompromised patients; and c) AII rooms for isolation of patients with airborne infections (e.g., those caused by *M. tuberculosis*, VZV, or measles virus). The number of rooms required for PE and AII are determined by a risk assessment of the health-care facility.⁶ Continuous, visual monitoring of air flow direction is required for new or renovated pressurized rooms.^{120, 256}

a. Protective Environments (PE)

Although the exact configuration and specifications of PEs might differ among hospitals, these care areas for high-risk, immunocompromised patients are designed to minimize fungal spore counts in air by maintaining a) filtration of incoming air by using central or point-of-use HEPA filters; b) directed room air flow [i.e., from supply on one side of the room, across the patient, and out through the exhaust on the opposite side of the room]; c) positive room air pressure of 2.5 Pa [0.01" water gauge] relative to the corridor; d) well-sealed rooms; and e) ≥ 12 ACH.^{44, 120, 251, 254, 316–319} Air flow rates must be adjusted accordingly to ensure sufficient ACH, and these rates vary depending on certain factors (e.g., room air leakage area). For example, to provide ≥ 12 ACH in a typical patient room with 0.5 sq. ft. air leakage, the air flow rate will be minimally 125 cubic feet/min (cfm).^{320, 321} Higher air flow rates may be needed. A general ventilation diagram for a positive-pressure room is given in Figure 2. Directed room air flow in PE rooms is not laminar; parallel air streams are not generated. Studies attempting to demonstrate patient benefit from laminar air flow in a PE setting are equivocal.^{316, 318, 319, 322–327}

Air flow direction at the entrances to these areas should be maintained and verified, preferably on a daily basis, using either a visual means of indication (e.g., smoke tubes and flutter strips) or manometers. Permanent installation of a visual monitoring device is indicated for new PE construction and renovation.¹²⁰ Facility service structures can interfere with the proper unidirectional air flow from the patients' rooms to the adjacent corridor. In one outbreak investigation, *Aspergillus* spp. infections in a critical care unit may have been associated with a pneumatic specimen transport system, a textile disposal duct system, and central vacuum lines for housekeeping, all of which disrupted proper air flow from the patients' rooms to the outside and allowed entry of fungal spores into the unit (M.McNeil, CDC, 2000).

Figure 2. Example of positive-pressure room control for protection from airborne environmental microbes (PE)* + §



* Stacked black boxes represent patient's bed. Long open box with cross-hatch represents supply air. Open boxes with single, diagonal slashes represent air exhaust registers. Arrows indicate directions of air flow.

+ Possible uses include immunocompromised patient rooms (e.g., hematopoietic stem cell transplant or solid organ transplant procedure rooms) and orthopedic operating rooms.

§ Positive-pressure room engineering features include

- positive pressure (greater supply than exhaust air volume);
- pressure differential range of 2.5–8 Pa (0.01–0.03-in. water gauge), ideal at 8 Pa;
- air flow volume differential >125-cfm supply versus exhaust;
- sealed room, approximately 0.5-sq. ft. leakage;
- clean to dirty air flow;
- monitoring;
- ≥ 12 air changes per hour (ACH); and
- return air if refiltered.

¶ This diagram is a generic illustration of air flow in a typical installation. Alternative air flow arrangements are recognized. Adapted and used with permission from A. Streifel and the publisher of reference 328 (Penton Media, Inc.)

The use of surface fungicide treatments is becoming more common, especially for building materials.³²⁹ Copper-based compounds have demonstrated anti-fungal activity and are often applied to wood or paint. Copper-8-quinolinolate was used on environmental surfaces contaminated with *Aspergillus* spp. to control one reported outbreak of aspergillosis.³¹⁰ The compound was also incorporated into the fireproofing material of a newly constructed hospital to help decrease the environmental spore burden.³¹⁶

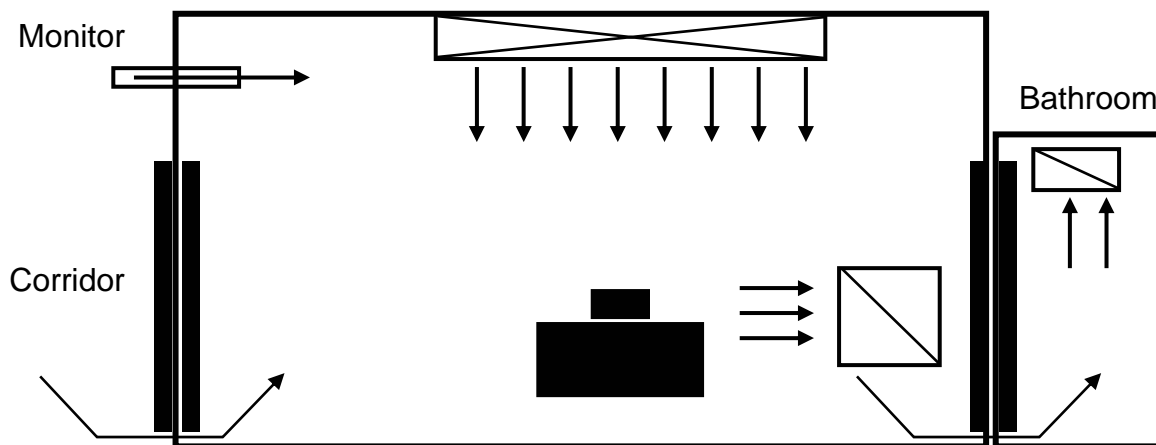
b. Airborne Infection Isolation (AII)

Acute-care inpatient facilities need at least one room equipped to house patients with airborne infectious disease. Every health-care facility, including ambulatory and long-term care facilities, should undertake an ICRA to identify the need for AII areas. Once the need is established, the appropriate ventilation equipment can be identified. Air handling systems for this purpose need not be restricted to central systems. Guidelines for the prevention of health-care-acquired TB have been published in response to multiple reports of health-care-associated transmission of multi-drug resistant strains.^{4, 330} In reports documenting health-care-acquired TB, investigators have noted a failure to comply fully with prevention measures in established guidelines.³³¹⁻³⁴⁵ These gaps highlight the importance of prompt recognition of the disease, isolation of patients, proper treatment, and engineering controls. AII rooms

are also appropriate for the care and management of smallpox patients.⁶ Environmental infection control with respect to smallpox is currently being revisited (see Appendix E).

Salient features of engineering controls for AII areas include a) use of negative pressure rooms with close monitoring of air flow direction using manometers or temporary or installed visual indicators [e.g., smoke tubes and flutter strips] placed in the room with the door closed; b) minimum 6 ACH for existing facilities, ≥ 12 ACH for areas under renovation or for new construction; and c) air from negative pressure rooms and treatment rooms exhausted directly to the outside if possible.^{4, 120, 248} As with PE, airflow rates need to be determined to ensure the proper numbers of ACH.^{320, 321} AII rooms can be constructed either with (Figure 3) or without (Figure 4) an anteroom. When the recirculation of air from AII rooms is unavoidable, HEPA filters should be installed in the exhaust duct leading from the room to the general ventilation system. In addition to UVGI fixtures in the room, UVGI can be placed in the ducts as an adjunct measure to HEPA filtration, but it can not replace the HEPA filter.^{4, 346} A UVGI fixture placed in the upper room, coupled with a minimum of 6 ACH, also provides adequate air cleaning.²⁴⁸

Figure 3. Example of negative-pressure room control for airborne infection isolation (AII)* + §¶



* Stacked black boxes represent patient's bed. Long open box with cross-hatch represents supply air. Open boxes with single, diagonal slashes represent air exhaust registers. Arrows indicate direction of air flow.

+ Possible uses include treatment or procedure rooms, bronchoscopy rooms, and autopsy.

§ Negative-pressure room engineering features include

- negative pressure (greater exhaust than supply air volume);
- pressure differential of 2.5 Pa (0.01-in. water gauge);
- air flow volume differential >125 -cfm exhaust versus supply;
- sealed room, approximately 0.5-sq. ft. leakage;
- clean to dirty air flow;
- monitoring;
- ≥ 12 air changes per hour (ACH) new or renovation, 6 ACH existing; and
- exhaust to outside or HEPA-filtered if recirculated.

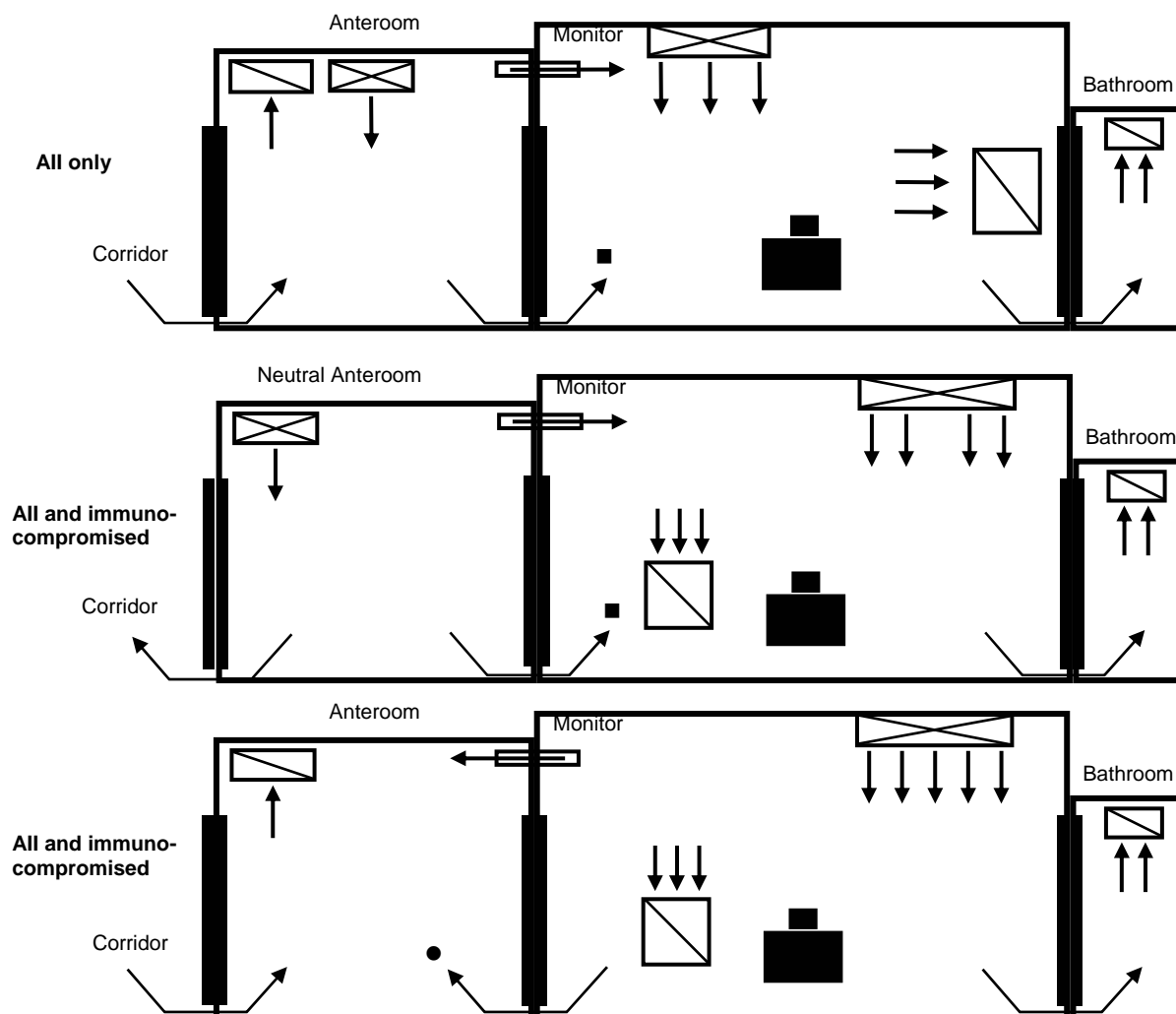
¶ This diagram is a generic illustration of air flow in a typical installation. Alternative air flow arrangements are recognized. Adapted and used with permission from A. Streifel and the publisher of reference 328 (Penton Media, Inc.)

One of the components of airborne infection isolation is respiratory protection for health-care workers and visitors when entering AII rooms.^{4, 6, 347} Recommendations of the type of respiratory protection are dependent on the patient's airborne infection (indicating the need for AII) and the risk of infection to

persons entering the AII room. A more in-depth discussion of respiratory protection in this instance is presented in the current isolation guideline;⁶ a revision of this guideline is in development. Cough-inducing procedures (e.g., endotracheal intubation and suctioning of known or suspected TB patients, diagnostic sputum induction, aerosol treatments, and bronchoscopy) require similar precautions.^{348–350}

Additional engineering measures are necessary for the management of patients requiring PE (i.e., allogeneic HSCT patients) who concurrently have airborne infection. For this type of patient treatment, an anteroom (Figure 4) is required in new construction and renovation as per AIA guidelines.¹²⁰

Figure 4. Example of airborne infection isolation (AII) room with anteroom and neutral anteroom* + §



* The top diagram indicates air flow patterns when patient with only airborne infectious disease occupies room. Middle and bottom diagrams indicate recommended air flow patterns when room is occupied by immunocompromised patient with airborne infectious disease. Stacked black boxes represent patient beds. Long open boxes with cross-hatches represent supply air. Open boxes with single, diagonal slashes represent air exhaust registers. Arrows indicate directions of air flow.

+ AII isolation room with anteroom engineering features include

- pressure differential of 2.5 Pa (0.01-in. water gauge) measured at the door between patient room and anteroom;
- air flow volume differential >125-cfm, depending on anteroom air flow direction (pressurized versus depressurized);

- sealed room with approximately 0.5-sq. ft. leakage;
- clean to dirty air flow
- monitoring;
- ≥ 12 air changes per hour (ACH) new or renovation, 6 ACH existing; and
- anteroom air flow patterns. The small ■ in panels 1 and 2 indicate the anteroom is pressurized (supply versus exhaust), while the small • in panel 3 indicates the anteroom is depressurized (exhaust versus supply).

§ Used with permission of A. Streifel, University of Minnesota

The pressure differential of an anteroom can be positive or negative relative to the patient in the room.¹²⁰ An anteroom can act as an airlock (Figure 4). If the anteroom is positive relative to the air space in the patient's room, staff members do not have to mask prior to entry into the anteroom if air is directly exhausted to the outside and a minimum of 10 ACH (Figure 4, top diagram).¹²⁰ When an anteroom is negative relative to both the AII room and the corridor, health-care workers must mask prior to entering the anteroom (Figure 4, bottom diagram). If an AII room with an anteroom is not available, use of a portable, industrial-grade HEPA filter unit may help to increase the number of ACHs while facilitating the removal of fungal spores; however, a fresh air source must be present to achieve the proper air exchange rate. Incoming ambient air should receive HEPA filtration.

c. Operating Rooms

Operating room air may contain microorganisms, dust, aerosol, lint, skin squamous epithelial cells, and respiratory droplets. The microbial level in operating room air is directly proportional to the number of people moving in the room.³⁵¹ One study documented lower infection rates with coagulase-negative staphylococci among patients when operating room traffic during the surgical procedure was limited.³⁵² Therefore, efforts should be made to minimize personnel traffic during operations. Outbreaks of SSIs caused by group A beta-hemolytic streptococci have been traced to airborne transmission from colonized operating-room personnel to patients.^{150–154} Several potential health-care-associated pathogens (e.g., *Staphylococcus aureus* and *Staphylococcus epidermidis*) and drug-resistant organisms have also been recovered from areas adjacent to the surgical field,³⁵³ but the extent to which the presence of bacteria near the surgical field influences the development of postoperative SSIs is not clear.³⁵⁴

Proper ventilation, humidity (<68%), and temperature control in the operating room is important for the comfort of surgical personnel and patients, but also in preventing environmental conditions that encourage growth and transmission of microorganisms.³⁵⁵ Operating rooms should be maintained at positive pressure with respect to corridors and adjacent areas.³⁵⁶ Operating rooms typically do not have a variable air handling system. Variable air handling systems are permitted for use in operating rooms only if they continue to provide a positive pressure with respect to the corridors and adjacent areas and the proper ACHs are maintained when the room is occupied. Conventional operating-room ventilation systems produce a minimum of about 15 ACH of filtered air for thermal control, three (20%) of which must be fresh air.^{120, 357, 358} Air should be introduced at the ceiling and exhausted near the floor.^{357, 359}

Laminar airflow and UVGI have been suggested as adjunct measures to reduce SSI risk for certain operations. Laminar airflow is designed to move particle-free air over the aseptic operating field at a uniform velocity (0.3–0.5 m/sec), sweeping away particles in its path. This air flow can be directed vertically or horizontally, and recirculated air is passed through a HEPA filter.^{360–363} Neither laminar airflow nor UV light, however, has been conclusively shown to decrease overall SSI risk.^{356, 364–370}

Elective surgery on infectious TB patients should be postponed until such patients have received adequate drug therapy. The use of general anesthesia in TB patients poses infection-control challenges because intubation can induce coughing, and the anesthesia breathing circuit apparatus potentially can become contaminated.³⁷¹ Although operating room suites at 15 ACH exceed the air exchanges required

for TB isolation, the positive air flow relative to the corridor could result in health-care-associated transmission of TB to operating-room personnel. If feasible, intubation and extubation of the TB surgical patient should be performed in AII. AIA currently does not recommend changing pressure from positive to negative or setting it to neutral; most facilities lack the capability to do so.¹²⁰ When emergency surgery is indicated for a suspected/diagnosed infectious TB patient, taking specific infection-control measures is prudent (Box 8).

Box 8. Strategy for managing TB patients and preventing airborne transmission in operating rooms*

1. If emergency surgery is indicated for a patient with active TB, schedule the TB patient as the last surgical case to provide maximum time for adequate ACH.
2. Operating room personnel should use NIOSH-approved N95 respirators without exhalation valves.³⁴⁷
3. Keep the operating room door closed after the patient is intubated, and allow adequate time for sufficient ACH to remove 99% of airborne particles (Appendix B, Table B.1.):
 - a) after the patient is intubated and particularly if intubation produces coughing;
 - b) if the door to the operating suite must be opened, and intubation induces coughing in the patient; or
 - c) after the patient is extubated and suctioned [unless a closed suctioning system is present].
4. Extubate the patient in the operating room or allow the patient to recover in AII rather than in the regular open recovery facilities.
5. Temporary use of a portable, industrial grade HEPA filter may expedite removal of airborne contaminants (fresh-air exchange requirements for proper ventilation must still be met).+
6. Breathing circuit filters with 0.1–0.2 μm pore size can be used as an adjunct infection-control measure.^{373, 374}

* Material in this table was compiled from references 4, 347, and 372–374.

+ The placement of portable HEPA filter units in the operating room must be carefully evaluated for potential disruptions in normal air flow. The portable unit should be turned off while the surgical procedure is underway and turned on following extubation. Portable HEPA filter units previously placed in construction areas may be used in subsequent patient care, provided that all internal and external surfaces are cleaned and the filter's performance is verified with appropriate particle testing and is changed, if needed.

Table 10. Summary of ventilation specifications in selected areas of health-care facilities*

Specifications	AII room+	PE room	Critical care room§	Isolation anteroom	Operating room
Air pressure¶	Negative	Positive	Positive, negative, or neutral	Positive or negative	Positive
Room air changes	≥ 6 ACH (for existing rooms); ≥ 12 ACH (for renovation or new construction)	≥ 12 ACH	≥ 6 ACH	≥ 10 ACH	≥ 15 ACH
Sealed**	Yes	Yes	No	Yes	Yes
Filtration supply	90% (dust-spot ASHRAE 52.1 1992)	99.97%++	$\geq 90\%$	$\geq 90\%$	90%
Recirculation	No§§	Yes	Yes	No	Yes

* Material in this table is compiled from references 35 and 120.

+ Includes bronchoscopy suites.

§ Positive pressure and HEPA filters may be preferred in some rooms in intensive care units (ICUs) caring for large numbers of immunocompromised patients.

¶ Clean-to-dirty: negative to an infectious patient, positive away from an immunocompromised patient.

** Minimized infiltration for ventilation control; pertains to windows, closed doors, and surface joints.

++ Fungal spore filter at point of use (HEPA at 99.97% of 0.3 μm particles).

§§ Recirculated air may be used if the exhaust air is first processed through a HEPA filter.

¶¶ Table used with permission of the publisher of reference 35 (Lippincott Williams and Wilkins).

6. Other Aerosol Hazards in Health-Care Facilities

In addition to infectious bioaerosols, several crucial non-infectious, indoor air-quality issues must be addressed by health-care facilities. The presence of sensitizing and allergenic agents and irritants in the workplace (e.g., ethylene oxide, glutaraldehyde, formaldehyde, hexachlorophene, and latex allergens³⁷⁵) is increasing. Asthma and dermatologic and systemic reactions often result with exposure to these chemicals. Anesthetic gases and aerosolized medications (e.g., ribavirin, pentamidine, and aminoglycosides) represent some of the emerging potentially hazardous exposures to health-care workers. Containment of the aerosol at the source is the first level of engineering control, but personal protective equipment (e.g., masks, respirators, and glove liners) that distances the worker from the hazard also may be needed.

Laser plumes and surgical smoke represent another potential risk for health-care workers.^{376–378} Lasers transfer electromagnetic energy into tissues, resulting in the release of a heated plume that includes particles, gases, tissue debris, and offensive smells. One concern is that aerosolized infectious material in the laser plume might reach the nasal mucosa of surgeons and adjacent personnel. Although some viruses (i.e., varicella-zoster virus, pseudorabies virus, and herpes simplex virus) do not aerosolize efficiently,^{379, 380} other viruses and bacteria (e.g., human papilloma virus [HPV], HIV, coagulase-negative *Staphylococcus*, *Corynebacterium* spp., and *Neisseria* spp.) have been detected in laser plumes.^{381–387} The presence of an infectious agent in a laser plume may not, however, be sufficient to cause disease from airborne exposure, especially if the normal mode of transmission for the agent is not airborne. No evidence indicated that HIV or hepatitis B virus (HBV) has been transmitted via aerosolization and inhalation.³⁸⁸

Although continuing studies are needed to fully evaluate the risk of laser plumes to surgical personnel, the prevention measures in these other guidelines should be followed: a) NIOSH recommendations,³⁷⁸ b) the *Recommended Practices for Laser Safety in Practice Settings* developed by the Association of periOperative Registered Nurses [AORN],³⁸⁹ c) the assessments of ECRI,^{390–392} and d) the ANSI standard.³⁹³ These guidelines recommend the use of a) respirators (N95 or N100) or full face shields and masks,²⁶⁰ b) central wall-suction units with in-line filters to collect particulate matter from minimal plumes, and c) dedicated mechanical smoke exhaust systems with a high-efficiency filter to remove large amounts of laser plume. Although transmission of TB has occurred as a result of abscess management practices that lacked airborne particulate control measures and respiratory protection, use of a smoke evacuator or needle aspirator and a high degree of clinical awareness can help protect health-care workers when excising and draining an extrapulmonary TB abscess.¹³⁷

D. Water

1. Modes of Transmission of Waterborne Diseases

Moist environments and aqueous solutions in health-care settings have the potential to serve as reservoirs for waterborne microorganisms. Under favorable environmental circumstances (e.g., warm temperature and the presence of a source of nutrition), many bacterial and some protozoal microorganisms can either proliferate in active growth or remain for long periods in highly stable, environmentally resistant (yet infectious) forms. Modes of transmission for waterborne infections

include a) direct contact [e.g., that required for hydrotherapy]; b) ingestion of water [e.g., through consuming contaminated ice]; c) indirect-contact transmission [e.g., from an improperly reprocessed medical device];⁶ d) inhalation of aerosols dispersed from water sources;³ and e) aspiration of contaminated water. The first three modes of transmission are commonly associated with infections caused by gram-negative bacteria and nontuberculous mycobacteria (NTM). Aerosols generated from water sources contaminated with *Legionella* spp. often serve as the vehicle for introducing legionellae to the respiratory tract.³⁹⁴

2. Waterborne Infectious Diseases in Health-Care Facilities

a. Legionellosis

Legionellosis is a collective term describing infection produced by *Legionella* spp., whereas Legionnaires disease is a multi-system illness with pneumonia.³⁹⁵ The clinical and epidemiologic aspects of these diseases (Table 11) are discussed extensively in another guideline.³ Although Legionnaires disease is a respiratory infection, infection-control measures intended to prevent health-care-associated cases center on the quality of water—the principal reservoir for *Legionella* spp.

Table 11. Clinical and epidemiologic characteristics of legionellosis/Legionnaires disease

		References
Causative agent	<i>Legionella pneumophila</i> (90% of infections); <i>L. micdadei</i> , <i>L. bozemanii</i> , <i>L. dumoffii</i> , <i>L. longbeachii</i> , (14 additional species can cause infection in humans)	395–399
Mode of transmission	Aspiration of water, direct inhalation or water aerosols	3, 394–398, 400
Source of exposure	Exposure to environmental sources of <i>Legionella</i> spp. (i.e., water or water aerosols)	31, 33, 401–414
Clinical syndromes and diseases	Two distinct illnesses: a) Pontiac fever [a milder, influenza-like illness]; and b) progressive pneumonia that may be accompanied by cardiac, renal, and gastrointestinal involvement	3, 397–399, 415–422
Populations at greatest risk	Immunosuppressed patients (e.g., transplant patients, cancer patients, and patients receiving corticosteroid therapy); immunocompromised patients (e.g., surgical patients, patients with underlying chronic lung disease, and dialysis patients); elderly persons; and patients who smoke	395–397, 423–433
Occurrence	Proportion of community-acquired pneumonia caused by <i>Legionella</i> spp. ranges from 1%–5%; estimated annual incidence among the general population is 8,000–18,000 cases in the United States; the incidence of health-care-associated pneumonia (0%–14%) may be underestimated if appropriate laboratory diagnostic methods are unavailable.	396, 397, 434–444
Mortality rate	Mortality declined markedly during 1980–1998, from 34% to 12% for all cases; the mortality rate is higher among persons with health-care-associated pneumonia compared with the rate among community-acquired pneumonia patients (14% for health-care-associated pneumonia versus 10% for community-acquired pneumonia [1998 data]).	395–397, 445

Legionella spp. are commonly found in various natural and man-made aquatic environments^{446, 447} and can enter health-care facility water systems in low or undetectable numbers.^{448, 449} Cooling towers, evaporative condensers, heated potable water distribution systems, and locally-produced distilled water can provide environments for multiplication of legionellae.^{450–454} In several hospital outbreaks, patients have been infected through exposure to contaminated aerosols generated by cooling towers, showers, faucets, respiratory therapy equipment, and room-air humidifiers.^{401–410, 455} Factors that enhance

colonization and amplification of legionellae in man-made water environments include a) temperatures of 77°F–107.6°F [25°C–42°C],^{456–460} b) stagnation,⁴⁶¹ c) scale and sediment,⁴⁶² and d) presence of certain free-living aquatic amoebae that can support intracellular growth of legionellae.^{462, 463} The bacteria multiply within single-cell protozoa in the environment and within alveolar macrophages in humans.

b. Other Gram-Negative Bacterial Infections

Other gram-negative bacteria present in potable water also can cause health-care–associated infections. Clinically important, opportunistic organisms in tap water include *Pseudomonas aeruginosa*, *Pseudomonas* spp., *Burkholderia cepacia*, *Ralstonia pickettii*, *Stenotrophomonas maltophilia*, and *Sphingomonas* spp. (Tables 12 and 13). Immunocompromised patients are at greatest risk of developing infection. Medical conditions associated with these bacterial agents range from colonization of the respiratory and urinary tracts to deep, disseminated infections that can result in pneumonia and bloodstream bacteremia. Colonization by any of these organisms often precedes the development of infection. The use of tap water in medical care (e.g., in direct patient care, as a diluent for solutions, as a water source for medical instruments and equipment, and during the final stages of instrument disinfection) therefore presents a potential risk for exposure. Colonized patients also can serve as a source of contamination, particularly for moist environments of medical equipment (e.g., ventilators).

In addition to *Legionella* spp., *Pseudomonas aeruginosa* and *Pseudomonas* spp. are among the most clinically relevant, gram-negative, health-care–associated pathogens identified from water. These and other gram-negative, non-fermentative bacteria have minimal nutritional requirements (i.e., these organisms can grow in distilled water) and can tolerate a variety of physical conditions. These attributes are critical to the success of these organisms as health-care–associated pathogens. Measures to prevent the spread of these organisms and other waterborne, gram-negative bacteria include hand hygiene, glove use, barrier precautions, and eliminating potentially contaminated environmental reservoirs.^{464, 465}

Table 12. *Pseudomonas aeruginosa* infections in health-care facilities

		References
Clinical syndromes and diseases	Septicemia, pneumonia (particularly ventilator-associated), chronic respiratory infections among cystic fibrosis patients, urinary tract infections, skin and soft-tissue infections (e.g., tissue necrosis and hemorrhage), burn-wound infections, folliculitis, endocarditis, central nervous system infections (e.g., meningitis and abscess), eye infections, and bone and joint infections	466–503
Modes of transmission	Direct contact with water, aerosols; aspiration of water and inhalation of water aerosols; and indirect transfer from moist environmental surfaces via hands of health-care workers	28, 502–506
Environmental sources of pseudomonads in health-care settings	Potable (tap) water, distilled water, antiseptic solutions contaminated with tap water, sinks, hydrotherapy pools, whirlpools and whirlpool spas, water baths, lithotripsy therapy tanks, dialysis water, eyewash stations, flower vases, and endoscopes with residual moisture in the channels	28, 29, 466, 468, 507–520
Environmental sources of pseudomonads in the community	Fomites (e.g., drug injection equipment stored in contaminated water)	494, 495
Populations at greatest risk	Intensive care unit (ICU) patients (including neonatal ICU), transplant patients (organ and hematopoietic stem cell), neutropenic patients, burn therapy and hydrotherapy patients, patients with malignancies, cystic fibrosis patients, patients with underlying medical conditions, and dialysis patients	28, 466, 467, 472, 477, 493, 506–508, 511, 512, 521–526

Table 13. Other gram-negative bacteria associated with water and moist environments

Implicated contaminated environmental vehicle	References
<i>Burkholderia cepacia</i>	
Distilled water	527
Contaminated solutions and disinfectants	528, 529
Dialysis machines	527
Nebulizers	530–532
Water baths	533
Intrinsically-contaminated mouthwash*	534
Ventilator temperature probes	535
<i>Stenotrophomonas maltophilia, Sphingomonas spp.</i>	
Distilled water	536, 537
Contaminated solutions and disinfectants	529
Dialysis machines	527
Nebulizers	530–532
Water	538
Ventilator temperature probes	539
<i>Ralstonia pickettii</i>	
Fentanyl solutions	540
Chlorhexidine	541
Distilled water	541
Contaminated respiratory therapy solution	541, 542
<i>Serratia marcescens</i>	
Potable water	543
Contaminated antiseptics (i.e., benzalkonium chloride and chlorhexidine)	544–546
Contaminated disinfectants (i.e., quaternary ammonium compounds and glutaraldehyde)	547, 548
<i>Acinetobacter spp.</i>	
Medical equipment that collects moisture (e.g., mechanical ventilators, cool mist humidifiers, vaporizers, and mist tents)	549–556
Room humidifiers	553, 555
Environmental surfaces	557–564
<i>Enterobacter spp.</i>	
Humidifier water	565
Intravenous fluids	566–578
Unsterilized cotton swabs	573
Ventilators	565, 569
Rubber piping on a suctioning machine	565, 569
Blood gas analyzers	570

* This report describes intrinsic contamination (i.e., occurring during manufacture) prior to use by the health-care facility staff. All other entries reflect extrinsic sources of contamination.

Two additional gram-negative bacterial pathogens that can proliferate in moist environments are *Acinetobacter spp.* and *Enterobacter spp.*^{571, 572} Members of both genera are responsible for health-care-associated episodes of colonization, bloodstream infections, pneumonia, and urinary tract infections among medically compromised patients, especially those in ICUs and burn therapy units.^{566, 572–583} Infections caused by *Acinetobacter spp.* represent a significant clinical problem. Average infection rates are higher from July through October compared with rates from November through June.⁵⁸⁴ Mortality rates associated with *Acinetobacter* bacteremia are 17%–52%, and rates as high as 71% have been reported for pneumonia caused by infection with either *Acinetobacter spp.* or

Pseudomonas spp.^{574–576} Multi-drug resistance, especially in third generation cephalosporins for *Enterobacter* spp., contributes to increased morbidity and mortality.^{569, 572}

Patients and health-care workers contribute significantly to the environmental contamination of surfaces and equipment with *Acinetobacter* spp. and *Enterobacter* spp., especially in intensive care areas, because of the nature of the medical equipment (e.g., ventilators) and the moisture associated with this equipment.^{549, 571, 572, 585} Hand carriage and hand transfer are commonly associated with health-care–associated transmission of these organisms and for *S. marcescens*.⁵⁸⁶ *Enterobacter* spp. are primarily spread in this manner among patients by the hands of health-care workers.^{567, 587} *Acinetobacter* spp. have been isolated from the hands of 4%–33% of health-care workers in some studies,^{585–590} and transfer of an epidemic strain of *Acinetobacter* from patients’ skin to health-care workers’ hands has been demonstrated experimentally.⁵⁹¹ *Acinetobacter* infections and outbreaks have also been attributed to medical equipment and materials (e.g., ventilators, cool mist humidifiers, vaporizers, and mist tents) that may have contact with water of uncertain quality (e.g., rinsing a ventilator circuit in tap water).^{549–556} Strict adherence to hand hygiene helps prevent the spread of both *Acinetobacter* spp. and *Enterobacter* spp.^{577, 592}

Acinetobacter spp. have also been detected on dry environmental surfaces (e.g., bed rails, counters, sinks, bed cupboards, bedding, floors, telephones, and medical charts) in the vicinity of colonized or infected patients; such contamination is especially problematic for surfaces that are frequently touched.^{557–564} In two studies, the survival periods of *Acinetobacter baumannii* and *Acinetobacter calcoaceticus* on dry surfaces approximated that for *S. aureus* (e.g., 26–27 days).^{593, 594} Because *Acinetobacter* spp. may come from numerous sources at any given time, laboratory investigation of health-care–associated *Acinetobacter* infections should involve techniques to determine biotype, antibiotype, plasmid profile, and genomic fingerprinting (i.e., macrorestriction analysis) to accurately identify sources and modes of transmission of the organism(s).⁵⁹⁵

c. Infections and Pseudo-Infections Due to Nontuberculous Mycobacteria

NTM are acid-fast bacilli (AFB) commonly found in potable water. NTM include both saprophytic and opportunistic organisms. Many NTM are of low pathogenicity, and some measure of host impairment is necessary to enhance clinical disease.⁵⁹⁶ The four most common forms of human disease associated with NTM are a) pulmonary disease in adults; b) cervical lymph node disease in children; c) skin, soft tissue, and bone infections; and d) disseminated disease in immunocompromised patients.^{596, 597} Person-to-person acquisition of NTM infection, especially among immunocompetent persons, does not appear to occur, and close contacts of patients are not readily infected, despite the high numbers of organisms harbored by such patients.^{596, 598–600} NTM are spread via all modes of transmission associated with water. In addition to health-care–associated outbreaks of clinical disease, NTM can colonize patients in health-care facilities through consumption of contaminated water or ice or through inhalation of aerosols.^{601–605} Colonization following NTM exposure, particularly of the respiratory tract, occurs when a patient’s local defense mechanisms are impaired; overt clinical disease does not develop.⁶⁰⁶ Patients may have positive sputum cultures in the absence of clinical disease.

Using tap water during patient procedures and specimen collection and in the final steps of instrument reprocessing can result in pseudo-outbreaks of NTM contamination.^{607–609} NTM pseudo-outbreaks of *Mycobacterium chelonae*, *M. gordonae*, and *M. xenopi* have been associated with both bronchoscopy and gastrointestinal endoscopy when a) tap water is used to provide irrigation to the site or to rinse off the viewing tip *in situ* or b) the instruments are inappropriately reprocessed with tap water in the final steps.^{610–612}

Table 14. Nontuberculous mycobacteria—environmental vehicles

Vehicles associated with infections or colonizations	References
<i>Mycobacterium abscessus</i>	
Inadequately sterilized medical instruments	613
<i>Mycobacterium avium</i> complex (MAC)	
Potable water	614–616
<i>Mycobacterium chelonae</i>	
Dialysis, reprocessed dialyzers	31, 32
Inadequately-sterilized medical instruments, jet injectors	617, 618
Contaminated solutions	619, 620
Hydrotherapy tanks	621
<i>Mycobacterium fortuitum</i>	
Aerosols from showers or other water sources	605, 606
Ice	602
Inadequately sterilized medical instruments	603
Hydrotherapy tanks	622
<i>Mycobacterium marinum</i>	
Hydrotherapy tanks	623
<i>Mycobacterium ulcerans</i>	
Potable water	624
Vehicles associated with pseudo-outbreaks	References
<i>Mycobacterium chelonae</i>	
Potable water used during bronchoscopy and instrument reprocessing	610
<i>Mycobacterium fortuitum</i>	
Ice	607
<i>Mycobacterium gordonae</i>	
Deionized water	611
Ice	603
Laboratory solution (intrinsically contaminated)	625
Potable water ingestion prior to sputum specimen collection	626
<i>Mycobacterium kansasii</i>	
Potable water	627
<i>Mycobacterium terrae</i>	
Potable water	608
<i>Mycobacterium xenopi</i>	
Potable water	609, 612, 627

NTM can be isolated from both natural and man-made environments. Numerous studies have identified various NTM in municipal water systems and in hospital water systems and storage tanks.^{615, 616, 624, 627–632} Some NTM species (e.g., *Mycobacterium xenopi*) can survive in water at 113°F (45°C), and can be isolated from hot water taps, which can pose a problem for hospitals that lower the temperature of their hot water systems.⁶²⁷ Other NTM (e.g., *Mycobacterium kansasii*, *M. gordonae*, *M. fortuitum*, and *M. chelonae*) cannot tolerate high temperatures and are associated more often with cold water lines and taps.⁶²⁹

NTM have a high resistance to chlorine; they can tolerate free chlorine concentrations of 0.05–0.2 mg/L (0.05–0.2 ppm) found at the tap.^{598, 633, 634} They are 20–100 times more resistant to chlorine compared with coliforms; slow-growing strains of NTM (e.g., *Mycobacterium avium* and *M. kansasii*) appear to be

more resistant to chlorine inactivation compared to fast-growing NTM.⁶³⁵ Slow-growing NTM species have also demonstrated some resistance to formaldehyde and glutaraldehyde, which has posed problems for reuse of hemodialyzers.³¹ The ability of NTM to form biofilms at fluid-surface interfaces (e.g., interior surfaces of water pipes) contributes to the organisms' resistance to chemical inactivation and provides a microenvironment for growth and proliferation.^{636, 637}

d. Cryptosporidiosis

Cryptosporidium parvum is a protozoan parasite that causes self-limiting gastroenteritis in normal hosts but can cause severe, life-threatening disease in immunocompromised patients. First recognized as a human pathogen in 1976, *C. parvum* can be present in natural and finished waters after fecal contamination from either human or animal sources.^{638–641}

The health risks associated with drinking potable water contaminated with minimal numbers of *C. parvum* oocysts are unknown.⁶⁴² It remains to be determined if immunosuppressed persons are more susceptible to lower doses of oocysts than are immunocompetent persons. One study demonstrated that a median 50% infectious dose (ID₅₀) of 132 oocysts of calf origin was sufficient to cause infection among healthy volunteers.⁶⁴³ In a second study, the same researchers found that oocysts obtained from infected foals (newborn horses) were infectious for human volunteers at median ID₅₀ of 10 oocysts, indicating that different strains or species of *Cryptosporidium* may vary in their infectivity for humans.⁶⁴⁴ In a small study population of 17 healthy adults with pre-existing antibody to *C. parvum*, the ID₅₀ was determined to be 1,880 oocysts, more than 20-fold higher than in seronegative persons.⁶⁴⁵ These data suggest that pre-existing immunity derived from previous exposures to *Cryptosporidium* offers some protection from infection and illness that ordinarily would result from exposure to low numbers of oocysts.^{645, 646}

Oocysts, particularly those with thick walls, are environmentally resistant, but their survival under natural water conditions is poorly understood. Under laboratory conditions, some oocysts remain viable and infectious in cold (41°F [5°C]) for months.⁶⁴¹ The prevalence of *Cryptosporidium* in the U.S. drinking water supply is notable. Two surveys of approximately 300 surface water supplies revealed that 55%–77% of the water samples contained *Cryptosporidium* oocysts.^{647, 648} Because the oocysts are highly resistant to common disinfectants (e.g., chlorine) used to treat drinking water, filtration of the water is important in reducing the risk of waterborne transmission. Coagulation-flocculation and sedimentation, when used with filtration, can collectively achieve approximately a 2.5 log₁₀ reduction in the number of oocysts.⁶⁴⁹ However, outbreaks have been associated with both filtered and unfiltered drinking water systems (e.g., the 1993 outbreak in Milwaukee, Wisconsin that affected 400,000 people).^{641, 650–652} The presence of oocysts in the water is not an absolute indicator that infection will occur when the water is consumed, nor does the absence of detectable oocysts guarantee that infection will not occur. Health-care-associated outbreaks of cryptosporidiosis primarily have been described among groups of elderly patients and immunocompromised persons.⁶⁵³

3. Water Systems in Health-Care Facilities

a. Basic Components and Point-of-Use Fixtures

Treated municipal water enters a health-care facility via the water mains and is distributed throughout the building(s) by a network of pipes constructed of galvanized iron, copper, and polyvinylchloride (PVC). The pipe runs should be as short as is practical. Where recirculation is employed, the pipe runs should be insulated and long dead legs avoided in efforts to minimize the potential for water stagnation, which favors the proliferation of *Legionella* spp. and NTM. In high-risk applications (e.g., PE areas for severely immunosuppressed patients), insulated recirculation loops should be incorporated as a design

feature. Recirculation loops prevent stagnation and insulation maintains return water temperature with minimal loss.

Each water service main, branch main, riser, and branch (to a group of fixtures) has a valve and a means to reach the valves via an access panel.¹²⁰ Each fixture has a stop valve. Valves permit the isolation of a portion of the water system within a facility during repairs or maintenance. Vacuum breakers and other similar devices in the lines prevent water from back-flowing into the system. All systems that supply water should be evaluated to determine risk for potential back siphonage and cross connections.

Health-care facilities generate hot water from municipal water using a boiler system. Hot water heaters and storage vessels for such systems should have a drainage facility at the lowest point, and the heating element should be located as close as possible to the bottom of the vessel to facilitate mixing and to prevent water temperature stratification. Those hot or cold water systems that incorporate an elevated holding tank should be inspected and cleaned annually. Lids should fit securely to exclude foreign materials.

The most common point-of-use fixtures for water in patient-care areas are sinks, faucets, aerators, showers, and toilets; eye-wash stations are found primarily in laboratories. The potential for these fixtures to serve as a reservoir for pathogenic microorganisms has long been recognized (Table 15).^{509, 654–656} Wet surfaces and the production of aerosols facilitate the multiplication and dispersion of microbes. The level of risk associated with aerosol production from point-of-use fixtures varies. Aerosols from shower heads and aerators have been linked to a limited number of clusters of gram-negative bacterial colonizations and infections, including Legionnaires disease, especially in areas where immunocompromised patients are present (e.g., surgical ICUs, transplant units, and oncology units).^{412, 415, 656–659} In one report, clinical infection was not evident among immunocompetent persons (e.g., hospital staff) who used hospital showers when *Legionella pneumophila* was present in the water system.⁶⁶⁰ Given the infrequency of reported outbreaks associated with faucet aerators, consensus has not been reached regarding the disinfection of or removal of these devices from general use. If additional clusters of infections or colonizations occur in high-risk patient-care areas, it may be prudent to clean and decontaminate the aerators or to remove them.^{658, 659} ASHRAE recommends cleaning and monthly disinfection of aerators in high-risk patient-care areas as part of *Legionella* control measures.⁶⁶¹ Although aerosols are produced with toilet flushing,^{662, 663} no epidemiologic evidence suggests that these aerosols pose a direct infection hazard.

Although not considered a standard point-of-use fixture, decorative fountains are being installed in increasing numbers in health-care facilities and other public buildings. Aerosols from a decorative fountain have been associated with transmission of *Legionella pneumophila* serogroup 1 infection to a small cluster of older adults.⁶⁶⁴ This hotel lobby fountain had been irregularly maintained, and water in the fountain may have been heated by submersed lighting, all of which favored the proliferation of *Legionella* in the system.⁶⁶⁴ Because of the potential for generations of infectious aerosols, a prudent prevention measure is to avoid locating these fixtures in or near high-risk patient-care areas and to adhere to written policies for routine fountain maintenance.¹²⁰

Table 15. Water and point-of-use fixtures as sources and reservoirs of waterborne pathogens*

Reservoir	Associated pathogens	Transmission	Strength of evidence+	Prevention and control	References
Potable water	<i>Pseudomonas</i> , gram-negative bacteria, NTM	Contact	Moderate	Follow public health guidelines.	(See Tables 12–14)

Reservoir	Associated pathogens	Transmission	Strength of evidence+	Prevention and control	References
Potable water	<i>Legionella</i>	Aerosol inhalation	Moderate	Provide supplemental treatment for water.	(See Table 11)
Holy water	Gram-negative bacteria	Contact	Low	Avoid contact with severe burn injuries. Minimize use among immunocompromised patients.	665
Dialysis water	Gram-negative bacteria	Contact	Moderate	Dialysate should be $\leq 2,000$ cfu/mL; water should be ≤ 200 cfu/mL.	2, 527, 666–668
Automated endoscope reprocessors and rinse water	Gram-negative bacteria	Contact	Moderate	Use and maintain equipment according to instructions; eliminate residual moisture by drying the channels (e.g., through alcohol rinse and forced air drying).	669–675
Water baths	<i>Pseudomonas</i> , <i>Burkholderia</i> , <i>Acinetobacter</i>	Contact	Moderate	Add germicide to the water; wrap transfusion products in protective plastic wrap if using the bath to modulate the temperature of these products.	29, 533, 676, 677
Tub immersion	<i>Pseudomonas</i> , <i>Enterobacter</i> , <i>Acinetobacter</i>	Contact	Moderate	Drain and disinfect tub after each use; consider adding germicide to the water; water in large hydrotherapy pools should be properly disinfected and filtered.	678–683
Ice and ice machines	NTM, <i>Enterobacter</i> , <i>Pseudomonas</i> , <i>Cryptosporidium</i>	Ingestion, contact	Moderate	Clean periodically; use automatic dispenser (avoid open chest storage compartments in patient areas).	601, 684–687
			Low		
Faucet aerators	<i>Legionella</i>	Aerosol inhalation	Moderate	Clean and disinfect monthly in high-risk patient areas; consider removing if additional infections occur.	415, 661
Faucet aerators	<i>Pseudomonas</i> , <i>Acinetobacter</i> , <i>Stenotrophomonas</i> , <i>Chryseobacterium</i>	Contact, droplet	Low	No precautions are necessary at present in immunocompetent patient-care areas.	658, 659, 688, 689
Sinks	<i>Pseudomonas</i>	Contact, droplet	Moderate	Use separate sinks for handwashing and disposal of contaminated fluids.	509, 653, 685–693
Showers	<i>Legionella</i>	Aerosol inhalation	Low	Provide sponge baths for hematopoietic stem cell transplant patients; avoid shower use for immunocompromised patients when <i>Legionella</i> is detected in facility water.	656

Reservoir	Associated pathogens	Transmission	Strength of evidence+	Prevention and control	References
Dental unit water lines	<i>Pseudomonas</i> , <i>Legionella</i> , <i>Sphingomonas</i> , <i>Acinetobacter</i>	Contact	Low	Clean water systems according to system manufacturer's instructions.	636, 694–696
Ice baths for thermodilution catheters	<i>Ewingella</i> , <i>Staphylococcus</i>	Contact	Low	Use sterile water.	697, 698
Decorative fountains	<i>Legionella</i>	Aerosol inhalation	Low	Perform regular maintenance, including water disinfection; avoid use in or near high-risk patient-care areas.	664
Eyewash stations	<i>Pseudomonas</i> , amoebae, <i>Legionella</i>	Contact	Low Minimum	Flush eyewash stations weekly; have sterile water available for eye flushes.	518, 699, 700
Toilets	Gram-negative bacteria	–	Minimum	Clean regularly; use good hand hygiene.	662
Flowers	Gram-negative bacteria, <i>Aspergillus</i>	–	Minimum	Avoid use in intensive care units and in immunocompromised patient-care settings.	515, 701, 702

* Modified from reference 654 and used with permission of the publisher (Slack, Inc.)

+ **Moderate:** occasional well-described outbreaks. **Low:** few well-described outbreaks. **Minimal:** actual infections not demonstrated.

b. Water Temperature and Pressure

Hot water temperature is usually measured at the point of use or at the point at which the water line enters equipment requiring hot water for proper operation.¹²⁰ Generally, the hot water temperature in hospital patient-care areas is no greater than a temperature within the range of 105°F–120°F (40.6°C–49°C), depending on the AIA guidance issued at the year in which the facility was built.¹²⁰ Hot water temperature in patient-care areas of skilled nursing-care facilities is set within a slightly lower range of 95°F–110°F (35°C–43.3°C) depending on the AIA guidance at the time of facility construction.¹²⁰ Many states have adopted a temperature setting in these ranges into their health-care regulations and building codes. ASHRAE, however, has recommended higher settings.⁶⁶¹ Steam jets or booster heaters are usually needed to meet the hot water temperature requirements in certain service areas of the hospital (e.g., the kitchen [120°F (49°C)] or the laundry [160°F (71°C)]).¹²⁰ Additionally, water lines may need to be heated to a particular temperature specified by manufacturers of specific hospital equipment. Hot-water distribution systems serving patient-care areas are generally operated under constant recirculation to provide continuous hot water at each hot-water outlet.¹²⁰ If a facility is or has a hemodialysis unit, then continuously circulated, cold treated water is provided to that unit.¹²⁰

To minimize the growth and persistence of gram-negative waterborne bacteria (e.g., thermophilic NTM and *Legionella* spp.),^{627, 703–709} cold water in health-care facilities should be stored and distributed at temperatures below 68°F (20°C); hot water should be stored above 140°F (60°C) and circulated with a minimum return temperature of 124°F (51°C),⁶⁶¹ or the highest temperature specified in state regulations and building codes. If the return temperature setting of 124°F (51°C) is permitted, then installation of preset thermostatic mixing valves near the point-of-use can help to prevent scalding. Valve maintenance is especially important in preventing valve failure, which can result in scalding. New shower systems in large buildings, hospitals, and nursing homes should be designed to permit mixing of hot and cold water near the shower head. The warm water section of pipe between the control valve and shower head should be self-draining. Where buildings can not be retrofitted, other

approaches to minimize the growth of *Legionella* spp. include a) periodically increasing the temperature to at least 150°F [66°C] at the point of use [i.e., faucets] and b) adding additional chlorine and flushing the water.^{661, 710, 711} Systems should be inspected annually to ensure that thermostats are functioning properly.

Adequate water pressure ensures sufficient water supplies for a) direct patient care; b) operation of water-cooled instruments and equipment [e.g., lasers, computer systems, telecommunications systems, and automated endoscope reprocessors⁷¹²]; c) proper function of vacuum suctioning systems; d) indoor climate control; and e) fire-protection systems. Maintaining adequate pressure also helps to ensure the integrity of the piping system.

c. Infection-Control Impact of Water System Maintenance and Repair

Corrective measures for water-system failures have not been studied in well-designed experiments; these measures are instead based on empiric engineering and infection-control principles. Health-care facilities can occasionally sustain both intentional cut-offs by the municipal water authority to permit new construction project tie-ins and unintentional disruptions in service when a water main breaks as a result of aging infrastructure or a construction accident. Vacuum breakers or other similar devices can prevent backflow of water in the facility's distribution system during water-disruption emergencies.¹¹ To be prepared for such an emergency, all health-care facilities need contingency plans that identify a) the total demand for potable water, b) the quantity of replacement water [e.g., bottled water] required for a minimum of 24 hours when the water system is down, c) mechanisms for emergency water distribution, and 4) procedures for correcting drops in water pressure that affect operation of essential devices and equipment that are driven or cooled by a water system [Table 16].⁷¹³

Table 16. Water demand in health-care facilities during water disruption emergencies

	Potable water	Bottled, sterile water
Water use needs	Drinking water Handwashing Cafeteria services Ice Manual flushing of toilets Patient baths, hygiene Hemodialysis Hydrotherapy Fire prevention (e.g., sprinkler systems) Surgery and critical care areas Laboratory services Laundry and central sterile services* Cooling towers+ Steam generation	Surgical scrub Emergency surgical procedures Pharmaceutical preparations Patient-care equipment (e.g., ventilators)§

* Arrange to have a contingency provision of these services from another resource, if possible (e.g., another health-care facility or contractor).

+ Some cooling towers may use a potable water source, but most units use non-potable water.

§ This item is included in the table under the assumption that electrical power is available during the water emergency.

Detailed, up-to-date plans for hot and cold water piping systems should be readily available for maintenance and repair purposes in case of system problems. Opening potable water systems for repair or construction and subjecting systems to water-pressure changes can result in water discoloration and dramatic increases in the concentrations of *Legionella* spp. and other gram-negative bacteria. The maintenance of a chlorine residual at all points within the piping system also offers some protection from entry of contamination to the pipes in the event of inadvertent cross-connection between potable and non-potable water lines. As a minimum preventive measure, ASHRAE recommends a thorough flushing of the system.⁶⁶¹ High-temperature flushing or hyperchlorination may also be appropriate

strategies to decrease potentially high concentrations of waterborne organisms. The decision to pursue either of these remediation strategies, however, should be made on a case-by-case basis. If only a portion of the system is involved, high temperature flushing or chlorination can be used on only that portion of the system.⁶⁶¹

When shock decontamination of hot water systems is necessary (e.g., after disruption caused by construction and after cross-connections), the hot water temperature should be raised to 160°F–170°F (71°C–77°C) and maintained at that level while each outlet around the system is progressively flushed. A minimum flush time of 5 minutes has been recommended;³ the optimal flush time is not known, however, and longer flush times may be necessary.⁷¹⁴ The number of outlets that can be flushed simultaneously depends on the capacity of the water heater and the flow capability of the system. Appropriate safety procedures to prevent scalding are essential. When possible, flushing should be performed when the fewest building occupants are present (e.g., during nights and weekends).

When thermal shock treatment is not possible, shock chlorination may serve as an alternative method.⁶⁶¹ Experience with this method of decontamination is limited, however, and high levels of free chlorine can corrode metals. Chlorine should be added, preferably overnight, to achieve a free chlorine residual of at least 2 mg/L (2 ppm) throughout the system.⁶⁶¹ This may require chlorination of the water heater or tank to levels of 20–50 mg/L (20–50 ppm). The pH of the water should be maintained at 7.0–8.0.⁶⁶¹ After completion of the decontamination, recolonization of the hot water system is likely to occur unless proper temperatures are maintained or a procedure such as continuous supplemental chlorination is continued.

Interruptions of the water supply and sewage spills are situations that require immediate recovery and remediation measures to ensure the health and safety of patients and staff.⁷¹⁵ When delivery of potable water through the municipal distribution system has been disrupted, the public water supplier must issue a “boil water” advisory if microbial contamination presents an immediate public health risk to customers. The hospital engineer should oversee the restoration of the water system in the facility and clear it for use when appropriate. Hospitals must maintain a high level of surveillance for waterborne disease among patients and staff after the advisory is lifted.⁶⁴²

Flooding from either external (e.g., from a hurricane) or internal sources (e.g., a water system break) usually results in property damage and a temporary loss of water and sanitation.^{716–718} JCAHO requires all hospitals to have plans that address facility response for recovery from both internal and external disasters.^{713, 719} The plans are required to discuss a) general emergency preparedness, b) staffing, c) regional planning among area hospitals, d) emergency supply of potable water, e) infection control and medical services needs, f) climate control, and g) remediation. The basic principles of structural recovery from flooding are similar to those for recovery from sewage contamination (Box 9 and 10). Following a major event (e.g., flooding), facilities may elect to conduct microbial sampling of water after the system is restored to verify that water quality has been returned to safe levels (<500 CFU/mL, heterotrophic plate count). This approach may help identify point-of-use fixtures that may harbor contamination as a result of design or engineering features.⁷²⁰ Medical records should be allowed to dry and then either photocopied or placed in plastic covers before returning them to the record.

Moisture meters can be used to assess water-damaged structural materials. If porous structural materials for walls have a moisture content of >20% after 72 hours, the affected material should be removed.^{266, 278, 313} The management of water-damaged structural materials is not strictly limited to major water catastrophes (e.g., flooding and sewage intrusions); the same principles are used to evaluate the damage from leaking roofs, point-of-use fixtures, and equipment. Additional sources of moisture include condensate on walls from boilers and poorly engineered humidification in HVAC systems.

Box 9. Recovery and remediation measures for water-related emergencies*

Potable water disruptions

Contingency plan items

- Ensure access to plumbing network so that repairs can be easily made.
- Provide sufficient potable water, either from bottled sources or truck delivery.
- Post advisory notices against consuming tap water, ice, or beverages made with water.
- Rope off or bag drinking fountains to designate these as being “out of service” until further notice.
- Rinse raw foods as needed in disinfected water.
- Disconnect ice machines whenever possible.+
- Postpone laundry services until after the water system is restored.

Water treatment

- Heat water to a rolling boil for ≥ 1 minute.

Remediation of the water system after the “boil water” advisory is rescinded

- Flush fixtures (e.g., faucets and drinking fountains) and equipment for several minutes and restart.
 - Run water softeners through a regeneration cycle.
 - Drain, disinfect, and refill water storage tanks, if needed.
 - Change pretreatment filters and disinfect the dialysis water system.
-

Sewage spills/malfunction

Overall strategy

- Move patients and clean/sterile supplies out of the area.
- Redirect traffic away from the area.
- Close the doors or use plastic sheeting to isolate the area prior to clean-up.
- Restore sewage system function first, then the potable water system (if both are malfunctioning).
- Remove sewage solids, drain the area, and let dry.

Remediation of the structure

- Hard surfaces: clean with detergent/disinfectant after the area has been drained.
- Carpeting, loose tiles, buckled flooring: remove and allow the support surface to dry; replace the items; wet down carpeting with a low-level disinfectant or sanitizer prior to removal to minimize dust dispersion to the air.
- Wallboard and other porous structural materials: remove and replace if they cannot be cleaned and dried within 72 hours.§

Furniture

- Hard surface furniture (e.g., metal or plastic furniture): clean and allow to dry.
- Wood furniture: let dry, sand the wood surface, and reapply varnish.
- Cloth furniture: replace.

Electrical equipment

- Replace if the item cannot be easily dismantled, cleaned, and reassembled.
-

* Material in this box is compiled from references 266, 278, 315, 713, 716–719, 721–729.

+ Ice machines should always be disconnected from the water source in advance of planned water disruptions.

§ Moisture meter readings should be <20% moisture content.

An exception to these recommendations is made for hemodialysis units where water is further treated either by portable water treatment or large-scale water treatment systems usually involving reverse osmosis (RO). In the United States, >97% of dialysis facilities use RO treatment for their water.⁷²¹ However, changing pre-treatment filters and disinfecting the system to prevent colonization of the RO membrane and microbial contamination down-stream of the pre-treatment filter are prudent measures.

Box 10. Contingency planning for flooding

General emergency preparedness

- Ensure that emergency electrical generators are not located in flood-prone areas of the facility.
- Develop alternative strategies for moving patients, water containers, medical records, equipment, and supplies in the event that the elevators are inoperable.
- Establish in advance a centralized base of operations with batteries, flashlights, and cellular phones.
- Ensure sufficient supplies of sandbags to place at the entrances and the area around boilers, incinerators, and generators.
- Establish alternative strategies for bringing core employees to the facility if high water prevents travel.

Staffing Patterns

- Temporarily reassign licensed staff as needed to critical care areas to provide manual ventilation and to perform vital assessments on patients.
- Designate a core group of employees to remain on site to keep all services operational if the facility remains open.
- Train all employees in emergency preparedness procedures.

Regional planning among are facilities for disaster management

- Incorporate community support and involvement (e.g., media alerts, news, and transportation).
- Develop in advance strategies for transferring patients, as needed.
- Develop strategies for sharing supplies and providing essential services among participating facilities (e.g., central sterile department services, and laundry services).
- Identify sources for emergency provisions (e.g., blood, emergency vehicles, and bottled water).

Medical services and infection control

- Use alcohol-based hand rubs in general patient-care areas.
- Postpone elective surgeries until full services are restored, or transfer these patients to other facilities.
- Consider using portable dialysis machines.+
- Provide an adequate supply of tetanus and hepatitis A immunizations for patients and staff.

Climate control

- Provide adequate water for cooling towers.§
-

* Material in this box was compiled from references 713, 716–719.

+ Portable dialysis machines require less water compared to the larger units situated in dialysis settings.

§ Water for cooling towers may need to be trucked in, especially if the tower uses a potable water source.

4. Strategies for Controlling Waterborne Microbial Contamination

a. Supplemental Treatment of Water with Heat and/or Chemicals

In addition to using supplemental treatment methods as remediation measures after inadvertent contamination of water systems, health-care facilities sometimes use special measures to control waterborne microorganisms on a sustained basis. This decision is most often associated with outbreaks of legionellosis and subsequent efforts to control legionellae,⁷²² although some facilities have tried supplemental measures to better control thermophilic NTM.⁶²⁷

The primary disinfectant for both cold and hot water systems is chlorine. However, chlorine residuals are expected to be low, and possibly nonexistent, in hot water tanks because of extended retention time in the tank and elevated water temperature. Flushing, especially that which removes sludge from the bottom of the tank, probably provides the most effective treatment of water systems. Unlike the situation for disinfecting cooling towers, no equivalent recommendations have been made for potable water systems, although specific intervention strategies have been published.^{403, 723} The principal approaches to disinfection of potable systems are heat flushing using temperatures 160°F–170°F (71°C–77°C), hyperchlorination, and physical cleaning of hot-water tanks.^{3, 403, 661} Potable systems are easily recolonized and may require continuous intervention (e.g., raising of hot water temperatures or continuous chlorination).^{403, 711} Chlorine solutions lose potency over time, thereby rendering the stocking of large quantities of chlorine impractical.

Some hospitals with hot water systems identified as the source of *Legionella* spp. have performed emergency decontamination of their systems by pulse (i.e., one-time) thermal disinfection/superheating or hyperchlorination.^{711, 714, 724, 725} After either of these procedures, hospitals either maintain their heated water with a minimum return temperature of 124°F (51°C) and cold water at <68°F (<20°C) or chlorinate their hot water to achieve 1–2 mg/L (1–2 ppm) of free residual chlorine at the tap.^{26, 437, 709–711, 726, 727} Additional measures (e.g., physical cleaning or replacement of hot-water storage tanks, water heaters, faucets, and shower heads) may be required to help eliminate accumulations of scale and sediment that protect organisms from the biocidal effects of heat and chlorine.^{457, 711} Alternative methods for controlling and eradicating legionellae in water systems (e.g., treating water with chlorine dioxide, heavy metal ions [i.e., copper/silver ions], ozone, and UV light) have limited the growth of legionellae under laboratory and operating conditions.^{728–742} Further studies on the long-term efficacy of these treatments are needed before these methods can be considered standard applications.

Renewed interest in the use of chloramines stems from concerns about adverse health effects associated with disinfectants and disinfection by-products.⁷⁴³ Monochloramine usage minimizes the formation of disinfection by-products, including trihalomethanes and haloacetic acids. Monochloramine can also reach distal points in a water system and can penetrate into bacterial biofilms more effectively than free chlorine.⁷⁴⁴ However, monochloramine use is limited to municipal water treatment plants and is currently not available to health-care facilities as a supplemental water-treatment approach. A recent study indicated that 90% of Legionnaires disease outbreaks associated with drinking water could have been prevented if monochloramine rather than free chlorine has been used for residual disinfection.⁷⁴⁵ In a retrospective comparison of health-care–associated Legionnaires disease incidence in central Texas hospitals, the same research group documented an absence of cases in facilities located in communities with monochloramine-treated municipal water.⁷⁴⁶ Additional data are needed regarding the effectiveness of using monochloramine before its routine use as a disinfectant in water systems can be recommended. No data have been published regarding the effectiveness of monochloramine installed at the level of the health-care facility.

Additional filtration of potable water systems is not routinely necessary. Filters are used in water lines in dialysis units, however, and may be inserted into the lines for specific equipment (e.g., endoscope washers and disinfectors) for the purpose of providing bacteria-free water for instrument reprocessing. Additionally, an RO unit is usually added to the distribution system leading to PE areas.

b. Primary Prevention of Legionnaires Disease (No Cases Identified)

The primary and secondary environmental infection-control strategies described in this section on the guideline pertain to health-care facilities without transplant units. Infection-control measures specific to PE or transplant units (i.e., patient-care areas housing patients at the highest risk for morbidity and mortality from *Legionella* spp. infection) are described in the subsection titled *Preventing Legionnaires Disease in Protective Environments*.

Health-care facilities use at least two general strategies to prevent health-care–associated legionellosis when no cases or only sporadic cases have been detected. The first is an environmental surveillance approach involving periodic culturing of water samples from the hospital’s potable water system to monitor for *Legionella* spp.^{747–750} If any sample is culture-positive, diagnostic testing is recommended for all patients with health-care–associated pneumonia.^{748, 749} In-house testing is recommended for facilities with transplant programs as part of a comprehensive treatment/management program. If $\geq 30\%$ of the samples are culture-positive for *Legionella* spp., decontamination of the facility’s potable water system is warranted.⁷⁴⁸ The premise for this approach is that no cases of health-care–associated legionellosis can occur if *Legionella* spp. are not present in the potable water system, and, conversely, cases of health-care–associated legionellosis could potentially occur if *Legionella* spp. are cultured from the water.^{26, 751} Physicians who are informed that the hospital’s potable water system is culture-positive

for *Legionella* spp. are more likely to order diagnostic tests for legionellosis.

A potential advantage of the environmental surveillance approach is that periodic culturing of water is less costly than routine laboratory diagnostic testing for all patients who have health-care-associated pneumonia. The primary argument against this approach is that, in the absence of cases, the relationship between water-culture results and legionellosis risk remains undefined.³ *Legionella* spp. can be present in the water systems of buildings⁷⁵² without being associated with known cases of disease.^{437, 707, 753} In a study of 84 hospitals in Québec, 68% of the water systems were found to be colonized with *Legionella* spp., and 26% were colonized at >30% of sites sampled; cases of Legionnaires disease, however, were infrequently reported from these hospitals.⁷⁰⁷

Other factors also argue against environmental surveillance. Interpretation of results from periodic water culturing might be confounded by differing results among the sites sampled in a single water system and by fluctuations in the concentration of *Legionella* spp. at the same site.^{709, 754} In addition, the risk for illness after exposure to a given source might be influenced by several factors other than the presence or concentration of organisms, including a) the degree to which contaminated water is aerosolized into respirable droplets, b) the proximity of the infectious aerosol to the potential host, c) the susceptibility of the host, and d) the virulence properties of the contaminating strain.^{755–757} Thus, data are insufficient to assign a level of disease risk even on the basis of the number of colony-forming units detected in samples from areas for immunocompetent patients. Conducting environmental surveillance would obligate hospital administrators to initiate water-decontamination programs if *Legionella* spp. are identified. Therefore, periodic monitoring of water from the hospital's potable water system and from aerosol-producing devices is not widely recommended in facilities that have not experienced cases of health-care-associated legionellosis.^{661, 758}

The second strategy to prevent and control health-care-associated legionellosis is a clinical approach, in which providers maintain a high index of suspicion for legionellosis and order appropriate diagnostic tests (i.e., culture, urine antigen, and direct fluorescent antibody [DFA] serology) for patients with health-care-associated pneumonia who are at high risk for legionellosis and its complications.^{437, 759, 760} The testing of autopsy specimens can be included in this strategy should a death resulting from health-care-associated pneumonia occur. Identification of one case of definite or two cases of possible health-care-associated Legionnaires disease should prompt an epidemiologic investigation for a hospital source of *Legionella* spp., which may involve culturing the facility's water for *Legionella*. Routine maintenance of cooling towers, and use of sterile water for the filling and terminal rinsing of nebulization devices and ventilation equipment can help to minimize potential sources of contamination. Circulating potable water temperatures should match those outlined in the subsection titled *Water Temperature and Pressure*, as permitted by state code.

c. Secondary prevention of Legionnaires Disease (With Identified Cases)

The indications for a full-scale environmental investigation to search for and subsequently decontaminate identified sources of *Legionella* spp. in health-care facilities without transplant units have not been clarified; these indications would likely differ depending on the facility. Case categories for health-care-associated Legionnaires disease in facilities without transplant units include definite cases (i.e., laboratory-confirmed cases of legionellosis that occur in patients who have been hospitalized continuously for ≥ 10 days before the onset of illness) and possible cases (i.e., laboratory-confirmed infections that occur 2–9 days after hospital admission).³ In settings in which as few as one to three health-care-associated cases are recognized over several months, intensified surveillance for Legionnaires disease has frequently identified numerous additional cases.^{405, 408, 432, 453, 739, 759, 760} This finding suggests the need for a low threshold for initiating an investigation after laboratory confirmation of cases of health-care-associated legionellosis. When developing a strategy for responding to such a finding, however, infection-control personnel should consider the level of risk for health-care-

associated acquisition of, and mortality from, *Legionella* spp. infection at their particular facility.

An epidemiologic investigation conducted to determine the source of *Legionella* spp. involves several important steps (Box 11). Laboratory assessment is crucial in supporting epidemiologic evidence of a link between human illness and a specific environmental source.⁷⁶¹ Strain determination from subtype analysis is most frequently used in these investigations.^{410, 762–764} Once the environmental source is established and confirmed with laboratory support, supplemental water treatment strategies can be initiated as appropriate.

Box 11. Steps in an epidemiologic investigation for legionellosis

Review medical and microbiologic records.

Initiate active surveillance to identify all recent or ongoing cases.

Develop a line listing of cases by time, place, and person.

Determine the type of epidemiologic investigation needed for assessing risk factors:

- Case-control study,
- Cohort study.

Gather and analyze epidemiologic information:

- Evaluate risk factors associated with potential environmental exposures (e.g., showers, cooling towers, and respiratory-therapy equipment).

Collect water samples:

- Sample environmental sources implicated by epidemiologic investigation,
- Sample other potential source of water aerosols.

Subtype strains of *Legionella* spp. cultured from both patients and environmental sources.

Review autopsy records and include autopsy specimens in diagnostic testing.

The decision to search for hospital environmental sources of *Legionella* spp. and the choice of procedures to eradicate such contamination are based on several considerations, as follows: a) the hospital's patient population; b) the cost of an environmental investigation and institution of control measures to eradicate *Legionella* spp. from the water supply,^{765–768} and c) the differential risk, based on host factors, for acquiring health-care-associated legionellosis and developing severe and fatal infection.

d. Preventing Legionnaires Disease in Protective Environments

This subsection outlines infection-control measures applicable to those health-care facilities providing care to severely immunocompromised patients. Indigenous microorganisms in the tap water of these facilities may pose problems for such patients. These measures are designed to prevent the generation of potentially infectious aerosols from water and the subsequent exposure of PE patients or other immunocompromised patients (e.g., transplant patients) (Table 17). Infection-control measures that address the use of water with medical equipment (e.g., ventilators, nebulizers, and equipment humidifiers) are described in other guidelines and publications.^{3, 455}

If one case of laboratory-confirmed, health-care-associated Legionnaires disease is identified in a patient in a solid-organ transplant program or in PE (i.e., an inpatient in PE for all or part of the 2–10 days prior to onset of illness) or if two or more laboratory-confirmed cases occur among patients who had visited an outpatient PE setting, the hospital should report the cases to the local and state health departments. The hospital should then initiate a thorough epidemiologic and environmental investigation to determine the likely environmental sources of *Legionella* spp.⁹ The source of *Legionella* should be decontaminated or removed. Isolated cases may be difficult to investigate. Because transplant recipients are at substantially higher risk for disease and death from legionellosis

compared with other hospitalized patients, periodic culturing for *Legionella* spp. in water samples from the potable water in the solid-organ transplant and/or PE unit can be performed as part of an overall strategy to prevent Legionnaires disease in PE units.^{9, 431, 710, 769} The optimal methodology (i.e., frequency and number of sites) for environmental surveillance cultures in PE units has not been determined, and the cost-effectiveness of this strategy has not been evaluated. Because transplant recipients are at high risk for Legionnaires disease and because no data are available to determine a safe concentration of legionellae organisms in potable water, the goal of environmental surveillance for *Legionella* spp. should be to maintain water systems with no detectable organisms.^{9, 431} Culturing for legionellae may be used to assess the effectiveness of water treatment or decontamination methods, a practice that provides benefits to both patients and health-care workers.^{767, 770}

Table 17. Additional infection-control measures to prevent exposure of high-risk patients to waterborne pathogens

Measures	References
<ul style="list-style-type: none"> • Restrict patients from taking showers if the water is contaminated with <i>Legionella</i> spp. • Use water that is not contaminated with <i>Legionella</i> spp. for patients' sponge baths. • Provide sterile water for drinking, tooth brushing, or for flushing nasogastric tubes. • Perform supplemental treatment of the water for the unit. • Consider periodic monitoring (i.e., culturing) of the unit water supply for <i>Legionella</i> spp. • Remove shower heads and faucet aerators monthly for cleaning.* • Use a 500–600 ppm (1:100 v/v dilution) solution of sodium hypochlorite to disinfect shower heads and faucet aerators.* • Do not use large-volume room air humidifiers that create aerosols unless these are subjected to cleaning and high-level disinfection daily and filled with distilled water. • Eliminate water-containing bath toys.+ 	<ul style="list-style-type: none"> • 407, 412, 654, 655, 658 • 9 • 9, 412 • 732 • 9, 431 • 661 • 661 • 3 • 30

* These measures can be considered in settings where legionellosis cases have occurred. These measures are not generally recommended in routine patient-care setting..

+ These items have been associated with outbreaks of *Pseudomonas*.

Protecting patient-care devices and instruments from inadvertent tap water contamination during room cleaning procedures is also important in any immunocompromised patient-care area. In a recent outbreak of gram-negative bacteremias among open-heart-surgery patients, pressure-monitoring equipment that was assembled and left uncovered overnight prior to the next day's surgeries was inadvertently contaminated with mists and splashing water from a hose-disinfectant system used for cleaning.⁷⁷¹

5. Cooling Towers and Evaporative Condensers

Modern health-care facilities maintain indoor climate control during warm weather by use of cooling towers (large facilities) or evaporative condensers (smaller buildings). A cooling tower is a wet-type, evaporative heat transfer device used to discharge to the atmosphere waste heat from a building's air conditioning condensers (Figure 5).^{772, 773} Warm water from air-conditioning condensers is piped to the cooling tower where it is sprayed downward into a counter- or cross-current air flow. To accelerate heat transfer to the air, the water passes over the fill, which either breaks water into droplets or causes it to spread into a thin film.^{772, 773} Most systems use fans to move air through the tower, although some large industrial cooling towers rely on natural draft circulation of air. The cooled water from the tower is piped back to the condenser, where it again picks up heat generated during the process of chilling the system's refrigerant. The water is cycled back to the cooling tower to be cooled. Closed-circuit cooling towers and evaporative condensers are also evaporative heat-transfer devices. In these systems, the

has been linked to cooling tower aerosol exposure.^{404, 405} Contaminated aerosols from cooling towers on hospital premises gained entry to the buildings either through open windows or via air handling system intakes located near the tower equipment.

Cooling towers and evaporative condensers provide ideal ecological niches for *Legionella* spp. The typical temperature of the water in cooling towers ranges from 85°F–95°F (29°C–35°C), although temperatures can be above 120°F (49°C) and below 70°F (21°C) depending on system heat load, ambient temperature, and operating strategy.⁶⁶¹ An Australian study of cooling towers found that legionellae colonized or multiplied in towers with basin temperatures above 60.8°F (16°C), and multiplication became explosive at temperatures above 73.4°F (23°C).⁷⁸³ Water temperature in closed-circuit cooling towers and evaporative condensers is similar to that in cooling towers. Considerable variation in the piping arrangement occurs. In addition, stagnant areas or dead legs may be difficult to clean or penetrate with biocides.

Several documents address the routine maintenance of cooling towers, evaporative condensers, and whirlpool spas.^{661, 784–787} They suggest following manufacturer's recommendations for cleaning and biocide treatment of these devices; all health-care facilities should ensure proper maintenance for their cooling towers and evaporative condensers, even in the absence of *Legionella* spp (Appendix C). Because cooling towers and evaporative condensers can be shut down during periods when air conditioning is not needed, this maintenance cleaning and treatment should be performed before starting up the system for the first time in the warm season.⁷⁸² Emergency decontamination protocols describing cleaning procedures and hyperchlorination for cooling towers have been developed for towers implicated in the transmission of legionellosis.^{786, 787}

6. Dialysis Water Quality and Dialysate

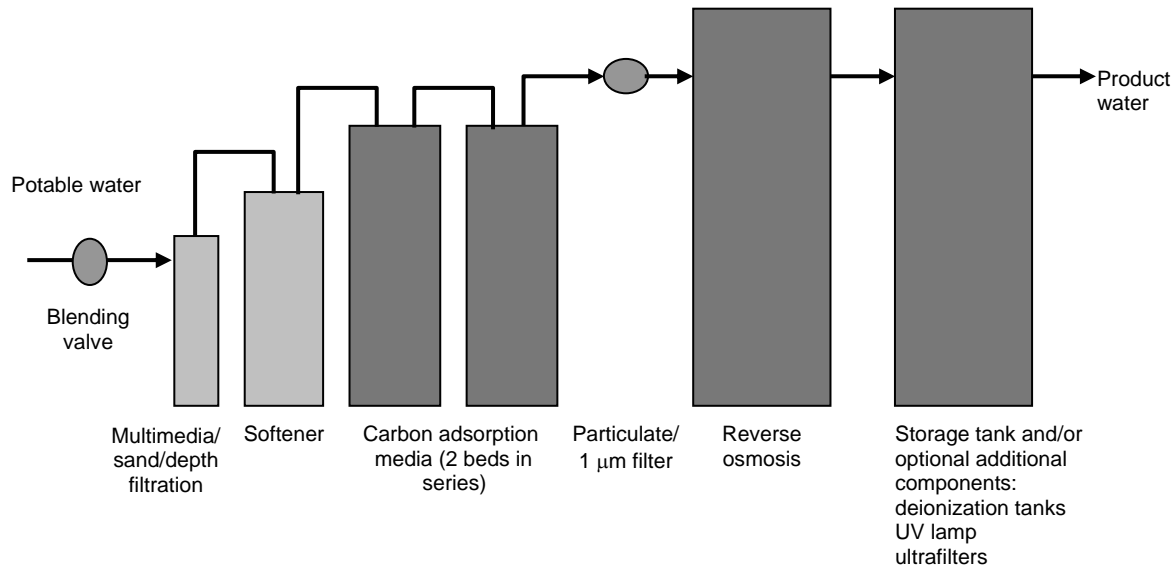
a. Rationale for Water Treatment in Hemodialysis

Hemodialysis, hemofiltration, and hemodiafiltration require special water-treatment processes to prevent adverse patient outcomes of dialysis therapy resulting from improper formulation of dialysate with water containing high levels of certain chemical or biological contaminants. The Association for the Advancement of Medical Instrumentation (AAMI) has established chemical and microbiologic standards for the water used to prepare dialysate, substitution fluid, or to reprocess hemodialyzers for renal replacement therapy.^{788–792} The AAMI standards address: a) equipment and processes used to purify water for the preparation of concentrates and dialysate and the reprocessing of dialyzers for multiple use and b) the devices used to store and distribute this water. Future revisions to these standards may include hemofiltration and hemodiafiltration.

Water treatment systems used in hemodialysis employ several physical and/or chemical processes either singly or in combination (Figure 6). These systems may be portable units or large systems that feed several rooms. In the United States, >97% of maintenance hemodialysis facilities use RO alone or in combination with deionization.⁷⁹³ Many acute-care facilities use portable hemodialysis machines with attached portable water treatment systems that use either deionization or RO. These machines were exempted from earlier versions of AAMI recommendations, but given current knowledge about toxic exposures to and inflammatory processes in patients new to dialysis, these machines should now come into compliance with current AAMI recommendations for hemodialysis water and dialysate quality.^{788, 789} Previous recommendations were based on the assumption that acute-care patients did not experience the same degree of adverse effects from short-term, cumulative exposures to either chemicals or microbiologic agents present in hemodialysis fluids compared with the effects encountered by patients during chronic, maintenance dialysis.^{788, 789} Additionally, JCAHO is reviewing inpatient

practices and record-keeping for dialysis (acute and maintenance) for adherence to AAMI standards and recommended practices.

Figure 6. Dialysis water treatment system*



* See text for description of the placement and function of these components.

Neither the water used to prepare dialysate nor the dialysate itself needs to be sterile, but tap water can not be used without additional treatment. Infections caused by rapid-growing NTM (e.g., *Mycobacterium chelonae* and *M. abscessus*) present a potential risk to hemodialysis patients (especially those in hemodialyzer reuse programs) if disinfection procedures to inactivate mycobacteria in the water (low-level disinfection) and the hemodialyzers (high-level disinfection) are inadequate.^{31, 32, 633} Other factors associated with microbial contamination in dialysis systems could involve the water treatment system, the water and dialysate distribution systems, and the type of hemodialyzer.^{666, 667, 794-799} Understanding the various factors and their influence on contamination levels is the key to preventing high levels of microbial contamination in dialysis therapy.

In several studies, pyrogenic reactions were demonstrated to have been caused by lipopolysaccharide or endotoxin associated with gram-negative bacteria.^{794, 800-803} Early studies demonstrated that parenteral exposure to endotoxin at a concentration of 1 ng/kg body weight/hour was the threshold dose for producing pyrogenic reactions in humans, and that the relative potencies of endotoxin differ by bacterial species.^{804, 805} Gram-negative water bacteria (e.g., *Pseudomonas* spp.) have been shown to multiply rapidly in a variety of hospital-associated fluids that can be used as supply water for hemodialysis (e.g., distilled water, deionized water, RO water, and softened water) and in dialysate (a balanced salt solution made with this water).⁸⁰⁶ Several studies have demonstrated that the attack rates of pyrogenic reactions are directly associated with the number of bacteria in dialysate.^{666, 667, 807} These studies provided the rationale for setting the heterotrophic bacteria standards in the first AAMI hemodialysis guideline at $\leq 2,000$ CFU/mL in dialysate and one log lower (≤ 200 CFU/mL) for the water used to prepare dialysate.^{668, 788} If the level of bacterial contamination exceeded 200 CFU/mL in water, this level could be amplified in the system and effectively constitute a high inoculum for dialysate at the start of a

dialysis treatment.^{807, 808} Pyrogenic reactions did not appear to occur when the level of contamination was below 2,000 CFU/mL in dialysate unless the source of the endotoxin was exogenous to the dialysis system (i.e., present in the community water supply). Endotoxins in a community water supply have been linked to the development of pyrogenic reactions among dialysis patients.⁷⁹⁴

Whether endotoxin actually crosses the dialyzer membrane is controversial. Several investigators have shown that bacteria growing in dialysate-generated products that could cross the dialyzer membrane.^{809,}

⁸¹⁰ Gram-negative bacteria growing in dialysate have produced endotoxins that in turn stimulated the production of anti-endotoxin antibodies in hemodialysis patients;^{801, 811} these data suggest that bacterial endotoxins, although large molecules, cross dialyzer membranes either intact or as fragments. The use of the very permeable membranes known as high-flux membranes (which allow large molecules [e.g., β_2 microglobulin] to traverse the membrane) increases the potential for passage of endotoxins into the blood path. Several studies support this contention. In one such study, an increase in plasma endotoxin concentrations during dialysis was observed when patients were dialyzed against dialysate containing 10^3 – 10^4 CFU/mL *Pseudomonas* spp.⁸¹² *In vitro* studies using both radiolabeled lipopolysaccharide and biologic assays have demonstrated that biologically active substances derived from bacteria found in dialysate can cross a variety of dialyzer membranes.^{802, 813–816} Patients treated with high-flux membranes have had higher levels of anti-endotoxin antibodies than subjects or patients treated with conventional membranes.⁸¹⁷ Finally, since 1989, 19%–22% of dialysis centers have reported pyrogenic reactions in the absence of septicemia.^{818, 819}

Investigations of adverse outcomes among patients using reprocessed dialyzers have demonstrated a greater risk for developing pyrogenic reactions when the water used to reprocess these devices contained >6 ng/mL endotoxin and $>10^4$ CFU/mL bacteria.⁸²⁰ In addition to the variability in endotoxin assays, host factors also are involved in determining whether a patient will mount a response to endotoxin.⁸⁰³ Outbreak investigations of pyrogenic reactions and bacteremias associated with hemodialyzer reuse have demonstrated that pyrogenic reactions are prevented once the endotoxin level in the water used to reprocess the dialyzers is returned to below the AAMI standard level.⁸²¹

Reuse of dialyzers and use of bicarbonate dialysate, high-flux dialyzer membranes, or high-flux dialysis may increase the potential for pyrogenic reactions if the water in the dialysis setting does not meet standards.^{796–798} Although investigators have been unable to demonstrate endotoxin transfer across dialyzer membranes,^{803, 822, 823} the preponderance of reports now supports the ability of endotoxin to transfer across at least some high-flux membranes under some operating conditions. In addition to the acute risk of pyrogenic reactions, indirect evidence is increasingly demonstrating that chronic exposure to low amounts of endotoxin may play a role in some of the long-term complications of hemodialysis therapy. Patients treated with ultrafiltered dialysate for 5–6 months have demonstrated a decrease in serum β_2 microglobulin concentrations and a decrease in markers of an inflammatory response.^{824–826} In studies of longer duration, use of microbiologically ultrapure dialysate has been associated with a decreased incidence of β_2 microglobulin-associated amyloidosis.^{827, 828}

Although patient benefit likely is associated with the use of ultrapure dialysate, no consensus has been reached regarding the potential adoption of this as standard in the United States. Debate continues regarding the bacterial and endotoxin limits for dialysate. As advances in water treatment and hemodialysis processes occur, efforts are underway to move improved technology from the manufacturer out into the user community. Cost-benefit studies, however, have not been done, and substantially increased costs to implement newer water treatment modalities are anticipated.

To reconcile AAMI documents with current International Organization for Standardization (ISO) format, AAMI has determined that its hemodialysis standards will be discussed in the following four installments: RD 5 for hemodialysis equipment, RD 62 for product water quality, RD 47 for dialyzer

reprocessing, and RD 52 for dialysate quality. The Renal Diseases and Dialysis Committee of AAMI is expected to finalize and promulgated the dialysate standard pertinent to the user community (RD 52), adopting by reference the bacterial and endotoxin limits in product water as currently outlined in the AAMI standard that applies to systems manufacturers (RD 62). At present, the user community should continue to observe water quality and dialysate standards as outlined in AAMI RD 5 (Hemodialysis Systems, 1992) and AAMI RD 47 (Reuse of Hemodialyzers, 1993) until the new RD 52 standard becomes available (Table 18).^{789, 791}

Table 18. Microbiologic limits for hemodialysis fluids*

Hemodialysis fluid	Maximum total heterotrophs (CFU/mL)+	Maximum endotoxin level (EU/mL)§
<i>Present standard</i>		
Product water¶		
Used to prepare dialysate	200	No standard
Used to reprocess dialyzers	200	5
Dialysate	2,000	No standard
<i>Proposed standard**</i>		
Product water	200	2
Dialysate	200	2

* The material in this table was compiled from references 789 and 791 (ANSI/AAMI standards RD 5-1992 and ANSI/AAMI RD 47-1993).

+ Colony forming units per milliliter.

§ Endotoxin units per milliliter.

¶ Product water presently includes water used to prepare dialysate and water used to reprocess dialyzers.

** Dialysate for hemodialysis, RD 52, under development, American National Standards Institute, Association for the Advancement of Medical Instrumentation (AAMI).

The current AAMI standard directed at systems manufacturers (RD 62 [Water Treatment Equipment for Hemodialysis Applications, 2001]) now specifies that all product water used to prepare dialysate or to reprocess dialyzers for multiple use should contain <2 endotoxin units per milliliter (EU/mL).⁷⁹² A level of 2 EU/mL was chosen as the upper limit for endotoxin because this level is easily achieved with contemporary water treatment systems using RO and/or ultrafiltration. CDC has advocated monthly endotoxin testing along with microbiologic assays of water, because endotoxin activity may not correspond to the total heterotrophic plate counts.⁸²⁹ Additionally, the current AAMI standard RD 62 for manufacturers includes action levels for product water. Because 48 hours can elapse between the time of sampling water for microbial contamination and the time when results are received, and because bacterial proliferation can be rapid, action levels for microbial counts and endotoxin concentrations are reported as 50 CFU/mL and 1 EU/mL, respectively, in this revision of the standard.⁷⁹² These recommendations will allow users to initiate corrective action before levels exceed the maximum levels established by the standard.

In hemodialysis, the net movement of water is from the blood to the dialysate, although within the dialyzer, local movement of water from the dialysate to the blood through the phenomenon of back-filtration may occur, particularly in dialyzers with highly permeable membranes.⁸³⁰ In contrast, hemofiltration and hemodiafiltration feature infusion of large volumes of electrolyte solution (20–70 L) into the blood. Increasingly, this electrolyte solution is being prepared on-line from water and concentrate. Because of the large volumes of fluid infused, AAMI considered the necessity of setting more stringent requirements for water to be used in this application, but this organization has not yet established these because of lack of expert consensus and insufficient experience with on-line therapies in the United States. On-line hemofiltration and hemodiafiltration systems use sequential ultrafiltration as the final step in the preparation of infusion fluid. Several experts from AAMI concur that these

point-of-use ultrafiltration systems should be capable of further reducing the bacteria and endotoxin burden of solutions prepared from water meeting the requirements of the AAMI standard to a safe level for infusion.

b. Microbial Control Strategies

The strategy for controlling massive accumulations of gram-negative water bacteria and NTM in dialysis systems primarily involves preventing their growth through proper disinfection of water-treatment systems and hemodialysis machines. Gram-negative water bacteria, their associated lipopolysaccharides (bacterial endotoxins), and NTM ultimately come from the community water supply, and levels of these bacteria can be amplified depending on the water treatment system, dialysate distribution system, type of dialysis machine, and method of disinfection (Table 19).^{634, 794, 831} Control strategies are designed to reduce levels of microbial contamination in water and dialysis fluid to relatively low levels but not to completely eradicate it.

Table 19. Factors influencing microbial contamination in hemodialysis systems

Factors	Comments
<u>Water supply</u> Source of community water Ground water Surface water	Contains endotoxin and bacteria Contains high levels of endotoxin and bacteria
<u>Water treatment at the dialysis center</u> None Filtration Prefilter Absolute filter (depth or membrane filter) Activated carbon filter	Not recommended Particulate filter to protect equipment; does not remove microorganisms Removes bacteria, however, unless the filter is changed frequently or disinfected, bacteria will accumulate and grow through the filter; acts as a significant reservoir of bacteria and endotoxin Removes organics and available chlorine or chloramines; acts as a significant reservoir of bacteria and endotoxin
<u>Water treatment devices</u> Deionization/ion-exchange softener Reverse osmosis (RO) Ultraviolet light Ultrafilter	Both softeners and deionizers are significant reservoirs of bacteria and do not remove endotoxin. Removes bacteria and endotoxin, but must be disinfected; operates at high water pressure Kills some bacteria, but there is no residual; ultraviolet-resistant bacteria can develop if the unit is not properly maintained Removes bacteria and endotoxin; operates on normal line pressure; can be positioned distal to deionizer; must be disinfected
<u>Water and dialysate distribution system</u> Distribution pipes Size Construction Elevation Storage tanks	Oversized diameter and length decrease fluid flow and increase bacterial reservoir for both treated water and centrally-prepared dialysate. Rough joints, dead ends, unused branches, and polyvinyl chloride (PVC) piping can act as bacterial reservoirs. Outlet taps should be located at the highest elevation to prevent loss of disinfectant; keep a recirculation loop in the system; flush unused ports routinely. Tanks are undesirable because they act as a reservoir for water bacteria; if tanks are present, they must be routinely scrubbed and disinfected.
<u>Dialysis machines</u> Single-pass Recirculating single-pass or recirculating (batch)	Disinfectant should have contact with all parts of the machine that are exposed to water or dialysis fluid. Recirculating pumps and machine design allow for massive contamination levels if not properly disinfected; overnight chemical germicide treatment is recommended.

Two components of hemodialysis water distribution systems – pipes (particularly those made of polyvinyl chloride [PVC]) and storage tanks – can serve as reservoirs of microbial contamination. Hemodialysis systems frequently use pipes that are wider and longer than are needed to handle the required flow, which slows the fluid velocity and increases both the total fluid volume and the wetted surface area of the system. Gram-negative bacteria in fluids remaining in pipes overnight multiply rapidly and colonize the wet surfaces, producing bacterial populations and endotoxin quantities in proportion to the volume and surface area. Such colonization results in formation of protective biofilm that is difficult to remove and protects the bacteria from disinfection.⁸³² Routine (i.e., monthly), low-level disinfection of the pipes can help to control bacterial contamination of the distribution system. Additional measures to protect pipes from contaminations include a) situating all outlet taps at equal elevation and at the highest point of the system so that the disinfectant cannot drain from pipes by gravity before adequate contact time has elapsed and b) eliminating rough joints, dead-end pipes, and unused branches and taps that can trap fluid and serve as reservoirs of bacteria capable of continuously inoculating the entire volume of the system.⁸⁰⁰ Maintain a flow velocity of 3–5 ft/sec.

A storage tank in the distribution system greatly increases the volume of fluid and surface area available and can serve as a niche for water bacteria. Storage tanks are therefore not recommended for use in dialysis systems unless they are frequently drained and adequately disinfected, including scrubbing the sides of the tank to remove bacterial biofilm. An ultrafilter should be used distal to the storage tank.^{808, 833}

Microbiologic sampling of dialysis fluids is recommended because gram-negative bacteria can proliferate rapidly in water and dialysate in hemodialysis systems; high levels of these organisms place patients at risk for pyrogenic reactions or health-care–associated infection.^{667, 668, 808}

Health-care facilities are advised to sample dialysis fluids at least monthly using standard microbiologic assay methods for waterborne microorganisms.^{788, 793, 799, 834–836} Product water used to prepare dialysate and to reprocess hemodialyzers for reuse on the same patient should also be tested for bacterial endotoxin on a monthly basis.^{792, 829, 837} (See Appendix C for information about water sampling methods for dialysis.)

Cross-contamination of dialysis machines and inadequate disinfection measures can facilitate the spread of waterborne organisms to patients. Steps should be taken to ensure that dialysis equipment is performing correctly and that all connectors, lines, and other components are specific for the equipment, in good repair, and properly in place. A recent outbreak of gram-negative bacteremias among dialysis patients was attributed to faulty valves in a drain port of the machine that allowed backflow of saline used to flush the dialyzer before patient use.^{838, 839} This backflow contaminated the drain priming connectors, which contaminated the blood lines and exposed the patients to high concentrations of gram-negative bacteria. Environmental infection control in dialysis settings also includes low-level disinfection of housekeeping surfaces and spot decontamination of spills of blood (see Environmental Services in Part I of this guideline for further information).

c. Infection-Control Issues in Peritoneal Dialysis

Peritoneal dialysis (PD), most commonly administered as continuous ambulatory peritoneal dialysis (CAPD) and continual cycling peritoneal dialysis (CCPD), is the third most common treatment for end-stage renal disease (ESRD) in the United States, accounting for 12% of all dialysis patients.⁸⁴⁰ Peritonitis is the primary complication of CAPD, with coagulase-negative staphylococci the most clinically significant causative organisms.⁸⁴¹ Other organisms that have been found to produce peritonitis include *Staphylococcus aureus*, *Mycobacterium fortuitum*, *M. mucogenicum*, *Stenotrophomonas maltophilia*, *Burkholderia cepacia*, *Corynebacterium jeikeium*, *Candida* spp., and

other fungi.⁸⁴²⁻⁸⁵⁰ Substantial morbidity is associated with peritoneal dialysis infections. Removal of peritoneal dialysis catheters usually is required for treatment of peritonitis caused by fungi, NTM, or other bacteria that are not cleared within the first several days of effective antimicrobial treatment. Furthermore, recurrent episodes of peritonitis may lead to fibrosis and loss of the dialysis membrane.

Many reported episodes of peritonitis are associated with exit-site or tunneled catheter infections. Risk factors for the development of peritonitis in PD patients include a) under dialysis, b) immune suppression, c) prolonged antimicrobial treatment, d) patient age [more infections occur in younger patients and older hospitalized patients], e) length of hospital stay, and f) hypoalbuminemia.^{844, 851, 852} Concern has been raised about infection risk associated with the use of automated cyclers in both inpatient and outpatient settings; however, studies suggest that PD patients who use automated cyclers have much lower infection rates.⁸⁵³ One study noted that a closed-drainage system reduced the incidence of system-related peritonitis among intermittent peritoneal dialysis (IPD) patients from 3.6 to 1.5 cases/100 patient days.⁸⁵⁴ The association of peritonitis with management of spent dialysate fluids requires additional study. Therefore, ensuring that the tip of the waste line is not submerged beneath the water level in a toilet or in a drain is prudent.

7. Ice Machines and Ice

Microorganisms may be present in ice, ice-storage chests, and ice-making machines. The two main sources of microorganisms in ice are the potable water from which it is made and a transferral of organisms from hands (Table 20). Ice from contaminated ice machines has been associated with patient colonization, blood stream infections, pulmonary and gastrointestinal illnesses, and pseudoinfections.^{602, 603, 683, 684, 854, 855} Microorganisms in ice can secondarily contaminate clinical specimens and medical solutions that require cold temperatures for either transport or holding.^{601, 620} An outbreak of surgical-site infections was interrupted when sterile ice was used in place of tap water ice to cool cardioplegia solutions.⁶⁰¹

Table 20. Microorganisms and their sources in ice and ice machines

Sources of microorganisms	References
From potable water	
<i>Legionella</i> spp.	684, 685, 857, 858
Nontuberculous mycobacteria (NTM)	602, 603, 859
<i>Pseudomonas aeruginosa</i>	859
<i>Burkholderia cepacia</i>	859, 860
<i>Stenotrophomonas maltophilia</i>	860
<i>Flavobacterium</i> spp.	860
From fecally-contaminated water	
Norwalk virus	861-863
<i>Giardia lamblia</i>	864
<i>Cryptosporidium parvum</i>	685
From hand-transfer of organisms	
<i>Acinetobacter</i> spp.	859
Coagulase-negative staphylococci	859
<i>Salmonella enteritidis</i>	865
<i>Cryptosporidium parvum</i>	685

In a study comparing the microbial populations of hospital ice machines with organisms recovered from ice samples gathered from the community, samples from 27 hospital ice machines yielded low numbers (<10 CFU/mL) of several potentially opportunistic microorganisms, mainly gram-negative bacilli.⁸⁵⁹ During the survey period, no health-care-associated infections were attributed to the use of ice. Ice from community sources had higher levels of microbial contamination (75%–95% of 194 samples had total heterotrophic plate counts <500 CFU/mL, with the proportion of positive cultures dependent on the incubation temperature) and showed evidence of fecal contamination from the source water.⁸⁵⁹ Thus, ice machines in health-care settings are no more heavily contaminated compared with ice machines in the community. If the source water for ice in a health-care facility is not fecally contaminated, then ice from clean ice machines and chests should pose no increased hazard for immunocompetent patients. Some waterborne bacteria found in ice could potentially be a risk to immunocompromised patients if they consume ice or drink beverages with ice. For example, *Burkholderia cepacia* in ice could present an infection risk for cystic fibrosis patients.^{859, 860} Therefore, protecting immunosuppressed and otherwise medically at-risk patients from exposure to tap water and ice potentially contaminated with opportunistic pathogens is prudent.⁹

No microbiologic standards for ice, ice-making machines, or ice storage equipment have been established, although several investigators have suggested the need for such standards.^{859, 866} Culturing of ice machines is not routinely recommended, but it may be useful as part of an epidemiologic investigation.^{867–869} Sampling might also help determine the best schedule for cleaning open ice-storage chests. Recommendations for a regular program of maintenance and disinfection have been published.^{866–869} Health-care facilities are advised to clean ice-storage chests on a regular basis. Open ice chests may require a more frequent cleaning schedule compared with chests that have covers. Portable ice chests and containers require cleaning and low-level disinfection before the addition of ice intended for consumption. Ice-making machines may require less frequent cleaning, but their maintenance is important to proper performance. The manufacturer's instructions for both the proper method of cleaning and/or maintenance should be followed. These instructions may also recommend an EPA-registered disinfectant to ensure chemical potency, materials compatibility, and safety. In the event that instructions and suitable EPA-registered disinfectants are not available for this process, then a generic approach to cleaning, disinfecting, and maintaining ice machines and dispensers can be used (Box 12).

Ice and ice-making machines also may be contaminated via improper storage or handling of ice by patients and/or staff.^{684–686, 855–858, 870} Suggested steps to avoid this means of contamination include a) minimizing or avoiding direct hand contact with ice intended for consumption, b) using a hard-surface scoop to dispense ice, and c) installing machines that dispense ice directly into portable containers at the touch of a control.^{687, 869}

Box 12. General steps for cleaning and maintaining ice machines, dispensers, and storage chests*+

-
1. Disconnect unit from power supply.
 2. Remove and discard ice from bin or storage chest.
 3. Allow unit to warm to room temperature.
 4. Disassemble removable parts of machine that make contact with water to make ice.
 5. Thoroughly clean machine and parts with water and detergent.
 6. Dry external surfaces of removable parts before reassembling.
 7. Check for any needed repair.
 8. Replace feeder lines, as appropriate (e.g., when damaged, old, or difficult to clean).
 9. Ensure presence of an air space in tubing leading from water inlet into water distribution system of machine.

(Box 12. continued)

10. Inspect for rodent or insect infestations under the unit and treat, as needed.
11. Check door gaskets (open compartment models) for evidence of leakage or dripping into the storage chest.
12. Clean the ice-storage chest or bin with fresh water and detergent; rinse with fresh tap water.
13. Sanitize machine by circulating a 50–100 parts per million (ppm) solution of sodium hypochlorite (i.e., 4–8 mL sodium hypochlorite/gallon of water) through the ice-making and storage systems for 2 hours (100 ppm solution), or 4 hours (50 ppm solution).
14. Drain sodium hypochlorite solutions and flush with fresh tap water.
15. Allow all surfaces of equipment to dry before returning to service.

* Material in this box is adapted from reference 869.

+ These general guidelines should be used only where manufacturer-recommended methods and EPA-registered disinfectants are not available.

8. Hydrotherapy Tanks and Pools

a. General Information

Hydrotherapy equipment (e.g., pools, whirlpools, whirlpool spas, hot tubs, and physiotherapy tanks) traditionally has been used to treat patients with certain medical conditions (e.g., burns,^{871, 872} septic ulcers, lesions, amputations,⁸⁷³ orthopedic impairments and injuries, arthritis,⁸⁷⁴ and kidney lithotripsy).⁶⁵⁴ Wound-care medicine is increasingly moving away from hydrotherapy, however, in favor of bedside pulsed-lavage therapy using sterile solutions for cleaning and irrigation.^{492, 875–878}

Several episodes of health-care-associated infections have been linked to use of hydrotherapy equipment (Table 21). Potential routes of infection include incidental ingestion of the water, sprays and aerosols, and direct contact with wounds and intact skin (folliculitis). Risk factors for infection include a) age and sex of the patient, b) underlying medical conditions, c) length of time spent in the hydrotherapy water, and d) portals of entry.⁸⁷⁹

Table 21. Infections associated with use of hydrotherapy equipment

Microorganisms	Medical conditions	References
<i>Acinetobacter baumannii</i>	Sepsis	572
<i>Citrobacter freundii</i>	Cellulitis	880
<i>Enterobacter cloacae</i>	Sepsis	881
<i>Legionella</i> spp.	Legionellosis	882
<i>Mycobacterium abscessus</i> , <i>Mycobacterium fortuitum</i> , <i>Mycobacterium marinum</i>	Skin ulcers and soft tissue infections	621–623, 883
<i>Pseudomonas aeruginosa</i>	Sepsis, soft tissue infections, folliculitis, and wound infections	492, 493, 506, 679, 884–888
Adenovirus, adeno-associated virus	Conjunctivitis	889

Infection control for hydrotherapy tanks, pools, or birthing tanks presents unique challenges because indigenous microorganisms are always present in the water during treatments. In addition, some studies have found free living amoebae (i.e., *Naegleria lovaniensis*), which are commonly found in association with *Naegleria fowleri*, in hospital hydrotherapy pools.⁸⁹⁰ Although hydrotherapy is at times appropriate for patients with wounds, burns, or other types of non-intact skin conditions (determined on a case-by-case basis), this equipment should not be considered “semi-critical” in accordance with the Spaulding classification.⁸⁹¹ Microbial data to evaluate the risk of infection to patients using hydrotherapy pools and birthing tanks are insufficient. Nevertheless, health-care facilities should maintain stringent cleaning and disinfection practices in accordance with the manufacturer’s instructions

and with relevant scientific literature until data supporting more rigorous infection-control measures become available. Factors that should be considered in therapy decisions in this situation would include a) availability of alternative aseptic techniques for wound management and b) a risk-benefit analysis of using traditional hydrotherapy.

b. Hydrotherapy Tanks

Hydrotherapy tanks (e.g., whirlpools, Hubbard tanks and whirlpool bath tubs) are shallow tanks constructed of stainless steel, plexiglass, or tile. They are closed-cycle water systems with hydrojets to circulate, aerate, and agitate the water. The maximum water temperature range is 50°F–104°F (10°C–40°C). The warm water temperature, constant agitation and aeration, and design of the hydrotherapy tanks provide ideal conditions for bacterial proliferation if the equipment is not properly maintained, cleaned, and disinfected. The design of the hydrotherapy equipment should be evaluated for potential infection-control problems that can be associated with inaccessible surfaces that can be difficult to clean and/or remain wet in between uses (i.e., recessed drain plates with fixed grill plates).⁸⁸⁷ Associated equipment (e.g., parallel bars, plinths, Hoyer lifts, and wheelchairs) can also be potential reservoirs of microorganisms, depending on the materials used in these items (i.e., porous vs. non-porous materials) and the surfaces that may become wet during use. Patients with active skin colonizations and wound infections can serve as sources of contamination for the equipment and the water. Contamination from spilled tub water can extend to drains, floors, and walls.^{680–683} Health-care-associated colonization or infection can result from exposure to endogenous sources of microorganisms (autoinoculation) or exogenous sources (via cross-contamination from other patients previously receiving treatment in the unit).

Although some facilities have used tub liners to minimize environmental contamination of the tanks, the use of a tub liner does not eliminate the need for cleaning and disinfection. Draining these small pools and tanks after each patient use, thoroughly cleaning with a detergent, and disinfecting according to manufacturers' instructions have reduced bacterial contamination levels in the water from 10⁴ CFU/mL to <10 CFU/mL.⁸⁹² A chlorine residual of 15 ppm in the water should be obtained prior to the patient's therapy session (e.g., by adding 15 grams of calcium hypochlorite 70% [e.g., HTH®] per 100 gallons of water).⁸⁹² A study of commercial and residential whirlpools found that superchlorination or draining, cleaning, disinfection, and refilling of whirlpools markedly reduced densities of *Pseudomonas aeruginosa* in whirlpool water.⁸⁹³ The bacterial populations were rapidly replenished, however, when disinfectant concentrations dropped below recommended levels for recreational use (i.e., chlorine at 3.0 ppm or bromine at 6.0 ppm). When using chlorine, however, knowing whether the community drinking-water system is disinfected with chloramine is important, because municipal utilities adjust the pH of the water to the basic side to enhance chloramine formation. Because chlorine is not very effective at pH levels above 8, it may be necessary to re-adjust the pH of the water to a more acidic level.⁸⁹⁴

A few reports describe the addition of antiseptic chemicals to hydrotherapy tank water, especially for burn patient therapy.^{895–897} One study involving a minimal number of participants demonstrated a reduction in the number of *Pseudomonas* spp. and other gram-negative bacteria from both patients and equipment surfaces when chloramine-T ("chlorazene") was added to the water.⁸⁹⁸ Chloramine-T has not, however, been approved for water treatment in the United States.

c. Hydrotherapy Pools

Hydrotherapy pools typically serve large numbers of patients and are usually heated to 91.4°F–98.6°F (31°C–37°C). The temperature range is more narrow (94°F–96.8°F [35°C–36°C]) for pediatric and geriatric patient use.⁸⁹⁹ Because the size of hydrotherapy pools precludes draining after patient use, proper management is required to maintain the proper balance of water conditioning (i.e., alkalinity, hardness, and temperature) and disinfection. The most widely used chemicals for disinfection of pools

are chlorine and chlorine compounds – calcium hypochlorite, sodium hypochlorite, lithium hypochlorite, chloroisocyanurates, and chlorine gas. Solid and liquid formulations of chlorine chemicals are the easiest and safest to use.⁹⁰⁰ Other halogenated compounds have also been used for pool-water disinfection, albeit on a limited scale. Bromine, which forms bactericidal bromamines in the presence of ammonia, has limited use because of its association with contact dermatitis.⁹⁰¹ Iodine does not bleach hair, swim suits, or cause eye irritation, but when introduced at proper concentrations, it gives water a greenish-yellowish cast.⁸⁹²

In practical terms, maintenance of large hydrotherapy pools (e.g., those used for exercise) is similar to that for indoor public pools (i.e., continuous filtration, chlorine residuals no less than 0.4 ppm, and pH of 7.2–7.6).^{902,903} Supply pipes and pumps also need to be maintained to eliminate the possibility of this equipment serving as a reservoir for waterborne organisms.⁹⁰⁴ Specific standards for chlorine residual and pH of the water are addressed in local and state regulations. Patients who are fecally incontinent or who have draining wounds should refrain from using these pools until their condition improves.

d. Birthing Tanks and Other Equipment

The use of birthing tanks, whirlpool spas, and whirlpools is a recent addition to obstetrical practice.⁹⁰⁵ Few studies on the potential risks associated with these pieces of equipment have been conducted. In one study of 32 women, a newborn contracted a *Pseudomonas* infection after being birthed in such a tank, the strain of which was identical to the organism isolated from the tank water.⁹⁰⁶ Another report documented identical strains of *P. aeruginosa* isolates from a newborn with sepsis and on the environmental surfaces of a tub that the mother used for relaxation while in labor.⁹⁰⁷ Other studies have shown no significant increases in the rates of post-immersion infections among mothers and infants.^{908,909}

Because the water and the tub surfaces routinely become contaminated with the mother's skin flora and blood during labor and delivery, birthing tanks and other tub equipment must be drained after each patient use and the surfaces thoroughly cleaned and disinfected. Health-care facilities are advised to follow the manufacturer's instructions for selection of disinfection method and chemical germicide. The range of chlorine residuals for public whirlpools and whirlpool spas is 2–5 ppm.⁹¹⁰ Use of an inflatable tub is an alternative solution, but this item must be cleaned and disinfected between patients if it is not considered a single-use unit.

Recreational tanks and whirlpool spas are increasingly being used as hydrotherapy equipment. Although such home equipment appears to be suitable for hydrotherapy, they are neither designed nor constructed to function in this capacity. Additionally, manufacturers generally are not obligated to provide the health-care facility with cleaning and disinfecting instructions appropriate for medical equipment use, and the U.S. Food and Drug Administration (FDA) does not evaluate recreational equipment. Health-care facilities should therefore carefully evaluate this “off-label” use of home equipment before proceeding with a purchase.

9. Miscellaneous Medical/Dental Equipment Connected to Main Water Systems

a. Automated Endoscope Reprocessors

The automated endoscopic reprocessor (AER) is classified by the FDA as an accessory for the flexible endoscope.⁶⁵⁴ A properly operating AER can provide a more consistent, reliable method of decontaminating and terminal reprocessing for endoscopes between patient procedures than manual reprocessing methods alone.⁹¹¹ An endoscope is generally subjected to high-level disinfection using a

liquid chemical sterilant or a high-level disinfectant. Because the instrument is a semi-critical device, the optimal rinse fluid for a disinfected endoscope would be sterile water.³ Sterile water, however, is expensive and difficult to produce in sufficient quantities and with adequate quality assurance for instrument rinsing in an AER.^{912, 913} Therefore, one option to be used for AERs is rinse water that has been passed through filters with a pore size of 0.1–0.2 μm to render the water “bacteria-free.” These filters usually are located in the water line at or near the port where the mains water enters the equipment. The product water (i.e., tap water passing through these filters) in these applications is not considered equivalent in microbial quality to that for membrane-filtered water as produced by pharmaceutical firms. Membrane filtration in pharmaceutical applications is intended to ensure the microbial quality of polished product water.

Water has been linked to the contamination of flexible fiberoptic endoscopes in the following two scenarios: a) rinsing a disinfected endoscope with unfiltered tap water, followed by storage of the instrument without drying out the internal channels and b) contamination of AERs from tap water inadvertently introduced into the equipment. In the latter instance, the machine’s water reservoirs and fluid circuitry become contaminated with waterborne, heterotrophic bacteria (e.g., *Pseudomonas aeruginosa* and NTM), which can survive and persist in biofilms attached to these components.^{914–917} Colonization of the reservoirs and water lines of the AER becomes problematic if the required cleaning, disinfection, and maintenance are not performed on the equipment as recommended by the manufacturer.^{669, 916, 917} Use of the 0.1–0.2- μm filter in the water line helps to keep bacterial contamination to a minimum,^{670, 911, 917} but filters may fail and allow bacteria to pass through to the equipment and then to the instrument undergoing reprocessing.^{671–674, 913, 918} Filters also require maintenance for proper performance.^{670, 911, 912, 918, 919} Heightened awareness of the proper disinfection of the connectors that hook the instrument to the AER may help to further reduce the potential for contaminating endoscopes during reprocessing.⁹²⁰ An emerging issue in the field of endoscopy is that of the possible role of rinse water monitoring and its potential to help reduce endoscopy/bronchoscopy-associated infections.⁹¹⁸

Studies have linked deficiencies in endoscope cleaning and/or disinfecting processes to the incidence of post-endoscopic adverse outcomes.^{921–924} Several clusters have been traced to AERs of older designs and these were associated with water quality.^{675, 914–916} Regardless of whether manual or automated terminal reprocessing is used for endoscopes, the internal channels of the instrument should be dried before storage.⁹²⁵ The presence of residual moisture in the internal channels encourages the proliferation of waterborne microorganisms, some of which may be pathogenic. One of the most frequently used methods employs 70% isopropyl alcohol to flush the internal channels, followed by forced air drying of these channels and hanging the endoscope vertically in a protected cabinet; this method ensures internal drying of the endoscope, lessens the potential for proliferation of waterborne microorganisms,^{669, 913, 917, 922, 926, 927} and is consistent with professional organization guidance for endoscope reprocessing.⁹²⁸

An additional problem with waterborne microbial contamination of AERs centers on increased microbial resistance to alkaline glutaraldehyde, a widely used liquid chemical sterilant/high-level disinfectant.^{669, 929} Opportunistic waterborne microorganisms (e.g., *Mycobacterium chelonae*, *Methylobacterium* spp.) have been associated with pseudo-outbreaks and colonization; infection caused by these organisms has been associated with procedures conducted in clinical settings (e.g., bronchoscopy).^{669, 913, 929–931} Increasing microbial resistance to glutaraldehyde has been attributed to improper use of the disinfectant in the equipment, allowing the dilution of glutaraldehyde to fall below the manufacturer’s recommended minimal use concentration.⁹²⁹

b. Dental Unit Water Lines

Dental unit water lines (DUWLs) consist of small-bore plastic tubing that delivers water used for general, non-surgical irrigation and as a coolant to dental handpieces, sonic and ultrasonic scalers, and air-water syringes; municipal tap water is the source water for these lines. The presence of biofilms of waterborne bacteria and fungi (e.g., *Legionella* spp., *Pseudomonas aeruginosa*, and NTM) in DUWLs has been established.^{636, 637, 694, 695, 932–954} Biofilms continually release planktonic microorganisms into the water, the titers of which can exceed 1×10^6 CFU/mL.⁶⁹⁴ However, scientific evidence indicates that immunocompetent persons are only at minimal risk for substantial adverse health effects after contact with water from a dental unit. Nonetheless, exposing patients or dental personnel to water of uncertain microbiological quality is not consistent with universally accepted infection-control principles.⁹³⁵

In 1993, CDC issued guidelines relative to water quality in a dental setting. These guidelines recommend that all dental instruments that use water (including high-speed handpieces) should be run to discharge water for 20–30 seconds after each patient and for several minutes before the start of each clinic day.⁹³⁶ This practice can help to flush out any patient materials that may have entered the turbine, air, or waterlines.^{937, 938} The 1993 guidance also indicated that waterlines be flushed at the beginning of the clinic day. Although these guidelines are designed to help reduce the number of microorganisms present in treatment water, they do not address the issue of reducing or preventing biofilm formation in the waterlines. Research published subsequent to the 1993 dental infection control guideline suggests that flushing the lines at the beginning of the day has only minimal effect on the status of the biofilm in the lines and does not reliably improve the quality of water during dental treatment.^{939–941} Updated recommendations on infection-control practices for water line use in dentistry will be available in late 2003.⁹⁴²

The numbers of microorganisms in water used as coolant or irrigant for non-surgical dental treatment should be as low as reasonably achievable and, at a minimum, should meet nationally recognized standards for safe drinking water.^{935, 943} Only minimal evidence suggests that water meeting drinking water standards poses a health hazard for immunocompetent persons. The EPA, the American Public Health Association (APHA), and the American Water Works Association (AWWA) have set a maximum limit of 500 CFU/mL for aerobic, heterotrophic, mesophilic bacteria in drinking water in municipal distribution systems.^{944, 945} This standard is achievable, given improvements in water-line technology. Dentists should consult with the manufacturer of their dental unit to determine the best equipment and method for maintaining and monitoring good water quality.^{935, 946}

E. Environmental Services

1. Principles of Cleaning and Disinfecting Environmental Surfaces

Although microbiologically contaminated surfaces can serve as reservoirs of potential pathogens, these surfaces generally are not directly associated with transmission of infections to either staff or patients. The transferral of microorganisms from environmental surfaces to patients is largely via hand contact with the surface.^{947, 948} Although hand hygiene is important to minimize the impact of this transfer, cleaning and disinfecting environmental surfaces as appropriate is fundamental in reducing their potential contribution to the incidence of healthcare-associated infections.

The principles of cleaning and disinfecting environmental surfaces take into account the intended use of the surface or item in patient care. CDC retains the Spaulding classification for medical and surgical instruments, which outlines three categories based on the potential for the instrument to transmit infection if the instrument is microbiologically contaminated before use.^{949, 950} These categories are

“critical,” “semicritical,” and “noncritical.” In 1991, CDC proposed an additional category designated “environmental surfaces” to Spaulding’s original classification⁹⁵¹ to represent surfaces that generally do not come into direct contact with patients during care. Environmental surfaces carry the least risk of disease transmission and can be safely decontaminated using less rigorous methods than those used on medical instruments and devices. Environmental surfaces can be further divided into medical equipment surfaces (e.g., knobs or handles on hemodialysis machines, x-ray machines, instrument carts, and dental units) and housekeeping surfaces (e.g., floors, walls, and tabletops).⁹⁵¹

The following factors influence the choice of disinfection procedure for environmental surfaces: a) the nature of the item to be disinfected, b) the number of microorganisms present, c) the innate resistance of those microorganisms to the inactivating effects of the germicide, d) the amount of organic soil present, e) the type and concentration of germicide used, f) duration and temperature of germicide contact, and g) if using a proprietary product, other specific indications and directions for use.^{952, 953}

Cleaning is the necessary first step of any sterilization or disinfection process. Cleaning is a form of decontamination that renders the environmental surface safe to handle or use by removing organic matter, salts, and visible soils, all of which interfere with microbial inactivation.⁹⁵⁴⁻⁹⁶⁰ The physical action of scrubbing with detergents and surfactants and rinsing with water removes large numbers of microorganisms from surfaces.⁹⁵⁷ If the surface is not cleaned before the terminal reprocessing procedures are started, the success of the sterilization or disinfection process is compromised.

Spaulding proposed three levels of disinfection for the treatment of devices and surfaces that do not require sterility for safe use. These disinfection levels are “high-level,” “intermediate-level,” and “low-level.”^{949, 950} The basis for these levels is that microorganisms can usually be grouped according to their innate resistance to a spectrum of physical or chemical germicidal agents (Table 22). This information, coupled with the instrument/surface classification, determines the appropriate level of terminal disinfection for an instrument or surface.

Table 22. Levels of disinfection by type of microorganism*

Disinfection level	Bacteria			Fungi+	Viruses	
	Vegetative	Tubercle bacillus	Spores		Lipid and medium size	Nonlipid and small size
High	+ §	+	+ ¶	+	+	+
Intermediate	+	+	—**	+	+	± ⁺⁺
Low	+	—	—	±	+	±

* Material in this table compiled from references 2 and 951.

+ This class of microorganisms includes asexual spores but not necessarily chlamydo spores or sexual spores.

§ The “plus” sign indicates that a killing effect can be expected when the normal use-concentrations of chemical disinfectants or pasteurization are properly employed; a “negative” sign indicates little or no killing effect.

¶ Only with extended exposure times are high-level disinfectant chemicals capable of killing high numbers of bacterial spores in laboratory tests; they are, however, capable of sporicidal activity.

** Some intermediate-level disinfectants (e.g., hypochlorites) can exhibit some sporicidal activity; others (e.g., alcohols and phenolics) have no demonstrable sporicidal activity.

++ Some intermediate-level disinfectants, although they are tuberculocidal, may have limited virucidal activity.

The process of high-level disinfection, an appropriate standard of treatment for heat-sensitive, semi-critical medical instruments (e.g., flexible, fiberoptic endoscopes), inactivates all vegetative bacteria, mycobacteria, viruses, fungi, and some bacterial spores. High-level disinfection is accomplished with powerful, sporicidal chemicals (e.g., glutaraldehyde, peracetic acid, and hydrogen peroxide) that are not appropriate for use on housekeeping surfaces. These liquid chemical sterilants/high-level disinfectants

are highly toxic.^{961–963} Use of these chemicals for applications other than those indicated in their label instructions (i.e., as immersion chemicals for treating heat-sensitive medical instruments) is not appropriate.⁹⁶⁴ Intermediate-level disinfection does not necessarily kill bacterial spores, but it does inactivate *Mycobacterium tuberculosis* var. *bovis*, which is substantially more resistant to chemical germicides than ordinary vegetative bacteria, fungi, and medium to small viruses (with or without lipid envelopes). Chemical germicides with sufficient potency to achieve intermediate-level disinfection include chlorine-containing compounds (e.g., sodium hypochlorite), alcohols, some phenolics, and some iodophors. Low-level disinfection inactivates vegetative bacteria, fungi, enveloped viruses (e.g., human immunodeficiency virus [HIV], and influenza viruses), and some non-enveloped viruses (e.g., adenoviruses). Low-level disinfectants include quaternary ammonium compounds, some phenolics, and some iodophors. Sanitizers are agents that reduce the numbers of bacterial contaminants to safe levels as judged by public health requirements, and are used in cleaning operations, particularly in food service and dairy applications. Germicidal chemicals that have been approved by FDA as skin antiseptics are not appropriate for use as environmental surface disinfectants.⁹⁵¹

The selection and use of chemical germicides are largely matters of judgment, guided by product label instructions, information, and regulations. Liquid sterilant chemicals and high-level disinfectants intended for use on critical and semi-critical medical/dental devices and instruments are regulated exclusively by the FDA as a result of recent memoranda of understanding between FDA and the EPA that delineates agency authority for chemical germicide regulation.^{965, 966} Environmental surface germicides (i.e., primarily intermediate- and low-level disinfectants) are regulated by the EPA and labeled with EPA registration numbers. The labels and technical data or product literature of these germicides specify indications for product use and provide claims for the range of antimicrobial activity. The EPA requires certain pre-registration laboratory potency tests for these products to support product label claims. EPA verifies (through laboratory testing) manufacturers' claims to inactivate microorganisms for selected products and organisms. Germicides labeled as "hospital disinfectant" have passed the potency tests for activity against three representative microorganisms – *Pseudomonas aeruginosa*, *Staphylococcus aureus*, and *Salmonella cholerae suis*. Low-level disinfectants are often labeled "hospital disinfectant" without a tuberculocidal claim, because they lack the potency to inactivate mycobacteria. Hospital disinfectants with demonstrated potency against mycobacteria (i.e., intermediate-level disinfectants) may list "tuberculocidal" on the label as well. Other claims (e.g., "fungicidal," "pseudomonocidal," and "virucidal") may appear on labels of environmental surface germicides, but the designations of "tuberculocidal hospital disinfectant" and "hospital disinfectant" correlate directly to Spaulding's assessment of intermediate-level disinfectants and low-level disinfectants, respectively.⁹⁵¹

A common misconception in the use of surface disinfectants in health-care settings relates to the underlying purpose for use of proprietary products labeled as a "tuberculocidal" germicide. Such products will not interrupt and prevent the transmission of TB in health-care settings because TB is not acquired from environmental surfaces. The tuberculocidal claim is used as a benchmark by which to measure germicidal potency. Because mycobacteria have the highest intrinsic level of resistance among the vegetative bacteria, viruses, and fungi, any germicide with a tuberculocidal claim on the label (i.e., an intermediate-level disinfectant) is considered capable of inactivating a broad spectrum of pathogens, including much less resistant organisms such as the bloodborne pathogens (e.g., hepatitis B virus [HBV], hepatitis C virus [HCV], and HIV). It is this broad spectrum capability, rather than the product's specific potency against mycobacteria, that is the basis for protocols and OSHA regulations indicating the appropriateness of using tuberculocidal chemicals for surface disinfection.⁹⁶⁷

2. General Cleaning Strategies for Patient-Care Areas

The number and types of microorganisms present on environmental surfaces are influenced by the following factors: a) number of people in the environment, b) amount of activity, c) amount of moisture, d) presence of material capable of supporting microbial growth, e) rate at which organisms suspended in the air are removed, and f) type of surface and orientation [i.e., horizontal or vertical].⁹⁶⁸ Strategies for cleaning and disinfecting surfaces in patient-care areas take into account a) potential for direct patient contact, b) degree and frequency of hand contact, and c) potential contamination of the surface with body substances or environmental sources of microorganisms (e.g., soil, dust, and water).

a. Cleaning of Medical Equipment

Manufacturers of medical equipment should provide care and maintenance instructions specific to their equipment. These instructions should include information about a) the equipments' compatibility with chemical germicides, b) whether the equipment is water-resistant or can be safely immersed for cleaning, and c) how the equipment should be decontaminated if servicing is required.⁹⁶⁷ In the absence of manufacturers' instructions, non-critical medical equipment (e.g., stethoscopes, blood pressure cuffs, dialysis machines, and equipment knobs and controls) usually only require cleansing followed by low- to intermediate-level disinfection, depending on the nature and degree of contamination. Ethyl alcohol or isopropyl alcohol in concentrations of 60%–90% (v/v) is often used to disinfect small surfaces (e.g., rubber stoppers of multiple-dose medication vials, and thermometers)^{952, 969} and occasionally external surfaces of equipment (e.g., stethoscopes and ventilators). However, alcohol evaporates rapidly, which makes extended contact times difficult to achieve unless items are immersed, a factor that precludes its practical use as a large-surface disinfectant.⁹⁵¹ Alcohol may cause discoloration, swelling, hardening, and cracking of rubber and certain plastics after prolonged and repeated use and may damage the shellac mounting of lenses in medical equipment.⁹⁷⁰

Barrier protection of surfaces and equipment is useful, especially if these surfaces are a) touched frequently by gloved hands during the delivery of patient care, b) likely to become contaminated with body substances, or c) difficult to clean. Impervious-backed paper, aluminum foil, and plastic or fluid-resistant covers are suitable for use as barrier protection. An example of this approach is the use of plastic wrapping to cover the handle of the operatory light in dental-care settings.^{936, 942} Coverings should be removed and discarded while the health-care worker is still gloved.^{936, 942} The health-care worker, after ungloving and performing hand hygiene, must cover these surfaces with clean materials before the next patient encounter.

b. Cleaning Housekeeping Surfaces

Housekeeping surfaces require regular cleaning and removal of soil and dust. Dry conditions favor the persistence of gram-positive cocci (e.g., coagulase-negative *Staphylococcus* spp.) in dust and on surfaces, whereas moist, soiled environments favor the growth and persistence of gram-negative bacilli.^{948, 971, 972} Fungi are also present on dust and proliferate in moist, fibrous material.

Most, if not all, housekeeping surfaces need to be cleaned only with soap and water or a detergent/disinfectant, depending on the nature of the surface and the type and degree of contamination. Cleaning and disinfection schedules and methods vary according to the area of the health-care facility, type of surface to be cleaned, and the amount and type of soil present. Disinfectant/detergent formulations registered by EPA are used for environmental surface cleaning, but the actual physical removal of microorganisms and soil by wiping or scrubbing is probably as important, if not more so, than any antimicrobial effect of the cleaning agent used.⁹⁷³ Therefore, cost, safety, product-surface compatibility, and acceptability by housekeepers can be the main criteria for selecting a registered agent. If using a proprietary detergent/disinfectant, the manufacturers' instructions for appropriate use

of the product should be followed.⁹⁷⁴ Consult the products' material safety data sheets (MSDS) to determine appropriate precautions to prevent hazardous conditions during product application. Personal protective equipment (PPE) used during cleaning and housekeeping procedures should be appropriate to the task.

Housekeeping surfaces can be divided into two groups – those with minimal hand-contact (e.g., floors, and ceilings) and those with frequent hand-contact (“high touch surfaces”). The methods, thoroughness, and frequency of cleaning and the products used are determined by health-care facility policy.⁶ However, high-touch housekeeping surfaces in patient-care areas (e.g., doorknobs, bedrails, light switches, wall areas around the toilet in the patient's room, and the edges of privacy curtains) should be cleaned and/or disinfected more frequently than surfaces with minimal hand contact. Infection-control practitioners typically use a risk-assessment approach to identify high-touch surfaces and then coordinate an appropriate cleaning and disinfecting strategy and schedule with the housekeeping staff.

Horizontal surfaces with infrequent hand contact (e.g., window sills and hard-surface flooring) in routine patient-care areas require cleaning on a regular basis, when soiling or spills occur, and when a patient is discharged from the facility.⁶ Regular cleaning of surfaces and decontamination, as needed, is also advocated to protect potentially exposed workers.⁹⁶⁷ Cleaning of walls, blinds, and window curtains is recommended when they are visibly soiled.^{972, 973, 975} Disinfectant fogging is not recommended for general infection control in routine patient-care areas.^{2, 976} Further, paraformaldehyde, which was once used in this application, is no longer registered by EPA for this purpose. Use of paraformaldehyde in these circumstances requires either registration or an exemption issued by EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). Infection control, industrial hygienists, and environmental services supervisors should assess the cleaning procedures, chemicals used, and the safety issues to determine if a temporary relocation of the patient is needed when cleaning in the room.

Extraordinary cleaning and decontamination of floors in health-care settings is unwarranted. Studies have demonstrated that disinfection of floors offers no advantage over regular detergent/water cleaning and has minimal or no impact on the occurrence of health-care-associated infections.^{947, 948, 977–980} Additionally, newly cleaned floors become rapidly recontaminated from airborne microorganisms and those transferred from shoes, equipment wheels, and body substances.^{971, 975, 981} Nevertheless, health-care institutions or contracted cleaning companies may choose to use an EPA-registered detergent/disinfectant for cleaning low-touch surfaces (e.g., floors) in patient-care areas because of the difficulty that personnel may have in determining if a spill contains blood or body fluids (requiring a detergent/disinfectant for clean-up) or when a multi-drug resistant organism is likely to be in the environment. Methods for cleaning non-porous floors include wet mopping and wet vacuuming, dry dusting with electrostatic materials, and spray buffing.^{973, 982–984} Methods that produce minimal mists and aerosols or dispersion of dust in patient-care areas are preferred.^{9, 20, 109, 272}

Part of the cleaning strategy is to minimize contamination of cleaning solutions and cleaning tools. Bucket solutions become contaminated almost immediately during cleaning, and continued use of the solution transfers increasing numbers of microorganisms to each subsequent surface to be cleaned.^{971, 981, 985} Cleaning solutions should be replaced frequently. A variety of “bucket” methods have been devised to address the frequency with which cleaning solutions are replaced.^{986, 987} Another source of contamination in the cleaning process is the cleaning cloth or mop head, especially if left soaking in dirty cleaning solutions.^{971, 988–990} Laundering of cloths and mop heads after use and allowing them to dry before re-use can help to minimize the degree of contamination.⁹⁹⁰ A simplified approach to cleaning involves replacing soiled cloths and mop heads with clean items each time a bucket of detergent/disinfectant is emptied and replaced with fresh, clean solution (B. Stover, Kosair Children's Hospital, 2000). Disposable cleaning cloths and mop heads are an alternative option, if costs permit.

Another reservoir for microorganisms in the cleaning process may be dilute solutions of the detergents or disinfectants, especially if the working solution is prepared in a dirty container, stored for long periods of time, or prepared incorrectly.⁵⁴⁷ Gram-negative bacilli (e.g., *Pseudomonas* spp. and *Serratia marcescens*) have been detected in solutions of some disinfectants (e.g., phenolics and quaternary ammonium compounds).^{547, 991} Contemporary EPA registration regulations have helped to minimize this problem by asking manufacturers to provide potency data to support label claims for detergent/disinfectant properties under real-use conditions (e.g., diluting the product with tap water instead of distilled water). Application of contaminated cleaning solutions, particularly from small-quantity aerosol spray bottles or with equipment that might generate aerosols during operation, should be avoided, especially in high-risk patient areas.^{992, 993} Making sufficient fresh cleaning solution for daily cleaning, discarding any remaining solution, and drying out the container will help to minimize the degree of bacterial contamination. Containers that dispense liquid as opposed to spray-nozzle dispensers (e.g., quart-sized dishwashing liquid bottles) can be used to apply detergent/disinfectants to surfaces and then to cleaning cloths with minimal aerosol generation. A pre-mixed, “ready-to-use” detergent/disinfectant solution may be used if available.

c. Cleaning Special Care Areas

Guidelines have been published regarding cleaning strategies for isolation areas and operating rooms.^{6, 7} The basic strategies for areas housing immunosuppressed patients include a) wet dusting horizontal surfaces daily with cleaning cloths pre-moistened with detergent or an EPA-registered hospital disinfectant or disinfectant wipes;^{94, 98463} b) using care when wet dusting equipment and surfaces above the patient to avoid patient contact with the detergent/disinfectant; c) avoiding the use of cleaning equipment that produces mists or aerosols; d) equipping vacuums with HEPA filters, especially for the exhaust, when used in any patient-care area housing immunosuppressed patients,^{9, 94, 986} and e) regular cleaning and maintenance of equipment to ensure efficient particle removal. When preparing the cleaning cloths for wet-dusting, freshly prepared solutions of detergents or disinfectants should be used rather than cloths that have soaked in such solutions for long periods of time. Dispersal of microorganisms in the air from dust or aerosols is more problematic in these settings than elsewhere in health-care facilities. Vacuum cleaners can serve as dust disseminators if they are not operating properly.⁹⁹⁴ Doors to immunosuppressed patients’ rooms should be closed when nearby areas are being vacuumed.⁹ Bacterial and fungal contamination of filters in cleaning equipment is inevitable, and these filters should be cleaned regularly or replaced as per equipment manufacturer instructions.

Mats with tacky surfaces placed in operating rooms and other patient-care areas only slightly minimize the overall degree of contamination of floors and have little impact on the incidence rate of health-care-associated infection in general.^{351, 971, 983} An exception, however, is the use of tacky mats inside the entry ways of cordoned-off construction areas inside the health-care facility; these mats help to minimize the intrusion of dust into patient-care areas.

Special precautions for cleaning incubators, mattresses, and other nursery surfaces have been recommended to address reports of hyperbilirubinemia in newborns linked to inadequately diluted solutions of phenolics and poor ventilation.⁹⁹⁵⁻⁹⁹⁷ These medical conditions have not, however, been associated with the use of properly prepared solutions of phenolics. Non-porous housekeeping surfaces in neonatal units can be disinfected with properly diluted or pre-mixed phenolics, followed by rinsing with clean water.⁹⁹⁷ However, phenolics are not recommended for cleaning infant bassinets and incubators during the stay of the infant. Infants who remain in the nursery for an extended period should be moved periodically to freshly cleaned and disinfected bassinets and incubators.⁹⁹⁷ If phenolics are used for cleaning bassinets and incubators after they have been vacated, the surfaces should be rinsed thoroughly with water and dried before either piece of equipment is reused. Cleaning

and disinfecting protocols should allow for the full contact time specified for the product used. Bassinet mattresses should be replaced, however, if the mattress cover surface is broken.⁹⁹⁷

3. Cleaning Strategies for Spills of Blood and Body Substances

Neither HBV, HCV, nor HIV has ever been transmitted from a housekeeping surface (i.e., floors, walls, or countertops). Nonetheless, prompt removal and surface disinfection of an area contaminated by either blood or body substances are sound infection-control practices and OSHA requirements.⁹⁶⁷

Studies have demonstrated that HIV is inactivated rapidly after being exposed to commonly used chemical germicides at concentrations that are much lower than those used in practice.^{998–1003} HBV is readily inactivated with a variety of germicides, including quaternary ammonium compounds.¹⁰⁰⁴ Embalming fluids (e.g., formaldehyde) are also capable of completely inactivating HIV and HBV.^{1005, 1006} OSHA has revised its regulation for disinfecting spills of blood or other potentially infectious material to include proprietary products whose label includes inactivation claims for HBV and HIV, provided that such surfaces have not become contaminated with agent(s) or volumes of or concentrations of agent(s) for which a higher level of disinfection is recommended.¹⁰⁰⁷ These registered products are listed in EPA's List D – *Registered Antimicrobials Effective Against Hepatitis B Virus and Human HIV-1*, which may include products tested against duck hepatitis B virus (DHBV) as a surrogate for HBV.^{1008, 1009} Additional lists of interest include EPA's List C – *Registered Antimicrobials Effective Against Human HIV-1* and EPA's List E – *Registered Antimicrobials Effective Against Mycobacterium spp., Hepatitis B Virus, and Human HIV-1*.

Sodium hypochlorite solutions are inexpensive and effective broad-spectrum germicidal solutions.^{1010, 1011} Generic sources of sodium hypochlorite include household chlorine bleach or reagent grade chemical. Concentrations of sodium hypochlorite solutions with a range of 5,000–6,150 ppm (1:10 v/v dilution of household bleaches marketed in the United States) to 500–615 ppm (1:100 v/v dilution) free chlorine are effective depending on the amount of organic material (e.g., blood, mucus, and urine) present on the surface to be cleaned and disinfected.^{1010, 1011} EPA-registered chemical germicides may be more compatible with certain materials that could be corroded by repeated exposure to sodium hypochlorite, especially the 1:10 dilution. Appropriate personal protective equipment (e.g., gloves and goggles) should be worn when preparing and using hypochlorite solutions or other chemical germicides.⁹⁶⁷

Despite laboratory evidence demonstrating adequate potency against bloodborne pathogens (e.g., HIV and HBV), many chlorine bleach products available in grocery and chemical-supply stores are not registered by the EPA for use as surface disinfectants. Use of these chlorine products as surface disinfectants is considered by the EPA to be an “unregistered use.” EPA encourages the use of registered products because the agency reviews them for safety and performance when the product is used according to label instructions. When unregistered products are used for surface disinfection, users do so at their own risk.

Strategies for decontaminating spills of blood and other body fluids differ based on the setting in which they occur and the volume of the spill.¹⁰¹⁰ In patient-care areas, workers can manage small spills with cleaning and then disinfecting using an intermediate-level germicide or an EPA-registered germicide from the EPA List D or E.^{967, 1007} For spills containing large amounts of blood or other body substances, workers should first remove visible organic matter with absorbent material (e.g., disposable paper towels discarded into leak-proof, properly labeled containment) and then clean and decontaminate the area.^{1002, 1003, 1012} If the surface is nonporous and a generic form of a sodium hypochlorite solution is used (e.g., household bleach), a 1:100 dilution is appropriate for decontamination assuming that a) the

worker assigned to clean the spill is wearing gloves and other personal protective equipment appropriate to the task, b) most of the organic matter of the spill has been removed with absorbent material, and c) the surface has been cleaned to remove residual organic matter. A recent study demonstrated that even strong chlorine solutions (i.e., 1:10 dilution of chlorine bleach) may fail to totally inactivate high titers of virus in large quantities of blood, but in the absence of blood these disinfectants can achieve complete viral inactivation.¹⁰¹¹ This evidence supports the need to remove most organic matter from a large spill before final disinfection of the surface. Additionally, EPA-registered proprietary disinfectant label claims are based on use on a pre-cleaned surface.^{951, 954}

Managing spills of blood, body fluids, or other infectious materials in clinical, public health, and research laboratories requires more stringent measures because of a) the higher potential risk of disease transmission associated with large volumes of blood and body fluids and b) high numbers of microorganisms associated with diagnostic cultures. The use of an intermediate-level germicide for routine decontamination in the laboratory is prudent.⁹⁵⁴ Recommended practices for managing large spills of concentrated infectious agents in the laboratory include a) confining the contaminated area, b) flooding the area with a liquid chemical germicide before cleaning, and c) decontaminating with fresh germicidal chemical of at least intermediate-level disinfectant potency.¹⁰¹⁰ A suggested technique when flooding the spill with germicide is to lay absorbent material down on the spill and apply sufficient germicide to thoroughly wet both the spill and the absorbent material.¹⁰¹³ If using a solution of household chlorine bleach, a 1:10 dilution is recommended for this purpose. EPA-registered germicides should be used according to the manufacturers' instructions for use dilution and contact time. Gloves should be worn during the cleaning and decontamination procedures in both clinical and laboratory settings. PPE in such a situation may include the use of respiratory protection (e.g., an N95 respirator) if clean-up procedures are expected to generate infectious aerosols. Protocols for cleaning spills should be developed and made available on record as part of good laboratory practice.¹⁰¹³ Workers in laboratories and in patient-care areas of the facility should receive periodic training in environmental-surface infection-control strategies and procedures as part of an overall infection-control and safety curriculum.

4. Carpeting and Cloth Furnishings

a. Carpeting

Carpeting has been used for more than 30 years in both public and patient-care areas of health-care facilities. Advantages of carpeting in patient-care areas include a) its noise-limiting characteristics; b) the "humanizing" effect on health care; and c) its contribution to reductions in falls and resultant injuries, particularly for the elderly.¹⁰¹⁴⁻¹⁰¹⁶ Compared to hard-surface flooring, however, carpeting is harder to keep clean, especially after spills of blood and body substances. It is also harder to push equipment with wheels (e.g., wheelchairs, carts, and gurneys) on carpeting.

Several studies have documented the presence of diverse microbial populations, primarily bacteria and fungi, in carpeting;^{111, 1017-1024} the variety and number of microorganisms tend to stabilize over time. New carpeting quickly becomes colonized, with bacterial growth plateauing after about 4 weeks.¹⁰¹⁹ Vacuuming and cleaning the carpeting can temporarily reduce the numbers of bacteria, but these populations soon rebound and return to pre-cleaning levels.^{1019, 1020, 1023} Bacterial contamination tends to increase with higher levels of activity.^{1018-1020, 1025} Soiled carpeting that is or remains damp or wet provides an ideal setting for the proliferation and persistence of gram-negative bacteria and fungi.¹⁰²⁶ Carpeting that remains damp should be removed, ideally within 72 hours.

Despite the evidence of bacterial growth and persistence in carpeting, only limited epidemiologic evidence demonstrates that carpets influence health-care-associated infection rates in areas housing

immunocompetent patients.^{1023, 1025, 1027} This guideline, therefore, includes no recommendations against the use of carpeting in these areas. Nonetheless, avoiding the use of carpeting is prudent in areas where spills are likely to occur (e.g., laboratories, areas around sinks, and janitor closets) and where patients may be at greater risk of infection from airborne environmental pathogens (e.g., HSCT units, burn units, ICUs, and ORs).^{111, 1028} An outbreak of aspergillosis in an HSCT unit was recently attributed to carpet contamination and a particular method of carpet cleaning.¹¹¹ A window in the unit had been opened repeatedly during the time of a nearby building fire, which allowed fungal spore intrusion into the unit. After the window was sealed, the carpeting was cleaned using a “bonnet buffing” machine, which dispersed *Aspergillus* spores into the air.¹¹¹ Wet vacuuming was instituted, replacing the dry cleaning method used previously; no additional cases of invasive aspergillosis were identified.

The care setting and the method of carpet cleaning are important factors to consider when attempting to minimize or prevent production of aerosols and dispersal of carpet microorganisms into the air.^{94, 111} Both vacuuming and shampooing or wet cleaning with equipment can disperse microorganisms to the air.^{111, 994} Vacuum cleaners should be maintained to minimize dust dispersal in general, and be equipped with HEPA filters, especially for use in high-risk patient-care areas.^{9, 94, 986} Some formulations of carpet-cleaning chemicals, if applied or used improperly, can be dispersed into the air as a fine dust capable of causing respiratory irritation in patients and staff.¹⁰²⁹ Cleaning equipment, especially those that engage in wet cleaning and extraction, can become contaminated with waterborne organisms (e.g., *Pseudomonas aeruginosa*) and serve as a reservoir for these organisms if this equipment is not properly maintained. Substantial numbers of bacteria can then be transferred to carpeting during the cleaning process.¹⁰³⁰ Therefore, keeping the carpet cleaning equipment in good repair and allowing such equipment to dry between uses is prudent.

Carpet cleaning should be performed on a regular basis determined by internal policy. Although spills of blood and body substances on non-porous surfaces require prompt spot cleaning using standard cleaning procedures and application of chemical germicides,⁹⁶⁷ similar decontamination approaches to blood and body substance spills on carpeting can be problematic from a regulatory perspective.¹⁰³¹ Most, if not all, modern carpet brands suitable for public facilities can tolerate the activity of a variety of liquid chemical germicides. However, according to OSHA, carpeting contaminated with blood or other potentially infectious materials can not be fully decontaminated.¹⁰³² Therefore, facilities electing to use carpeting for high-activity patient-care areas may choose carpet tiles in areas at high risk for spills.^{967, 1032} In the event of contamination with blood or other body substances, carpet tiles can be removed, discarded, and replaced. OSHA also acknowledges that only minimal direct skin contact occurs with carpeting, and therefore, employers are expected to make reasonable efforts to clean and sanitize carpeting using carpet detergent/cleaner products.¹⁰³²

Over the last few years, some carpet manufacturers have treated their products with fungicidal and/or bactericidal chemicals. Although these chemicals may help to reduce the overall numbers of bacteria or fungi present in carpet, their use does not preclude the routine care and maintenance of the carpeting. Limited evidence suggests that chemically treated carpet may have helped to keep health-care–associated aspergillosis rates low in one HSCT unit,¹¹¹ but overall, treated carpeting has not been shown to prevent the incidence of health-care–associated infections in care areas for immunocompetent patients.

b. Cloth Furnishings

Upholstered furniture and furnishings are becoming increasingly common in patient-care areas. These furnishings range from simple cloth chairs in patients’ rooms to a complete decorating scheme that gives the interior of the facility more the look of an elegant hotel.¹⁰³³ Even though pathogenic microorganisms have been isolated from the surfaces of cloth chairs, no epidemiologic evidence suggests that general patient-care areas with cloth furniture pose increased risks of health-care–

associated infection compared with areas that contain hard-surfaced furniture.^{1034, 1035} Allergens (e.g., dog and cat dander) have been detected in or on cloth furniture in clinics and elsewhere in hospitals in concentrations higher than those found on bed linens.^{1034, 1035} These allergens presumably are transferred from the clothing of visitors. Researchers have therefore suggested that cloth chairs should be vacuumed regularly to keep the dust and allergen levels to a minimum. This recommendation, however, has generated concerns that aerosols created from vacuuming could place immunocompromised patients or patients with preexisting lung disease (e.g., asthma) at risk for development of health-care–associated, environmental airborne disease.^{9, 20, 109, 988} Recovering worn, upholstered furniture (especially the seat cushion) with covers that are easily cleaned (e.g., vinyl), or replacing the item is prudent; minimizing the use of upholstered furniture and furnishings in any patient-care areas where immunosuppressed patients are located (e.g., HSCT units) reduces the likelihood of disease.⁹

5. Flowers and Plants in Patient-Care Areas

Fresh flowers, dried flowers, and potted plants are common items in health-care facilities. In 1974, clinicians isolated an *Erwinia* sp. post mortem from a neonate diagnosed with fulminant septicemia, meningitis, and respiratory distress syndrome.¹⁰³⁸ Because *Erwinia* spp. are plant pathogens, plants brought into the delivery room were suspected to be the source of the bacteria, although the case report did not definitively establish a direct link. Several subsequent studies evaluated the numbers and diversity of microorganisms in the vase water of cut flowers. These studies revealed that high concentrations of bacteria, ranging from 10^4 – 10^{10} CFU/mL, were often present, especially if the water was changed infrequently.^{515, 702, 1039} The major group of microorganisms in flower vase water was gram-negative bacteria, with *Pseudomonas aeruginosa* the most frequently isolated organism.^{515, 702, 1039, 1040} *P. aeruginosa* was also the primary organism directly isolated from chrysanthemums and other potted plants.^{1041, 1042} However, flowers in hospitals were not significantly more contaminated with bacteria compared with flowers in restaurants or in the home.⁷⁰² Additionally, no differences in the diversity and degree of antibiotic resistance of bacteria have been observed in samples isolated from hospital flowers versus those obtained from flowers elsewhere.⁷⁰²

Despite the diversity and large numbers of bacteria associated with flower-vase water and potted plants, minimal or no evidence indicates that the presence of plants in immunocompetent patient-care areas poses an increased risk of health-care–associated infection.⁵¹⁵ In one study involving a limited number of surgical patients, no correlation was observed between bacterial isolates from flowers in the area and the incidence and etiology of postoperative infections among the patients.¹⁰⁴⁰ Similar conclusions were reached in a study that examined the bacteria found in potted plants.¹⁰⁴² Nonetheless, some precautions for general patient-care settings should be implemented, including a) limiting flower and plant care to staff with no direct patient contact, b) advising health-care staff to wear gloves when handling plants, c) washing hands after handling plants, d) changing vase water every 2 days and discharging the water into a sink outside the immediate patient environment, and e) cleaning and disinfecting vases after use.⁷⁰²

Some researchers have examined the possibility of adding a chemical germicide to vase water to control bacterial populations. Certain chemicals (e.g., hydrogen peroxide and chlorhexidine) are well tolerated by plants.^{1040, 1043, 1044} Use of these chemicals, however, was not evaluated in studies to assess impact on health-care–associated infection rates. Modern florists now have a variety of products available to add to vase water to extend the life of cut flowers and to minimize bacterial clouding of the water.

Flowers (fresh and dried) and ornamental plants, however, may serve as a reservoir of *Aspergillus* spp., and dispersal of conidiospores into the air from this source can occur.¹⁰⁹ Health-care–associated outbreaks of invasive aspergillosis reinforce the importance of maintaining an environment as free of

Aspergillus spp. spores as possible for patients with severe, prolonged neutropenia. Potted plants, fresh-cut flowers, and dried flower arrangements may provide a reservoir for these fungi as well as other fungal species (e.g., *Fusarium* spp.).^{109, 1045, 1046} Researchers in one study of bacteria and flowers suggested that flowers and vase water should be avoided in areas providing care to medically at-risk patients (e.g., oncology patients and transplant patients), although this study did not attempt to correlate the observations of bacterial populations in the vase water with the incidence of health-care–associated infections.⁵¹⁵ Another study using molecular epidemiology techniques demonstrated identical *Aspergillus terreus* types among environmental and clinical specimens isolated from infected patients with hematological malignancies.¹⁰⁴⁶ Therefore, attempts should be made to exclude flowers and plants from areas where immunosuppressed patients are located (e.g., HSCT units).^{9, 1046}

6. Pest Control

Cockroaches, flies and maggots, ants, mosquitoes, spiders, mites, midges, and mice are among the typical arthropod and vertebrate pest populations found in health-care facilities. Insects can serve as agents for the mechanical transmission of microorganisms, or as active participants in the disease transmission process by serving as a vector.^{1047–1049} Arthropods recovered from health-care facilities have been shown to carry a wide variety of pathogenic microorganisms.^{1050–1056} Studies have suggested that the diversity of microorganisms associated with insects reflects the microbial populations present in the indoor health-care environment; some pathogens encountered in insects from hospitals were either absent from or present to a lesser degree in insects trapped from residential settings.^{1057–1060} Some of the microbial populations associated with insects in hospitals have demonstrated resistance to antibiotics.^{1048, 1059, 1061–1063}

Insect habitats are characterized by warmth, moisture, and availability of food.¹⁰⁶⁴ Insects forage in and feed on substrates, including but not limited to food scraps from kitchens/cafeteria, foods in vending machines, discharges on dressings either in use or discarded, other forms of human detritus, medical wastes, human wastes, and routine solid waste.^{1057–1061} Cockroaches, in particular, have been known to feed on fixed sputum smears in laboratories.^{1065, 1066} Both cockroaches and ants are frequently found in the laundry, central sterile supply departments, and anywhere in the facility where water or moisture is present (e.g., sink traps, drains and janitor closets). Ants will often find their way into sterile packs of items as they forage in a warm, moist environment.¹⁰⁵⁷ Cockroaches and other insects frequent loading docks and other areas with direct access to the outdoors.

Although insects carry a wide variety of pathogenic microorganisms on their surfaces and in their gut, the direct association of insects with disease transmission (apart from vector transmission) is limited, especially in health-care settings; the presence of insects in itself likely does not contribute substantially to health-care–associated disease transmission in developed countries. However, outbreaks of infection attributed to microorganisms carried by insects may occur because of infestation coupled with breaks in standard infection-control practices.¹⁰⁶³ Studies have been conducted to examine the role of houseflies as possible vectors for shigellosis and other forms of diarrheal disease in non-health–care settings.^{1046, 1067} When control measures aimed at reducing the fly population density were implemented, a concomitant reduction in the incidence of diarrheal infections, carriage of *Shigella* organisms, and mortality caused by diarrhea among infants and young children was observed.

Myiasis is defined as a parasitosis in which the larvae of any of a variety of flies use living or necrotic tissue or body substances of the host as a nutritional source.¹⁰⁶⁸ Larvae from health-care–acquired myiasis have been observed in nares, wounds, eyes, ears, sinuses, and the external urogenital structures.^{1069–1071} Patients with this rare condition are typically older adults with underlying medical conditions (e.g., diabetes, chronic wounds, and alcoholism) who have a decreased capacity to ward off

the flies. Persons with underlying conditions who live or travel to tropical regions of the world are especially at risk.^{1070, 1071} Cases occur in the summer and early fall months in temperate climates when flies are most active.¹⁰⁷¹ An environmental assessment and review of the patient's history are necessary to verify that the source of the myiasis is health-care-acquired and to identify corrective measures.^{1069, 1072} Simple prevention measures (e.g., installing screens on windows) are important in reducing the incidence of myiasis.¹⁰⁷²

From a public health and hygiene perspective, arthropod and vertebrate pests should be eradicated from all indoor environments, including health-care facilities.^{1073, 1074} Modern approaches to institutional pest management usually focus on a) eliminating food sources, indoor habitats, and other conditions that attract pests; b) excluding pests from the indoor environments; and c) applying pesticides as needed.¹⁰⁷⁵ Sealing windows in modern health-care facilities helps to minimize insect intrusion. When windows need to be opened for ventilation, ensuring that screens are in good repair and closing doors to the outside can help with pest control. Insects should be kept out of all areas of the health-care facility, especially ORs and any area where immunosuppressed patients are located. A pest-control specialist with appropriate credentials can provide a regular insect-control program that is tailored to the needs of the facility and uses approved chemicals and/or physical methods. Industrial hygienists can provide information on possible adverse reactions of patients and staff to pesticides and suggest alternative methods for pest control, as needed.

7. Special Pathogen Concerns

a. Antibiotic-Resistant Gram-Positive Cocci

Vancomycin-resistant enterococci (VRE), methicillin-resistant *Staphylococcus aureus* (MRSA), and *S. aureus* with intermediate levels of resistance to glycopeptide antibiotics (vancomycin intermediate resistant *S. aureus* [VISA] or glycopeptide intermediate resistant *S. aureus* [GISA]) represent crucial and growing concerns for infection control. Although the term GISA is technically a more accurate description of the strains isolated to date (most of which are classified as having intermediate resistance to both vancomycin and teicoplanin), the term “glycopeptide” may not be recognized by many clinicians. Thus, the label of VISA, which emphasizes a change in minimum inhibitory concentration (MICs) to vancomycin, is similar to that of VRE and is more meaningful to clinicians.¹⁰⁷⁶ According to National Nosocomial Infection Surveillance (NNIS) statistics for infections acquired among ICU patients in the United States in 1999, 52.3% of infections resulting from *S. aureus* were identified as MRSA infections, and 25.2% of enterococcal infections were attributed to VRE. These figures reflect a 37% and a 43% increase, respectively, since 1994–1998.¹⁰⁷⁷

People represent the primary reservoir of *S. aureus*.¹⁰⁷⁸ Although *S. aureus* has been isolated from a variety of environmental surfaces (e.g., stethoscopes, floors, charts, furniture, dry mops, and hydrotherapy tanks), the role of environmental contamination in transmission of this organism in health care appears to be minimal.^{1079–1082} *S. aureus* contamination of surfaces and tanks within burn therapy units, however, may be a major factor in the transmission of infection among burn patients.¹⁰⁸³

Colonized patients are the principal reservoir of VRE, and patients who are immunosuppressed (e.g., transplant patients) or otherwise medically at-risk (e.g., ICU patients, cardio-thoracic surgical patients, patients previously hospitalized for extended periods, and those having received multi-antimicrobial or vancomycin therapy) are at greatest risk for VRE colonization.^{1084–1087} The mechanisms by which cross-colonization take place are not well defined, although recent studies have indicated that both MRSA and VRE may be transmitted either a) directly from patient to patient, b) indirectly by transient carriage on the hands of health-care workers,^{1088–1091} or c) by hand transfer of these gram-positive organisms from contaminated environmental surfaces and patient-care equipment.^{1084, 1087, 1092–1097} In

one survey, hand carriage of VRE in workers in a long-term care facility ranged from 13%–41%.¹⁰⁹⁸ Many of the environmental surfaces found to be contaminated with VRE in outbreak investigations have been those that are touched frequently by the patient or the health-care worker.¹⁰⁹⁹ Such high-touch surfaces include bedrails, doorknobs, bed linens, gowns, overbed tables, blood pressure cuffs, computer table, bedside tables, and various medical equipment.^{22, 1087, 1094, 1095, 1100–1102} Contamination of environmental surfaces with VRE generally occurs in clinical laboratories and areas where colonized patients are present,^{1087, 1092, 1094, 1095, 1103} but the potential for contamination increases when such patients have diarrhea¹⁰⁸⁷ or have multiple body-site colonization.¹¹⁰⁴ Additional factors that can be important in the dispersion of these pathogens to environmental surfaces are misuse of glove techniques by health-care workers (especially when cleaning fecal contamination from surfaces) and patient, family, and visitor hygiene.

Interest in the importance of environmental reservoirs of VRE increased when laboratory studies demonstrated that enterococci can persist in a viable state on dry environmental surfaces for extended periods of time (7 days to 4 months)^{1099, 1105} and multiple strains can be identified during extensive periods of surveillance.¹¹⁰⁴ VRE can be recovered from inoculated hands of health-care workers (with or without gloves) for up to 60 minutes.²² The presence of either MRSA, VISA, or VRE on environmental surfaces, however, does not mean that patients in the contaminated areas will become colonized. Strict adherence to hand hygiene/handwashing and the proper use of barrier precautions help to minimize the potential for spread of these pathogens. Published recommendations for preventing the spread of vancomycin resistance address isolation measures, including patient cohorting and management of patient-care items.⁵ Direct patient-care items (e.g., blood pressure cuffs) should be disposable whenever possible when used in contact isolation settings for patients with multiply resistant microorganisms.¹¹⁰²

Careful cleaning of patient rooms and medical equipment contributes substantially to the overall control of MRSA, VISA, or VRE transmission. The major focus of a control program for either VRE or MRSA should be the prevention of hand transfer of these organisms. Routine cleaning and disinfection of the housekeeping surfaces (e.g., floors and walls) and patient-care surfaces (e.g., bedrails) should be adequate for inactivation of these organisms. Both MRSA and VRE are susceptible to several EPA-registered low- and intermediate-level disinfectants (e.g., alcohols, sodium hypochlorite, quaternary ammonium compounds, phenolics, and iodophors) at recommended use dilutions for environmental surface disinfection.^{1103, 1106–1109} Additionally, both VRE and vancomycin-sensitive enterococci are equally sensitive to inactivation by chemical germicides,^{1106, 1107, 1109} and similar observations have been made when comparing the germicidal resistance of MRSA to that of either methicillin-sensitive *S. aureus* (MSSA) or VISA.¹¹¹⁰ The use of stronger solutions of disinfectants for inactivation of either VRE, MRSA, or VISA is not recommended based on the organisms' resistance to antibiotics.^{1110–1112} VRE from clinical specimens have exhibited some measure of increased tolerance to heat inactivation in temperature ranges <212°F (<100°C),^{1106, 1113} however, the clinical significance of these observations is unclear because the role of cleaning the surface or item prior to heat treatment was not evaluated. Although routine environmental sampling is not recommended, laboratory surveillance of environmental surfaces during episodes when VRE contamination is suspected can help determine the effectiveness of the cleaning and disinfecting procedures. Environmental culturing should be approved and supervised by the infection-control program in collaboration with the clinical laboratory.^{1084, 1087, 1088, 1092, 1096}

Two cases of wound infections associated with vancomycin-resistant *Staphylococcus aureus* (VRSA) determined to be resistant by NCCLS standards for sensitivity/resistance testing were identified in Michigan and Pennsylvania in 2002.^{1114, 1115} These represented isolated cases, and neither the family members nor the health-care providers of these case-patients had evidence of colonization or infection with VRSA. Conventional environmental infection-control measures (i.e., cleaning and then

disinfecting surfaces using EPA-registered disinfectants with label claims for *S. aureus*) were used during the environmental investigation of these two cases;^{1110–1112} however, studies have yet to evaluate the potential intrinsic resistance of these VRSA strains to surface disinfectants.

Standard procedures during terminal cleaning and disinfection of surfaces, if performed incorrectly, may be inadequate for the elimination of VRE from patient rooms.^{1113, 1116–1118} Given the sensitivity of VRE to hospital disinfectants, current disinfecting protocols should be effective if they are diligently carried out and properly performed. Health-care facilities should be sure that housekeeping staff use correct procedures for cleaning and disinfecting surfaces in VRE-contaminated areas, which include using sufficient amounts of germicide at proper use dilution and allowing adequate contact time.¹¹¹⁸

b. Clostridium difficile

Clostridium difficile is the most frequent etiologic agent for health-care–associated diarrhea.^{1119, 1120} In one hospital, 30% of adults who developed health-care–associated diarrhea were positive for *C. difficile*.¹¹²¹ One recent study employing PCR-ribotyping techniques demonstrated that cases of *C. difficile*-acquired diarrhea occurring in the hospital included patients whose infections were attributed to endogenous *C. difficile* strains and patients whose illnesses were considered to be health-care–associated infections.¹¹²² Most patients remain asymptomatic after infection, but the organism continues to be shed in their stools. Risk factors for acquiring *C. difficile*-associated infection include a) exposure to antibiotic therapy, particularly with beta-lactam agents;¹¹²³ b) gastrointestinal procedures and surgery;¹¹²⁴ c) advanced age; and d) indiscriminate use of antibiotics.^{1125–1128} Of all the measures that have been used to prevent the spread of *C. difficile*-associated diarrhea, the most successful has been the restriction of the use of antimicrobial agents.^{1129, 1130}

C. difficile is an anaerobic, gram-positive bacterium. Normally fastidious in its vegetative state, it is capable of sporulating when environmental conditions no longer support its continued growth. The capacity to form spores enables the organism to persist in the environment (e.g., in soil and on dry surfaces) for extended periods of time. Environmental contamination by this microorganism is well known, especially in places where fecal contamination may occur.¹¹³¹ The environment (especially housekeeping surfaces) rarely serves as a direct source of infection for patients.^{1024, 1132–1136} However, direct exposure to contaminated patient-care items (e.g., rectal thermometers) and high-touch surfaces in patients' bathrooms (e.g., light switches) have been implicated as sources of infection.^{1130, 1135, 1136, 1138}

Transfer of the pathogen to the patient via the hands of health-care workers is thought to be the most likely mechanism of exposure.^{24, 1133, 1139} Standard isolation techniques intended to minimize enteric contamination of patients, health-care–workers' hands, patient-care items, and environmental surfaces have been published.¹¹⁴⁰ Handwashing remains the most effective means of reducing hand contamination. Proper use of gloves is an ancillary measure that helps to further minimize transfer of these pathogens from one surface to another.

The degree to which the environment becomes contaminated with *C. difficile* spores is proportional to the number of patients with *C. difficile*-associated diarrhea,^{24, 1132, 1135} although asymptomatic, colonized patients may also serve as a source of contamination. Few studies have examined the use of specific chemical germicides for the inactivation of *C. difficile* spores, and no well-controlled trials have been conducted to determine efficacy of surface disinfection and its impact on health-care–associated diarrhea. Some investigators have evaluated the use of chlorine-containing chemicals (e.g., 1,000 ppm hypochlorite at recommended use-dilution, 5,000 ppm sodium hypochlorite [1:10 v/v dilution], 1:100 v/v dilutions of unbuffered hypochlorite, and phosphate-buffered hypochlorite [1,600 ppm]). One of the studies demonstrated that the number of contaminated environmental sites was reduced by half,¹¹³⁵ whereas another two studies demonstrated declines in health-care–associated *C. difficile* infections in a HSCT unit¹¹⁴¹ and in two geriatric medical units¹¹⁴² during a period of hypochlorite use. The presence

of confounding factors, however, was acknowledged in one of these studies.¹¹⁴² The recommended approach to environmental infection control with respect to *C. difficile* is meticulous cleaning followed by disinfection using hypochlorite-based germicides as appropriate.^{952, 1130, 1143} However, because no EPA-registered surface disinfectants with label claims for inactivation of *C. difficile* spores are available, the recommendation is based on the best available evidence from the scientific literature.

c. Respiratory and Enteric Viruses in Pediatric-Care Settings

Although the viruses mentioned in this guideline are not unique to the pediatric-care setting in health-care facilities, their prevalence in these areas, especially during the winter months, is substantial. Children (particularly neonates) are more likely to develop infection and substantial clinical disease from these agents compared with adults and therefore are more likely to require supportive care during their illness.

Common respiratory viruses in pediatric-care areas include rhinoviruses, respiratory syncytial virus (RSV), adenoviruses, influenza viruses, and parainfluenza viruses. Transmission of these viruses occurs primarily via direct contact with small-particle aerosols or via hand contamination with respiratory secretions that are then transferred to the nose or eyes. Because transmission primarily requires close personal contact, contact precautions are appropriate to interrupt transmission.⁶ Hand contamination can occur from direct contact with secretions or indirectly from touching high-touch environmental surfaces that have become contaminated with virus from large droplets. The indirect transfer of virus from one person to other via hand contact with frequently-touched fomites was demonstrated in a study using a bacteriophage whose environmental stability approximated that of human viral pathogens (e.g., poliovirus and parvovirus).¹¹⁴⁴ The impact of this mode of transmission with respect to human respiratory- and enteric viruses is dependent on the ability of these agents to survive on environmental surfaces. Infectious RSV has been recovered from skin, porous surfaces, and non-porous surfaces after 30 minutes, 1 hour, and 7 hours, respectively.¹¹⁴⁵ Parainfluenza viruses are known to persist for up to 4 hours on porous surfaces and up to 10 hours on non-porous surfaces.¹¹⁴⁶ Rhinoviruses can persist on porous surfaces and non-porous surfaces for approximately 1 and 3 hours respectively; study participants in a controlled environment became infected with rhinoviruses after first touching a surface with dried secretions and then touching their nasal or conjunctival mucosa.¹¹⁴⁷ Although the efficiency of direct transmission of these viruses from surfaces in uncontrolled settings remains to be defined, these data underscore the basis for maintaining regular protocols for cleaning and disinfecting of high-touch surfaces.

The clinically important enteric viruses encountered in pediatric care settings include enteric adenovirus, astroviruses, caliciviruses, and rotavirus. Group A rotavirus is the most common cause of infectious diarrhea in infants and children. Transmission of this virus is primarily fecal-oral, however, the role of fecally contaminated surfaces and fomites in rotavirus transmission is unclear. During one epidemiologic investigation of enteric disease among children attending day care, rotavirus contamination was detected on 19% of inanimate objects in the center.^{1148, 1149} In an outbreak in a pediatric unit, secondary cases of rotavirus infection clustered in areas where children with rotaviral diarrhea were located.¹¹⁵⁰ Astroviruses cause gastroenteritis and diarrhea in newborns and young children and can persist on fecally contaminated surfaces for several months during periods of relatively low humidity.^{1151, 1152} Outbreaks of small round-structured viruses (i.e., caliciviruses [Norwalk virus and Norwalk-like viruses]) can affect both patients and staff, with attack rates of $\geq 50\%$.¹¹⁵³ Routes of person-to-person transmission include fecal-oral spread and aerosols generated from vomiting.^{1154–1156} Fecal contamination of surfaces in care settings can spread large amounts of virus to the environment. Studies that have attempted to use low- and intermediate-level disinfectants to inactivate rotavirus suspended in feces have demonstrated a protective effect of high concentrations of organic matter.^{1157, 1158} Intermediate-level disinfectants (e.g., alcoholic quaternary ammonium compounds, and chlorine solutions) can be effective in inactivating enteric viruses provided that a cleaning step to remove most of

the organic matter precedes terminal disinfection.¹¹⁵⁸ These findings underscore the need for proper cleaning and disinfecting procedures where contamination of environmental surfaces with body substances is likely. EPA-registered surface disinfectants with label claims for these viral agents should be used in these settings. Using disposable, protective barrier coverings may help to minimize the degree of surface contamination.⁹³⁶

d. Severe Acute Respiratory Syndrome (SARS) Virus

In November 2002 an atypical pneumonia of unknown etiology emerged in Asia and subsequently developed into an international outbreak of respiratory illness among persons in 29 countries during the first six months of 2003. “Severe acute respiratory syndrome” (SARS) is a viral upper respiratory infection associated with a newly described coronavirus (SARS-associated Co-V [SARS-CoV]). SARS-CoV is an enveloped RNA virus. It is present in high titers in respiratory secretions, stool, and blood of infected persons. The modes of transmission determined from epidemiologic investigations were primarily forms of direct contact (i.e., large droplet aerosolization and person-to-person contact). Respiratory secretions were presumed to be the major source of virus in these situations; airborne transmission of virus has not been completely ruled out. Little is known about the impact of fecal-oral transmission and SARS.

The epidemiology of SARS-CoV infection is not completely understood, and therefore recommended infection control and prevention measures to contain the spread of SARS will evolve as new information becomes available.¹¹⁵⁹ At present there is no indication that established strategies for cleaning (i.e., to remove the majority of bioburden) and disinfecting equipment and environmental surfaces need to be changed for the environmental infection control of SARS. In-patient rooms housing SARS patients should be cleaned and disinfected at least daily and at the time of patient transfer or discharge. More frequent cleaning and disinfection may be indicated for high-touch surfaces and following aerosol-producing procedures (e.g., intubation, bronchoscopy, and sputum production). While there are presently no disinfectant products registered by EPA specifically for inactivation of SARS-CoV, EPA-registered hospital disinfectants that are equivalent to low- and intermediate-level germicides may be used on pre-cleaned, hard, non-porous surfaces in accordance with manufacturer’s instructions for environmental surface disinfection. Monitoring adherence to guidelines established for cleaning and disinfection is an important component of environmental infection control to contain the spread of SARS.

e. Creutzfeldt-Jakob Disease (CJD) in Patient-Care Areas

Creutzfeldt-Jakob disease (CJD) is a rare, invariably fatal, transmissible spongiform encephalopathy (TSE) that occurs worldwide with an average annual incidence of 1 case per million population.¹¹⁶⁰⁻¹¹⁶² CJD is one of several TSEs affecting humans; other diseases in this group include kuru, fatal familial insomnia, and Gerstmann-Sträussler-Scheinker syndrome. A TSE that affects a younger population (compared to the age range of CJD cases) has been described primarily in the United Kingdom since 1996.¹¹⁶³ This variant form of CJD (vCJD) is clinically and neuropathologically distinguishable from classic CJD; epidemiologic and laboratory evidence suggests a causal association for bovine spongiform encephalopathy (BSE [Mad Cow disease]) and vCJD.¹¹⁶³⁻¹¹⁶⁶

The agent associated with CJD is a prion, which is an abnormal isoform of a normal protein constituent of the central nervous system.¹¹⁶⁷⁻¹¹⁶⁹ The mechanism by which the normal form of the protein is converted to the abnormal, disease-causing prion is unknown. The tertiary conformation of the abnormal prion protein appears to confer a heightened degree of resistance to conventional methods of sterilization and disinfection.^{1170, 1171}

Although about 90% of CJD cases occur sporadically, a limited number of cases are the result of a direct exposure to prion-containing material (usually central nervous system tissue or pituitary

hormones) acquired as a result of health care (iatrogenic cases). These cases have been linked to a) pituitary hormone therapy [from human sources as opposed to hormones prepared through the use of recombinant technology],^{1170–1174} b) transplants of either dura mater or corneas,^{1175–1181} and c) neurosurgical instruments and depth electrodes.^{1182–1185} In the cases involving instruments and depth electrodes, conventional cleaning and terminal reprocessing methods of the day failed to fully inactivate the contaminating prions and are considered inadequate by today's standards.

Prion inactivation studies involving whole tissues and tissue homogenates have been conducted to determine the parameters of physical and chemical methods of sterilization or disinfection necessary for complete inactivation,^{1170, 1186–1191} however, the application of these findings to environmental infection control in health-care settings is problematic. No studies have evaluated the effectiveness of medical instrument reprocessing in inactivating prions. Despite a consensus that abnormal prions display some extreme measure of resistance to inactivation by either physical or chemical methods, scientists disagree about the exact conditions needed for sterilization. Inactivation studies utilizing whole tissues present extraordinary challenges to any sterilizing method.¹¹⁹² Additionally, the experimental designs of these studies preclude the evaluation of surface cleaning as a part of the total approach to pathogen inactivation.^{951, 1192}

Some researchers have recommended the use of either a 1:2 v/v dilution of sodium hypochlorite (approximately 20,000 ppm), full-strength sodium hypochlorite (50,000–60,000 ppm), or 1–2 N sodium hydroxide (NaOH) for the inactivation of prions on certain surfaces (e.g., those found in the pathology laboratory).^{1170, 1188} Although these chemicals may be appropriate for the decontamination of laboratory, operating-room, or autopsy-room surfaces that come into contact with central nervous system tissue from a known or suspected patient, this approach is not indicated for routine or terminal cleaning of a room previously occupied by a CJD patient. Both chemicals pose hazards for the health-care worker doing the decontamination. NaOH is caustic and should not make contact with the skin. Sodium hypochlorite solutions (i.e., chlorine bleach) can corrode metals (e.g., aluminum). MSDS information should be consulted when attempting to work with concentrated solutions of either chemical. Currently, no EPA-registered products have label claims for prion inactivation; therefore, this guidance is based on the best available evidence from the scientific literature.

Environmental infection-control strategies must be based on the principles of the “chain of infection,” regardless of the disease of concern.¹³ Although CJD is transmissible, it is not highly contagious. All iatrogenic cases of CJD have been linked to a direct exposure to prion-contaminated central nervous system tissue or pituitary hormones. The six documented iatrogenic cases associated with instruments and devices involved neurosurgical instruments and devices that introduced residual contamination directly to the recipient's brain. No evidence suggests that vCJD has been transmitted iatrogenically or that either CJD or vCJD has been transmitted from environmental surfaces (e.g., housekeeping surfaces). Therefore, routine procedures are adequate for terminal cleaning and disinfection of a CJD patient's room. Additionally, in epidemiologic studies involving highly transfused patients, blood was not identified as a source for prion transmission.^{1193–1198} Routine procedures for containing, decontaminating, and disinfecting surfaces with blood spills should be adequate for proper infection control in these situations.^{951, 1199}

Guidance for environmental infection control in ORs and autopsy areas has been published.^{1197, 1199} Hospitals should develop risk-assessment procedures to identify patients with known or suspected CJD in efforts to implement prion-specific infection-control measures for the OR and for instrument reprocessing.¹²⁰⁰ This assessment also should be conducted for older patients undergoing non-lesionous neurosurgery when such procedures are being done for diagnosis. Disposable, impermeable coverings should be used during these autopsies and neurosurgeries to minimize surface contamination. Surfaces that have become contaminated with central nervous system tissue or cerebral spinal fluid should be

cleaned and decontaminated by a) removing most of the tissue or body substance with absorbent materials, b) wetting the surface with a sodium hypochlorite solution containing $\geq 5,000$ ppm or a 1 N NaOH solution, and c) rinsing thoroughly.^{951, 1197–1199, 1201} The optimum duration of contact exposure in these instances is unclear. Some researchers recommend a 1-hour contact time on the basis of tissue-inactivation studies,^{1197, 1198, 1201} whereas other reviewers of the subject draw no conclusions from this research.¹¹⁹⁹ Factors to consider before cleaning a potentially contaminated surface are a) the degree to which gross tissue/body substance contamination can be effectively removed and b) the ease with which the surface can be cleaned.

F. Environmental Sampling

This portion of Part I addresses the basic principles and methods of sampling environmental surfaces and other environmental sources for microorganisms. The applied strategies of sampling with respect to environmental infection control have been discussed in the appropriate preceding subsections.

1. General Principles: Microbiologic Sampling of the Environment

Before 1970, U.S. hospitals conducted regularly scheduled culturing of the air and environmental surfaces (e.g., floors, walls, and table tops).¹²⁰² By 1970, CDC and the American Hospital Association (AHA) were advocating the discontinuation of routine environmental culturing because rates of health-care–associated infection had not been associated with levels of general microbial contamination of air or environmental surfaces, and because meaningful standards for permissible levels of microbial contamination of environmental surfaces or air did not exist.^{1203–1205} During 1970–1975, 25% of U.S. hospitals reduced the extent of such routine environmental culturing — a trend that has continued.^{1206, 1207}

Random, undirected sampling (referred to as “routine” in previous guidelines) differs from the current practice of targeted sampling for defined purposes.^{2, 1204} Previous recommendations against routine sampling were not intended to discourage the use of sampling in which sample collection, culture, and interpretation are conducted in accordance with defined protocols.² In this guideline, targeted microbiologic sampling connotes a monitoring process that includes a) a written, defined, multidisciplinary protocol for sample collection and culturing; b) analysis and interpretation of results using scientifically determined or anticipatory baseline values for comparison; and c) expected actions based on the results obtained. Infection control, in conjunction with laboratorians, should assess the health-care facility’s capability to conduct sampling and determine when expert consultation and/or services are needed.

Microbiologic sampling of air, water, and inanimate surfaces (i.e., environmental sampling) is an expensive and time-consuming process that is complicated by many variables in protocol, analysis, and interpretation. It is therefore indicated for only four situations.¹²⁰⁸ The first is to support an investigation of an outbreak of disease or infections when environmental reservoirs or fomites are implicated epidemiologically in disease transmission.^{161, 1209, 1210} It is important that such culturing be supported by epidemiologic data. Environmental sampling, as with all laboratory testing, should not be conducted if there is no plan for interpreting and acting on the results obtained.^{11, 1211, 1212} Linking microorganisms from environmental samples with clinical isolates by molecular epidemiology is crucial whenever it is possible to do so.

The second situation for which environmental sampling may be warranted is in research. Well-designed and controlled experimental methods and approaches can provide new information about the spread of health-care–associated diseases.^{126, 129} A classic example is the study of environmental microbial

contamination that compared health-care–associated infection rates in an old hospital and a new facility before and shortly after occupancy.⁹⁴⁷

The third indication for sampling is to monitor a potentially hazardous environmental condition, confirm the presence of a hazardous chemical or biological agent, and validate the successful abatement of the hazard. This type of sampling can be used to: a) detect bioaerosols released from the operation of health-care equipment (e.g., an ultrasonic cleaner) and determine the success of repairs in containing the hazard,¹²¹³ b) detect the release of an agent of bioterrorism in an indoor environmental setting and determine its successful removal or inactivation, and c) sample for industrial hygiene or safety purposes (e.g., monitoring a “sick building”).

The fourth indication is for quality assurance to evaluate the effects of a change in infection-control practice or to ensure that equipment or systems perform according to specifications and expected outcomes. Any sampling for quality-assurance purposes must follow sound sampling protocols and address confounding factors through the use of properly selected controls. Results from a single environmental sample are difficult to interpret in the absence of a frame of reference or perspective. Evaluations of a change in infection-control practice are based on the assumption that the effect will be measured over a finite period, usually of short duration. Conducting quality-assurance sampling on an extended basis, especially in the absence of an adverse outcome, is usually unjustified. A possible exception might be the use of air sampling during major construction periods to qualitatively detect breaks in environmental infection-control measures. In one study, which began as part of an investigation of an outbreak of health-care–associated aspergillosis, airborne concentrations of *Aspergillus* spores were measured in efforts to evaluate the effectiveness of sealing hospital doors and windows during a period of construction of a nearby building.⁵⁰ Other examples of sampling for quality-assurance purposes may include commissioning newly constructed space in special care areas (i.e., ORs and units for immunosuppressed patients) or assessing a change in housekeeping practice. However, the only types of routine environmental microbiologic sampling recommended as part of a quality-assurance program are a) the biological monitoring of sterilization processes by using bacterial spores¹²¹⁴ and b) the monthly culturing of water used in hemodialysis applications and for the final dialysate use dilution. Some experts also advocate periodic environmental sampling to evaluate the microbial/particulate quality for regular maintenance of the air handling system (e.g., filters) and to verify that the components of the system meet manufacturer’s specifications (A. Streifel, University of Minnesota, 2000). Certain equipment in health-care settings (e.g., biological safety cabinets) may also be monitored with air flow and particulate sampling to determine performance or as part of adherence to a certification program; results can then be compared with a predetermined standard of performance. These measurements, however, usually do not require microbiologic testing.

2. Air Sampling

Biological contaminants occur in the air as aerosols and may include bacteria, fungi, viruses, and pollens.^{1215, 1216} Aerosols are characterized as solid or liquid particles suspended in air. Talking for 5 minutes and coughing each can produce 3,000 droplet nuclei; sneezing can generate approximately 40,000 droplets which then evaporate to particles in the size range of 0.5–12 μm .^{137, 1217} Particles in a biological aerosol usually vary in size from $<1 \mu\text{m}$ to $\geq 50 \mu\text{m}$. These particles may consist of a single, unattached organism or may occur in the form of clumps composed of a number of bacteria. Clumps can also include dust and dried organic or inorganic material. Vegetative forms of bacterial cells and viruses may be present in the air in a lesser number than bacterial spores or fungal spores. Factors that determine the survival of microorganisms within a bioaerosol include a) the suspending medium, b) temperature, c) relative humidity, d) oxygen sensitivity, and e) exposure to UV or electromagnetic radiation.¹²¹⁵ Many vegetative cells will not survive for lengthy periods of time in the air unless the

relative humidity and other factors are favorable for survival and the organism is enclosed within some protective cover (e.g., dried organic or inorganic matter).¹²¹⁶ Pathogens that resist drying (e.g., *Staphylococcus* spp., *Streptococcus* spp., and fungal spores) can survive for long periods and can be carried considerable distances via air and still remain viable. They may also settle on surfaces and become airborne again as secondary aerosols during certain activities (e.g., sweeping and bed making).^{1216, 1218}

Microbiologic air sampling is used as needed to determine the numbers and types of microorganisms, or particulates, in indoor air.²⁸⁹ Air sampling for quality control is, however, problematic because of lack of uniform air-quality standards. Although airborne spores of *Aspergillus* spp. can pose a risk for neutropenic patients, the critical number (i.e., action level) of these spores above which outbreaks of aspergillosis would be expected to occur has not been defined. Health-care professionals considering the use of air sampling should keep in mind that the results represent indoor air quality at singular points in time, and these may be affected by a variety of factors, including a) indoor traffic, b) visitors entering the facility, c) temperature, d) time of day or year, e) relative humidity, f) relative concentration of particles or organisms, and g) the performance of the air-handling system components. To be meaningful, air-sampling results must be compared with those obtained from other defined areas, conditions, or time periods.

Several preliminary concerns must be addressed when designing a microbiologic air sampling strategy (Box 13). Because the amount of particulate material and bacteria retained in the respiratory system is largely dependent on the size of the inhaled particles, particle size should be determined when studying airborne microorganisms and their relation to respiratory infections. Particles $>5\ \mu\text{m}$ are efficiently trapped in the upper respiratory tract and are removed primarily by ciliary action.¹²¹⁹ Particles $\leq 5\ \mu\text{m}$ in diameter reach the lung, but the greatest retention in the alveoli is of particles 1–2 μm in diameter.^{1220–1222}

Box 13. Preliminary concerns for conducting air sampling

-
- Consider the possible characteristics and conditions of the aerosol, including size range of particles, relative amount of inert material, concentration of microorganisms, and environmental factors.
 - Determine the type of sampling instruments, sampling time, and duration of the sampling program.
 - Determine the number of samples to be taken.
 - Ensure that adequate equipment and supplies are available.
 - Determine the method of assay that will ensure optimal recovery of microorganisms.
 - Select a laboratory that will provide proper microbiologic support.
 - Ensure that samples can be refrigerated if they cannot be assayed in the laboratory promptly.
-

Bacteria, fungi, and particulates in air can be identified and quantified with the same methods and equipment (Table 23). The basic methods include a) impingement in liquids, b) impaction on solid surfaces, c) sedimentation, d) filtration, e) centrifugation, f) electrostatic precipitation, and g) thermal precipitation.¹²¹⁸ Of these, impingement in liquids, impaction on solid surfaces, and sedimentation (on settle plates) have been used for various air-sampling purposes in health-care settings.²⁸⁹

Several instruments are available for sampling airborne bacteria and fungi (Box 14). Some of the samplers are self-contained units requiring only a power supply and the appropriate collecting medium, but most require additional auxiliary equipment (e.g., a vacuum pump and an airflow measuring device [i.e., a flowmeter or anemometer]). Sedimentation or depositional methods use settle plates and

therefore need no special instruments or equipment. Selection of an instrument for air sampling requires a clear understanding of the type of information desired and the particular determinations that must be made (Box 14). Information may be needed regarding a) one particular organism or all organisms that may be present in the air, b) the concentration of viable particles or of viable organisms, c) the change in concentration with time, and d) the size distribution of the collected particles. Before sampling begins, decisions should be made regarding whether the results are to be qualitative or quantitative. Comparing quantities of airborne microorganisms to those of outdoor air is also standard operating procedure. Infection-control professionals, hospital epidemiologists, industrial hygienists, and laboratory supervisors, as part of a multidisciplinary team, should discuss the potential need for microbial air sampling to determine if the capacity and expertise to conduct such sampling exists within the facility and when it is appropriate to enlist the services of an environmental microbiologist consultant.

Table 23. Air sampling methods and examples of equipment*

Method	Principle	Suitable for measuring:	Collection media or surface	Rate of collection (L/min.)	Auxilliary equipment needed+	Points to consider	Prototype samplers§
Impingement in liquids	Air drawn through a small jet and directed against a liquid surface	Viable organisms, and concentration over time. Example use: sampling water aerosols to <i>Legionella</i> spp.	Buffered gelatin, tryptose saline, peptone, nutrient broth	12.5	Yes	Antifoaming agent may be needed. Ambient temperature and humidity will influence length of collection time	Chemical Corps. All Glass Impinger (AGI)
Impaction on solid surfaces	Air drawn into the sampler; particles deposited on a dry surface	Viable particles; viable organisms (on non-nutrient surfaces, limited to organisms that resist drying and spores); size measurement, and concentration over time. Example use: sampling air for <i>Aspergillus</i> spp., fungal spores	Dry surface, coated surfaces, and agar	28 (sieve) 30–800 (slit)	Yes	Available as sieve impactors or slit impactors. Sieve impactors can be set up to measure particle size. Slit impactors have a rotating support stage for agar plates to allow for measurement of concentration over time.	Andersen Air Sampler (sieve impactor); TDL, Cassella MK-2 (slit impactors)
Sedimentation	Particles and micro-organisms settle onto surfaces via gravity	Viable particles. Example uses: sampling air for bacteria in the vicinity of and during a medical procedure; general measurements of microbial air quality.	Nutrient media (agars) on plates or slides	—	No	Simple and inexpensive; best suited for qualitative sampling; significant airborne fungal spores are too buoyant to settle efficiently for collection using this method.	Settle plates

Method	Principle	Suitable for measuring:	Collection media or surface	Rate of collection (L/min.)	Auxilliary equipment needed+	Points to consider	Prototype samplers§
Filtration	Air drawn through a filter unit; particles trapped; 0.2 µm pore size	Viable particles; viable organisms (on non-nutrient surfaces, limited to spores and organisms that resist drying); concentration over time. Example use: air sampling for <i>Aspergillus</i> spp., fungal spores, and dust	Paper, cellulose, glass wool, gelatin foam, and membrane filters	1–50	Yes	Filter must be agitated first in rinse fluid to remove and disperse trapped micro-organisms; rinse fluid is assayed; used more for sampling dust and chemicals.	—
Centrifugation	Aerosols subjected to centrifugal force; particles impacted onto a solid surface	Viable particles; viable organisms (on non-nutrient surfaces, limited to spores and organisms that resist drying); concentration over time. Example use: air sampling for <i>Aspergillus</i> spp., and fungal spores	Coated glass or plastic slides, and agar surfaces	40–50	Yes	Calibration is difficult and is done only by the factory; relative comparison of airborne contamination is its general use.	Biotest RCS Plus
Electrostatic precipitation	Air drawn over an electrostatically charged surface; particles become charged	Viable particles; viable organisms (on non-nutrient surfaces, limited to spores and organisms that resist drying); concentration over time	Solid collecting surfaces (glass, and agar)	85	Yes	High volume sampling rate, but equipment is complex and must be handled carefully; not practical for use in health-care settings.	—
Thermal precipitation	Air drawn over a thermal gradient; particles repelled from hot surfaces, settle on colder surfaces	Size measurements	Glass coverslip, and electron microscope grid	0.003–0.4	Yes	Determine particle size by direct observation; not frequently used because of complex adjustments and low sampling rates.	—

* Material in this table is compiled from references 289, 1218, 1223, and 1224.

+ Most samplers require a flow meter or anemometer and a vacuum source as auxiliary equipment.

§ Trade names listed are for identification purposes only and are not intended as endorsements by the U.S. Public Health Service.

Box 14. Selecting an air sampling device*

The following factors must be considered when choosing an air sampling instrument:

- Viability and type of the organism to be sampled
- Compatibility with the selected method of analysis
- Sensitivity of particles to sampling
- Assumed concentrations and particle size
- Whether airborne clumps must be broken (i.e., total viable organism count vs. particle count)
- Volume of air to be sampled and length of time sampler is to be continuously operated
- Background contamination
- Ambient conditions
- Sampler collection efficiency
- Effort and skill required to operate sampler
- Availability and cost of sampler, plus back-up samplers in case of equipment malfunction
- Availability of auxiliary equipment and utilities (e.g., vacuum pumps, electricity, and water)

* Material in this box is compiled from reference 1218.

Liquid impinger and solid impactor samplers are the most practical for sampling bacteria, particles, and fungal spores, because they can sample large volumes of air in relatively short periods of time.²⁸⁹ Solid impactor units are available as either “slit” or “sieve” designs. Slit impactors use a rotating disc as support for the collecting surface, which allows determinations of concentration over time. Sieve impactors commonly use stages with calibrated holes of different diameters. Some impactor-type samplers use centrifugal force to impact particles onto agar surfaces. The interior of either device must be made sterile to avoid inadvertent contamination from the sampler. Results obtained from either sampling device can be expressed as organisms or particles per unit volume of air (CFU/m³).

Sampling for bacteria requires special attention, because bacteria may be present as individual organisms, as clumps, or mixed with or adhering to dust or covered with a protective coating of dried organic or inorganic substances. Reports of bacterial concentrations determined by air sampling therefore must indicate whether the results represent individual organisms or particles bearing multiple cells. Certain types of samplers (e.g., liquid impingers) will completely or partially disintegrate clumps and large particles; the sampling result will therefore reflect the total number of individual organisms present in the air.

The task of sizing a bioaerosol is simplified through the use of sieves or slit impactors because these samplers will separate the particles and microorganisms into size ranges as the sample is collected. These samplers must, however, be calibrated first by sampling aerosols under similar use conditions.¹²²⁵

The use of settle plates (i.e., the sedimentation or depositional method) is not recommended when sampling air for fungal spores, because single spores can remain suspended in air indefinitely.²⁸⁹ Settle plates have been used mainly to sample for particulates and bacteria either in research studies or during epidemiologic investigations.^{161, 1226–1229} Results of sedimentation sampling are typically expressed as numbers of viable particles or viable bacteria per unit area per the duration of sampling time (i.e., CFU/area/time); this method can not quantify the volume of air sampled. Because the survival of microorganisms during air sampling is inversely proportional to the velocity at which the air is taken into the sampler,¹²¹⁵ one advantage of using a settle plate is its reliance on gravity to bring organisms and particles into contact with its surface, thus enhancing the potential for optimal survival of collected organisms. This process, however, takes several hours to complete and may be impractical for some situations.

Air samplers are designed to meet differing measurement requirements. Some samplers are better suited for one form of measurement than others. No one type of sampler and assay procedure can be used to collect and enumerate 100% of airborne organisms. The sampler and/or sampling method chosen should, however, have an adequate sampling rate to collect a sufficient number of particles in a reasonable time period so that a representative sample of air is obtained for biological analysis. Newer analytical techniques for assaying air samples include PCR methods and enzyme-linked immunosorbent assays (ELISAs).

3. Water Sampling

A detailed discussion of the principles and practices of water sampling has been published.⁹⁴⁵ Water sampling in health-care settings is used to detect waterborne pathogens of clinical significance or to determine the quality of finished water in a facility's distribution system. Routine testing of the water in a health-care facility is usually not indicated, but sampling in support of outbreak investigations can help determine appropriate infection-control measures. Water-quality assessments in dialysis settings have been discussed in this guideline (see Water, Dialysis Water Quality and Dialysate, and Appendix C).

Health-care facilities that conduct water sampling should have their samples assayed in a laboratory that uses established methods and quality-assurance protocols. Water specimens are not "static specimens" at ambient temperature; potential changes in both numbers and types of microbial populations can occur during transport. Consequently, water samples should be sent to the testing laboratory cold (i.e., at approximately 39.2°F [4°C]) and testing should be done as soon as practical after collection (preferably within 24 hours).

Because most water sampling in health-care facilities involves the testing of finished water from the facility's distribution system, a reducing agent (i.e., sodium thiosulfate [Na₂S₂O₃]) needs to be added to neutralize residual chlorine or other halogen in the collected sample. If the water contains elevated levels of heavy metals, then a chelating agent should be added to the specimen. The minimum volume of water to be collected should be sufficient to complete any and all assays indicated; 100 mL is considered a suitable minimum volume. Sterile collection equipment should always be used.

Sampling from a tap requires flushing of the water line before sample collection. If the tap is a mixing faucet, attachments (e.g., screens and aerators) must be removed, and hot and then cold water must be run through the tap before collecting the sample.⁹⁴⁵ If the cleanliness of the tap is questionable, disinfection with 500–600 ppm sodium hypochlorite (1:100 v/v dilution of chlorine bleach) and flushing the tap should precede sample collection.

Microorganisms in finished or treated water often are physically damaged ("stressed") to the point that growth is limited when assayed under standard conditions. Such situations lead to false-negative readings and misleading assessments of water quality. Appropriate neutralization of halogens and chelation of heavy metals are crucial to the recovery of these organisms. The choice of recovery media and incubation conditions will also affect the assay. Incubation temperatures should be closer to the ambient temperature of the water rather than at 98.6°F (37°C), and recovery media should be formulated to provide appropriate concentrations of nutrients to support organisms exhibiting less than rigorous growth.⁹⁴⁵ High-nutrient content media (e.g., blood agar and tryptic soy agar [TSA]) may actually inhibit the growth of these damaged organisms. Reduced nutrient media (e.g., diluted peptone and R2A) are preferable for recovery of these organisms.⁹⁴⁵

Use of aerobic, heterotrophic plate counts allows both a qualitative and quantitative measurement for water quality. If bacterial counts in water are expected to be high in number (e.g., during waterborne outbreak investigations), assaying small quantities using pour plates or spread plates is appropriate.⁹⁴⁵ Membrane filtration is used when low-count specimens are expected and larger sampling volumes are required (≥ 100 mL). The sample is filtered through the membrane, and the filter is applied directly face-up onto the surface of the agar plate and incubated.

Unlike the testing of potable water supplies for coliforms (which uses standardized test and specimen collection parameters and conditions), water sampling to support epidemiologic investigations of disease outbreaks may be subjected to modifications dictated by the circumstances present in the facility. Assay methods for waterborne pathogens may also not be standardized. Therefore, control or comparison samples should be included in the experimental design. Any departure from a standard method should be fully documented and should be considered when interpreting results and developing strategies. Assay methods specific for clinically significant waterborne pathogens (e.g., *Legionella* spp., *Aeromonas* spp, *Pseudomonas* spp., and *Acinetobacter* spp.) are more complicated and costly compared with both methods used to detect coliforms and other standard indicators of water quality.

4. Environmental Surface Sampling

Routine environmental-surface sampling (e.g., surveillance cultures) in health-care settings is neither cost-effective nor warranted.^{951, 1225} When indicated, surface sampling should be conducted with multidisciplinary approval in adherence to carefully considered plans of action and policy (Box 15).

Box 15. Undertaking environmental-surface sampling*

The following factors should be considered before engaging in environmental-surface sampling:

- **Background information from the literature and present activities (i.e., preliminary results from an epidemiologic investigation)**
 - **Location of surfaces to be sampled**
 - **Method of sample collection and the appropriate equipment for this task**
 - **Number of replicate samples needed and which control or comparison samples are required**
 - **Parameters of the sample assay method and whether the sampling will be qualitative, quantitative, or both**
 - **An estimate of the maximum allowable microbial numbers or types on the surface(s) sampled (refer to the Spaulding classification for devices and surfaces)**
 - **Some anticipation of a corrective action plan**
-

* The material in this box is compiled from reference 1214.

Surface sampling is used currently for research, as part of an epidemiologic investigation, or as part of a comprehensive approach for specific quality assurance purposes. As a research tool, surface sampling has been used to determine a) potential environmental reservoirs of pathogens,^{564, 1230–1232} b) survival of microorganisms on surfaces,^{1232, 1233} and c) the sources of the environmental contamination.¹⁰²³ Some or all of these approaches can also be used during outbreak investigations.¹²³² Discussion of surface sampling of medical devices and instruments is beyond the scope of this document and is deferred to future guidelines on sterilization and disinfection issues.

Meaningful results depend on the selection of appropriate sampling and assay techniques.¹²¹⁴ The media, reagents, and equipment required for surface sampling are available from any well-equipped

microbiology laboratory and laboratory supplier. For quantitative assessment of surface organisms, non-selective, nutrient-rich agar media and broth (e.g., TSA and brain-heart infusion broth [BHI] with or without 5% sheep or rabbit blood supplement) are used for the recovery of aerobic bacteria. Broth media are used with membrane-filtration techniques. Further sample work-up may require the use of selective media for the isolation and enumeration of specific groups of microorganisms. Examples of selective media are MacConkey agar (MAC [selects for gram-negative bacteria]), Cetrimide agar (selects for *Pseudomonas aeruginosa*), or Sabouraud dextrose- and malt extract agars and broths (select for fungi). Qualitative determinations of organisms from surfaces require only the use of selective or non-selective broth media.

Effective sampling of surfaces requires moisture, either already present on the surface to be sampled or via moistened swabs, sponges, wipes, agar surfaces, or membrane filters.^{1214, 1234–1236} Dilution fluids and rinse fluids include various buffers or general purpose broth media (Table 24). If disinfectant residuals are expected on surfaces being sampled, specific neutralizer chemicals should be used in both the growth media and the dilution or rinse fluids. Lists of the neutralizers, the target disinfectant active ingredients, and the use concentrations have been published.^{1214, 1237} Alternatively, instead of adding neutralizing chemicals to existing culture media (or if the chemical nature of the disinfectant residuals is unknown), the use of either a) commercially available media including a variety of specific and non-specific neutralizers or b) double-strength broth media will facilitate optimal recovery of microorganisms. The inclusion of appropriate control specimens should be included to rule out both residual antimicrobial activity from surface disinfectants and potential toxicity caused by the presence of neutralizer chemicals carried over into the assay system.¹²¹⁴

Table 24. Examples of eluents and diluents for environmental-surface sampling* +

Solutions	Concentration in water
Ringer	¼ strength
Peptone water	0.1%–1.0%
Buffered peptone water	0.067 M phosphate, 0.43% NaCl, 0.1% peptone
Phosphate-buffered saline	0.02 M phosphate, 0.9% NaCl
Sodium chloride (NaCl)	0.25%–0.9%
Calgon Ringer§	¼ strength
Thiosulfate Ringer¶	¼ strength
Water	–
Tryptic soy broth (TSB)	–
Brain-heart infusion broth (BHI) supplemented with 0.5% beef extract	–

* Material in this table is compiled from references 1214 and 1238.

+ A surfactant (e.g., polysorbate [i.e., Tween® 80]) may be added to eluents and diluents. A concentration ranging from 0.01%–0.1% is generally used, depending on the specific application. Foaming may occur during use.

§ This solution is used for dissolution of calcium alginate swabs.

¶ This solution is used for neutralization of residual chlorine.

Several methods can be used for collecting environmental surface samples (Table 25). Specific step-by-step discussions of each of the methods have been published.^{1214, 1239} For best results, all methods should incorporate aseptic techniques, sterile equipment, and sterile recovery media.

Table 25. Methods of environmental-surface sampling

Method	Suitable for appropriate surface(s)	Assay technique	Procedural notes	Points of interpretation	Available standards	References
Sample/rinse Moistened swab/rinse	Non-absorbent surfaces, corners, crevices, devices, and instruments	Dilutions; qualitative or quantitative assays	Assay multiple measures areas or devices with separate swabs	Report results per measured areas or if assaying an object, per the entire sample site	YES – food industry; NO – health care	1214, 1239–1242
Moistened sponge/rinse	Large areas and housekeeping surfaces (e.g., floors or walls)	Dilutions; qualitative or quantitative assays	Vigorously rub a sterile sponge over the surface	Report results per measured area	YES – food industry; NO – health care	1214, 1239–1242
Moistened wipe/rinse	Large areas and housekeeping surfaces (e.g., countertops)	Dilutions; qualitative or quantitative assays	Use a sterile wipe	Report results per measured area	YES – food industry; NO – health care	1214, 1239–1242
Direct immersion	Small items capable of being immersed	Dilutions; qualitative or quantitative assays	Use membrane filtration if rinse volume is large and anticipated microbiological concentration is low	Report results per item	NO	1214
Containment	Interior surfaces of containers, tubes, or bottles	Dilutions; qualitative or quantitative assays	Use membrane filtration if rinse volume is large	Evaluate both the types and numbers of microorganisms	YES – food and industrial applications for containers prior to fill	1214
RODAC*	Previously cleaned and sanitized flat, non-absorbent surfaces; not suitable for irregular surfaces	Direct assay	Overgrowth occurs if used on heavily contaminated surfaces; use neutralizers in the agar if surface disinfectant residuals are present	Provides direct, quantitative results; use a minimum of 15 plates per an average hospital room	NO	1214, 1237, 1239, 1243, 1244

* RODAC stands for “replicate organism direct agar contact.”

Sample/rinse methods are frequently chosen because of their versatility. However, these sampling methods are the most prone to errors caused by manipulation of the swab, gauze pad, or sponge.¹²³⁸ Additionally, no microbiocidal or microbiostatic agents should be present in any of these items when used for sampling.¹²³⁸ Each of the rinse methods requires effective elution of microorganisms from the item used to sample the surface. Thorough mixing of the rinse fluids after elution (e.g., via manual or mechanical mixing using a vortex mixer, shaking with or without glass beads, and ultrasonic bath) will help to remove and suspend material from the sampling device and break up clumps of organisms for a more accurate count.¹²³⁸ In some instances, the item used to sample the surface (e.g., gauze pad and sponge) may be immersed in the rinse fluids in a sterile bag and subjected to stomaching.¹²³⁸ This technique, however, is suitable only for soft or absorbent items that will not puncture the bag during the elution process.

If sampling is conducted as part of an epidemiologic investigation of a disease outbreak, identification of isolates to species level is mandatory, and characterization beyond the species level is preferred.¹²¹⁴ When interpreting the results of the sampling, the expected degree of microbial contamination

associated with the various categories of surfaces in the Spaulding classification must be considered. Environmental surfaces should be visibly clean; recognized pathogens in numbers sufficient to result in secondary transfer to other animate or inanimate surfaces should be absent from the surface being sampled.¹²¹⁴ Although the interpretation of a sample with positive microbial growth is self-evident, an environmental surface sample, especially that obtained from housekeeping surfaces, that shows no growth does not represent a “sterile” surface. Sensitivities of the sampling and assay methods (i.e., level of detection) must be taken into account when no-growth samples are encountered. Properly collected control samples will help rule out extraneous contamination of the surface sample.

G. Laundry and Bedding

1. General Information

Laundry in a health-care facility may include bed sheets and blankets, towels, personal clothing, patient apparel, uniforms, scrub suits, gowns, and drapes for surgical procedures.¹²⁴⁵ Although contaminated textiles and fabrics in health-care facilities can be a source of substantial numbers of pathogenic microorganisms, reports of health-care–associated diseases linked to contaminated fabrics are so few in number that the overall risk of disease transmission during the laundry process likely is negligible. When the incidence of such events are evaluated in the context of the volume of items laundered in health-care settings (estimated to be 5 billion pounds annually in the United States),¹²⁴⁶ existing control measures (e.g., standard precautions) are effective in reducing the risk of disease transmission to patients and staff. Therefore, use of current control measures should be continued to minimize the contribution of contaminated laundry to the incidence of health-care–associated infections. The control measures described in this section of the guideline are based on principles of hygiene, common sense, and consensus guidance; they pertain to laundry services utilized by health-care facilities, either in-house or contract, rather than to laundry done in the home.

2. Epidemiology and General Aspects of Infection Control

Contaminated textiles and fabrics often contain high numbers of microorganisms from body substances, including blood, skin, stool, urine, vomitus, and other body tissues and fluids. When textiles are heavily contaminated with potentially infective body substances, they can contain bacterial loads of 10^6 – 10^8 CFU/100 cm² of fabric.¹²⁴⁷ Disease transmission attributed to health-care laundry has involved contaminated fabrics that were handled inappropriately (i.e., the shaking of soiled linens). Bacteria (*Salmonella* spp., *Bacillus cereus*), viruses (hepatitis B virus [HBV]), fungi (*Microsporium canis*), and ectoparasites (scabies) presumably have been transmitted from contaminated textiles and fabrics to workers via a) direct contact or b) aerosols of contaminated lint generated from sorting and handling contaminated textiles.^{1248–1252} In these events, however, investigations could not rule out the possibility that some of these reported infections were acquired from community sources. Through a combination of soil removal, pathogen removal, and pathogen inactivation, contaminated laundry can be rendered hygienically clean. Hygienically clean laundry carries negligible risk to health-care workers and patients, provided that the clean textiles, fabric, and clothing are not inadvertently contaminated before use.

OSHA defines contaminated laundry as “laundry which has been soiled with blood or other potentially infectious materials or may contain sharps.”⁹⁶⁷ The purpose of the laundry portion of the standard is to protect the worker from exposure to potentially infectious materials during collection, handling, and sorting of contaminated textiles through the use of personal protective equipment, proper work practices, containment, labeling, hazard communication, and ergonomics.

Experts are divided regarding the practice of transporting clothes worn at the workplace to the health-care worker's home for laundering. Although OSHA regulations prohibit home laundering of items that are considered personal protective apparel or equipment (e.g., laboratory coats),⁹⁶⁷ experts disagree about whether this regulation extends to uniforms and scrub suits that are not contaminated with blood or other potentially infectious material. Health-care facility policies on this matter vary and may be inconsistent with recommendations of professional organizations.^{1253, 1254} Uniforms without blood or body substance contamination presumably do not differ appreciably from street clothes in the degree and microbial nature of soilage. Home laundering would be expected to remove this level of soil adequately. However, if health-care facilities require the use of uniforms, they should either make provisions to launder them or provide information to the employee regarding infection control and cleaning guidelines for the item based on the tasks being performed at the facility. Health-care facilities should address the need to provide this service and should determine the frequency for laundering these items. In a recent study examining the microbial contamination of medical students' white coats, the students perceived the coats as "clean" as long as the garments were not visibly contaminated with body substances, even after wearing the coats for several weeks.¹²⁵⁵ The heaviest bacterial load was found on the sleeves and the pockets of these garments; the organisms most frequently isolated were *Staphylococcus aureus*, diphtheroids, and *Acinetobacter* spp.¹²⁵⁵ Presumably, the sleeves of the coat may make contact with a patient and potentially serve to transfer environmentally stable microorganisms among patients. In this study, however, surveillance was not conducted among patients to detect new infections or colonizations. The students did, however, report that they would likely replace their coats more frequently and regularly if clean coats were provided.¹²⁵⁵ Apart from this study, which documents the presence of pathogenic bacteria on health-care facility clothing, reports of infections attributed to either the contact with such apparel or with home laundering have been rare.^{1256, 1257}

Laundry services for health-care facilities are provided either in-house (i.e., on-premise laundry [OPL]), co-operatives (i.e., those entities owned and operated by a group of facilities), or by off-site commercial laundries. In the latter, the textiles may be owned by the health-care facility, in which case the processor is paid for laundering only. Alternatively, the textiles may be owned by the processor who is paid for every piece laundered on a "rental" fee. The laundry facility in a health-care setting should be designed for efficiency in providing hygienically clean textiles, fabrics, and apparel for patients and staff. Guidelines for laundry construction and operation for health-care facilities, including nursing facilities, have been published.^{120, 1258} The design and engineering standards for existing facilities are those cited in the AIA edition in effect during the time of the facility's construction.¹²⁰ A laundry facility is usually partitioned into two separate areas - a "dirty" area for receiving and handling the soiled laundry and a "clean" area for processing the washed items.¹²⁵⁹ To minimize the potential for recontaminating cleaned laundry with aerosolized contaminated lint, areas receiving contaminated textiles should be at negative air pressure relative to the clean areas.¹²⁶⁰⁻¹²⁶² Laundry areas should have handwashing facilities readily available to workers. Laundry workers should wear appropriate personal protective equipment (e.g., gloves and protective garments) while sorting soiled fabrics and textiles.⁹⁶⁷ Laundry equipment should be used and maintained according to the manufacturer's instructions to prevent microbial contamination of the system.^{1250, 1263} Damp textiles should not be left in machines overnight.¹²⁵⁰

3. Collecting, Transporting, and Sorting Contaminated Textiles and Fabrics

The laundry process starts with the removal of used or contaminated textiles, fabrics, and/or clothing from the areas where such contamination occurred, including but not limited to patients' rooms, surgical/operating areas, and laboratories. Handling contaminated laundry with a minimum of agitation

can help prevent the generation of potentially contaminated lint aerosols in patient-care areas.^{967, 1259} Sorting or rinsing contaminated laundry at the location where contamination occurred is prohibited by OSHA.⁹⁶⁷ Contaminated textiles and fabrics are placed into bags or other appropriate containment in this location; these bags are then securely tied or otherwise closed to prevent leakage.⁹⁶⁷ Single bags of sufficient tensile strength are adequate for containing laundry, but leak-resistant containment is needed if the laundry is wet and capable of soaking through a cloth bag.¹²⁶⁴ Bags containing contaminated laundry must be clearly identified with labels, color-coding, or other methods so that health-care workers handle these items safely, regardless of whether the laundry is transported within the facility or destined for transport to an off-site laundry service.⁹⁶⁷

Typically, contaminated laundry originating in isolation areas of the hospital is segregated and handled with special practices; however, few, if any, cases of health-care–associated infection have been linked to this source.¹²⁶⁵ Single-blinded studies have demonstrated that laundry from isolation areas is no more heavily contaminated with microorganisms than laundry from elsewhere in the hospital.¹²⁶⁶ Therefore, adherence to standard precautions when handling contaminated laundry in isolation areas and minimizing agitation of the contaminated items are considered sufficient to prevent the dispersal of potentially infectious aerosols.⁶

Contaminated textiles and fabrics in bags can be transported by cart or chute.^{1258, 1262} Laundry chutes require proper design, maintenance, and use, because the piston-like action of a laundry bag traveling in the chute can propel airborne microbial contaminants throughout the facility.^{1267–1269} Laundry chutes should be maintained under negative air pressure to prevent the spread of microorganisms from floor to floor. Loose, contaminated pieces of laundry should not be tossed into chutes, and laundry bags should be closed or otherwise secured to prevent the contents from falling out into the chute.¹²⁷⁰ Health-care facilities should determine the point in the laundry process at which textiles and fabrics should be sorted. Sorting after washing minimizes the exposure of laundry workers to infective material in soiled fabrics, reduces airborne microbial contamination in the laundry area, and helps to prevent potential percutaneous injuries to personnel.¹²⁷¹ Sorting laundry before washing protects both the machinery and fabrics from hard objects (e.g., needles, syringes, and patients' property) and reduces the potential for recontamination of clean textiles.¹²⁷² Sorting laundry before washing also allows for customization of laundry formulas based on the mix of products in the system and types of soils encountered. Additionally, if work flow allows, increasing the amount of segregation by specific product types will usually yield the greatest amount of work efficiency during inspection, folding, and pack-making operations.¹²⁵³ Protective apparel for the workers and appropriate ventilation can minimize these exposures.^{967, 1258–1260} Gloves used for the task of sorting laundry should be of sufficient thickness to minimize sharps injuries.⁹⁶⁷ Employee safety personnel and industrial hygienists can help to determine the appropriate glove choice.

4. Parameters of the Laundry Process

Fabrics, textiles, and clothing used in health-care settings are disinfected during laundering and generally rendered free of vegetative pathogens (i.e., hygienically clean), but they are not sterile.¹²⁷³ Laundering cycles consist of flush, main wash, bleaching, rinsing, and souring.¹²⁷⁴ Cleaned wet textiles, fabrics, and clothing are then dried, pressed as needed, and prepared (e.g., folded and packaged) for distribution back to the facility. Clean linens provided by an off-site laundry must be packaged prior to transport to prevent inadvertent contamination from dust and dirt during loading, delivery, and unloading. Functional packaging of laundry can be achieved in several ways, including a) placing clean linen in a hamper lined with a previously unused liner, which is then closed or covered; b) placing clean linen in a properly cleaned cart and covering the cart with disposable material or a properly cleaned reusable textile material that can be secured to the cart; and c) wrapping individual bundles of clean

textiles in plastic or other suitable material and sealing or taping the bundles.

The antimicrobial action of the laundering process results from a combination of mechanical, thermal, and chemical factors.^{1271, 1275, 1276} Dilution and agitation in water remove substantial quantities of microorganisms. Soaps and detergents function to suspend soils and also exhibit some microbiocidal properties. Hot water provides an effective means of destroying microorganisms.¹²⁷⁷ A temperature of at least 160°F (71°C) for a minimum of 25 minutes is commonly recommended for hot-water washing.² Water of this temperature can be provided by steam jet or separate booster heater.¹²⁰ The use of chlorine bleach assures an extra margin of safety.^{1278, 1279} A total available chlorine residual of 50–150 ppm is usually achieved during the bleach cycle.¹²⁷⁷ Chlorine bleach becomes activated at water temperatures of 135°F–145°F (57.2°C–62.7°C). The last of the series of rinse cycles is the addition of a mild acid (i.e., sour) to neutralize any alkalinity in the water supply, soap, or detergent. The rapid shift in pH from approximately 12 to 5 is an effective means to inactivate some microorganisms.¹²⁴⁷ Effective removal of residual alkali from fabrics is an important measure in reducing the risk for skin reactions among patients.

Chlorine bleach is an economical, broad-spectrum chemical germicide that enhances the effectiveness of the laundering process. Chlorine bleach is not, however, an appropriate laundry additive for all fabrics. Traditionally, bleach was not recommended for laundering flame-retardant fabrics, linens, and clothing because its use diminished the flame-retardant properties of the treated fabric.¹²⁷³ However, some modern-day flame retardant fabrics can now tolerate chlorine bleach. Flame-retardant fabrics, whether topically treated or inherently flame retardant, should be thoroughly rinsed during the rinse cycles, because detergent residues are capable of supporting combustion. Chlorine alternatives (e.g., activated oxygen-based laundry detergents) provide added benefits for fabric and color safety in addition to antimicrobial activity. Studies comparing the antimicrobial potencies of chlorine bleach and oxygen-based bleach are needed. Oxygen-based bleach and detergents used in health-care settings should be registered by EPA to ensure adequate disinfection of laundry. Health-care workers should note the cleaning instructions of textiles, fabrics, drapes, and clothing to identify special laundering requirements and appropriate hygienic cleaning options.¹²⁷⁸

Although hot-water washing is an effective laundry disinfection method, the cost can be substantial. Laundries are typically the largest users of hot water in hospitals. They consume 50%–75% of the total hot water,¹²⁸⁰ representing an average of 10%–15% of the energy used by a hospital. Several studies have demonstrated that lower water temperatures of 71°F–77°F (22°C–25°C) can reduce microbial contamination when the cycling of the washer, the wash detergent, and the amount of laundry additive are carefully monitored and controlled.^{1247, 1281–1285} Low-temperature laundry cycles rely heavily on the presence of chlorine- or oxygen-activated bleach to reduce the levels of microbial contamination. The selection of hot- or cold-water laundry cycles may be dictated by state health-care facility licensing standards or by other regulation. Regardless of whether hot or cold water is used for washing, the temperatures reached in drying and especially during ironing provide additional significant microbiocidal action.¹²⁴⁷ Dryer temperatures and cycle times are dictated by the materials in the fabrics. Man-made fibers (i.e., polyester and polyester blends) require shorter times and lower temperatures.

After washing, cleaned and dried textiles, fabrics, and clothing are pressed, folded, and packaged for transport, distribution, and storage by methods that ensure their cleanliness until use.² State regulations and/or accrediting standards may dictate the procedures for this activity. Clean/sterile and contaminated textiles should be transported from the laundry to the health-care facility in vehicles (e.g., trucks, vans, and carts) that allow for separation of clean/sterile and contaminated items. Clean/sterile textiles and contaminated textiles may be transported in the same vehicle, provided that the use of physical barriers and/or space separation can be verified to be effective in protecting the clean/sterile items from

contamination. Clean, uncovered/unwrapped textiles stored in a clean location for short periods of time (e.g., uncovered and used within a few hours) have not been demonstrated to contribute to increased levels of health-care–acquired infection. Such textiles can be stored in convenient places for use during the provision of care, provided that the textiles can be maintained dry and free from soil and body-substance contamination.

In the absence of microbiologic standards for laundered textiles, no rationale exists for routine microbiologic sampling of cleaned health-care textiles and fabrics.¹²⁸⁶ Sampling may be used as part of an outbreak investigation if epidemiologic evidence suggests that textiles, fabrics, or clothing are a suspected vehicle for disease transmission. Sampling techniques include aseptically macerating the fabric into pieces and adding these to broth media or using contact plates (RODAC plates) for direct surface sampling.^{1271, 1286} When evaluating the disinfecting properties of the laundering process specifically, placing pieces of fabric between two membrane filters may help to minimize the contribution of the physical removal of microorganisms.¹²⁸⁷

Washing machines and dryers in residential-care settings are more likely to be consumer items rather than the commercial, heavy-duty, large volume units typically found in hospitals and other institutional health-care settings. Although all washing machines and dryers in health-care settings must be properly maintained for performance according to the manufacturer's instructions, questions have been raised about the need to disinfect washers and dryers in residential-care settings. Disinfection of the tubs and tumblers of these machines is unnecessary when proper laundry procedures are followed; these procedures involve a) the physical removal of bulk solids (e.g., feces) before the wash/dry cycle and b) proper use of temperature, detergent, and laundry additives. Infection has not been linked to laundry procedures in residential-care facilities, even when consumer versions of detergents and laundry additives are used.

5. Special Laundry Situations

Some textile items (e.g., surgical drapes and reusable gowns) must be sterilized before use and therefore require steam autoclaving after laundering.⁷ Although the American Academy of Pediatrics in previous guidelines recommended autoclaving for linens in neonatal intensive care units (NICUs), studies on the microbial quality of routinely cleaned NICU linen have not identified any increased risk for infection among the neonates receiving care.¹²⁸⁸ Consequently, hygienically clean linens are suitable for use in this setting.⁹⁹⁷ The use of sterile linens in burn therapy units remains unresolved.

Coated or laminated fabrics are often used in the manufacture of PPE. When these items become contaminated with blood or other body substances, the manufacturer's instructions for decontamination and cleaning take into account the compatibility of the rubber backing with the chemical germicides or detergents used in the process. The directions for decontaminating these items should be followed as indicated; the item should be discarded when the backing develops surface cracks.

Dry cleaning, a cleaning process that utilizes organic solvents (e.g., perchloroethylene) for soil removal, is an alternative means of cleaning fabrics that might be damaged in conventional laundering and detergent washing. Several studies, however, have shown that dry cleaning alone is relatively ineffective in reducing the numbers of bacteria and viruses on contaminated linens;^{1289, 1290} microbial populations are significantly reduced only when dry-cleaned articles are heat pressed. Dry cleaning should therefore not be considered a routine option for health-care facility laundry and should be reserved for those circumstances in which fabrics can not be safely cleaned with water and detergent.¹²⁹¹

6. Surgical Gowns, Drapes, and Disposable Fabrics

An issue of recent concern involves the use of disposable (i.e., single use) versus reusable (i.e., multiple use) surgical attire and fabrics in health-care settings.¹²⁹² Regardless of the material used to manufacture gowns and drapes, these items must be resistant to liquid and microbial penetration.^{7, 1293–1297} Surgical gowns and drapes must be registered with FDA to demonstrate their safety and effectiveness. Repellency and pore size of the fabric contribute to gown performance, but performance capability can be influenced by the item's design and construction.^{1298, 1299} Reinforced gowns (i.e., gowns with double-layered fabric) generally are more resistant to liquid strike-through.^{1300, 1301} Reinforced gowns may, however, be less comfortable. Guidelines for selection and use of barrier materials for surgical gowns and drapes have been published.¹³⁰² When selecting a barrier product, repellency level and type of barrier should be compatible for the exposure expected.⁹⁶⁷ However, data are limited regarding the association between gown or drape characteristics and risk for surgical site infections.^{7, 1303} Health-care facilities must ensure optimal protection of patients and health-care workers. Not all fabric items in health care lend themselves to single-use. Facilities exploring options for gowns and drapes should consider the expense of disposable items and the impact on the facility's waste-management costs once these items are discarded. Costs associated with the use of durable goods involve the fabric or textile items; staff expenses to collect, sort, clean, and package the laundry; and energy costs to operate the laundry if on-site or the costs to contract with an outside service.^{1304, 1305}

7. Antimicrobial-Impregnated Articles and Consumer Items Bearing Antimicrobial Labeling

Manufacturers are increasingly incorporating antibacterial or antimicrobial chemicals into consumer and health-care items. Some consumer products bearing labels that indicate treatment with antimicrobial chemicals have included pens, cutting boards, toys, household cleaners, hand lotions, cat litter, soaps, cotton swabs, toothbrushes, and cosmetics. The “antibacterial” label on household cleaning products, in particular, gives consumers the impression that the products perform “better” than comparable products without this labeling, when in fact all household cleaners have antibacterial properties.

In the health-care setting, treated items may include children's pajamas, mattresses, and bed linens with label claims of antimicrobial properties. These claims require careful evaluation to determine whether they pertain to the use of antimicrobial chemicals as preservatives for the fabric or other components or whether they imply a health claim.^{1306, 1307} No evidence is available to suggest that use of these products will make consumers and patients healthier or prevent disease. No data support the use of these items as part of a sound infection-control strategy, and therefore, the additional expense of replacing a facility's bedding and sheets with these treated products is unwarranted.

EPA has reaffirmed its position that manufacturers who make public health claims for articles containing antimicrobial chemicals must provide evidence to support those claims as part of the registration process.¹³⁰⁸ Current EPA regulations outlined in the Treated Articles Exemption of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) require manufacturers to register both the antimicrobial chemical used in or on the product and the finished product itself if a public health claim is maintained for the item. The exemption applies to the use of antimicrobial chemicals for the purpose of preserving the integrity of the product's raw material(s). The U.S. Federal Trade Commission (FTC) is evaluating manufacturer advertising of products with antimicrobial claims.¹³⁰⁹

8. Standard Mattresses, Pillows, and Air-Fluidized Beds

Standard mattresses and pillows can become contaminated with body substances during patient care if the integrity of the covers of these items is compromised. The practice of sticking needles into the mattress should be avoided. A mattress cover is generally a fitted, protective material, the purpose of which is to prevent the mattress from becoming contaminated with body fluids and substances. A linen sheet placed on the mattress is not considered a mattress cover. Patches for tears and holes in mattress covers do not provide an impermeable surface over the mattress. Mattress covers should be replaced when torn; the mattress should be replaced if it is visibly stained. Wet mattresses, in particular, can be a substantial environmental source of microorganisms. Infections and colonizations caused by *Acinetobacter* spp., MRSA, and *Pseudomonas aeruginosa* have been described, especially among burn patients.^{1310–1315} In these reports, the removal of wet mattresses was an effective infection-control measure. Efforts were made to ensure that pads and covers were cleaned and disinfected between patients using disinfectant products compatible with mattress-cover materials to ensure that these covers remained impermeable to fluids.^{1310–1314} Pillows and their covers should be easily cleanable, preferably in a hot water laundry cycle.¹³¹⁵ These should be laundered between patients or if contaminated with body substances.

Air-fluidized beds are used for the care of patients immobilized for extended periods of time because of therapy or injury (e.g., pain, decubitus ulcers, and burns).¹³¹⁶ These specialized beds consist of a base unit filled with microsphere beads fluidized by warm, dry air flowing upward from a diffuser located at the bottom of the unit. A porous, polyester filter sheet separates the patient from direct contact with the beads but allows body fluids to pass through to the beads. Moist beads aggregate into clumps which settle to the bottom where they are removed as part of routine bed maintenance.

Because the beads become contaminated with the patient's body substances, concerns have been raised about the potential for these beds to serve as an environmental source of pathogens. Certain pathogens (e.g., *Enterococcus* spp., *Serratia marcescens*, *Staphylococcus aureus*, and *Streptococcus fecalis*) have been recovered either from the microsphere beads or the polyester sheet after cleaning.^{1317, 1318} Reports of cross-contamination of patients, however, are few.¹³¹⁸ Nevertheless, routine maintenance and between-patient decontamination procedures can minimize potential risks to patients. Regular removal of bead clumps, coupled with the warm, dry air of the bed, can help to minimize bacterial growth in the unit.^{1319–1321} Beads are decontaminated between patients by high heat (113°F–194°F [45°C–90°C], depending on the manufacturer's specifications) for at least 1 hour; this procedure is particularly important for the inactivation of *Enterococcus* spp. which are relatively resistant to heat.^{1322, 1323} The polyester filter sheet requires regular changing and thorough cleaning and disinfection, especially between patients.^{1317, 1318, 1322, 1323}

Microbial contamination of the air space in the immediate vicinity of a properly maintained air-fluidized bed is similar to that found in air around conventional bedding, despite the air flow out of the base unit and around the patient.^{1320, 1324, 1325} An operational air-fluidized bed can, however, interfere with proper pressure differentials, especially in negative-pressure rooms;¹³²⁶ the effect varies with the location of the bed relative to the room's configuration and supply and exhaust vent locations. Use of an air-fluidized bed in a negative-pressure room requires consultation with a facility engineer to determine appropriate placement of the bed.

H. Animals in Health-Care Facilities

1. General Information

Animals in health-care facilities traditionally have been limited to laboratories and research areas. However, their presence in patient-care areas is now more frequent, both in acute-care and long-term care settings, prompting consideration for the potential transmission of zoonotic pathogens from animals to humans in these settings. Although dogs and cats may be commonly encountered in health-care settings, other animals (e.g., fish, birds, non-human primates, rabbits, rodents, and reptiles) also can be present as research, resident, or service animals. These animals can serve as sources of zoonotic pathogens that could potentially infect patients and health-care workers (Table 26).¹³²⁷⁻¹³⁴⁰ Animals potentially can serve as reservoirs for antibiotic-resistant microorganisms, which can be introduced to the health-care setting while the animal is present. VRE have been isolated from both farm animals and pets,¹³⁴¹ and a cat in a geriatric care center was found to be colonized with MRSA.¹³⁴²

Table 26. Examples of diseases associated with zoonotic transmission*+

Infectious disease	Cats	Dogs	Fish	Birds	Rabbits	Reptiles§	Primates	Rodents§
Virus								
Lymphocytic choriomeningitis								+¶
Rabies	+	+						
Bacteria								
Campylobacteriosis	+	+				+	+	+
<i>Capnocytophaga canimorsus</i> infection	+	+						
Cat scratch disease (<i>Bartonella henselae</i>)	+							
Leptospirosis	+						+	+
Mycobacteriosis			+	+				
Pasteurellosis	+	+			+			
Plague	+			+			+	+
Psittacosis				+				
Q fever (<i>Coxiella burnetti</i>)	+							
Rat bite fever (<i>Spirillum minus</i> , <i>Streptobacillus moniliformis</i>)								+
Salmonellosis	+	+		+	+	+	+	+
Tularemia	+				+			+
Yersiniosis					+	+	+	+
Parasites								
Ancylostomiasis	+	+					+	
Cryptosporidiosis	+							
Giardiasis	+	+					+	
Toxocariasis	+	+					+	
Toxoplasmosis	+	+					+	
Fungi								
Blastomycosis		+						
Dermatophytosis		+			+		+	+

* Material in this table is adapted from reference 1331 and used with permission of the publisher (Lippincott Williams and Wilkins).

+ This table does not include vectorborne diseases.

§ Reptiles include lizards, snakes, and turtles. Rodents include hamsters, mice, and rats.

¶ The + symbol indicates that the pathogen associated with the infection has been isolated from animals and is considered to pose potential risk to humans.

Zoonoses can be transmitted from animals to humans either directly or indirectly via bites, scratches, aerosols, ectoparasites, accidental ingestion, or contact with contaminated soil, food, water, or unpasteurized milk.^{1331, 1332, 1343–1345} Colonization and hand transferral of pathogens acquired from pets in health-care workers' homes represent potential sources and modes of transmission of zoonotic pathogens in health-care settings. An outbreak of infections caused by a yeast (*Malassezia pachydermatis*) among newborns was traced to transfer of the yeast from the hands of health-care workers with pet dogs at home.¹³⁴⁶ In addition, an outbreak of ringworm in a NICU caused by *Microsporum canis* was associated with a nurse and her cat,¹³⁴⁷ and an outbreak of *Rhodococcus (Gordona) bronchialis* sternal SSIs after coronary-artery bypass surgery was traced to a colonized nurse whose dogs were culture-positive for the organism.¹³⁴⁸ In the latter outbreak, whether the dogs were the sole source of the organism and whether other environmental reservoirs contributed to the outbreak are unknown. Nonetheless, limited data indicate that outbreaks of infectious disease have occurred as a result of contact with animals in areas housing immunocompetent patients. However, the low frequency of outbreaks may result from a) the relatively limited presence of the animals in health-care facilities and b) the immunocompetency of the patients involved in the encounters. Formal scientific studies to evaluate potential risks of transmission of zoonoses in health-care settings outside of the laboratory are lacking.

2. Animal-Assisted Activities, Animal-Assisted Therapy, and Resident Animals

Animal-Assisted Activities (AAA) are those programs that enhance the patients' quality of life. These programs allow patients to visit animals in either a common, central location in the facility or in individual patient rooms. A group session with the animals enhances opportunities for ambulatory patients and facility residents to interact with caregivers, family members, and volunteers.^{1349–1351} Alternatively, allowing the animals access to individual rooms provides the same opportunity to non-ambulatory patients and patients for whom privacy or dignity issues are a consideration. The decision to allow this access to patients' rooms should be made on a case-by-case basis, with the consultation and consent of the attending physician and nursing staff.

Animal-Assisted Therapy (AAT) is a goal-directed intervention that incorporates an animal into the treatment process provided by a credentialed therapist.^{1330, 1331} The concept for AAT arose from the observation that some patients with pets at home recover from surgical and medical procedures more rapidly than patients without pets.^{1352, 1353} Contact with animals is considered beneficial for enhancing wellness in certain patient populations (e.g., children, the elderly, and extended-care hospitalized patients).^{1349, 1354–1357} However, evidence supporting this benefit is largely derived from anecdotal reports and observations of patient/animal interactions.^{1357–1359} Guidelines for establishing AAT programs are available for facilities considering this option.¹³⁶⁰

The incorporation of non-human primates into an AAA or AAT program is not encouraged because of concerns regarding potential disease transmission from and unpredictable behavior of these animals.^{1361, 1362} Animals participating in either AAA or AAT sessions should be in good health and up-to-date with recommended immunizations and prophylactic medications (e.g., heartworm prevention) as determined by a licensed veterinarian based on local needs and recommendations. Regular re-evaluation of the animal's health and behavior status is essential.¹³⁶⁰ Animals should be routinely screened for enteric parasites and/or have evidence of a recently completed antihelminthic regimen.¹³⁶³ They should also be free of ectoparasites (e.g., fleas and ticks) and should have no sutures, open wounds, or obvious dermatologic lesions that could be associated with bacterial, fungal, or viral infections or parasitic infestations. Incorporating young animals (i.e., those aged <1 year) into these programs is not encouraged because of issues regarding unpredictable behavior and elimination control. Additionally,

the immune systems of very young puppies and kittens is not completely developed, thereby placing the health of these animals at risk. Animals should be clean and well-groomed. The visits must be supervised by persons who know the animals and their behavior. Animal handlers should be trained in these activities and receive site-specific orientation to ensure that they work efficiently with the staff in the specific health-care environment.¹³⁶⁰ Additionally, animal handlers should be in good health.¹³⁶⁰

The most important infection-control measure to prevent potential disease transmission is strict enforcement of hand-hygiene measures (e.g., using either soap and water or an alcohol-based hand rub) for all patients, staff, and residents after handling the animals.^{1355, 1364} Care should also be taken to avoid direct contact with animal urine or feces. Clean-up of these substances from environmental surfaces requires gloves and the use of leak-resistant plastic bags to discard absorbent material used in the process.² The area must be cleaned after visits according to standard cleaning procedures.

The American Academy of Allergy, Asthma, and Immunology estimates that dog or cat allergies occur in approximately 15% of the population.¹³⁶⁵ Minimizing contact with animal saliva, dander, and/or urine helps to mitigate allergic responses.^{1365–1367} Some facilities may not allow animal visitation for patients with a) underlying asthma, b) known allergies to cat or dog hair, c) respiratory allergies of unknown etiology, and d) immunosuppressive disorders. Hair shedding can be minimized by processes that remove dead hair (e.g., grooming) and that prevent the shedding of dead hair (e.g., therapy capes for dogs). Allergens can be minimized by bathing therapy animals within 24 hours of a visit.^{1333, 1368}

Animal therapists and handlers must take precautions to prevent animal bites. Common pathogens associated with animal bites include *Capnocytophaga canimorsus*, *Pasteurella* spp., *Staphylococcus* spp., and *Streptococcus* spp. Selecting well-behaved and well-trained animals for these programs greatly decreases the incidence of bites. Rodents, exotic species, wild/domestic animals (i.e., wolf-dog hybrids), and wild animals whose behavior is unpredictable should be excluded from AAA or AAT programs. A well-trained animal handler should be able to recognize stress in the animal and to determine when to terminate a session to minimize risk. When an animal bites a person during AAA or AAT, the animal is to be permanently removed from the program. If a bite does occur, the wound must be cleansed immediately and monitored for subsequent infection. Most infections can be treated with antibiotics, and antibiotics often are prescribed prophylactically in these situations.

The health-care facility's infection-control staff should participate actively in planning for and coordinating AAA and AAT sessions. Many facilities do not offer AAA or AAT programs for severely immunocompromised patients (e.g., HSCT patients and patients on corticosteroid therapy).¹³³⁹ The question of whether family pets or companion animals can visit terminally-ill HSCT patients or other severely immunosuppressed patients is best handled on a case-by-case basis, although animals should not be brought into the HSCT unit or any other unit housing severely immunosuppressed patients. An in-depth discussion of this issue is presented elsewhere.¹³⁶⁶

Immunocompromised patients who have been discharged from a health-care facility may be at higher risk for acquiring some pet-related zoonoses. Although guidelines have been developed to minimize the risk of disease transmission to HIV-infected patients,⁸ these recommendations may be applicable for patients with other immunosuppressive disorders. In addition to handwashing or hand hygiene, these recommendations include avoiding contact with a) animal feces and soiled litter box materials, b) animals with diarrhea, c) very young animals (i.e., dogs <6 months of age and cats <1 year of age), and d) exotic animals and reptiles.⁸ Pets or companion animals with diarrhea should receive veterinary care to resolve their condition.

Many health-care facilities are adopting more home-like environments for residential-care or extended-stay patients in acute-care settings, and resident animals are one element of this approach.¹³⁶⁹ One

concept, the “Eden Alternative,” incorporates children, plants, and animals (e.g., dogs, cats, fish, birds, rabbits, and rodents) into the daily care setting.^{1370, 1371} The concept of working with resident animals has not been scientifically evaluated. Several issues beyond the benefits of therapy must be considered before embarking on such a program, including a) whether the animals will come into direct contact with patients and/or be allowed to roam freely in the facility; b) how the staff will provide care for the animals; c) the management of patients’ or residents’ allergies, asthma, and phobias; d) precautionary measures to prevent bites and scratches; and e) measures to properly manage the disposal of animal feces and urine, thereby preventing environmental contamination by zoonotic microorganisms (e.g., *Toxoplasma* spp., *Toxocara* spp., and *Ancylostoma* spp.).^{1372, 1373} Few data document a link between health-care–acquired infection rates and frequency of cleaning fish tanks or rodent cages. Skin infections caused by *Mycobacterium marinum* have been described among persons who have fish aquariums at home.^{1374, 1375} Nevertheless, immunocompromised patients should avoid direct contact with fish tanks and cages and the aerosols that these items produce. Further, fish tanks should be kept clean on a regular basis as determined by facility policy, and this task should be performed by gloved staff members who are not responsible for patient care. The use of the infection-control risk assessment can help determine whether a fish tank poses a risk for patient or resident safety and health in these situations. No evidence, however, links the incidence of health-care–acquired infections among immunocompetent patients or residents with the presence of a properly cleaned and maintained fish tank, even in dining areas. As a general preventive measure, resident animal programs are advised to restrict animals from a) food preparation kitchens, b) laundries, c) central sterile supply and any storage areas for clean supplies, and d) medication preparation areas. Resident-animal programs in acute-care facilities should not allow the animals into the isolation areas, protective environments, ORs, or any area where immunocompromised patients are housed. Patients and staff routinely should wash their hands or use waterless, alcohol-based hand-hygiene products after contact with animals.

3. Service Animals

Although this section provides an overview about service animals in health-care settings, it cannot address every situation or question that may arise (see Appendix E - Information Resources). A service animal is any animal individually trained to do work or perform tasks for the benefit of a person with a disability.^{1366, 1376} A service animal is not considered a pet but rather an animal trained to provide assistance to a person because of a disability. Title III of the “Americans with Disabilities Act” (ADA) of 1990 mandates that persons with disabilities accompanied by service animals be allowed access with their service animals into places of public accommodation, including restaurants, public transportation, schools, and health-care facilities.^{1366, 1376} In health-care facilities, a person with a disability requiring a service animal may be an employee, a visitor, or a patient.

An overview of the subject of service animals and their presence in health-care facilities has been published.¹³⁶⁶ No evidence suggests that animals pose a more significant risk of transmitting infection than people; therefore, service animals should not be excluded from such areas, unless an individual patient’s situation or a particular animal poses greater risk that cannot be mitigated through reasonable measures. If health-care personnel, visitors, and patients are permitted to enter care areas (e.g., in-patient rooms, some ICUs, and public areas) without taking additional precautions to prevent transmission of infectious agents (e.g., donning gloves, gowns, or masks), a clean, healthy, well-behaved service animal should be allowed access with its handler.¹³⁶⁶ Similarly, if immunocompromised patients are able to receive visitors without using protective garments or equipment, an exclusion of service animals from this area would not be justified.¹³⁶⁶

Because health-care facilities are covered by the ADA or the Rehabilitation Act, a person with a disability may be accompanied by a service animal within the facility unless the animal’s presence or

behavior creates a fundamental alteration in the nature of a facility's services in a particular area or a direct threat to other persons in a particular area.¹³⁶⁶ A "direct threat" is defined as a significant risk to the health or safety of others that cannot be mitigated or eliminated by modifying policies, practices, or procedures.¹³⁷⁶ The determination that a service animal poses a direct threat in any particular health-care setting must be based on an individualized assessment of the service animal, the patient, and the health-care situation. When evaluating risk in such situations, health-care personnel should consider the nature of the risk (including duration and severity); the probability that injury will occur; and whether reasonable modifications of policies, practices, or procedures will mitigate the risk (J. Wodatch, U.S. Department of Justice, 2000). The person with a disability should contribute to the risk-assessment process as part of a pre-procedure health-care provider/patient conference.

Excluding a service animal from an OR or similar special care areas (e.g., burn units, some ICUs, PE units, and any other area containing equipment critical for life support) is appropriate if these areas are considered to have "restricted access" with regards to the general public. General infection-control measures that dictate such limited access include a) the area is required to meet environmental criteria to minimize the risk of disease transmission, b) strict attention to hand hygiene and absence of dermatologic conditions, and c) barrier protective measures [e.g., using gloves, wearing gowns and masks] are indicated for persons in the affected space. No infection-control measures regarding the use of barrier precautions could be reasonably imposed on the service animal. Excluding a service animal that becomes threatening because of a perceived danger to its handler during treatment also is appropriate; however, exclusion of such an animal must be based on the actual behavior of the particular animal, not on speculation about how the animal might behave.

Another issue regarding service animals is whether to permit persons with disabilities to be accompanied by their service animals during all phases of their stay in the health-care facility. Health-care personnel should discuss all aspects of anticipatory care with the patient who uses a service animal. Health-care personnel may not exclude a service animal because health-care staff may be able to perform the same services that the service animal does (e.g., retrieving dropped items and guiding an otherwise ambulatory person to the restroom). Similarly, health-care personnel can not exclude service animals because the health-care staff perceive a lack of need for the service animal during the person's stay in the health-care facility. A person with a disability is entitled to independent access (i.e., to be accompanied by a service animal unless the animal poses a direct threat or a fundamental alteration in the nature of services); "need" for the animal is not a valid factor in either analysis. For some forms of care (e.g., ambulation as physical therapy following total hip replacement or knee replacement), the service animal should not be used in place of a credentialed health-care worker who directly provides therapy. However, service animals need not be restricted from being in the presence of its handler during this time; in addition, rehabilitation and discharge planning should incorporate the patient's future use of the animal. The health-care personnel and the patient with a disability should discuss both the possible need for the service animal to be separated from its handler for a period of time during non-emergency care and an alternate plan of care for the service animal in the event the patient is unable or unwilling to provide that care. This plan might include family members taking the animal out of the facility several times a day for exercise and elimination, the animal staying with relatives, or boarding off-site. Care of the service animal, however, remains the obligation of the person with the disability, not the health-care staff.

Although animals potentially carry zoonotic pathogens transmissible to man, the risk is minimal with a healthy, clean, vaccinated, well-behaved, and well-trained service animal, the most common of which are dogs and cats. No reports have been published regarding infectious disease that affects humans originating in service dogs. Standard cleaning procedures are sufficient following occupation of an area by a service animal.¹³⁶⁶ Clean-up of spills of animal urine, feces, or other body substances can be accomplished with blood/body substance procedures outlined in the Environmental Services section of

this guideline. No special bathing procedures are required prior to a service animal accompanying its handler into a health-care facility.

Providing access to exotic animals (e.g., reptiles and non-human primates) that are used as service animals is problematic. Concerns about these animals are discussed in two published reviews.^{1331, 1366} Because some of these animals exhibit high-risk behaviors that may increase the potential for zoonotic disease transmission (e.g., herpes B infection), providing health-care facility access to nonhuman primates used as service animals is discouraged, especially if these animals might come into contact with the general public.^{1361, 1362} Health-care administrators should consult the Americans with Disabilities Act for guidance when developing policies about service animals in their facilities.^{1366, 1376}

Requiring documentation for access of a service animal to an area generally accessible to the public would impose a burden on a person with a disability. When health-care workers are not certain that an animal is a service animal, they may ask the person who has the animal if it is a service animal required because of a disability; however, no certification or other documentation of service animal status can be required.¹³⁷⁷

4. Animals as Patients in Human Health-Care Facilities

The potential for direct and indirect transmission of zoonoses must be considered when rooms and equipment in human health-care facilities are used for the medical or surgical treatment or diagnosis of animals.¹³⁷⁸ Inquiries should be made to veterinary medical professionals to determine an appropriate facility and equipment to care for an animal.

The central issue associated with providing medical or surgical care to animals in human health-care facilities is whether cross-contamination occurs between the animal patient and the human health-care workers and/or human patients. The fundamental principles of infection control and aseptic practice should differ only minimally, if at all, between veterinary medicine and human medicine. Health-care-associated infections can and have occurred in both patients and workers in veterinary medical facilities when lapses in infection-control procedures are evident.^{1379–1384} Further, veterinary patients can be at risk for acquiring infection from veterinary health-care workers if proper precautions are not taken.¹³⁸⁵

The issue of providing care to veterinary patients in human health-care facilities can be divided into the following three areas of infection-control concerns: a) whether the room/area used for animal care can be made safe for human patients, b) whether the medical/surgical instruments used on animals can be subsequently used on human patients, and c) which disinfecting or sterilizing procedures need to be done for these purposes. Studies addressing these concerns are lacking. However, with respect to disinfection or sterilization in veterinary settings, only minimal evidence suggests that zoonotic microbial pathogens are unusually resistant to inactivation by chemical or physical agents (with the exception of prions). Ample evidence supports the contrary observation (i.e., that pathogens from human- and animal sources are similar in their relative intrinsic resistance to inactivation).^{1386–1391} Further, no evidence suggests that zoonotic pathogens behave differently from human pathogens with respect to ventilation. Despite this knowledge, an aesthetic and sociologic perception that animal care must remain separate from human care persists. Health-care facilities, however, are increasingly faced with requests from the veterinary medical community for access to human health-care facilities for reasons that are largely economical (e.g., costs of acquiring sophisticated diagnostic technology and complex medical instruments). If hospital guidelines allow treatment of animals, alternate veterinary resources (including veterinary hospitals, clinics, and universities) should be exhausted before using human health-care settings. Additionally, the hospital's public/media relations should be notified of the situation. The goal is to develop policies and procedures to proactively and positively discuss and

disclose this activity to the general public.

An infection-control risk assessment (ICRA) must be undertaken to evaluate the circumstances specific to providing care to animals in a human health-care facility. Individual hospital policies and guidelines should be reviewed before any animal treatment is considered in such facilities. Animals treated in human health-care facilities should be under the direct care and supervision of a licensed veterinarian; they also should be free of known infectious diseases, ectoparasites, and other external contaminants (e.g., soil, urine, and feces). Measures should be taken to avoid treating animals with a known or suspected zoonotic disease in a human health-care setting (e.g., lambs being treated for Q fever).

If human health-care facilities must be used for animal treatment or diagnostics, the following general infection-control actions are suggested: a) whenever possible, the use of ORs or other rooms used for invasive procedures should be avoided [e.g., cardiac catheterization labs and invasive nuclear medicine areas]; b) when all other space options are exhausted and use of the aforementioned rooms is unavoidable, the procedure should be scheduled late in the day as the last procedure for that particular area such that patients are not present in the department/unit/area; c) environmental surfaces should be thoroughly cleaned and disinfected using procedures discussed in the Environmental Services portion of this guideline after the animal is removed from the care area; d) sufficient time should be allowed for ACH to help prevent allergic reactions by human patients [Table B.1. in Appendix B]; e) only disposable equipment or equipment that can be thoroughly and easily cleaned, disinfected, or sterilized should be used; f) when medical or surgical instruments, especially those invasive instruments that are difficult to clean [e.g., endoscopes], are used on animals, these instruments should be reserved for future use only on animals; and g) standard precautions should be followed.

5. Research Animals in Health-Care Facilities

The risk of acquiring a zoonotic infection from research animals has decreased in recent years because many small laboratory animals (e.g., mice, rats, and rabbits) come from quality stock and have defined microbiologic profiles.¹³⁹² Larger animals (e.g., nonhuman primates) are still obtained frequently from the wild and may harbor pathogens transmissible to humans. Primates, in particular, benefit from vaccinations to protect their health during the research period provided the vaccination does not interfere with the study of the particular agent. Animals serving as models for human disease studies pose some risk for transmission of infection to laboratory or health-care workers from percutaneous or mucosal exposure. Exposures can occur either through a) direct contact with an infected animal or its body substances and secretions or b) indirect contact with infectious material on equipment, instruments, surfaces, or supplies.¹³⁹² Uncontained aerosols generated during laboratory procedures can also transmit infection.

Infection-control measures to prevent transmission of zoonotic infections from research animals are largely derived from the following basic laboratory safety principles: a) purchasing pathogen-free animals, b) quarantining incoming animals to detect any zoonotic pathogens, c) treating infected animals or removing them from the facility, d) vaccinating animal carriers and high-risk contacts if possible, e) using specialized containment caging or facilities, and f) using protective clothing and equipment [e.g., gloves, face shields, gowns, and masks].¹³⁹² An excellent resource for detailed discussion of these safety measures has been published.¹⁰¹³

The animal research unit within a health-care facility should be engineered to provide a) adequate containment of animals and pathogens; b) daily decontamination and transport of equipment and waste; c) proper ventilation and air filtration, which prevents recirculation of the air in the unit to other areas of the facility; and d) negative air pressure in the animal rooms relative to the corridors. To ensure

adequate security and containment, no through traffic to other areas of the health-care facility should flow through this unit; access should be restricted to animal-care staff, researchers, environmental services, maintenance, and security personnel.

Occupational health programs for animal-care staff, researchers, and maintenance staff should take into consideration the animals' natural pathogens and research pathogens. Components of such programs include a) prophylactic vaccines, b) TB skin testing when primates are used, c) baseline serums, and d) hearing and respiratory testing. Work practices, PPE, and engineering controls specific for each of the four animal biosafety levels have been published.^{1013, 1393} The facility's occupational or employee health clinic should be aware of the appropriate post-exposure procedures involving zoonoses and have available the appropriate post-exposure biologicals and medications.

Animal-research-area staff should also develop standard operating procedures for a) daily animal husbandry [e.g., protection of the employee while facilitating animal welfare]; b) pathogen containment and decontamination; c) management, cleaning, disinfecting and/or sterilizing equipment and instruments; and d) employee training for laboratory safety and safety procedures specific to animal research worksites.¹⁰¹³ The federal Animal Welfare Act of 1966 and its amendments serve as the regulatory basis for ensuring animal welfare in research.^{1394, 1395}

I. Regulated Medical Waste

1. Epidemiology

No epidemiologic evidence suggests that most of the solid- or liquid wastes from hospitals, other health-care facilities, or clinical/research laboratories is any more infective than residential waste. Several studies have compared the microbial load and the diversity of microorganisms in residential wastes and wastes obtained from a variety of health-care settings.¹³⁹⁹⁻¹⁴⁰² Although hospital wastes had a greater number of different bacterial species compared with residential waste, wastes from residences were more heavily contaminated.^{1397, 1398} Moreover, no epidemiologic evidence suggests that traditional waste-disposal practices of health-care facilities (whereby clinical and microbiological wastes were decontaminated on site before leaving the facility) have caused disease in either the health-care setting or the general community.^{1400, 1401} This statement excludes, however, sharps injuries sustained during or immediately after the delivery of patient care before the sharp is "discarded." Therefore, identifying wastes for which handling and disposal precautions are indicated is largely a matter of judgment about the relative risk of disease transmission, because no reasonable standards on which to base these determinations have been developed. Aesthetic and emotional considerations (originating during the early years of the HIV epidemic) have, however, figured into the development of treatment and disposal policies, particularly for pathology and anatomy wastes and sharps.¹⁴⁰²⁻¹⁴⁰⁵ Public concerns have resulted in the promulgation of federal, state, and local rules and regulations regarding medical waste management and disposal.¹⁴⁰⁶⁻¹⁴¹⁴

2. Categories of Medical Waste

Precisely defining medical waste on the basis of quantity and type of etiologic agents present is virtually impossible. The most practical approach to medical waste management is to identify wastes that represent a sufficient potential risk of causing infection during handling and disposal and for which some precautions likely are prudent.² Health-care facility medical wastes targeted for handling and disposal precautions include microbiology laboratory waste (e.g., microbiologic cultures and stocks of microorganisms), pathology and anatomy waste, blood specimens from clinics and laboratories, blood

products, and other body-fluid specimens.² Moreover, the risk of either injury or infection from certain sharp items (e.g., needles and scalpel blades) contaminated with blood also must be considered. Although any item that has had contact with blood, exudates, or secretions may be potentially infective, treating all such waste as infective is neither practical nor necessary. Federal, state, and local guidelines and regulations specify the categories of medical waste that are subject to regulation and outline the requirements associated with treatment and disposal. The categorization of these wastes has generated the term “regulated medical waste.” This term emphasizes the role of regulation in defining the actual material and as an alternative to “infectious waste,” given the lack of evidence of this type of waste’s infectivity. State regulations also address the degree or amount of contamination (e.g., blood-soaked gauze) that defines the discarded item as a regulated medical waste. The EPA’s *Manual for Infectious Waste Management* identifies and categorizes other specific types of waste generated in health-care facilities with research laboratories that also require handling precautions.¹⁴⁰⁶

3. Management of Regulated Medical Waste in Health-Care Facilities

Medical wastes require careful disposal and containment before collection and consolidation for treatment. OSHA has dictated initial measures for discarding regulated medical-waste items. These measures are designed to protect the workers who generate medical wastes and who manage the wastes from point of generation to disposal.⁹⁶⁷ A single, leak-resistant biohazard bag is usually adequate for containment of regulated medical wastes, provided the bag is sturdy and the waste can be discarded without contaminating the bag’s exterior. The contamination or puncturing of the bag requires placement into a second biohazard bag. All bags should be securely closed for disposal. Puncture-resistant containers located at the point of use (e.g., sharps containers) are used as containment for discarded slides or tubes with small amounts of blood, scalpel blades, needles and syringes, and unused sterile sharps.⁹⁶⁷ To prevent needlestick injuries, needles and other contaminated sharps should not be recapped, purposefully bent, or broken by hand. CDC has published general guidelines for handling sharps.^{6, 1415} Health-care facilities may need additional precautions to prevent the production of aerosols during the handling of blood-contaminated items for certain rare diseases or conditions (e.g., Lassa fever and Ebola virus infection).²⁰³

Transporting and storing regulated medical wastes within the health-care facility prior to terminal treatment is often necessary. Both federal and state regulations address the safe transport and storage of on- and off-site regulated medical wastes.^{1406–1408} Health-care facilities are instructed to dispose medical wastes regularly to avoid accumulation. Medical wastes requiring storage should be kept in labeled, leak-proof, puncture-resistant containers under conditions that minimize or prevent foul odors. The storage area should be well ventilated and be inaccessible to pests. Any facility that generates regulated medical wastes should have a regulated medical waste management plan to ensure health and environmental safety as per federal, state, and local regulations.

4. Treatment of Regulated Medical Waste

Regulated medical wastes are treated or decontaminated to reduce the microbial load in or on the waste and to render the by-products safe for further handling and disposal. From a microbiologic standpoint, waste need not be rendered “sterile” because the treated waste will not be deposited in a sterile site. In addition, waste need not be subjected to the same reprocessing standards as are surgical instruments. Historically, treatment methods involved steam-sterilization (i.e., autoclaving), incineration, or interment (for anatomy wastes). Alternative treatment methods developed in recent years include chemical disinfection, grinding/shredding/disinfection methods, energy-based technologies (e.g., microwave or radiowave treatments), and disinfection/encapsulation methods.¹⁴⁰⁹ State medical waste regulations specify appropriate treatment methods for each category of regulated medical waste.

The recommendations in this guideline for Ebola Virus Disease has been superseded by CDC’s Infection Prevention and Control Recommendations for Hospitalized Patients with Known or Suspected Ebola Virus Disease in U.S. Hospitals and by CDC’s Interim Guidance for Environmental Infection Control in Hospitals for Ebola Virus issued on August 1, 2014. [Click here for current information on how Ebola virus is transmitted.](#)

Of all the categories comprising regulated medical waste, microbiologic wastes (e.g., untreated cultures, stocks, and amplified microbial populations) pose the greatest potential for infectious disease transmission, and sharps pose the greatest risk for injuries. Untreated stocks and cultures of microorganisms are subsets of the clinical laboratory or microbiologic waste stream. If the microorganism must be grown and amplified in culture to high concentration to permit work with the specimen, this item should be considered for on-site decontamination, preferably within the laboratory unit. Historically, this was accomplished effectively by either autoclaving (steam sterilization) or incineration. If steam sterilization in the health-care facility is used for waste treatment, exposure of the waste for up to 90 minutes at 250°F (121°C) in an autoclave (depending on the size of the load and type container) may be necessary to ensure an adequate decontamination cycle.^{1416–1418} After steam sterilization, the residue can be safely handled and discarded with all other nonhazardous solid waste in accordance with state solid-waste disposal regulations. On-site incineration is another treatment option for microbiologic, pathologic, and anatomic waste, provided the incinerator is engineered to burn these wastes completely and stay within EPA emissions standards.¹⁴¹⁰ Improper incineration of waste with high moisture and low energy content (e.g., pathology waste) can lead to emission problems. State medical-waste regulatory programs identify acceptable methods for inactivating amplified stocks and cultures of microorganisms, some of which may employ technology rather than steam sterilization or incineration.

Concerns have been raised about the ability of modern health-care facilities to inactivate microbiologic wastes on-site, given that many of these institutions have decommissioned their laboratory autoclaves. Current laboratory guidelines for working with infectious microorganisms at biosafety level (BSL) 3 recommend that all laboratory waste be decontaminated before disposal by an approved method, preferably within the laboratory.¹⁰¹³ These same guidelines recommend that all materials removed from a BSL 4 laboratory (unless they are biological materials that are to remain viable) are to be decontaminated before they leave the laboratory.¹⁰¹³ Recent federal regulations for laboratories that handle certain biological agents known as “select agents” (i.e., those that have the potential to pose a severe threat to public health and safety) require these agents (and those obtained from a clinical specimen intended for diagnostic, reference, or verification purposes) to be destroyed on-site before disposal.¹⁴¹² Although recommendations for laboratory waste disposal from BSL 1 or 2 laboratories (e.g., most health-care clinical and diagnostic laboratories) allow for these materials to be decontaminated off-site before disposal, on-site decontamination by a known effective method is preferred to reduce the potential of exposure during the handling of infectious material.

A recent outbreak of TB among workers in a regional medical-waste treatment facility in the United States demonstrated the hazards associated with aerosolized microbiologic wastes.^{1419, 1420} The facility received diagnostic cultures of *Mycobacterium tuberculosis* from several different health-care facilities before these cultures were chemically disinfected; this facility treated this waste with a grinding/shredding process that generated aerosols from the material.^{1419, 1420} Several operational deficiencies facilitated the release of aerosols and exposed workers to airborne *M. tuberculosis*. Among the suggested control measures was that health-care facilities perform on-site decontamination of laboratory waste containing live cultures of microorganisms before release of the waste to a waste management company.^{1419, 1420} This measure is supported by recommendations found in the CDC/NIH guideline for laboratory workers.¹⁰¹³ This outbreak demonstrates the need to avoid the use of any medical-waste treatment method or technology that can aerosolize pathogens from live cultures and stocks (especially those of airborne microorganisms) unless aerosols can be effectively contained and workers can be equipped with proper PPE.^{1419–1421} Safe laboratory practices, including those addressing waste management, have been published.^{1013, 1422}

In an era when local, state, and federal health-care facilities and laboratories are developing bioterrorism

response strategies and capabilities, the need to reinstate in-laboratory capacity to destroy cultures and stocks of microorganisms becomes a relevant issue.¹⁴²³ Recent federal regulations require health-care facility laboratories to maintain the capability of destroying discarded cultures and stocks on-site if these laboratories isolate from a clinical specimen any microorganism or toxin identified as a “select agent” from a clinical specimen (Table 27).^{1412, 1413} As an alternative, isolated cultures of select agents can be transferred to a facility registered to accept these agents in accordance with federal regulations.¹⁴¹² State medical waste regulations can, however, complicate or completely prevent this transfer if these cultures are determined to be medical waste, because most states regulate the inter-facility transfer of untreated medical wastes.

Table 27. Microorganisms and biologicals identified as select agents*+

HHS Non-overlap select agents and toxins (42 CFR Part 73 §73.4)	
Viruses	Crimean-Congo hemorrhagic fever virus; Ebola viruses; Cercopithecine herpesvirus 1 (herpes B virus); Lassa fever virus; Marburg virus; monkeypox virus; South American hemorrhagic fever viruses (Junin, Machupo, Sabia, Flexal, Guanarito); tick-borne encephalitis complex (flavi) viruses (Central European tick-borne encephalitis, Far Eastern tick-borne encephalitis [Russian spring and summer encephalitis, Kyasnaur Forest disease, Omsk hemorrhagic fever]); variola major virus (smallpox virus); and variola minor virus (alastrim)
Exclusions¶	Vaccine strain of Junin virus (Candid. #1)
Bacteria	<i>Rickettsia prowazekii</i> , <i>R. rickettsii</i> , <i>Yersinia pestis</i>
Fungi	<i>Coccidioides posadasii</i>
Toxins	Abrin; conotoxins; diacetoxyscirpenol; ricin; saxitoxin; Shiga-like ribosome inactivating proteins; tetrodotoxin
Exclusions¶	The following toxins (in purified form or in combinations of pure and impure forms) if the aggregate amount under the control of a principal investigator does not, at any time, exceed the amount specified: 100 mg of abrin; 100 mg of conotoxins; 1,000 mg of diacetoxyscirpenol; 100 mg of ricin; 100 mg of saxitoxin; 100 mg of Shiga-like ribosome inactivating proteins; or 100 mg of tetrodotoxin
Genetic elements, recombinant nucleic acids, and recombinant organisms¶	<ul style="list-style-type: none"> • Select agent viral nucleic acids (synthetic or naturally-derived, contiguous or fragmented, in host chromosomes or in expression vectors) that can encode infectious and/or replication competent forms of any of the select agent viruses; • Nucleic acids (synthetic or naturally-derived) that encode for the functional form(s) of any of the toxins listed in this table if the nucleic acids: a) are in a vector or host chromosome; b) can be expressed <i>in vivo</i> or <i>in vitro</i>; or c) are in a vector or host chromosome and can be expressed <i>in vivo</i> or <i>in vitro</i>; • Viruses, bacteria, fungi, and toxins listed in this table that have been genetically modified.
High consequence livestock pathogens and toxins/select agents (overlap agents) (42 CFR Part 73 §73.5 and USDA regulation 9 CFR Part 121)	
Viruses	Eastern equine encephalitis virus; Nipah and Hendra complex viruses; Rift Valley fever virus; Venezuelan equine encephalitis virus
Exclusions¶	MP-12 vaccine strain of Rift Valley fever virus; TC-83 vaccine strain of Venezuelan equine encephalitis virus
Bacteria	<i>Bacillus anthracis</i> ; <i>Brucella abortus</i> , <i>B. melitensis</i> , <i>B. suis</i> ; <i>Burkholderia mallei</i> (formerly <i>Pseudomonas mallei</i>), <i>B. pseudomallei</i> (formerly <i>P. pseudomallei</i>); botulinum neurotoxin-producing species of <i>Clostridium</i> ; <i>Coxiella burnetii</i> ; <i>Francisella tularensis</i>
Fungi	<i>Coccidioides immitis</i>
Toxins	Botulinum neurotoxins; <i>Clostridium perfringens</i> epsilon toxin; Shigatoxin; staphylococcal enterotoxins; T-2 toxin
Exclusions¶	The following toxins (in purified form or in combinations of pure and impure forms) if the aggregate amount under the control of a principal investigator does not, at any time, exceed the amount specified: 0.5 mg of botulinum neurotoxins; 100 mg of <i>Clostridium perfringens</i> epsilon toxin; 100 mg of Shigatoxin; 5 mg of staphylococcal enterotoxins; or 1,000 mg of T-2 toxin

High consequence livestock pathogens and toxins/select agents (overlap agents) (42 CFR Part 73 §73.5 and USDA regulation 9 CFR Part 121) (continued)	
Genetic elements, recombinant nucleic acids, and recombinant organisms¶	<ul style="list-style-type: none"> • Select agent viral nuclei acids (synthetic or naturally derived, contiguous or fragmented, in host chromosomes or in expression vectors) that can encode infectious and/or replication competent forms of any of the select agent viruses; • Nucleic acids (synthetic or naturally derived) that encode for the functional form(s) of any of the toxins listed in this table if the nucleic acids: a) are in a vector or host chromosome; b) can be expressed <i>in vivo</i> or <i>in vitro</i>; or c) are in a vector or host chromosome and can be expressed <i>in vivo</i> or <i>in vitro</i>; • Viruses, bacteria, fungi, and toxins listed in this table that have been genetically modified

* Material in this table is compiled from references 1412, 1413, and 1424. Reference 1424 also contains lists of select agents that include plant pathogens and pathogens affecting livestock.

+ 42 CFR 73 §§73.4 and 73.5 do not include any select agent or toxin that is in its naturally-occurring environment, provided it has not been intentionally introduced, cultivated, collected, or otherwise extracted from its natural source. These sections also do not include non-viable select agent organisms or nonfunctional toxins. This list of select agents is current as of 3 October 2003 and is subject to change pending the final adoption of 42 CFR Part 73.

¶ These table entries are listed in reference 1412 and 1413, but were not included in reference 1424.

5. Discharging Blood, Fluids to Sanitary Sewers or Septic Tanks

The contents of all vessels that contain more than a few milliliters of blood remaining after laboratory procedures, suction fluids, or bulk blood can either be inactivated in accordance with state-approved treatment technologies or carefully poured down a utility sink drain or toilet.¹⁴¹⁴ State regulations may dictate the maximum volume allowable for discharge of blood/body fluids to the sanitary sewer. No evidence indicates that bloodborne diseases have been transmitted from contact with raw or treated sewage. Many bloodborne pathogens, particularly bloodborne viruses, are not stable in the environment for long periods of time;^{1425, 1426} therefore, the discharge of small quantities of blood and other body fluids to the sanitary sewer is considered a safe method of disposing of these waste materials.¹⁴¹⁴ The following factors increase the likelihood that bloodborne pathogens will be inactivated in the disposal process: a) dilution of the discharged materials with water; b) inactivation of pathogens resulting from exposure to cleaning chemicals, disinfectants, and other chemicals in raw sewage; and c) effectiveness of sewage treatment in inactivating any residual bloodborne pathogens that reach the treatment facility. Small amounts of blood and other body fluids should not affect the functioning of a municipal sewer system. However, large quantities of these fluids, with their high protein content, might interfere with the biological oxygen demand (BOD) of the system. Local municipal sewage treatment restrictions may dictate that an alternative method of bulk fluid disposal be selected. State regulations may dictate what quantity constitutes a small amount of blood or body fluids.

Although concerns have been raised about the discharge of blood and other body fluids to a septic tank system, no evidence suggests that septic tanks have transmitted bloodborne infections. A properly functioning septic system is adequate for inactivating bloodborne pathogens. System manufacturers' instructions specify what materials may be discharged to the septic tank without jeopardizing its proper operation.

6. Medical Waste and CJD

Concerns also have been raised about the need for special handling and treatment procedures for wastes generated during the care of patients with CJD or other transmissible spongiform encephalopathies (TSEs). Prions, the agents that cause TSEs, have significant resistance to inactivation by a variety of physical, chemical, or gaseous methods.¹⁴²⁷ No epidemiologic evidence, however, links acquisition of CJD with medical-waste disposal practices. Although handling neurologic tissue for pathologic examination and autopsy materials with care, using barrier precautions, and following specific

procedures for the autopsy are prudent measures,¹¹⁹⁷ employing extraordinary measures once the materials are discarded is unnecessary. Regulated medical wastes generated during the care of the CJD patient can be managed using the same strategies as wastes generated during the care of other patients. After decontamination, these wastes may then be disposed in a sanitary landfill or discharged to the sanitary sewer, as appropriate.

Part II. Recommendations for Environmental Infection Control in Health-Care Facilities

A. Rationale for Recommendations

As in previous CDC guidelines, each recommendation is categorized on the basis of existing scientific data, theoretic rationale, applicability, and possible economic benefit. The recommendations are evidence-based wherever possible. However, certain recommendations are derived from empiric infection-control or engineering principles, theoretic rationale, or from experience gained from events that cannot be readily studied (e.g., floods).

The HICPAC system for categorizing recommendations has been modified to include a category for engineering standards and actions required by state or federal regulations. Guidelines and standards published by the American Institute of Architects (AIA), American Society of Heating, Refrigeration, and Air-Conditioning Engineers (ASHRAE), and the Association for the Advancement in Medical Instrumentation (AAMI) form the basis of certain recommendations. These standards reflect a consensus of expert opinions and extensive consultation with agencies of the U.S. Department of Health and Human Services. Compliance with these standards is usually voluntary. However, state and federal governments often adopt these standards as regulations. For example, the standards from AIA regarding construction and design of new or renovated health-care facilities, have been adopted by reference by >40 states. Certain recommendations have two category ratings (e.g., Categories IA and IC or Categories IB and IC), indicating the recommendation is evidence-based as well as a standard or regulation.

B. Rating Categories

Recommendations are rated according to the following categories:

- **Category IA.** Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiologic studies.
- **Category IB.** Strongly recommended for implementation and supported by certain experimental, clinical, or epidemiologic studies and a strong theoretical rationale.
- **Category IC.** Required by state or federal regulation, or representing an established association standard. (Note: Abbreviations for governing agencies and regulatory citations are listed, where appropriate. Recommendations from regulations adopted at state levels are also noted. Recommendations from AIA guidelines cite the appropriate sections of the standard).
- **Category II.** Suggested for implementation and supported by suggestive clinical or epidemiologic studies, or a theoretical rationale.
- **Unresolved Issue.** No recommendation is offered. No consensus or insufficient evidence exists regarding efficacy.

C. Recommendations—Air

I. Air-Handling Systems in Health-Care Facilities

- A. Use AIA guidelines as minimum standards where state or local regulations are not in place for design and construction of ventilation systems in new or renovated health-care facilities. Ensure that existing structures continue to meet the specifications in effect at the time of construction.¹²⁰ **Category IC** (AIA: 1.1.A, 5.4)
- B. Monitor ventilation systems in accordance with engineers' and manufacturers' recommendations to ensure preventive engineering, optimal performance for removal of particulates, and elimination of excess moisture.^{18, 35, 106, 120, 220, 222, 333, 336} **Category IB, IC** (AIA: 7.2, 7.31.D, 8.31.D, 9.31.D, 10.31.D, 11.31.D, EPA guidance)
 1. Ensure that heating, ventilation, air conditioning (HVAC) filters are properly installed and maintained to prevent air leakages and dust overloads.^{17, 18, 106, 222} **Category IB**
 2. Monitor areas with special ventilation requirements (e.g., AII or PE) for ACH, filtration, and pressure differentials.^{21, 120, 249, 250, 273–275, 277, 333–344} **Category IB, IC** (AIA: 7.2.C7, 7.2.D6)
 - a. Develop and implement a maintenance schedule for ACH, pressure differentials, and filtration efficiencies using facility-specific data as part of the multidisciplinary risk assessment. Take into account the age and reliability of the system.
 - b. Document these parameters, especially the pressure differentials.
 3. Engineer humidity controls into the HVAC system and monitor the controls to ensure proper moisture removal.¹²⁰ **Category IC** (AIA: 7.31.D9)
 - a. Locate duct humidifiers upstream from the final filters.
 - b. Incorporate a water-removal mechanism into the system.
 - c. Locate all duct takeoffs sufficiently down-stream from the humidifier so that moisture is completely absorbed.
 4. Incorporate steam humidifiers, if possible, to reduce potential for microbial proliferation within the system, and avoid use of cool mist humidifiers. **Category II**
 5. Ensure that air intakes and exhaust outlets are located properly in construction of new facilities and renovation of existing facilities.^{3, 120} **Category IC** (AIA: 7.31.D3, 8.31.D3, 9.31.D3, 10.31.D3, 11.31.D3)
 - a. Locate exhaust outlets >25 ft. from air-intake systems.
 - b. Locate outdoor air intakes ≥6 ft. above ground or ≥3 ft. above roof level.
 - c. Locate exhaust outlets from contaminated areas above roof level to minimize recirculation of exhausted air.
 6. Maintain air intakes and inspect filters periodically to ensure proper operation.^{3, 120, 249, 250, 273–275, 277} **Category IC** (AIA: 7.31.D8)
 7. Bag dust-filled filters immediately upon removal to prevent dispersion of dust and fungal spores during transport within the facility.^{106, 221} **Category IB**
 - a. Seal or close the bag containing the discarded filter.
 - b. Discard spent filters as regular solid waste, regardless of the area from which they were removed.²²¹
 8. Remove bird roosts and nests near air intakes to prevent mites and fungal spores from entering the ventilation system.^{3, 98, 119} **Category IB**
 9. Prevent dust accumulation by cleaning air-duct grilles in accordance with facility-specific procedures and schedules when rooms are not occupied by patients.^{21, 120, 249, 250, 273–275, 277} **Category IC, II** (AIA: 7.31.D10)

10. Periodically measure output to monitor system function; clean ventilation ducts as part of routine HVAC maintenance to ensure optimum performance.^{120, 263, 264}
Category II (AIA: 7.31.D10)
- C. Use portable, industrial-grade HEPA filter units capable of filtration rates in the range of 300–800 ft³/min. to augment removal of respirable particles as needed.²¹⁹ **Category II**
 1. Select portable HEPA filters that can recirculate all or nearly all of the room air and provide the equivalent of ≥ 12 ACH.⁴ **Category II**
 2. Portable HEPA filter units previously placed in construction zones can be used later in patient-care areas, provided all internal and external surfaces are cleaned, and the filter's performance verified by appropriate particle testing. **Category II**
 3. Situate portable HEPA units with the advice of facility engineers to ensure that all room air is filtered.⁴ **Category II**
 4. Ensure that fresh-air requirements for the area are met.^{214, 219} **Category II**
- D. Follow appropriate procedures for use of areas with through-the-wall ventilation units.¹²⁰
Category IC (AIA: 8.31.D1, 8.31.D8, 9.31.D23, 10.31.D18, 11.31.D15)
 1. Do not use such areas as PE rooms.¹²⁰ **Category IC** (AIA: 7.2.D3)
 2. Do not use a room with a through-the-wall ventilation unit as an AII room unless it can be demonstrated that all required AII engineering controls required are met.^{4, 120}
Category IC (AIA: 7.2.C3)
- E. Conduct an infection-control risk assessment (ICRA) and provide an adequate number of AII and PE rooms (if required) or other areas to meet the needs of the patient population.^{4, 6, 9, 18, 19, 69, 94, 120, 142, 331–334, 336–338} **Category IA, IC** (AIA: 7.2.C, 7.2.D)
- F. When UVGI is used as a supplemental engineering control, install fixtures 1) on the wall near the ceiling or suspended from the ceiling as an upper air unit; 2) in the air-return duct of an AII room; or 3) in designated enclosed areas or booths for sputum induction.⁴
Category II
- G. Seal windows in buildings with centralized HVAC systems and especially with PE areas.^{35, 111, 120}
Category IB, IC (AIA: 7.2.D3)
- H. Keep emergency doors and exits from PE rooms closed except during an emergency; equip emergency doors and exits with alarms. **Category II**
- I. Develop a contingency plan for backup capacity in the event of a general power failure.⁷¹³
Category IC (Joint Commission on Accreditation of Healthcare Organizations [JCAHO]: Environment of Care [EC] 1.4)
 1. Emphasize restoration of proper air quality and ventilation conditions in AII rooms, PE rooms, operating rooms, emergency departments, and intensive care units.^{120, 713}
Category IC (AIA: 1.5.A1; JCAHO: EC 1.4)
 2. Deploy infection-control procedures to protect occupants until power and systems functions are restored.^{6, 120, 713} **Category IC** (AIA: 5.1, 5.2; JCAHO: EC 1.4)
- J. Do not shut down HVAC systems in patient-care areas except for maintenance, repair, testing of emergency backup capacity, or new construction.^{120, 206} **Category IB, IC** (AIA: 5.1, 5.2.B, C)
 1. Coordinate HVAC system maintenance with infection-control staff to allow for relocation of immunocompromised patients if necessary.¹²⁰ **Category IC** (AIA: 5.1, 5.2)
 2. Provide backup emergency power and air-handling and pressurization systems to maintain filtration, constant ACH, and pressure differentials in PE rooms, AII rooms, operating rooms, and other critical-care areas.^{9, 120, 278} **Category IC** (AIA: 1.5, 5.1, 5.2)
 3. For areas not served by installed emergency ventilation and backup systems, use portable units and monitor ventilation parameters and patients in those areas.²¹⁹
Category II
 4. Coordinate system startups with infection-control staff to protect patients in PE rooms from bursts of fungal spores.^{9, 35, 120, 278} **Category IC** (AIA: 5.1, 5.2)

5. Allow sufficient time for ACH to clean the air once the system is operational (Appendix B, Table B.1).^{4, 120} **Category IC** (AIA: 5.1, 5.2)
- K. HVAC systems serving offices and administration areas may be shut down for energy conservation purposes, but the shutdown must not alter or adversely affect pressure differentials maintained in laboratories or critical-care areas with specific ventilation requirements (i.e., PE rooms, AII rooms, operating rooms). **Category II**
- L. Whenever possible, avoid inactivating or shutting down the entire HVAC system at one time, especially in acute-care facilities. **Category II**
- M. Whenever feasible, design and install fixed backup ventilation systems for new or renovated construction for PE rooms, AII rooms, operating rooms, and other critical care areas identified by ICRA.¹²⁰ **Category IC** (AIA: 1.5.A1)

II. Construction, Renovation, Remediation, Repair, and Demolition

- A. Establish a multidisciplinary team that includes infection-control staff to coordinate demolition, construction, and renovation projects and consider proactive preventive measures at the inception; produce and maintain summary statements of the team's activities.^{17, 19, 20, 97, 109, 120, 249, 250, 273–277} **Category IB, IC** (AIA: 5.1)
- B. Educate both the construction team and the health-care staff in immunocompromised patient-care areas regarding the airborne infection risks associated with construction projects, dispersal of fungal spores during such activities, and methods to control the dissemination of fungal spores.^{3, 249, 250, 273–277, 1428–1432} **Category IB**
- C. Incorporate mandatory adherence agreements for infection control into construction contracts, with penalties for noncompliance and mechanisms to ensure timely correction of problems.^{3, 120, 249, 273–277} **Category IC** (AIA: 5.1)
- D. Establish and maintain surveillance for airborne environmental disease (e.g., aspergillosis) as appropriate during construction, renovation, repair, and demolition activities to ensure the health and safety of immunocompromised patients.^{3, 64, 65, 79} **Category IB**
 1. Using active surveillance, monitor for airborne fungal infections in immunocompromised patients.^{3, 9, 64, 65} **Category IB**
 2. Periodically review the facility's microbiologic, histopathologic, and postmortem data to identify additional cases.^{3, 9, 64, 65} **Category IB**
 3. If cases of aspergillosis or other health-care-associated airborne fungal infections occur, aggressively pursue the diagnosis with tissue biopsies and cultures as feasible.^{3, 64, 65, 79, 249, 273–277} **Category IB**
- E. Implement infection-control measures relevant to construction, renovation, maintenance, demolition, and repair.^{96, 97, 120, 276, 277} **Category IB, IC** (AIA: 5.1, 5.2)
 1. Before the project gets underway, perform an ICRA to define the scope of the project and the need for barrier measures.^{96, 97, 120, 249, 273–277} **Category IB, IC** (AIA: 5.1)
 - a. Determine if immunocompromised patients may be at risk for exposure to fungal spores from dust generated during the project.^{20, 109, 273–275, 277}
 - b. Develop a contingency plan to prevent such exposures.^{20, 109, 273–275, 277}
 2. Implement infection-control measures for external demolition and construction activities.^{50, 249, 273–277, 283} **Category IB**
 - a. Determine if the facility can operate temporarily on recirculated air; if feasible, seal off adjacent air intakes.
 - b. If this is not possible or practical, check the low-efficiency (roughing) filter banks frequently and replace as needed to avoid buildup of particulates.
 - c. Seal windows and reduce wherever possible other sources of outside air intrusion (e.g., open doors in stairwells and corridors), especially in PE areas.
 3. Avoid damaging the underground water distribution system (i.e., buried pipes) to prevent soil and dust contamination of the water.^{120, 305} **Category IB, IC** (AIA: 5.1)

4. Implement infection-control measures for internal construction activities.^{20, 49, 97, 120, 249, 273–277} **Category IB, IC** (AIA: 5.1, 5.2)
 - a. Construct barriers to prevent dust from construction areas from entering patient-care areas; ensure that barriers are impermeable to fungal spores and in compliance with local fire codes.^{20, 49, 97, 120, 284, 312, 713, 1431}
 - b. Block and seal off return air vents if rigid barriers are used for containment.^{120, 276, 277}
 - c. Implement dust control measures on surfaces and by diverting pedestrian traffic away from work zones.^{20, 49, 97, 120}
 - d. Relocate patients whose rooms are adjacent to work zones, depending upon their immune status, the scope of the project, the potential for generation of dust or water aerosols, and the methods used to control these aerosols.^{49, 120, 281}
5. Perform those engineering and work-site related infection-control measures as needed for internal construction, repairs, and renovations.^{20, 49, 97, 109, 120, 312} **Category IB, IC** (AIA: 5.1, 5.2)
 - a. Ensure proper operation of the air-handling system in the affected area after erection of barriers and before the room or area is set to negative pressure.^{49, 69, 276, 278} **Category IB**
 - b. Create and maintain negative air pressure in work zones adjacent to patient-care areas and ensure that required engineering controls are maintained.^{20, 49, 97, 109, 120, 312}
 - c. Monitor negative air flow inside rigid barriers.^{120, 281}
 - d. Monitor barriers and ensure the integrity of the construction barriers; repair gaps or breaks in barrier joints.^{120, 284, 307, 312}
 - e. Seal windows in work zones if practical; use window chutes for disposal of large pieces of debris as needed, but ensure that the negative pressure differential for the area is maintained.^{20, 120, 273}
 - f. Direct pedestrian traffic from construction zones away from patient-care areas to minimize the dispersion of dust.^{20, 49, 97, 109, 111, 120, 273–277}
 - g. Provide construction crews with 1) designated entrances, corridors, and elevators whenever practical; 2) essential services [e.g., toilet facilities], and convenience services [e.g., vending machines]; 3) protective clothing [e.g., coveralls, footwear, and headgear] for travel to patient-care areas; and 4) a space or anteroom for changing clothing and storing equipment.^{120, 249, 273–277}
 - h. Clean work zones and their entrances daily by 1) wet-wiping tools and tool carts before their removal from the work zone; 2) placing mats with tacky surfaces inside the entrance; and 3) covering debris and securing this covering before removing debris from the work zone.^{120, 249, 273–277}
 - i. In patient-care areas, for major repairs that include removal of ceiling tiles and disruption of the space above the false ceiling, use plastic sheets or prefabricated plastic units to contain dust; use a negative pressure system within this enclosure to remove dust; and either pass air through an industrial grade, portable HEPA filter capable of filtration rates ranging from 300–800 ft³/min., or exhaust air directly to the outside.^{49, 276, 277, 281, 309}
 - j. Upon completion of the project, clean the work zone according to facility procedures, and install barrier curtains to contain dust and debris before removal of rigid barriers.^{20, 97, 120, 249, 273–277}
 - k. Flush the water system to clear sediment from pipes to minimize waterborne microorganism proliferation.^{120, 305}
 - l. Restore appropriate ACH, humidity, and pressure differential; clean or replace air filters; dispose of spent filters.^{35, 106, 221, 278}

- F. Use airborne-particle sampling as a tool to evaluate barrier integrity.^{35, 100} **Category II**
- G. Commission the HVAC system for newly constructed health-care facilities and renovated spaces before occupancy and use, with emphasis on ensuring proper ventilation for operating rooms, AII rooms, and PE areas.^{100, 120, 288, 304} **Category IC** (AIA: 5.1; ASHRAE: 1-1996)
- H. **No recommendation is offered** on routine microbiologic air sampling before, during, or after construction or before or during occupancy of areas housing immunocompromised patients.^{17, 20, 49, 97, 109, 272, 1433} **Unresolved issue**
- I. If a case of health-care-acquired aspergillosis or other opportunistic environmental airborne fungal disease occurs during or immediately after construction, implement appropriate follow-up measures.^{20, 55, 62, 77, 94, 95} **Category IB**
1. Review pressure differential monitoring documentation to verify that pressure differentials in the construction zone and in PE rooms were appropriate for their settings.^{94, 95, 120} **Category IB, IC** (AIA: 5.1)
 2. Implement corrective engineering measures to restore proper pressure differentials as needed.^{94, 95, 120} **Category IB, IC** (AIA: 5.1)
 3. Conduct a prospective search for additional cases and intensify retrospective epidemiologic review of the hospital's medical and laboratory records.^{3, 20, 62, 63, 104} **Category IB**
 4. If there is no evidence of ongoing transmission, continue routine maintenance in the area to prevent health-care-acquired fungal disease.^{3, 55} **Category IB**
- J. If there is epidemiologic evidence of ongoing transmission of fungal disease, conduct an environmental assessment to determine and eliminate the source.^{3, 96, 97, 109, 111, 115, 249, 273-277} **Category IB**
1. Collect environmental samples from potential sources of airborne fungal spores, preferably using a high-volume air sampler rather than settle plates.^{3, 18, 44, 48, 49, 97, 106, 111, 112, 115, 249, 254, 273-277, 292, 312} **Category IB**
 2. If either an environmental source of airborne fungi or an engineering problem with filtration or pressure differentials is identified, promptly perform corrective measures to eliminate the source and route of entry.^{96, 97} **Category IB**
 3. Use an EPA-registered anti-fungal biocide (e.g., copper-8-quinolinolate) for decontaminating structural materials.^{50, 277, 312, 329} **Category IB**
 4. If an environmental source of airborne fungi is not identified, review infection control measures, including engineering controls, to identify potential areas for correction or improvement.^{73, 117} **Category IB**
 5. If possible, perform molecular subtyping of *Aspergillus* spp. isolated from patients and the environment to establish strain identities.^{252, 293-296} **Category II**
- K. If air-supply systems to high-risk areas (e.g., PE rooms) are not optimal, use portable, industrial-grade HEPA filters on a temporary basis until rooms with optimal air-handling systems become available.^{3, 120, 273-277} **Category II**

III. Infection-Control and Ventilation Requirements for PE Rooms

- A. Minimize exposures of severely immunocompromised patients (e.g., solid organ transplant patients or allogeneic neutropenic patients) to activities that might cause aerosolization of fungal spores (e.g., vacuuming or disruption of ceiling tiles).^{9, 20, 109, 272} **Category IB**
- B. Minimize the length of time that immunocompromised patients in PE are outside their rooms for diagnostic procedures and other activities.^{9, 283} **Category IB**
- C. Provide respiratory protection for severely immunocompromised patients when they must leave PE for diagnostic studies and other activities; consult the most recent revision of CDC's *Guidelines for Prevention of Health-Care-Associated Pneumonia* for information regarding the appropriate type of respiratory protection.^{3, 9} **Category II**

- D. Incorporate ventilation engineering specifications and dust-controlling processes into the planning and construction of new PE units. **Category IB, IC**
1. Install central or point-of-use HEPA filters for supply (incoming) air.^{3, 18, 20, 44, 99–104, 120, 254, 316–318, 1432, 1434} **Category IB, IC** (AIA: 5.1, 5.2, 7.2.D)
 2. Ensure that rooms are well sealed by 1) properly constructing windows, doors, and intake and exhaust ports; 2) maintaining ceilings that are smooth and free of fissures, open joints, and crevices; 3) sealing walls above and below the ceiling, and 4) monitoring for leakage and making necessary repairs.^{3, 111, 120, 317, 318} **Category IB, IC** (AIA: 7.2.D3)
 3. Ventilate the room to maintain ≥ 12 ACH.^{3, 9, 120, 241, 317, 318} **Category IC** (AIA: 7.2.D)
 4. Locate air supply and exhaust grilles so that clean, filtered air enters from one side of the room, flows across the patient's bed, and exits from the opposite side of the room.^{3, 120, 317, 318} **Category IC** (AIA: 7.31.D1)
 5. Maintain positive room air pressure (≥ 2.5 Pa [0.01-inch water gauge]) in relation to the corridor.^{3, 35, 120, 317, 318} **Category IB, IC** (AIA: Table 7.2)
 6. Maintain airflow patterns and monitor these on a daily basis by using permanently installed visual means of detecting airflow in new or renovated construction, or using other visual methods (e.g., flutter strips, or smoke tubes) in existing PE units. Document the monitoring results.^{120, 273} **Category IC** (AIA: 7.2.D6)
 7. Install self-closing devices on all room exit doors in protective environments.¹²⁰ **Category IC** (AIA: 7.2.D4)
- E. Do not use laminar air flow systems in newly constructed PE rooms.^{316, 318} **Category II**
- F. Take measures to protect immunocompromised patients who would benefit from a PE room and who also have an airborne infectious disease (e.g., acute VZV infection or tuberculosis).
1. Ensure that the patient's room is designed to maintain positive pressure.
 2. Use an anteroom to ensure appropriate air balance relationships and provide independent exhaust of contaminated air to the outside, or place a HEPA filter in the exhaust duct if the return air must be recirculated.^{120, 317} **Category IC** (AIA: 7.2.D1, A7.2.D)
 3. If an anteroom is not available, place the patient in AII and use portable, industrial-grade HEPA filters to enhance filtration of spores in the room.²¹⁹ **Category II**
- G. Maintain backup ventilation equipment (e.g., portable units for fans or filters) for emergency provision of ventilation requirements for PE areas and take immediate steps to restore the fixed ventilation system function.^{9, 120, 278} **Category IC** (AIA: 5.1)

IV. Infection-Control and Ventilation Requirements for AII Rooms

- A. Incorporate certain specifications into the planning, and construction or renovation of AII units.^{4, 107, 120, 317, 318} **Category IB, IC**
1. Maintain continuous negative air pressure (2.5 Pa [0.01-inch water gauge]) in relation to the air pressure in the corridor; monitor air pressure periodically, preferably daily, with audible manometers or smoke tubes at the door (for existing AII rooms) or with a permanently installed visual monitoring mechanism. Document the results of monitoring.^{120, 317, 318} **Category IB, IC** (AIA: 7.2.C7, Table 7.2)
 2. Ensure that rooms are well-sealed by properly constructing windows, doors, and air-intake and exhaust ports; when monitoring indicates air leakage, locate the leak and make necessary repairs.^{120, 317, 318} **Category IB, IC** (AIA: 7.2.C3)
 3. Install self-closing devices on all AII room exit doors.¹²⁰ **Category IC** (AIA: 7.2.C4)
 4. Provide ventilation to ensure ≥ 12 ACH for renovated rooms and new rooms, and ≥ 6 ACH for existing AII rooms.^{4, 107, 120} **Category IC** (AIA: Table 7.2)

5. Direct exhaust air to the outside, away from air-intake and populated areas. If this is not practical, air from the room can be recirculated after passing through a HEPA filter.^{4, 120} **Category IC** (AIA: Table 7.2)
- B. Where supplemental engineering controls for air cleaning are indicated from a risk assessment of the AII area, install UVGI units in the exhaust air ducts of the HVAC system to supplement HEPA filtration or install UVGI fixtures on or near the ceiling to irradiate upper room air.⁴ **Category II**
- C. Implement environmental infection-control measures for persons with known or suspected airborne infectious diseases.
 1. Use AII rooms for patients with or suspected of having an airborne infection who also require cough-inducing procedures, or use an enclosed booth that is engineered to provide 1) ≥ 12 ACH; 2) air supply and exhaust rate sufficient to maintain a 2.5 Pa [0.01-inch water gauge] negative pressure difference with respect to all surrounding spaces with an exhaust rate of ≥ 50 ft³/min.; and 3) air exhausted directly outside away from air intakes and traffic or exhausted after HEPA filtration prior to recirculation.^{4, 120, 348–350} **Category IB, IC** (AIA: 7.15.E, 7.31.D23, 9.10, Table 7.2)
 2. Although airborne spread of viral hemorrhagic fever (VHF) has not been documented in a health-care setting, prudence dictates placing a VHF patient in an AII room, preferably with an anteroom to reduce the risk of occupational exposure to aerosolized infectious material in blood, vomitus, liquid stool, and respiratory secretions present in large amounts during the end stage of a patient's illness.^{202–204} **Category II**
 - a. If an anteroom is not available, use portable, industrial-grade HEPA filters in the patient's room to provide additional ACH equivalents for removing airborne particulates.
 - b. Ensure that health-care workers wear face shields or goggles with appropriate respirators when entering the rooms of VHF patients with prominent cough, vomiting, diarrhea, or hemorrhage.²⁰³
 3. Place smallpox patients in negative pressure rooms at the onset of their illness, preferably using a room with an anteroom if available.⁶ **Category II**
- D. **No recommendation is offered** regarding negative pressure or isolation rooms for patients with *Pneumocystis carinii* pneumonia.^{126, 131, 152} **Unresolved issue**
- E. Maintain back-up ventilation equipment (e.g., portable units for fans or filters) for emergency provision of ventilation requirements for AII rooms and take immediate steps to restore the fixed ventilation system function.^{4, 120, 278} **Category IC** (AIA: 5.1)

V. Infection-Control and Ventilation Requirements for Operating Rooms

- A. Implement environmental infection-control and ventilation measures for operating rooms.
 1. Maintain positive-pressure ventilation with respect to corridors and adjacent areas.^{7, 120, 356} **Category IB, IC** (AIA: Table 7.2)
 2. Maintain ≥ 15 ACH, of which ≥ 3 ACH should be fresh air.^{120, 357, 358} **Category IC** (AIA: Table 7.2)
 3. Filter all recirculated and fresh air through the appropriate filters, providing 90% efficiency (dust-spot testing) at a minimum.^{120, 362} **Category IC** (AIA: Table 7.3)
 4. In rooms not engineered for horizontal laminar airflow, introduce air at the ceiling and exhaust air near the floor.^{120, 357, 359} **Category IC** (AIA: 7.31.D4)
 5. Do not use UV lights to prevent surgical-site infections.^{356, 364–370} **Category IB**
 6. Keep operating room doors closed except for the passage of equipment, personnel, and patients, and limit entry to essential personnel.^{351, 352} **Category IB**
- B. Follow precautionary procedures for TB patients who also require emergency surgery.^{4, 347, 371} **Category IB, IC**

The recommendations in this guideline for Ebola Virus Disease has been superseded by CDC's Infection Prevention and Control Recommendations for Hospitalized Patients with Known or Suspected Ebola Virus Disease in U.S. Hospitals and by CDC's Interim Guidance for Environmental Infection Control in Hospitals for Ebola Virus issued on August 1, 2014. [Click here for current information on how Ebola virus is transmitted.](#)

1. Use an N95 respirator approved by the National Institute for Occupational Safety and Health (NIOSH) without exhalation valves in the operating room.^{347, 372} **Category IC** (Occupational Safety and Health Administration [OSHA]; 29 Code of Federal Regulations [CFR] 1910.134,139)
 2. Intubate the patient in either the AII room or the operating room; if intubating the patient in the operating room, do not allow the doors to open until 99% of the airborne contaminants are removed (Appendix B, Table B.1).^{4, 358} **Category IB**
 3. When anesthetizing a patient with confirmed or suspected TB, place a bacterial filter between the anesthesia circuit and patient's airway to prevent contamination of anesthesia equipment or discharge of tubercle bacilli into the ambient air.^{371, 373}
Category IB
 4. Extubate and allow the patient to recover in an AII room.^{4, 358} **Category IB**
 5. If the patient has to be extubated in the operating room, allow adequate time for ACH to clean 99% of airborne particles from the air (Appendix B, Table B.1) because extubation is a cough-producing procedure.^{4, 358} **Category IB**
- C. Use portable, industrial-grade HEPA filters temporarily for supplemental air cleaning during intubation and extubation for infectious TB patients who require surgery.^{4, 219, 358}
Category II
1. Position the units appropriately so that all room air passes through the filter; obtain engineering consultation to determine the appropriate placement of the unit.⁴
Category II
 2. Switch the portable unit off during the surgical procedure. **Category II**
 3. Provide fresh air as per ventilation standards for operating rooms; portable units do not meet the requirements for the number of fresh ACH.^{120, 215, 219} **Category II**
- D. If possible, schedule infectious TB patients as the last surgical cases of the day to maximize the time available for removal of airborne contamination. **Category II**
- E. **No recommendation is offered** for performing orthopedic implant operations in rooms supplied with laminar airflow.^{362, 364} **Unresolved issue**
- F. Maintain backup ventilation equipment (e.g., portable units for fans or filters) for emergency provision of ventilation requirements for operating rooms, and take immediate steps to restore the fixed ventilation system function.^{68, 120, 278, 372} **Category IB, IC** (AIA: 5.1)

VI. Other Potential Infectious Aerosol Hazards in Health-Care Facilities

- A. In settings where surgical lasers are used, wear appropriate personal protective equipment, including N95 or N100 respirators, to minimize exposure to laser plumes.^{347, 378, 389}
Category IC (OSHA; 29 CFR 1910.134,139)
- B. Use central wall suction units with in-line filters to evacuate minimal laser plumes.^{378, 382, 386, 389}
Category II
- C. Use a mechanical smoke evacuation system with a high-efficiency filter to manage the generation of large amounts of laser plume, when ablating tissue infected with human papilloma virus (HPV) or performing procedures on a patient with extrapulmonary TB.^{4, 382, 389-392}
Category II

D. Recommendations—Water

I. Controlling the Spread of Waterborne Microorganisms

- A. Practice hand hygiene to prevent the hand transfer of waterborne pathogens, and use barrier precautions (e.g., gloves) as defined by other guidelines.^{6, 464, 577, 586, 592, 1364} **Category IA**

- B. Eliminate contaminated water or fluid environmental reservoirs (e.g., in equipment or solutions) wherever possible.^{464, 465} **Category IB**
- C. Clean and disinfect sinks and wash basins on a regular basis by using an EPA-registered product as set by facility policies. **Category II**
- D. Evaluate for possible environmental sources (e.g., potable water) of specimen contamination when waterborne microorganisms (e.g., NTM) of unlikely clinical importance are isolated from clinical cultures (e.g., specimens collected aseptically from sterile sites or, if post-procedural, colonization occurs after use of tap water in patient care).^{607, 610–612} **Category IB**
- E. Avoid placing decorative fountains and fish tanks in patient-care areas; ensure disinfection and fountain maintenance if decorative fountains are used in the public areas of the health-care facility.⁶⁶⁴ **Category IB**

II. Routine Prevention of Waterborne Microbial Contamination Within the Distribution System

- A. Maintain hot water temperature at the return at the highest temperature allowable by state regulations or codes, preferably $\geq 124^{\circ}\text{F}$ ($\geq 51^{\circ}\text{C}$), and maintain cold water temperature at $< 68^{\circ}\text{F}$ ($< 20^{\circ}\text{C}$).^{3, 661} **Category IC** (States; ASHRAE: 12:2000)
- B. If the hot water temperature can be maintained at $\geq 124^{\circ}\text{F}$ ($\geq 51^{\circ}\text{C}$), explore engineering options (e.g., install preset thermostatic valves in point-of-use fixtures) to help minimize the risk of scalding.⁶⁶¹ **Category II**
- C. When state regulations or codes do not allow hot water temperatures above the range of 105°F – 120°F (40.6°C – 49°C) for hospitals or 95°F – 110°F (35°C – 43.3°C) for nursing care facilities or when buildings cannot be retrofitted for thermostatic mixing valves, follow either of these alternative preventive measures to minimize the growth of *Legionella* spp. in water systems. **Category II**
 - 1. Periodically increase the hot water temperature to $\geq 150^{\circ}\text{F}$ ($\geq 66^{\circ}\text{C}$) at the point of use.⁶⁶¹ **Category II**
 - 2. Alternatively, chlorinate the water and then flush it through the system.^{661, 710, 711} **Category II**
- D. Maintain constant recirculation in hot-water distribution systems serving patient-care areas.¹²⁰ **Category IC** (AIA: 7.31.E.3)

III. Remediation Strategies for Distribution System Repair or Emergencies

- A. Whenever possible, disconnect the ice machine before planned water disruptions. **Category II**
- B. Prepare a contingency plan to estimate water demands for the entire facility in advance of significant water disruptions (i.e., those expected to result in extensive and heavy microbial or chemical contamination of the potable water), sewage intrusion, or flooding.^{713, 719} **Category IC** (JCAHO: EC 1.4)
- C. When a significant water disruption or an emergency occurs, adhere to any advisory to boil water issued by the municipal water utility.⁶⁴² **Category IB, IC** (Municipal order)
 - 1. Alert patients, families, staff, and visitors not to consume water from drinking fountains, ice, or drinks made from municipal tap water, while the advisory is in effect, unless the water has been disinfected (e.g., by bringing to a rolling boil for ≥ 1 minute).⁶⁴² **Category IB, IC** (Municipal order)
 - 2. After the advisory is lifted, run faucets and drinking fountains at full flow for ≥ 5 minutes, or use high-temperature water flushing or chlorination.^{642, 661} **Category IC, II** (Municipal order; ASHRAE 12:2000)
- D. Maintain a high level of surveillance for waterborne disease among patients after a boil water advisory is lifted. **Category II**

- E. Corrective decontamination of the hot water system might be necessary after a disruption in service or a cross-connection with sewer lines has occurred.
1. Decontaminate the system when the fewest occupants are present in the building (e.g., nights or weekends).^{3, 661} **Category IC** (ASHRAE: 12:2000)
 2. If using high-temperature decontamination, raise the hot-water temperature to 160°F–170°F (71°C–77°C) and maintain that level while progressively flushing each outlet around the system for ≥ 5 minutes.^{3, 661} **Category IC** (ASHRAE: 12:2000)
 3. If using chlorination, add enough chlorine, preferably overnight, to achieve a free chlorine residual of ≥ 2 mg/L (≥ 2 ppm) throughout the system.⁶⁶¹ **Category IC** (ASHRAE: 12:2000)
 - a. Flush each outlet until chlorine odor is detected.
 - b. Maintain the elevated chlorine concentration in the system for ≥ 2 hrs (but ≤ 24 hrs).
 4. Use a very thorough flushing of the water system instead of chlorination if a highly chlorine-resistant microorganism (e.g., *Cryptosporidium* spp.) is suspected as the water contaminant. **Category II**
- F. Flush and restart equipment and fixtures according to manufacturers' instructions. **Category II**
- G. Change the pretreatment filter and disinfect the dialysis water system with an EPA-registered product to prevent colonization of the reverse osmosis membrane and downstream microbial contamination.⁷²¹ **Category II**
- H. Run water softeners through a regeneration cycle to restore their capacity and function. **Category II**
- I. If the facility has a water-holding reservoir or water-storage tank, consult the facility engineer or local health department to determine whether this equipment needs to be drained, disinfected with an EPA-registered product, and refilled. **Category II**
- J. Implement facility management procedures to manage a sewage system failure or flooding (e.g., arranging with other health-care facilities for temporary transfer of patients or provision of services), and establish communications with the local municipal water utility and the local health department to ensure that advisories are received in a timely manner upon release.^{713, 719} **Category IC** (JCAHO: EC 1.4; Municipal order)
- K. Implement infection-control measures during sewage intrusion, flooding, or other water-related emergencies.
1. Relocate patients and clean or sterilize supplies from affected areas. **Category II**
 2. If hands are not visibly soiled or contaminated with proteinaceous material, include an alcohol-based hand rub in the hand hygiene process 1) before performing invasive procedures; 2) before and after each patient contact; and 3) whenever hand hygiene is indicated.¹³⁶⁴ **Category II**
 3. If hands are visibly soiled or contaminated with proteinaceous material, use soap and bottled water for handwashing.¹³⁶⁴ **Category II**
 4. If the potable water system is not affected by flooding or sewage contamination, process surgical instruments for sterilization according to standard procedures. **Category II**
 5. Contact the manufacturer of the automated endoscope reprocessor (AER) for specific instructions on the use of this equipment during a water advisory. **Category II**
- L. Remediate the facility after sewage intrusion, flooding, or other water-related emergencies.
1. Close off affected areas during cleanup procedures. **Category II**
 2. Ensure that the sewage system is fully functional before beginning remediation so contaminated solids and standing water can be removed. **Category II**

3. If hard-surface equipment, floors, and walls remain in good repair, ensure that these are dry within 72 hours; clean with detergent according to standard cleaning procedures. **Category II**
 4. Clean wood furniture and materials (if still in good repair); allow them to dry thoroughly before restoring varnish or other surface coatings. **Category II**
 5. Contain dust and debris during remediation and repair as outlined in air recommendations (Air: II G 4, 5). **Category II**
- M. Regardless of the original source of water damage (e.g., flooding versus water leaks from point-of-use fixtures or roofs), remove wet, absorbent structural items (e.g., carpeting, wallboard, and wallpaper) and cloth furnishings if they cannot be easily and thoroughly cleaned and dried within 72 hours (e.g., moisture content $\leq 20\%$ as determined by moisture meter readings); replace with new materials as soon as the underlying structure is declared by the facility engineer to be thoroughly dry.^{18, 266, 278, 1026} **Category IB**

IV. Additional Engineering Measures as Indicated by Epidemiologic Investigation for Controlling Waterborne, Health-Care–Associated Legionnaires Disease

- A. When using a pulse or one-time decontamination method, superheat the water by flushing each outlet for ≥ 5 minutes with water at 160°F – 170°F (71°C – 77°C) or hyperchlorinate the system by flushing all outlets for ≥ 5 minutes with water containing ≥ 2 mg/L (≥ 2 ppm) free residual chlorine using a chlorine-based product registered by the EPA for water treatment (e.g., sodium hypochlorite [chlorine bleach]).^{661, 711, 714, 724, 764, 766} **Category IB** (ASHRAE: 12:2000)
- B. After a pulse treatment, maintain both the heated water temperature at the return and the cold water temperature as per the recommendation (Water: IIA) wherever practical and permitted by state codes, or chlorinate heated water to achieve 1–2 mg/L (1–2 ppm) free residual chlorine at the tap using a chlorine-based product registered by the EPA for water treatment (e.g., sodium hypochlorite [bleach]).^{26, 437, 661, 709, 726, 727} **Category IC** (States; ASHRAE: 12:2000)
- C. Explore engineering or educational options (e.g., install preset thermostatic mixing valves in point-of-use fixtures or post warning signs at each outlet) to minimize the risk of scalding for patients, visitors, and staff. **Category II**
- D. **No recommendation is offered** for treating water in the facility’s distribution system with chlorine dioxide, heavy-metal ions (e.g., copper or silver), monochloramine, ozone, or UV light.^{728–746} **Unresolved issue**

V. General Infection-Control Strategies for Preventing Legionnaires Disease

- A. Conduct an infection-control risk assessment of the facility to determine if patients at risk or severely immunocompromised patients are present.^{3, 431, 432} **Category IB**
- B. Implement general strategies for detecting and preventing Legionnaires disease in facilities that do not provide care for severely immunocompromised patients (i.e., facilities that do not have HSCT or solid organ transplant programs).^{3, 431, 432} **Category IB**
 1. Establish a surveillance process to detect health-care–associated Legionnaires disease.^{3, 431, 432} **Category IB**
 2. Inform health-care personnel (e.g., infection control, physicians, patient-care staff, and engineering) regarding the potential for Legionnaires disease to occur and measures to prevent and control health-care–associated legionellosis.^{437, 759} **Category IB**
 3. Establish mechanisms to provide clinicians with laboratory tests (e.g., culture, urine antigen, direct fluorescence assay [DFA], and serology) for the diagnosis of Legionnaires disease.^{3, 431} **Category IB**

- C. Maintain a high index of suspicion for health-care-associated Legionnaires disease, and perform laboratory diagnostic tests for legionellosis on suspected cases, especially in patients at risk who do not require a PE for care (e.g., patients receiving systemic steroids; patients aged ≥ 65 years; or patients with chronic underlying disease [e.g., diabetes mellitus, congestive heart failure, or chronic obstructive lung disease]).^{3, 395, 417, 423–425, 432, 435, 437, 453}
Category IA
- D. Periodically review the availability and clinicians' use of laboratory diagnostic tests for Legionnaires disease in the facility; if clinicians' use of the tests on patients with diagnosed or suspected pneumonia is limited, implement measures (e.g., an educational campaign) to enhance clinicians' use of the test(s).⁴⁵³ **Category IB**
- E. If one case of laboratory-confirmed, health-care-associated Legionnaires disease is identified, or if two or more cases of laboratory-suspected, health-care-associated Legionnaires disease occur during a 6-month period, certain activities should be initiated.^{405, 408, 431, 453, 739, 759} **Category IB**
1. Report the cases to the state and local health departments where required. **Category IC** (States)
 2. If the facility does not treat severely immunocompromised patients, conduct an epidemiologic investigation, including retrospective review of microbiologic, serologic, and postmortem data to look for previously unidentified cases of health-care-associated Legionnaires disease, and begin intensive prospective surveillance for additional cases.^{3, 405, 408, 431, 453, 739, 759} **Category IB**
 3. If no evidence of continued health-care-associated transmission exists, continue intensive prospective surveillance for ≥ 2 months after the initiation of surveillance.^{3, 405, 408, 431, 453, 739, 759} **Category IB**
- F. If there is evidence of continued health-care-associated transmission (i.e., an outbreak), conduct an environmental assessment to determine the source of *Legionella* spp.^{403–410, 455} **Category IB**
1. Collect water samples from potential aerosolized water sources (Appendix C).¹²⁰⁹ **Category IB**
 2. Save and subtype isolates of *Legionella* spp. obtained from patients and the environment.^{403–410, 453, 763, 764} **Category IB**
 3. If a source is identified, promptly institute water system decontamination measures per recommendations (see Water IV).^{766, 767} **Category IB**
 4. If *Legionella* spp. are detected in ≥ 1 cultures (e.g., conducted at 2-week intervals during 3 months), reassess the control measures, modify them accordingly, and repeat the decontamination procedures; consider intensive use of techniques used for initial decontamination, or a combination of superheating and hyperchlorination.^{3, 767, 768} **Category IB**
- G. If an environmental source is not identified during a Legionnaires disease outbreak, continue surveillance for new cases for ≥ 2 months. Either defer decontamination pending identification of the source of *Legionella* spp., or proceed with decontamination of the hospital's water distribution system, with special attention to areas involved in the outbreak. **Category II**
- H. **No recommendation is offered** regarding routine culturing of water systems in health-care facilities that do not have patient-care areas (i.e., PE or transplant units) for persons at high risk for *Legionella* spp. infection.^{26, 453, 707, 709, 714, 747, 753} **Unresolved issue**
- I. **No recommendation is offered** regarding the removal of faucet aerators in areas for immunocompetent patients. **Unresolved issue**
- J. Keep adequate records of all infection-control measures and environmental test results for potable water systems. **Category II**

VI. Preventing Legionnaires Disease in Protective Environments and Transplant Units

- A. When implementing strategies for preventing Legionnaires disease among severely immunosuppressed patients housed in facilities with HSCT or solid-organ transplant programs, incorporate these specific surveillance and epidemiologic measures in addition to the steps previously outlined (Water: V and Appendix C).
1. Maintain a high index of suspicion for legionellosis in transplant patients even when environmental surveillance cultures do not yield legionellae.^{430, 431} **Category IB**
 2. If a case occurs in a severely immunocompromised patient, or if severely immunocompromised patients are present in high-risk areas of the hospital (e.g., PE or transplant units) and cases are identified elsewhere in the facility, conduct a combined epidemiologic and environmental investigation to determine the source of *Legionella* spp.^{431, 767} **Category IB**
- B. Implement culture strategies and potable water and fixture treatment measures in addition to those previously outlined (Water: V). **Category II**
1. Depending on state regulations on potable water temperature in public buildings,⁷²⁵ hospitals housing patients at risk for health-care-associated legionellosis should either maintain heated water with a minimum return temperature of $\geq 124^{\circ}\text{F}$ [$\geq 51^{\circ}\text{C}$] and cold water at $< 68^{\circ}\text{F}$ [$< 20^{\circ}\text{C}$], or chlorinate heated water to achieve 1–2 mg/L (1–2 ppm) of free residual chlorine at the tap.^{26, 441, 661, 709–711, 726, 727} **Category II**
 2. Periodic culturing for legionellae in potable water samples from HSCT or solid-organ transplant units can be performed as part of a comprehensive strategy to prevent Legionnaires disease in these units.^{9, 431, 710, 769} **Category II**
 3. **No recommendation is offered** regarding the optimal methodology (i.e., frequency or number of sites) for environmental surveillance cultures in HSCT or solid organ transplant units. **Unresolved issue**
 4. In areas with patients at risk, when *Legionella* spp. are not detectable in unit water, remove, clean, and disinfect shower heads and tap aerators monthly by using a chlorine-based, EPA-registered product. If an EPA-registered chlorine disinfectant is not available, use a chlorine bleach solution (500–615 ppm [1:100 v/v dilution]).^{661, 745} **Category II**
- C. If *Legionella* spp. are determined to be present in the water of a transplant unit, implement certain measures until *Legionella* spp. are no longer detected by culture.
1. Decontaminate the water supply as outlined previously (Water: IV).^{3, 9, 661, 766, 767} **Category IB**
 2. Do not use water from the faucets in patient-care rooms to avoid creating infectious aerosols.^{9, 412} **Category IB**
 3. Restrict severely immunocompromised patients from taking showers.^{9, 412} **Category IB**
 4. Use water that is not contaminated with *Legionella* spp. for HSCT patients' sponge baths.^{9, 412} **Category IB**
 5. Provide patients with sterile water for tooth brushing, drinking, and for flushing nasogastric tubing during legionellosis outbreaks.^{9, 412} **Category IB**
- D. Do not use large-volume room air humidifiers that create aerosols (e.g., by Venturi principle, ultrasound, or spinning disk) unless they are subjected to high-level disinfection and filled only with sterile water.^{3, 9, 402, 455} **Category IB**

VII. Cooling Towers and Evaporative Condensers

- A. When planning construction of new health-care facilities, locate cooling towers so that the drift is directed away from the air-intake system, and design the towers to minimize the volume of aerosol drift.^{404, 661, 786} **Category IC** (ASHRAE: 12:2000)

- B. Implement infection-control procedures for operational cooling towers.^{404, 661, 784}
Category IC (ASHRAE: 12:2000)
1. Install drift eliminators.^{404, 661, 784} **Category IC** (ASHRAE: 12:2000)
 2. Use an effective EPA-registered biocide on a regular basis.⁶⁶¹ **Category IC** (ASHRAE: 12:2000)
 3. Maintain towers according to manufacturers' recommendations, and keep detailed maintenance and infection control records, including environmental test results from legionellosis outbreak investigations.⁶⁶¹ **Category IC** (ASHRAE: 12:2000)
- C. If cooling towers or evaporative condensers are implicated in health-care-associated legionellosis, decontaminate the cooling-tower system.^{404, 405, 786, 787} **Category IB**

VIII. Dialysis Water Quality and Dialysate

- A. Adhere to current AAMI standards for quality assurance performance of devices and equipment used to treat, store, and distribute water in hemodialysis centers (both acute and maintenance [chronic] settings) and for the preparation of concentrates and dialysate.^{31, 32, 666-668, 789, 791, 800, 807, 809, 1454, 1455} **Category IA, IC** (AAMI: ANSI/AAMI RD5:1992, ANSI/AAMI RD 47:1993)
- B. **No recommendation is offered** regarding whether more stringent requirements for water quality should be imposed in hemofiltration and hemodiafiltration. **Unresolved issue**^{789, 791, 792, 834, 835}
- C. Conduct microbiological testing specific to water in dialysis settings. **Category IA, IC** (AAMI: ANSI/AAMI RD 5: 1992, ANSI/AAMI RD 47: 1993, ANSI/AAMI RD 62:2001)
1. Perform bacteriologic assays of water and dialysis fluids at least once a month and during outbreaks using standard quantitative methods.^{792, 834, 835} **Category IA, IC** (AAMI: ANSI/AAMI RD 62:2001)
 - a. Assay for heterotrophic, mesophilic bacteria (e.g., *Pseudomonas* spp).
 - b. Do not use nutrient-rich media (e.g., blood agar or chocolate agar).
 2. In conjunction with microbiological testing, perform endotoxin testing on product water used to reprocess dialyzers for multiple use.^{789, 791, 806, 811, 816, 829} **Category IA, IC** (AAMI: ANSI/AAMI RD 5:1992, ANSI/AAMI RD 47:1993)
 3. Ensure that water does not exceed the limits for microbial counts and endotoxin concentrations outlined in Table 18.^{789, 791, 800} **Category IA, IC** (AAMI: ANSI/AAMI RD 5:1992, ANSI/AAMI RD 47:1993)
- D. Disinfect water distribution systems in dialysis settings on a regular schedule. Monthly disinfection is recommended.^{666-668, 792, 800} **Category IA, IC** (AAMI: ANSI/AAMI RD62:2001)
- E. Whenever practical, design and engineer water systems in dialysis settings to avoid incorporating joints, dead-end pipes, and unused branches and taps that can harbor bacteria.^{666-668, 792, 800} **Category IA, IC** (AAMI: ANSI/AAMI RD62:2001)
- F. When storage tanks are used in dialysis systems, they should be routinely drained, disinfected with an EPA-registered product, and fitted with an ultrafilter or pyrogenic filter (membrane filter with a pore size sufficient to remove small particles and molecules ≥ 1 kilodalton) installed in the water line distal to the storage tank.⁷⁹² **Category IC** (AAMI: ANSI/AAMI RD62:2001)

IX. Ice Machines and Ice

- A. Do not handle ice directly by hand, and wash hands before obtaining ice. **Category II**
- B. Use a smooth-surface ice scoop to dispense ice.^{680, 863} **Category II**
1. Keep the ice scoop on a chain short enough the scoop cannot touch the floor, or keep the scoop on a clean, hard surface when not in use.^{680, 863} **Category II**
 2. Do not store the ice scoop in the ice bin. **Category II**
- C. Do not store pharmaceuticals or medical solutions on ice intended for consumption; use sterile ice to keep medical solutions cold, or use equipment specifically manufactured for this purpose.^{600, 863} **Category IB**

- D. Machines that dispense ice are preferred to those that require ice to be removed from bins or chests with a scoop.^{687, 869} **Category II**
- E. Limit access to ice-storage chests, and keep the container doors closed except when removing ice.⁸⁶³ **Category II**
- F. Clean, disinfect, and maintain ice-storage chests on a regular basis. **Category II**
 - 1. Follow the manufacturer's instructions for cleaning. **Category II**
 - 2. Use an EPA-registered disinfectant suitable for use on ice machines, dispensers, or storage chests in accordance with label instructions. **Category II**
 - 3. If instructions and EPA-registered disinfectants suitable for use on ice machines are not available, use a general cleaning/disinfecting regimen as outlined in Box 12.⁸⁶³ **Category II**
 - 4. Flush and clean the ice machines and dispensers if they have not been disconnected before anticipated lengthy water disruptions. **Category II**
- G. Install proper air gaps where the condensate lines meet the waste lines. **Category II**
- H. Conduct microbiologic sampling of ice, ice chests, and ice-making machines and dispensers where indicated during an epidemiologic investigation.^{861–863} **Category IB**

X. Hydrotherapy Tanks and Pools

- A. Drain and clean hydrotherapy equipment (e.g., Hubbard tanks, tubs, whirlpools, whirlpool spas, or birthing tanks) after each patient's use, and disinfect equipment surfaces and components by using an EPA-registered product in accordance with the manufacturer's instructions. **Category II**
- B. In the absence of an EPA-registered product for water treatment, add sodium hypochlorite to the water:
 - 1. Maintain a 15-ppm chlorine residual in the water of small hydrotherapy tanks, Hubbard tanks, and tubs.⁸⁸⁹ **Category II**
 - 2. Maintain a 2–5 ppm chlorine residual in the water of whirlpools and whirlpool spas.⁹⁰⁵ **Category II**
 - 3. If the pH of the municipal water is in the basic range (e.g., when chloramine is used as the primary drinking water disinfectant in the community), consult the facility engineer regarding the possible need to adjust the pH of the water to a more acid level before disinfection, to enhance the biocidal activity of chlorine.⁸⁹⁴ **Category II**
- C. Clean and disinfect hydrotherapy equipment after using tub liners. **Category II**
- D. Clean and disinfect inflatable tubs unless they are single-use equipment. **Category II**
- E. **No recommendation is offered** regarding the use of antiseptic chemicals (e.g., chloramine-T) in the water during hydrotherapy sessions. **Unresolved issue**
- F. Conduct a risk assessment of patients prior to their use of large hydrotherapy pools, deferring patients with draining wounds or fecal incontinence from pool use until their condition resolves. **Category II**
- G. For large hydrotherapy pools, use pH and chlorine residual levels appropriate for an indoor pool as provided by local and state health agencies. **Category IC** (States)
- H. **No recommendation is offered** regarding the use in health care of whirlpools or spa equipment manufactured for home or recreational use. **Unresolved issue**

XI. Miscellaneous Medical Equipment Connected to Water Systems

- A. Clean, disinfect, and maintain AER equipment according to the manufacturer's instructions and relevant scientific literature to prevent inadvertent contamination of endoscopes and bronchoscopes with waterborne microorganisms.^{911–915} **Category IB**
 - 1. To rinse disinfected endoscopes and bronchoscopes, use water of the highest quality practical for the system's engineering and design (e.g., sterile water or

- bacteriologically-filtered water [water filtered through 0.1–0.2- μm filters]).^{912, 914, 915, 918} **Category IB**
2. Dry the internal channels of the reprocessed endoscope or bronchoscope using a proven method (e.g., 70% alcohol followed by forced-air treatment) to lessen the potential for the proliferation of waterborne microorganisms and to help prevent biofilm formation.^{671, 921, 923, 925, 928} **Category IB**
- B. Use water that meets nationally recognized standards set by the EPA for drinking water (<500 CFU/mL for heterotrophic plate count) for routine dental treatment output water.^{935, 936, 943, 944} **Category IB, IC** (EPA: 40 CFR 1 Part 141, Subpart G).
- C. Take precautions to prevent waterborne contamination of dental unit water lines and instruments.
1. After each patient, discharge water and air for a minimum of 20–30 seconds from any dental device connected to the dental water system that enters the patient’s mouth (e.g., handpieces, ultrasonic scalers, and air/water syringe).^{936, 937} **Category II**
 2. Consult with dental water-line manufacturers to 1) determine suitable methods and equipment to obtain the recommended water quality; and 2) determine appropriate methods for monitoring the water to ensure quality is maintained.^{936, 946} **Category II**
 3. Consult with the dental unit manufacturer on the need for periodic maintenance of anti-retraction mechanisms.^{937, 946} **Category IB**

E. Recommendations—Environmental Services

I. Cleaning and Disinfecting Strategies for Environmental Surfaces in Patient-Care Areas

- A. Select EPA-registered disinfectants, if available, and use them in accordance with the manufacturer’s instructions.^{2, 974, 983} **Category IB, IC** (EPA: 7 United States Code [USC] § 136 et seq)
- B. Do not use high-level disinfectants/liquid chemical sterilants for disinfection of either noncritical instrument/devices or any environmental surfaces; such use is counter to label instructions for these toxic chemicals.^{951, 952, 961–964} **Category IB, IC** (FDA: 21 CFR 801.5, 807.87.e)
- C. Follow manufacturers’ instructions for cleaning and maintaining noncritical medical equipment. **Category II**
- D. In the absence of a manufacturer’s cleaning instructions, follow certain procedures.
 1. Clean noncritical medical equipment surfaces with a detergent/disinfectant. This may be followed with an application of an EPA-registered hospital disinfectant with or without a tuberculocidal claim (depending on the nature of the surface and the degree of contamination), in accordance with disinfectant label instructions.⁹⁵² **Category II**
 2. Do not use alcohol to disinfect large environmental surfaces.⁹⁵¹ **Category II**
 3. Use barrier protective coverings as appropriate for noncritical equipment surfaces that are 1) touched frequently with gloved hands during the delivery of patient care; 2) likely to become contaminated with blood or body substances; or 3) difficult to clean (e.g., computer keyboards).⁹³⁶ **Category II**
- E. Keep housekeeping surfaces (e.g., floors, walls, and tabletops) visibly clean on a regular basis and clean up spills promptly.⁹⁵⁴ **Category II**
 1. Use a one-step process and an EPA-registered hospital disinfectant/detergent designed for general housekeeping purposes in patient-care areas when 1) uncertainty exists as to the nature of the soil on these surfaces [e.g., blood or body fluid contamination versus routine dust or dirt]; or 2) uncertainty exists regarding the presence or absence of multi-drug resistant organisms on such surfaces.^{952, 983, 986, 987} **Category II**

2. Detergent and water are adequate for cleaning surfaces in nonpatient-care areas (e.g., administrative offices). **Category II**
3. Clean and disinfect high-touch surfaces (e.g., doorknobs, bed rails, light switches, and surfaces in and around toilets in patients' rooms) on a more frequent schedule than minimal touch housekeeping surfaces. **Category II**
4. Clean walls, blinds, and window curtains in patient-care areas when they are visibly dusty or soiled.^{2, 971, 972, 982} **Category II**
- F. Do not perform disinfectant fogging in patient-care areas.^{2, 976} **Category IB**
- G. Avoid large-surface cleaning methods that produce mists or aerosols or disperse dust in patient-care areas.^{9, 20, 109, 272} **Category IB**
- H. Follow proper procedures for effective use of mops, cloths, and solutions. **Category II**
 1. Prepare cleaning solutions daily or as needed, and replace with fresh solution frequently according to facility policies and procedures.^{986, 987} **Category II**
 2. Change the mop head at the beginning of the day and also as required by facility policy, or after cleaning up large spills of blood or other body substances. **Category II**
 3. Clean mops and cloths after use and allow to dry before reuse; or use single-use, disposable mop heads and cloths.^{971, 988-990} **Category II**
- I. After the last surgical procedure of the day or night, wet vacuum or mop operating room floors with a single-use mop and an EPA-registered hospital disinfectant.⁷ **Category IB**
- J. Do not use mats with tacky surfaces at the entrance to operating rooms or infection-control suites.⁷ **Category IB**
- K. Use appropriate dusting methods for patient-care areas designated for immunocompromised patients (e.g., HSCT patients).^{9, 94, 986} **Category IB**
 1. Wet-dust horizontal surfaces daily by moistening a cloth with a small amount of an EPA-registered hospital detergent/disinfectant.^{9, 94, 986} **Category IB**
 2. Avoid dusting methods that disperse dust (e.g., feather-dusting).⁹⁴ **Category IB**
- L. Keep vacuums in good repair, and equip vacuums with HEPA filters for use in areas with patients at risk.^{9, 94, 986, 994} **Category IB**
- M. Close the doors of immunocompromised patients' rooms when vacuuming, waxing, or buffing corridor floors to minimize exposure to airborne dust.^{9, 94, 994} **Category IB**
- N. When performing low- or intermediate-level disinfection of environmental surfaces in nurseries and neonatal units, avoid unnecessary exposure of neonates to disinfectant residues on environmental surfaces by using EPA-registered disinfectants in accordance with manufacturers' instructions and safety advisories.^{974, 995-997} **Category IB, IC** (EPA: 7 USC § 136 et seq.)
 1. Do not use phenolics or any other chemical germicide to disinfect bassinets or incubators during an infant's stay.^{952, 995-997} **Category IB**
 2. Rinse disinfectant-treated surfaces, especially those treated with phenolics, with water.⁹⁹⁵⁻⁹⁹⁷ **Category IB**
- O. When using phenolic disinfectants in neonatal units, prepare solutions to correct concentrations in accordance with manufacturers' instructions, or use premixed formulations.^{974, 995-997} **Category IB, IC** (EPA: 7 USC § 136 et seq.)

II. Cleaning Spills of Blood and Body Substances

- A. Promptly clean and decontaminate spills of blood or other potentially infectious materials.^{967, 998-1004} **Category IB, IC** (OSHA: 29 CFR 1910.1030 §d.4.ii.A)
- B. Follow proper procedures for site decontamination of spills of blood or blood-containing body fluids.^{967, 998-1004} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.ii.A)
 1. Use protective gloves and other PPE appropriate for this task.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.3.i, ii)

2. If the spill contains large amounts of blood or body fluids, clean the visible matter with disposable absorbent material, and discard the contaminated materials in appropriate, labeled containment.^{967, 1002, 1003, 1010, 1012} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iii.B)
3. Swab the area with a cloth or paper towels moderately wetted with disinfectant, and allow the surface to dry.^{967, 1010} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.ii.A)
- C. Use EPA-registered hospital disinfectants labeled tuberculocidal or registered germicides on the EPA Lists D and E (products with specific label claims for HIV or hepatitis B virus [HBV]) in accordance with label instructions to decontaminate spills of blood and other body fluids.^{967, 1007, 1010} **Category IC** (OSHA 29 CFR 1910.1030 § d.4.ii.A memorandum 2/28/97; compliance document CPL 2-2.44D [11/99])
- D. An EPA-registered sodium hypochlorite product is preferred, but if such products are not available, generic versions of sodium hypochlorite solutions (e.g., household chlorine bleach) may be used.
 1. Use a 1:100 dilution (500–615 ppm available chlorine) to decontaminate nonporous surfaces after cleaning a spill of either blood or body fluids in patient-care settings.^{1010, 1011} **Category II**
 2. If a spill involves large amounts of blood or body fluids, or if a blood or culture spill occurs in the laboratory, use a 1:10 dilution (5,000–6,150 ppm available chlorine) for the first application of germicide before cleaning.^{954, 1010} **Category II**

III. Carpeting and Cloth Furnishings

- A. Vacuum carpeting in public areas of health-care facilities and in general patient-care areas regularly with well-maintained equipment designed to minimize dust dispersion.⁹⁸⁶
Category II
- B. Periodically perform a thorough, deep cleaning of carpeting as determined by facility policy by using a method that minimizes the production of aerosols and leaves little or no residue.¹¹¹ **Category II**
- C. Avoid use of carpeting in high-traffic zones in patient-care areas or where spills are likely (e.g., burn therapy units, operating rooms, laboratories, and intensive care units).^{111, 1023, 1028}
Category IB
- D. Follow proper procedures for managing spills on carpeting.
 1. Spot-clean blood or body substance spills promptly.^{967, 1010, 1011, 1032} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.ii.A, interpretation)
 2. If a spill occurs on carpet tiles, replace any tiles contaminated by blood and body fluids or body substances.¹⁰³² **Category IC** (OSHA 29 CFR 1910.1030 § d.4.ii interpretation)
- E. Thoroughly dry wet carpeting to prevent the growth of fungi; replace carpeting that remains wet after 72 hours.^{9, 1026} **Category IB**
- F. **No recommendation is offered** regarding the routine use of fungicidal or bactericidal treatments for carpeting in public areas of a health-care facility or in general patient-care areas. **Unresolved issue**
- G. Do not use carpeting in hallways and patient rooms in areas housing immunosuppressed patients (e.g., PE areas).^{9, 111} **Category IB**
- H. Avoid the use of upholstered furniture and furnishings in high-risk patient-care areas and in areas with increased potential for body substance contamination (e.g., pediatrics units).⁹
Category II
- I. **No recommendation is offered** regarding whether upholstered furniture and furnishings should be avoided in general patient-care areas. **Unresolved issue**
- J. Maintain upholstered furniture in good repair. **Category II**
 1. Maintain the surface integrity of the upholstery by repairing tears and holes.
Category II

2. If upholstered furniture in a patient's room requires cleaning to remove visible soil or body substance contamination, move that item to a maintenance area where it can be adequately cleaned with a process appropriate for the type of upholstery and the nature of the soil. **Category II**

IV. Flowers and Plants in Patient-Care Areas

- A. Flowers and potted plants need not be restricted from areas for immunocompetent patients.^{515, 702, 1040, 1042} **Category II**
- B. Designate care and maintenance of flowers and potted plants to staff not directly involved with patient care.⁷⁰² **Category II**
- C. If plant or flower care by patient-care staff is unavoidable, instruct the staff to wear gloves when handling the plants and flowers and perform hand hygiene after glove removal.⁷⁰² **Category II**
- D. Do not allow fresh or dried flowers, or potted plants in patient-care areas for immunosuppressed patients.^{9, 109, 515, 1046} **Category II**

V. Pest Control

- A. Develop pest-control strategies, with emphasis on kitchens, cafeterias, laundries, central sterile supply areas, operating rooms, loading docks, construction activities, and other areas prone to infestations.^{1050, 1072, 1075} **Category II**
- B. Install screens on all windows that open to the outside; keep screens in good repair.¹⁰⁷² **Category IB**
- C. Contract for routine pest control service by a credentialed pest-control specialist who will tailor the application to the needs of a health-care facility.¹⁰⁷⁵ **Category II**
- D. Place laboratory specimens (e.g., fixed sputum smears) in covered containers for overnight storage.^{1065, 1066} **Category II**

VI. Special Pathogens

- A. Use appropriate hand hygiene, PPE (e.g., gloves), and isolation precautions during cleaning and disinfecting procedures.^{5, 952, 1130, 1364} **Category IB**
- B. Use standard cleaning and disinfection protocols to control environmental contamination with antibiotic-resistant gram-positive cocci (e.g., methicillin-resistant *Staphylococcus aureus*, vancomycin intermediate-resistant *Staphylococcus aureus*, or vancomycin-resistant *Enterococcus* [VRE]).^{5, 1116–1118} **Category IB**
 1. Pay close attention to cleaning and disinfection of high-touch surfaces in patient-care areas (e.g., bed rails, carts, bedside commodes, bedrails, doorknobs, or faucet handles).^{5, 1116–1118} **Category IB**
 2. Ensure compliance by housekeeping staff with cleaning and disinfection procedures.^{5, 1116–1118} **Category IB**
 3. Use EPA-registered hospital disinfectants appropriate for the surface to be disinfected (e.g., either low- or intermediate-level disinfection) as specified by the manufacturers' instructions.^{974, 1106–1110, 1118} **Category IB, IC** (EPA: 7 USC § 136 et seq.)
 4. When contact precautions are indicated for patient care, use disposable patient-care items (e.g., blood pressure cuffs) whenever possible to minimize cross-contamination with multiple-resistant microorganisms.¹¹⁰² **Category IB**
 5. Follow these same surface cleaning and disinfecting measures for managing the environment of VRSA patients.^{1110, 1116–1118} **Category II**
- C. Environmental-surface culturing can be used to verify the efficacy of hospital policies and procedures before and after cleaning and disinfecting rooms that house patients with VRE.^{5, 1084, 1087, 1088, 1092, 1096} **Category II**

1. Obtain prior approval from infection-control staff and the clinical laboratory before performing environmental surface culturing. **Category II**
 2. Infection-control staff, with clinical laboratory consultation, must supervise all environmental culturing. **Category II**
- D. Thoroughly clean and disinfect environmental and medical equipment surfaces on a regular basis using EPA-registered disinfectants in accordance with manufacturers' instructions.^{952, 974, 1130, 1143} **Category IB, IC** (EPA: 7 USC § 136 et seq.)
- E. Advise families, visitors, and patients about the importance of hand hygiene to minimize the spread of body substance contamination (e.g., respiratory secretions or fecal matter) to surfaces.⁹⁵² **Category II**
- F. Do not use high-level disinfectants (i.e., liquid chemical sterilants) on environmental surfaces; such use is inconsistent with label instructions and because of the toxicity of the chemicals.^{2, 951, 952, 964} **Category IC** (FDA: 21 CFR 801.5, 807.87.e)
- G. Because no EPA-registered products are specific for inactivating *Clostridium difficile* spores, use hypochlorite-based products for disinfection of environmental surfaces in those patient-care areas where surveillance and epidemiology indicate ongoing transmission of *C. difficile*.^{952, 1130, 1141} **Category II**
- H. **No recommendation is offered** regarding the use of specific EPA-registered hospital disinfectants with respect to environmental control of *C. difficile*. **Unresolved issue**
- I. Apply standard cleaning and disinfection procedures to control environmental contamination with respiratory and enteric viruses in pediatric-care units and care areas for immunocompromised patients.^{986, 1158} **Category IC** (EPA: 7 USC § 136 et seq.)
- J. Clean surfaces that have been contaminated with body substances; perform low- to intermediate-level disinfection on cleaned surfaces with an EPA-registered disinfectant in accordance with the manufacturer's instructions.^{967, 974, 1158} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.ii.A; EPA: 7 USC § 136 et seq.)
- K. Use disposable barrier coverings as appropriate to minimize surface contamination. **Category II**
- L. Develop and maintain cleaning and disinfection procedures to control environmental contamination with agents of Creutzfeldt-Jakob disease (CJD), for which no EPA-registered product exists. **Category II**
1. In the absence of contamination with central nervous system tissue, extraordinary measures (e.g., use of 2N sodium hydroxide [NaOH] or applying full-strength sodium hypochlorite) are not needed for routine cleaning or terminal disinfection of a room housing a confirmed or suspected CJD patient.^{951, 1199} **Category II**
 2. After removing gross tissue from the surface, use either 1N NaOH or a sodium hypochlorite solution containing approximately 10,000–20,000 ppm available chlorine (dilutions of 1:5 to 1:3 v/v, respectively, of U.S. household chlorine bleach; contact the manufacturers of commercially available sodium hypochlorite products for advice) to decontaminate operating room or autopsy surfaces with central nervous system or cerebral spinal fluid contamination from a diagnosed or suspected CJD patient.^{951, 1170, 1188, 1191, 1197–1199, 1201} **Category II**
 - a. The contact time for the chemical used during this process should be 30 min–1 hour.^{1191, 1197, 1201}
 - b. Blot up the chemical with absorbent material and rinse the treated surface thoroughly with water.
 - c. Discard the used, absorbent material into appropriate waste containment.
 3. Use disposable, impervious covers to minimize body substance contamination to autopsy tables and surfaces.^{1197, 1201} **Category IB**

- M. Use standard procedures for containment, cleaning, and decontamination of blood spills on surfaces as previously described (Environmental Services: II).⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 §d.4.ii.A)
1. Wear PPE appropriate for a surface decontamination and cleaning task.^{967, 1199}
Category IC (OSHA 29 CFR 1910.1030 §d.3.i, ii)
 2. Discard used PPE by using routine disposal procedures or decontaminate reusable PPE as appropriate.^{967, 1199} **Category IC** (OSHA 29 CFR 1910.1030 §d.3.viii)

F. Recommendations—Environmental Sampling

I. General Information

- A. Do not conduct random, undirected microbiologic sampling of air, water, and environmental surfaces in health-care facilities.^{2, 1214} **Category IB**
- B. When indicated, conduct microbiologic sampling as part of an epidemiologic investigation or during assessment of hazardous environmental conditions to detect contamination and verify abatement of a hazard.^{2, 1214} **Category IB**
- C. Limit microbiologic sampling for quality assurance purposes to 1) biological monitoring of sterilization processes; 2) monthly cultures of water and dialysate in hemodialysis units; and 3) short-term evaluation of the impact of infection-control measures or changes in infection-control protocols.^{2, 1214} **Category IB**

II. Air, Water, and Environmental-Surface Sampling

- A. When conducting any form of environmental sampling, identify existing comparative standards and fully document departures from standard methods.^{945, 1214, 1223, 1224, 1238}
Category II
- B. Select a high-volume air sampling device if anticipated levels of microbial airborne contamination are expected to be low.^{290, 1218, 1223, 1224} **Category II**
- C. Do not use settle plates to quantify the concentration of airborne fungal spores.²⁹⁰
Category II
- D. When sampling water, choose growth media and incubation conditions that will facilitate the recovery of waterborne organisms.⁹⁴⁵ **Category II**
- E. When using a sample/rinse method for sampling an environmental surface, develop and document a procedure for manipulating the swab, gauze, or sponge in a reproducible manner so that results are comparable.¹²³⁸ **Category II**
- F. When environmental samples and patient specimens are available for comparison, perform the laboratory analysis on the recovered microorganisms down to the species level at a minimum and beyond the species level if possible.¹²¹⁴ **Category II**

G. Recommendations—Laundry and Bedding

I. Employer Responsibilities

- A. Employers must launder workers' personal protective garments or uniforms that are contaminated with blood or other potentially infectious materials.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.3.iv)

II. Laundry Facilities and Equipment

- A. Maintain the receiving area for contaminated textiles at negative pressure compared with the clean areas of the laundry in accordance with AIA construction standards in effect during the time of facility construction.^{120, 1260–1262} **Category IC** (AIA: 7.23.B1, B2)
- B. Ensure that laundry areas have handwashing facilities and products and appropriate PPE available for workers.^{120, 967} **Category IC** (AIA: 7.23.D4; OSHA: 29 CFR 1910.1030 § d.2.iii)
- C. Use and maintain laundry equipment according to manufacturers' instructions.^{1250, 1263}
Category II
- D. Do not leave damp textiles or fabrics in machines overnight.¹²⁵⁰ **Category II**
- E. Disinfection of washing and drying machines in residential care is not needed as long as gross soil is removed before washing and proper washing and drying procedures are used.
Category II

III. Routine Handling of Contaminated Laundry

- A. Handle contaminated textiles and fabrics with minimum agitation to avoid contamination of air, surfaces, and persons.^{6, 967, 1258, 1259} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iv)
- B. Bag or otherwise contain contaminated textiles and fabrics at the point of use.⁹⁶⁷
Category IC (OSHA: 29 CFR 1910.1030 § d.4.iv)
 - 1. Do not sort or prerinse contaminated textiles or fabrics in patient-care areas.⁹⁶⁷
Category IC (OSHA: 29 CFR 1910.1030 § d.4.iv)
 - 2. Use leak-resistant containment for textiles and fabrics contaminated with blood or body substances.^{967, 1258} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iv)
 - 3. Identify bags or containers for contaminated textiles with labels, color coding, or other alternative means of communication as appropriate.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iv)
- C. Covers are not needed on contaminated textile hampers in patient-care areas. **Category II**
- D. If laundry chutes are used, ensure that they are properly designed, maintained, and used in a manner to minimize dispersion of aerosols from contaminated laundry.^{1253, 1267–1270}
Category IC (AAMI: ANSI/AAMI ST65:2000)
 - 1. Ensure that laundry bags are closed before tossing the filled bag into the chute.
Category II
 - 2. Do not place loose items in the chute. **Category II**
- E. Establish a facility policy to determine when textiles or fabrics should be sorted in the laundry facility (i.e., before or after washing).^{1271, 1272} **Category II**

IV. Laundry Process

- A. If hot-water laundry cycles are used, wash with detergent in water $\geq 160^{\circ}\text{F}$ ($\geq 71^{\circ}\text{C}$) for ≥ 25 minutes.^{2, 120} **Category IC** (AIA: 7.31.E3)
- B. **No recommendation is offered** regarding a hot-water temperature setting and cycle duration for items laundered in residence-style health-care facilities. **Unresolved issue**
- C. Follow fabric-care instructions and special laundering requirements for items used in the facility.¹²⁷⁸ **Category II**
- D. Choose chemicals suitable for low-temperature washing at proper use concentration if low-temperature ($< 160^{\circ}\text{F}$ [$< 71^{\circ}\text{C}$]) laundry cycles are used.^{1247, 1281–1285} **Category II**
- E. Package, transport, and store clean textiles and fabrics by methods that will ensure their cleanliness and protect them from dust and soil during interfacility loading, transport, and unloading.² **Category II**

V. Microbiologic Sampling of Textiles

- A. Do not conduct routine microbiological sampling of clean textiles.^{2, 1286} **Category IB**

- B. Use microbiological sampling during outbreak investigations if epidemiologic evidence suggests a role for health-care textiles and clothing in disease transmission.¹²⁸⁶ **Category IB**

VI. Special Laundry Situations

- A. Use sterilized textiles, surgical drapes, and gowns for situations requiring sterility in patient care.⁷ **Category IB**
- B. Use hygienically clean textiles (i.e., laundered, but not sterilized) in neonatal intensive care units.^{997, 1288} **Category IB**
- C. Follow manufacturers' recommendations for cleaning fabric products including those with coated or laminated surfaces. **Category II**
- D. Do not use dry cleaning for routine laundering in health-care facilities.^{1289–1291} **Category II**
- E. Use caution when considering the use of antimicrobial mattresses, textiles, and clothing as replacements for standard bedding and other fabric items; EPA has not approved public health claims asserting protection against human pathogens for treated articles.¹³⁰⁶ **Category II**
- F. **No recommendation is offered** regarding using disposable fabrics and textiles versus durable goods. **Unresolved issue**

VII. Mattresses and Pillows

- A. Keep mattresses dry; discard them if they become and remain wet or stained, particularly in burn units.^{1310–1315} **Category IB**
- B. Clean and disinfect mattress covers using EPA-registered disinfectants, if available, that are compatible with the cover materials to prevent the development of tears, cracks, or holes in the cover.^{1310–1315} **Category IB**
- C. Maintain the integrity of mattress and pillow covers. **Category II**
 - 1. Replace mattress and pillow covers if they become torn or otherwise in need of repair. **Category II**
 - 2. Do not stick needles into the mattress through the cover. **Category II**
- D. Clean and disinfect moisture-resistant mattress covers between patients using an EPA-registered product, if available.^{1310–1315} **Category IB**
- E. If using a mattress cover completely made of fabric, change these covers and launder between patients.^{1310–1315} **Category IB**
- F. Launder pillow covers and washable pillows in the hot-water cycle between patients or when they become contaminated with body substances.¹³¹⁵ **Category IB**

VIII. Air-Fluidized Beds

- A. Follow manufacturers' instructions for bed maintenance and decontamination. **Category II**
- B. Change the polyester filter sheet at least weekly or as indicated by the manufacturer.^{1317, 1318, 1322, 1323} **Category II**
- C. Clean and disinfect the polyester filter sheet thoroughly, especially between patients, using an EPA-registered product, if available.^{1317, 1318, 1322, 1323} **Category IB**
- D. Consult the facility engineer to determine the proper location of air-fluidized beds in negative-pressure rooms.¹³²⁶ **Category II**

H. Recommendations—Animals in Health-Care Facilities

I. General Infection-Control Measures for Animal Encounters

- A. Minimize contact with animal saliva, dander, urine, and feces.^{1365–1367} **Category II**
- B. Practice hand hygiene after any animal contact.^{2, 1364} **Category IB**
 - 1. Wash hands with soap and water, especially if hands are visibly soiled.¹³⁶⁴
Category IB
 - 2. Use either soap and water or alcohol-based hand rubs when hands are not visibly soiled.¹³⁶⁴ **Category IB**

II. Animal-Assisted Activities, Animal-Assisted Therapy, and Resident Animal Programs

- A. Avoid selection of nonhuman primates and reptiles in animal-assisted activities, animal-assisted therapy, or resident animal programs.^{1360–1362} **Category IB**
- B. Enroll animals that are fully vaccinated for zoonotic diseases and that are healthy, clean, well-groomed, and negative for enteric parasites or otherwise have completed recent antihelminthic treatment under the regular care of a veterinarian.^{1349, 1360} **Category II**
- C. Enroll animals that are trained with the assistance or under the direction of individuals who are experienced in this field.¹³⁶⁰ **Category II**
- D. Ensure that animals are handled by persons trained in providing activities or therapies safely, and who know the animals' health status and behavior traits.^{1349, 1360} **Category II**
- E. Take prompt action when an incident of biting or scratching by an animal occurs during an animal-assisted activity or therapy.
 - 1. Remove the animal permanently from these programs.¹³⁶⁰ **Category II**
 - 2. Report the incident promptly to appropriate authorities (e.g., infection-control staff, animal program coordinator, or local animal control).¹³⁶⁰ **Category II**
 - 3. Promptly clean and treat scratches, bites, or other accidental breaks in the skin.
Category II
- F. Perform an ICRA and work actively with the animal handler prior to conducting an animal-assisted activity or therapy to determine if the session should be held in a public area of the facility or in individual patient rooms.^{1349, 1360} **Category II**
- G. Take precautions to mitigate allergic responses to animals. **Category II**
 - 1. Minimize shedding of animal dander by bathing animals <24 hours before a visit.¹³⁶⁰
Category II
 - 2. Groom animals to remove loose hair before a visit, or using a therapy animal cape.¹³⁵⁸
Category II
- H. Use routine cleaning protocols for housekeeping surfaces after therapy sessions.
Category II
- I. Restrict resident animals, including fish in fish tanks, from access to or placement in patient-care areas, food preparation areas, dining areas, laundry, central sterile supply areas, sterile and clean supply storage areas, medication preparation areas, operating rooms, isolation areas, and PE areas. **Category II**
- J. Establish a facility policy for regular cleaning of fish tanks, rodent cages, bird cages, and any other animal dwellings and assign this cleaning task to a nonpatient-care staff member; avoid splashing tank water or contaminating environmental surfaces with animal bedding.
Category II

III. Protective Measures for Immunocompromised Patients

- A. Advise patients to avoid contact with animal feces and body fluids such as saliva, urine, or solid litter box material.⁸ **Category II**

- B. Promptly clean and treat scratches, bites, or other wounds that break the skin.⁸ **Category II**
- C. Advise patients to avoid direct or indirect contact with reptiles.¹³⁴⁰ **Category IB**
- D. Conduct a case-by-case assessment to determine if animal-assisted activities or animal-assisted therapy programs are appropriate for immunocompromised patients.¹³⁴⁹ **Category II**
- E. **No recommendation is offered** regarding permitting pet visits to terminally ill immunosuppressed patients outside their PE units. **Unresolved issue**

IV. Service Animals

- A. Avoid providing access to nonhuman primates and reptiles as service animals.^{1340, 1362} **Category IB**
- B. Allow service animals access to the facility in accordance with the Americans with Disabilities Act of 1990, unless the presence of the animal creates a direct threat to other persons or a fundamental alteration in the nature of services.^{1366, 1376} **Category IC** (U.S. Department of Justice: 28 CFR § 36.302)
- C. When a decision must be made regarding a service animal's access to any particular area of the health-care facility, evaluate the service animal, the patient, and the health-care situation on a case-by-case basis to determine whether significant risk of harm exists and whether reasonable modifications in policies and procedures will mitigate this risk.¹³⁷⁶ **Category IC** (Justice: 28 CFR § 36.208 and App.B)
- D. If a patient must be separated from his or her service animal while in the health-care facility
 - 1) ascertain from the person what arrangements have been made for supervision or care of the animal during this period of separation; and 2) make appropriate arrangements to address the patient's needs in the absence of the service animal. **Category II**

V. Animals as Patients in Human Health-Care Facilities

- A. Develop health-care facility policies to address the treatment of animals in human health-care facilities.
 1. Use the multidisciplinary team approach to policy development, including public media relations in order to disclose and discuss these activities. **Category II**
 2. Exhaust all veterinary facility, equipment, and instrument options before undertaking the procedure. **Category II**
 3. Ensure that the care of the animal is supervised by a licensed veterinarian. **Category II**
- B. When animals are treated in human health-care facilities, avoid treating animals in operating rooms or other patient-care areas where invasive procedures are performed (e.g., cardiac catheterization laboratories, or invasive nuclear medicine areas). **Category II**
- C. Schedule the animal procedure for the last case of the day for the area, at a time when human patients are not scheduled to be in the vicinity. **Category II**
- D. Adhere strictly to standard precautions. **Category II**
- E. Clean and disinfect environmental surfaces thoroughly using an EPA-registered product in the room after the animal is removed. **Category II**
- F. Allow sufficient ACH to clean the air and help remove airborne dander, microorganisms, and allergens [Appendix B, Table B.1.]). **Category II**
- G. Clean and disinfect using EPA-registered products or sterilize equipment that has been in contact with animals, or use disposable equipment. **Category II**
- H. If reusable medical or surgical instruments are used in an animal procedure, restrict future use of these instruments to animals only. **Category II**

VI. Research Animals in Health-Care Facilities

- A. Use animals obtained from quality stock, or quarantine incoming animals to detect zoonotic diseases. **Category II**
- B. Treat sick animals or remove them from the facility. **Category II**
- C. Provide prophylactic vaccinations, as available, to animal handlers and contacts at high risk. **Category II**
- D. Ensure proper ventilation through appropriate facility design and location.¹³⁹⁵ **Category IC** (U.S. Department of Agriculture [USDA]: 7 USC 2131)
 - 1. Keep animal rooms at negative pressure relative to corridors.¹³⁹⁵ **Category IC** (USDA: 7 USC 2131)
 - 2. Prevent air in animal rooms from recirculating elsewhere in the health-care facility.¹³⁹⁵ **Category IC** (USDA: 7 USC 2131)
- E. Keep doors to animal research rooms closed. **Category II**
- F. Restrict access to animal facilities to essential personnel. **Category II**
- G. Establish employee occupational health programs specific to the animal research facility, and coordinate management of postexposure procedures specific for zoonoses with occupational health clinics in the health-care facility.^{1013, 1378} **Category IC** (U.S. Department of Health and Human Services [DHHS]: BMBL; OSHA: 29 CFR 1910.1030.132-139)
- H. Document standard operating procedures for the unit.¹⁰¹³ **Category IC** (DHHS: BMBL)
- I. Conduct routine employee training on worker safety issues relevant to the animal research facility (e.g., working safely with animals and animal handling).^{1013, 1393} **Category IC** (DHHS: BMBL; OSHA: 29 CFR 1910.1030.132-139)
- J. Use precautions to prevent the development of animal-induced asthma in animal workers.¹⁰¹³ **Category IC** (DHHS: BMBL)

I. Recommendations—Regulated Medical Waste

I. Categories of Regulated Medical Waste

- A. Designate the following as major categories of medical waste that require special handling and disposal precautions: 1) microbiology laboratory wastes [e.g., cultures and stocks of microorganisms]; 2) bulk blood, blood products, blood, and bloody body fluid specimens; 3) pathology and anatomy waste; and 4) sharps [e.g., needles and scalpels].² **Category II**
- B. Consult federal, state, and local regulations to determine if other waste items are considered regulated medical wastes.^{967, 1407, 1408} **Category IC** (States; Authorities having jurisdiction [AHJ]; OSHA: 29 CFR 1910.1030 §g.2.1; U.S. Department of Transportation [DOT]: 49 CFR 171-180; U.S. Postal Service: CO23.8)

II. Disposal Plan for Regulated Medical Wastes

- A. Develop a plan for the collection, handling, predisposal treatment, and terminal disposal of regulated medical wastes.^{967, 1409} **Category IC** (States; AHJ; OSHA: 29 CFR 1910.1030 §g.2.i)
- B. Designate a person or persons to be responsible for establishing, monitoring, reviewing, and administering the plan. **Category II**

III. Handling, Transporting, and Storing Regulated Medical Wastes

- A. Inform personnel involved in the handling and disposal of potentially infective waste of the possible health and safety hazards; ensure that they are trained in appropriate handling and disposal methods.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § g.2.i)
- B. Manage the handling and disposal of regulated medical wastes generated in isolation areas by using the same methods as for regulated medical wastes from other patient-care areas.² **Category II**
- C. Use proper sharps disposal strategies.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iii.A)

1. Use a sharps container capable of maintaining its impermeability after waste treatment to avoid subsequent physical injuries during final disposal.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iii.A)
 2. Place disposable syringes with needles, including sterile sharps that are being discarded, scalpel blades, and other sharp items into puncture-resistant containers located as close as practical to the point of use.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iii.A)
 3. Do not bend, recap, or break used syringe needles before discarding them into a container.^{6, 967, 1415} **Category IC** (OSHA: 29 CFR 1910.1030 § d.2.vii and § d.2.vii.A)
- D. Store regulated medical wastes awaiting treatment in a properly ventilated area that is inaccessible to vertebrate pests; use waste containers that prevent the development of noxious odors. **Category IC** (States; AHJ)
- E. If treatment options are not available at the site where the medical waste is generated, transport regulated medical wastes in closed, impervious containers to the on-site treatment location or to another facility for treatment as appropriate. **Category IC** (States; AHJ)

IV. Treatment and Disposal of Regulated Medical Wastes

- A. Treat regulated medical wastes by using a method (e.g., steam sterilization, incineration, interment, or an alternative treatment technology) approved by the appropriate authority having jurisdiction (AHJ) (e.g., states, Indian Health Service [IHS], Veterans Affairs [VA]) before disposal in a sanitary landfill. **Category IC** (States, AHJ)
- B. Follow precautions for treating microbiological wastes (e.g., amplified cultures and stocks of microorganisms).¹⁰¹³ **Category IC** (DHHS: BMBL)
1. Biosafety level 4 laboratories must inactivate microbiological wastes in the laboratory by using an approved inactivation method (e.g., autoclaving) before transport to and disposal in a sanitary landfill.¹⁰¹³ **Category IC** (DHHS: BMBL)
 2. Biosafety level 3 laboratories must inactivate microbiological wastes in the laboratory by using an approved inactivation method (e.g., autoclaving) or incinerate them at the facility before transport to and disposal in a sanitary landfill.¹⁰¹³ **Category IC** (DHHS: BMBL)
- C. Biosafety levels 1 and 2 laboratories should develop strategies to inactivate amplified microbial cultures and stocks onsite by using an approved inactivation method (e.g., autoclaving) instead of packaging and shipping untreated wastes to an offsite facility for treatment and disposal.^{1013, 1419–1421} **Category II**
- D. Laboratories that isolate select agents from clinical specimens must comply with federal regulations for the receipt, transfer, management, and appropriate disposal of these agents.¹⁴¹² **Category IC** (DHHS: 42 CFR 73 § 73.6)
- E. Sanitary sewers may be used for the safe disposal of blood, suctioned fluids, ground tissues, excretions, and secretions, provided that local sewage discharge requirements are met and that the state has declared this to be an acceptable method of disposal.¹⁴¹⁴ **Category II**

V. Special Precautions for Wastes Generated During Care of Patients with Rare Diseases

- A. When discarding items contaminated with blood and body fluids from VHF patients, contain these regulated medical wastes with minimal agitation during handling.^{6, 203} **Category II**
- B. Manage properly contained wastes from areas providing care to VHF patients in accordance with recommendations for other isolation areas (Regulated Medical Waste: III B).^{2, 6, 203} **Category II**
- C. Decontaminate bulk blood and body fluids from VHF patients using approved inactivation methods (e.g., autoclaving or chemical treatment) before disposal.^{6, 203} **Category IC, II** (States; AHJ)

- D. When discarding regulated medical waste generated during the routine (i.e., non-surgical) care of CJD patients, contain these wastes and decontaminate them using approved inactivation methods (e.g., autoclaving or incineration) appropriate for the medical waste category (e.g., blood, sharps, pathological waste).^{2, 6, 948, 1199} **Category IC, II** (States; AHJ)
- E. Incinerate medical wastes (e.g., central nervous system tissues or contaminated disposable materials) from brain autopsy or biopsy procedures of diagnosed or suspected CJD patients.^{1197, 1201} **Category IB**

Part III. References

Note: The bold item in parentheses indicated the citation number or the location of this reference listed in the MMWR version of this guideline.

1. Simmons BP. Guideline for hospital environmental control. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, 1981.
2. **(270)** Garner JS, Favero MS. Guideline for handwashing and hospital environmental control. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, 1985. Document No. 99-1117 (Also available at Infect Control 1986; 7: 231-43.)
3. **(27)** CDC. Guidelines for prevention of nosocomial pneumonia. MMWR 1997;46(No. RR-1):1-79.
4. **(34)** CDC. Guidelines for preventing the transmission of *Mycobacterium tuberculosis* in health-care facilities. MMWR 1994;43(No. RR-13):1-132.
5. **(318)** CDC. Recommendations for preventing the spread of vancomycin resistance. Recommendations of the Hospital Infection Control Practices Advisory Committee (HICPAC). MMWR 1995;44(No. RR-12):1-13.
6. **(36)** Garner JS, Hospital Infection Control Practices Advisory Committee. Guideline for isolation precautions in hospitals. Infect Control Hosp Epidemiol 1996;17:53-80.
7. **(114)** Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR, Hospital Infection Control Practices Advisory Committee. Guideline for prevention of surgical site infection, 1999. Infect Control Hosp Epidemiol 1999;20:247-80.
8. **(396)** CDC. USPHS/IDSA guidelines for the prevention of opportunistic infections in persons infected with human immunodeficiency virus. MMWR 1999;48(No. RR-10):1-66.
9. **(37, Appendix; 5)** CDC. CDC/IDSA/ASBMT guidelines for the prevention of opportunistic infections in hematopoietic stem cell transplant recipients. MMWR 2000;49(No. RR-10):1-128.
10. Garner JS. The CDC Hospital Infection Control Practice Advisory Committee. Am J Infect Control 1993;21:160-2.
11. Bennett JV, Brachman PS, eds. The inanimate environment. In: Rhame FS. Hospital Infections, 4th ed. Philadelphia, PA: Lippincott-Raven, 1998;299-324.
12. Weber DJ, Rutala WA. Environmental issues and nosocomial infections. In: Wenzel RP, ed. Prevention and control of nosocomial infections, 3rd ed. Baltimore, MD: Williams & Wilkins, 1997;491-514.
13. Greene VW. Microbiological contamination control in hospitals. Hospitals JAHA 1969;43:78-88.
14. American Hospital Association. Hospital statistics, 2000 ed. Historical trends in utilization, personnel, and finances for selected years from 1946 through 1998 [Table]. Chicago, IL: Health Forum LLC, 2000;2-3.
15. McKee B. Neither bust nor boom. Architecture 1998. Available at: www.britannica.com/bcom/magazine/article/0,5744,39579,00.html
16. Crowell CL. Better, not bigger: construction costs soar on wings of patient demand, construction and design survey finds. Mod Healthc 1999;29:23-6, 28-34, 36-8.
17. **(9)** Sarubbi FA Jr, Kopf BB, Wilson NO, McGinnis MR, Rutala WA. Increased recovery of *Aspergillus flavus* from respiratory specimens during hospital construction. Am Rev Respir Dis 1982;125:33-8.
18. **(2)** Arnow PM, Sadigh MC, Weil D, Chudy R. Endemic and epidemic aspergillosis associated with inhospital replication of *Aspergillus* organisms. J Infect Dis 1991;164:998-1002.

19. (38) Flynn PM, Williams BG, Hethrington SV, Williams BF, Giannini MA, Pearson TA. *Aspergillus terreus* during hospital renovation [letter]. *Infect Control Hosp Epidemiol* 1993;14:363–5.
20. (48) Weems JJ Jr, Davis BJ, Tablan OC, Kaufman L, Martone WJ. Construction activity: an independent risk factor for invasive aspergillosis and zygomycosis in patients with hematologic malignancy. *Infect Control* 1987;8:71–5.
21. (10) Streifel AJ, Stevens PP, Rhame FS. In-hospital source of airborne *Penicillium* species spores. *J Clin Microbiol* 1987;25:1–4.
22. Noskin GA, Stosor V, Cooper J, Peterson L. Recovery of vancomycin-resistant enterococci on fingertips and environmental surfaces. *Infect Control Hosp Epidemiol* 1995;16:577–81.
23. Manian FA, Meyer L, Jenne J. *Clostridium difficile* contamination of blood pressure cuffs: a call for a closer look at gloving practices in the era of universal precautions. *Infect Control Hosp Epidemiol* 1996;17:180–2.
24. McFarland LV, Mulligan NE, Kwok RYY, et al. Nosocomial acquisition of *Clostridium difficile* infection. *N Engl J Med* 1989;320:204–10.
25. Nath SK, Thomely JH, Kelly M, et al. A sustained outbreak of *Clostridium difficile* in a general hospital: persistence of a toxigenic clone in four units. *Infect Control Hosp Epidemiol* 1994;15:382–9.
26. (165) Johnson JT, Yu VL, Best MG, et al. Nosocomial legionellosis in surgical patients with head and neck cancer: implications for epidemiological reservoir and mode of transmission. *Lancet* 1985;2:298–300.
27. Blatt SP, Parkinson MD, Pace E, et al. Nosocomial Legionnaires' disease: aspiration as a primary mode of disease acquisition. *Am J Med* 1993;95:16–22.
28. Bert F, Maubec E, Bruneau B, Berry P, Lambert-Zechovsky N. Multi-resistant *Pseudomonas aeruginosa* associated with contaminated tap-water in a neurosurgery intensive care unit. *J Hosp Infect* 1998;39:53–62.
29. Muylldermans G, de Smet F, Perrard D, et al. Neonatal infections with *Pseudomonas aeruginosa* associated with a water-bath used to thaw fresh frozen plasma. *J Hosp Infect* 1998;39:309–14.
30. Buttery JP, Alabaster SJ, Heine FG, et al. Multi-resistant *Pseudomonas aeruginosa* outbreak in a pediatric oncology ward related to bath toys. *Pediatr Infect Dis J* 1998;17:509–13.
31. (224) Bolan G, Reingold AL, Carson LA, et al. Infections with *Mycobacterium chelonae* in patients receiving dialysis and using processed hemodialyzers. *J Infect Dis* 1985;152:1013–9.
32. (225) Lowry PW, Beck-Sague CM, Bland LA, et al. *Mycobacterium chelonae* infection among patients receiving high-flux dialysis in a hemodialysis clinic in California. *J Infect Dis* 1990;161:85–90.
33. Schaal KP. Medical and microbiological problems arising from airborne infection in hospitals. *J Hosp Infect* 1991;18 (Suppl A):451–9.
34. Osterholm MT, Hedberg CW, Moore KA. Epidemiology of infectious diseases. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and practice of infectious diseases*, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;156–67.
35. (3) Streifel AJ. Design and maintenance of hospital ventilation systems and prevention of airborne nosocomial infections. In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999:1211–21.
36. Bodey GP, Vartivarian S. Aspergillosis. *Eur J Clin Microbiol Infect Dis* 1989;8:413–37.
37. Latgé JP. *Aspergillus fumigatus* and aspergillosis. *Clin Microbiol Rev* 1999;12:310–50.
38. Derouin F. Special issue on aspergillosis. *Pathol Biol* 1990;42:625–736.
39. Dixon DD, Walsh TJ. Human pathogenesis. In: Bennett JW, Klich MA, eds. *Aspergillus*, biology and industrial application. Boston, MA: Butterworth-Heinemann, 1992;249–67.
40. Kurup VP, Kumar A. Immunodiagnosis of aspergillosis. *Clin Microbiol Rev* 1991;4:439–56.
41. Latgé JP, Paris S, Sarfati J, et al. Infectivity of *Aspergillus fumigatus*. In: Vanden Bossche H, Stevens DA, Odds FC, eds. *Host-Fungus Interplay*. Bethesda, MD: National Foundation for Infectious Diseases, 1997;99–110.
42. Schaffner A. Host defense in aspergillosis. In: Bennett E, Hay RJ, Peterson PK, eds. *New Strategies in Fungal Disease*. Edinburgh, United Kingdom: Churchill Livingstone, 1992;98–112.
43. Vanden Bossche H, Mackenzie DWR, Cauwenbergh G, eds. *Aspergillus* and aspergillosis. New York, NY: Plenum Press, 1988.
44. (82) Sherertz RJ, Belani A, Kramer BS, et al. Impact of air filtration on nosocomial *Aspergillus* infections: unique risk of bone marrow transplant recipients. *Am J Med* 1987;83:709–18.
45. Young RC, Bennett JE, Vogel CL, Carbone PP, DeVita VT. Aspergillosis: the spectrum of the disease in 98 patients. *Medicine* 1970;49:147–73.
46. Rhame FS. Lessons from the Roswell Park bone marrow transplant aspergillosis outbreak. *Infect Control* 1985;6:345–6.

47. Rotsein C, Cummings KM, Tidings J, et al. An outbreak of invasive aspergillosis among allogeneic bone marrow transplants: a case-control study. *Infect Control* 1985;6:347-55.
48. (83) Aisner J, Schimpff SC, Bennett JE, Young VM, Wiernik PH. Aspergillus infections in cancer patients: association with fireproofing materials in a new hospital. *JAMA* 1976;235:411-2.
49. (64) Arnow PM, Anderson RL, Mainous PD, Smith EJ. Pulmonary aspergillosis during hospital renovation. *Am Rev Respir Dis* 1978;118:49-53.
50. (61) Streifel AJ, Lauer JL, Vesley D, Juni B, Rhame FS. *Aspergillus fumigatus* and other thermotolerant fungi generated by hospital building demolition. *Appl Environ Microbiol* 1983;46:375-8.
51. Hopkins CC, Weber DJ, Rubin RH. Invasive aspergillosis infection: possible non-ward common source within the hospital environment. *J Hosp Infect* 1989;13:19-25.
52. Denning DW. Invasive aspergillosis. *Clin Infect Dis* 1998;26:781-805.
53. Manuel RJ, Kibbler CC. The epidemiology and prevention of invasive aspergillosis. *J Hosp Infect* 1998;39:95-109.
54. Kennedy HF, Simpson EM, Wilson N, Richardson MD, Michie JR. *Aspergillus flavus* endocarditis in a child with neuroblastoma. *J Infect* 1998;36:126-7.
55. (75) McCarty JM, Flam MS, Pullen G, Jones R, Kassel SH. Outbreak of primary cutaneous aspergillosis related to intravenous arm boards. *J Pediatr* 1986;108(Pt.1):721-4.
56. Goldberg B, Eversmann WW, Eitzen EM Jr. Invasive aspergillosis of the hand. *J Hand Surg* 1982;7:38-42.
57. Grossman ME, Fithian EC, Behrens C, Bissinger J, Fracaro M, Neu HC. Primary cutaneous aspergillosis in six leukemic children. *J Am Acad Dermatol* 1985;12:313-8.
58. Panke TW, McManus AT, Spebar MJ. Infection of a burn wound by *Aspergillus niger*: gross appearance simulating ecthyma gangrenosa. *Am J Clin Pathol* 1979;72:230-2.
59. Fraser DW, Ward JL, Ajello L, Plikaytis BD. Aspergillosis and other systemic mycoses: the growing problem. *JAMA* 1979;242:1631-5.
60. Iwen PC, Reed EC, Armitage JO, et al. Nosocomial invasive aspergillosis in lymphoma patients treated with bone marrow or peripheral stem cell transplants. *Infect Control Hosp Epidemiol* 1993;14:131-9.
61. Cordonnier C, Bernaudin JF, Bierling P, Huet Y, Vernant JP. Pulmonary complications occurring after allogeneic bone marrow transplantation: a study of 130 consecutively transplanted patients. *Cancer* 1986;58:1047-54.
62. (76) Klimowski LL, Rotstein C, Cummings KM. Incidence of nosocomial aspergillosis in patients with leukemia over a twenty-year period. *Infect Control Hosp Epidemiol* 1989;10:299-305.
63. (79) Walmsley S, Devi S, King S, Schneider R, Richardson S, Ford-Jones L. Invasive *Aspergillus* infections in a pediatric hospital: a ten-year review. *Pediatr Infect Dis* 1993;12:673-82.
64. (57) Pannuti CS, Gingrich RD, Pfaller MA, Wenzel RP. Nosocomial pneumonia in adult patients undergoing bone marrow transplantation: a 9-year study. *J Clin Oncol* 1991;9:77-84.
65. (58) Wingard JR, Beals SU, Santos GW, Mertz WG, Saral R. *Aspergillus* infections in bone marrow transplant recipients. *Bone Marrow Transplant* 1987;2:175-81.
66. Humphreys H, Johnson EM, Warnock DW, Willatts SM, Winter RJ, Speller DCE. An outbreak of aspergillosis in a general ITU. *J Hosp Infect* 1991;18:167-77.
67. Sessa A, Meroni M, Battini G, et al. Nosocomial outbreak of *Aspergillus fumigatus* infection among patients in a renal unit? *Nephrol Dial Transplant* 1996;11:1322-4.
68. (134) Anderson K, Morris G, Kennedy H, et al. Aspergillosis in immunocompromised pediatric patients: associations with building hygiene, design, and indoor air. *Thorax* 1996;51:256-61.
69. (39) Tabbara KF, al Jabarti A. Hospital construction-associated outbreak of ocular aspergillosis after cataract surgery. *Ophthalmology* 1998;105:522-26.
70. Ferre A, Domingo P, Alonso C, Franquet T, Gurgui M, Verger G. Invasive pulmonary aspergillosis: A study of 33 cases. *Med Clin (Barc)* 1998;110:421-5. (Spanish)
71. Ewig S, Paar WD, Pakos E et al. Nosocomial ventilator-associated pneumonias caused by *Aspergillus fumigatus* in non-immunosuppressed, non-neutropenic patients. *Pneumologie* 1998;52:85-90. (German)
72. Singer S, Singer D, Ruchel R, Mergeryan H, Schmidt U, Harms K. Outbreak of systemic aspergillosis in a neonatal intensive care unit. *Mycoses* 1998;41:223-7.
73. (88) Allo MD, Miller J, Townsend T, Tan C. Primary cutaneous aspergillosis associated with Hickman intravenous catheters. *N Engl J Med* 1987;317:1105-8.
74. Boon AP, Adams DH, Buckels J, McMaster P. Cerebral aspergillosis in liver transplantation. *J Clin Pathol* 1990;43:114-8.

75. Pla MP, Berenguer J, Arzuaga JA, Banares R, Polo JR, Bouza E. Surgical wound infection by *Aspergillus fumigatus* in liver transplant recipients. *Diagn Microbiol Infect Dis* 1992;15:703–6.
76. Kanj SS, Welty-Wolf K, Madden J, et al. Fungal infections in lung and heart-lung transplant recipients: report of 9 cases and review of the literature. *Medicine* 1996;75:142–56.
77. (77) Pfundstein J. *Aspergillus* infection among solid organ transplant recipients: a case study. *J Transpl Coord* 1997;7:187–9.
78. Brenier-Pinchart MP, Lebeau B, Devouassoux G, et al. *Aspergillus* and lung transplant recipients: a mycologic and molecular epidemiologic study. *J Heart Lung Transplant* 1998;17:972–9.
79. (59) Gerson SL, Talbot GH, Hurwitz S, Strom B, Lusk EJ, Cassileth PA. Prolonged granulocytopenia: the major risk factor for invasive pulmonary aspergillosis in patients with leukemia. *Ann Intern Med* 1984;100:345–51.
80. Weber SF, Peacock JE Jr, Do KA, Cruz JM, Powell BL, Capizzi RL. Interaction of granulocytopenia and construction activity as risk factors for nosocomial invasive filamentous fungal disease in patients with hematologic disorders. *Infect Control Hosp Epidemiol* 1990;11:235–42.
81. Rees JR, Pinner RW, Hajjeh RA, Brandt ME, Reingold AL. The epidemiological features of invasive mycotic infections in the San Francisco Bay area, 1992–1993: results of population-based laboratory active surveillance. *Clin Infect Dis* 1998;27:1138–47.
82. McNeil MM, Nash SL, Hajjeh RA, Conn LA, Plikaytis BD. Trends in mortality due to invasive mycoses in the United States [abstract]. In: Program & Abstracts of the International Conference on Emerging Infectious Diseases. Atlanta, GA, 1998. Abstract No. S7.3.
83. Wald A, Leisenring W, van Burik JA, Bowden RA. Epidemiology of *Aspergillus* infections in a large cohort of patients undergoing bone marrow transplantation. *J Infect Dis* 1997;175:1459–66.
84. Gurwith MJ, Stinson EB, Remington JS. *Aspergillus* infection complicating cardiac transplantation: Report of five cases. *Arch Intern Med* 1971;128:541–5.
85. Weiland D, Ferguson RM, Peterson PK, Snover DC, Simmons RL, Najarian JS. Aspergillosis in 25 renal transplant patients. *Ann Surg* 1983;198:622–9.
86. Hofflin JM, Potasman I, Baldwin JC, Oyster PE, Stinson EB, Remington JS. Infectious complications in heart transplant recipients receiving cyclosporine and corticosteroids. *Ann Intern Med* 1987;106:209–16.
87. Schulman LL, Smith CR, Drusin R, Rose EA, Enson Y, Reemtsma K. Respiratory complications of cardiac transplantation. *Am J Med Sci* 1988;296:1–10.
88. Gustafson TL, Schaffner W, Lavelly GB, Stratton CW, Johnson HK, Hutcheson RH. Invasive aspergillosis in renal transplant recipients: correlation with corticosteroid therapy. *J Infect Dis* 1983;148:230–8.
89. Denning DW, Stevens DA. Antifungal and surgical treatment of invasive aspergillosis: review of 2121 published cases. *Rev Infect Dis* 1990;12:1147–201.
90. Weinberger M, Elattaar I, Marshall D, et al. Patterns of infection in patients with aplastic anemia and the emergence of *Aspergillus* as a major cause of death. *Medicine* 1992;71:24–43.
91. Noble WC, Clayton YM. Fungi in the air of hospital wards. *J Gen Microbiol* 1963;32:397–402.
92. Solomon WR, Burge HP, Boise JR. Airborne *Aspergillus fumigatus* levels outside and within a large clinical center. *J Allergy Clin Immunol* 1978;62:56–60.
93. Streifel AJ, Rhame FS. Hospital air filamentous fungal spore and particle counts in a specially designed hospital. In: *Indoor Air '93: Proceedings of the Sixth International Conference on Indoor Air and Climate*, Vol. 4. Helsinki, Finland:161–5.
94. (40) Rhame FS, Streifel AJ, Kersey JH Jr, McGlave PB. Extrinsic risk factors for pneumonia in the patient at high risk for infection. *Am J Med* 1984;76(5A):42–52.
95. (78) Rhame FS, Streifel A, Stevens P, et al. Endemic *Aspergillus* airborne spore levels are a major risk factor for aspergillosis in bone marrow transplant patients [abstract]. In: *Abstracts of the 25th Interscience Conference on Antimicrobial Agents and Chemotherapy 1985*. Abstract No. 147.
96. (60) Lentino JR, Rosenkranz MA, Michaels JA, Kurup VP, Rose HD, Rytel MW. Nosocomial aspergillosis: a retrospective review of airborne disease secondary to road construction and contaminated air conditioners. *Am J Epidemiol* 1982;116:430–7.
97. (49) Krasinski K, Holzman RS, Hanna B, Greco MA, Graff M, Bhogal M. Nosocomial fungal infection during hospital renovation. *Infect Control* 1985;6:278–82.
98. (29) Gage AA, Dean DC, Schimert G, Minsley N. *Aspergillus* infection after cardiac surgery. *Arch Surg* 1970;101:384–87.

99. **(95)** Siegler L, Kennedy MJ. *Aspergillus, Fusarium*, and other opportunistic moniliaceous fungi. In: Murray PR, Baron EJ, Pfaller MA, Tenoer FC, Tenover RH, eds. Manual of clinical microbiology, 7th ed. Washington, DC: American Society for Microbiology Press, 1999;1212–41.
100. **(70)** Overberger PA, Wadowsky RM, Schaper MM. Evaluation of airborne particulates and fungi during hospital renovation. *Am Ind Hyg Assoc J* 1995;56:706–12.
101. **(96)** Breton P, Germaud P, Morin O, Audoin AF, Milpied N, Harousseau JL. Unusual pulmonary mycoses in patients with hematologic disease. *Rev Pneumol Clin* 1998;54:253–7. (French)
102. **(97)** Guarro J, Nucci M, Akiti T, Gené J, Barreiro MDGC, Gonçalves RT. Fungemia due to *Fusarium sacchari* in an immunosuppressed patient. *J Clin Microbiol* 2000;38:419–21.
103. **(98)** Burton JR, Zachery JB, Bessin R, et al. Aspergillosis in four renal transplant patients: diagnosis and effective treatment with amphotericin B. *Ann Intern Med* 1972;77:383–8.
104. **(80)** Kyriakides GK, Zinneman HHA, Hall WH, et al. Immunologic monitoring and aspergillosis in renal transplant patients. *Am J Surg* 1976;131:246–52.
105. Simmons RB, Price DL, Noble JA, Crow SA, Ahearn DG. Fungal colonization of air filters from hospitals. *Am Ind Hyg Assoc J* 1997;58:900–4.
106. **(4)** Pittet D, Huguenin T, Dharan S, et al. Unusual case of lethal pulmonary aspergillosis in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 1996;154(2 Pt 1):541–4.
107. **(104)** Mahoney DH Jr, Steuber CP, Starling KA, Barrett FF, Goldberg J, Fernbach DJ. An outbreak of aspergillosis in children with acute leukemia. *J Pediatr* 1979;95:70–2.
108. Ruutu P, Valtonen V, Tiitonen L, et al. An outbreak of invasive aspergillosis in a hematologic unit. *Scand J Infect Dis* 1987;19:347–51.
109. **(51)** Walsh TJ, Dixon DM. Nosocomial aspergillosis: environmental microbiology, hospital epidemiology, diagnosis, and treatment. *Eur J Epidemiol* 1989;5:131–42.
110. Buffington J, Reporter R, Lasker BA, et al. Investigation of an epidemic of invasive aspergillosis: utility of molecular typing with the use of random amplified polymorphic DNA probes. *Pediatr Infect Dis J* 1994;13:386–93.
111. **(44)** Gerson SL, Parker P, Jacobs MR, Creger R, Lazarus HM. Aspergillosis due to carpet contamination [letter]. *Infect Control Hosp Epidemiol* 1994;15:221–3.
112. **(84)** Fox BC, Chamberlin L, Kulich P, Rae EJ, Webster LR. Heavy contamination of operating room air by *Penicillium* species: identification of the source and attempts at decontamination. *Am J Infect Control* 1990;18:300–6.
113. Chazalet V, Debeauvais J-P, Sarfati J, et al. Molecular typing of environmental and patient isolates of *Aspergillus fumigatus* from various hospital settings. *J Clin Microbiol* 1998;36:1494–500.
114. Loudon KW, Coke AP, Burnie JP, Shaw AJ, Oppenheim BA, Morris CQ. Kitchens as a source of *Aspergillus niger* infection. *J Hosp Infect* 1996;32:191–8.
115. **(81)** Abzug MJ, Gardner S, Glode MP, Cymanski M, Roe MH, Odom LF. Heliport-associated nosocomial mucormycoses [letter]. *Infect Control Hosp Epidemiol* 1992;13:325–6.
116. Alvarez M, Lopez Ponga B, Rayon C, et al. Nosocomial outbreak caused by *Scedosporium prolificans (inflatum)*: four fatal cases in leukemic patients. *J Clin Microbiol* 1995;33:3290–5.
117. **(89)** Schleupner CJ, Hamilton JR. A pseudoepidemic of pulmonary fungal infections related to fiberoptic bronchoscopy. *Infect Control* 1980;1:38–42.
118. Jackson L, Klotz SA, Normand RE. A pseudoepidemic of *Sporothrix cyanescens* pneumonia occurring during renovation of a bronchoscopy suite. *J Med Vet Mycol* 1990;28:455–9.
119. **(30)** Vargo JA, Ginsberg MM, Mizrahi M. Human infestation by the pigeon mite: a case report. *Am J Infect Control* 1983;11:24–5.
120. **(1)** American Institute of Architects. Guidelines for design and construction of hospital and health care facilities, 2001. Washington, DC: American Institute of Architects Press, 2001.
121. Diamond RD. *Cryptococcus neoformans*. In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of Infectious Diseases, 5th Ed. Philadelphia, PA: Churchill Livingstone, 2000;2707–18.
122. Deepe GS Jr. *Histoplasma capsulatum*. In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of Infectious Diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2718–33.
123. Brodsky AL, Gregg MB, Loewenstein MS, et al. Outbreak of histoplasmosis associated with the 1970 earth day activities. *Am J Med* 1973;54:333–42.
124. Ward JI, Weeks M, Allen D, et al. Acute histoplasmosis: clinical, epidemiologic, and serologic findings of an outbreak associated with exposure to a fallen tree. *Am J Med* 1979;66:587–95.

125. Galgiani JN. Coccidioidomycoses. In: Remington JS, Swartz MN, eds. Current clinical topics in infectious disease. Malden, MA: Blackwell Science, 1997;188–204.
126. **(111)** Gerberding JL. Nosocomial transmission of opportunistic infections. *Infect Control Hosp Epidemiol* 1998;19:574–7.
127. Hughes WT. Natural mode of acquisition for de novo infection with *Pneumocystis carinii*. *J Infect Dis* 1982;145:842–8.
128. Olsson M, Sukura A, Lindberg LA, et al. Detections of *Pneumocystis carinii* DNA by filtration of air. *Scand J Infect Dis* 1996;28:279–82.
129. Bartlett MS, Vermund SH, Jacobs R, et al. Detection of *Pneumocystis carinii* DNA in air samples: likely environmental risk to susceptible persons. *J Clin Microbiol* 1997;35:2511–3.
130. Lundgren B, Elvin K, Rothman LP, Ljungstrom I, Lidman C, Lundgren JD. Transmission of *Pneumocystis carinii* from patients to hospital staff. *Thorax* 1997;52:422–4.
131. **(112)** Vargas SL, Ponce CA, Gigliotti F, et al. Transmission of *Pneumocystis carinii* DNA from a patient with *P. carinii* pneumonia to immunocompetent contact health care workers. *J Clin Microbiol* 2000;38:1536–8.
132. **(113)** Walzer PD. *Pneumocystis carinii*. In: Mandell GL, Bennett JE, Dolin R. eds. Principles and practice of infectious diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2781–95.
133. CDC. Screening for tuberculosis and tuberculosis infection in high-risk populations: recommendations of the Advisory Committee for Elimination of Tuberculosis. *MMWR* 1995;44(No. RR-11):18–34.
134. CDC. Targeted tuberculin testing and treatment of latent tuberculosis infection. *MMWR* 2000;49(No. RR-6):1–51.
135. D'Agata EMC, Wise S, Stewart A, Lefkowitz LB Jr. Nosocomial transmission of *Mycobacterium tuberculosis* from an extrapulmonary site. *Infect Control Hosp Epidemiol* 2001;22:10–2.
136. CDC. Summary of notifiable diseases, United States 2001. *MMWR* 2001;50(53):1–108.
137. Haas DW. *Mycobacterium tuberculosis*. In: Mandell GL, Bennett JE, Dolin R. eds. Principles and practice of infectious diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2576–607.
138. American Public Health Association. Tuberculosis. In: Chin J, ed. Control of communicable diseases manual, 17th ed. Washington, DC: American Public Health Association Press, 2000:521–30.
139. American Thoracic Society/CDC. Treatment of tuberculosis. *Am J Respir Crit Care Med* 2003;167:603–62.
140. Matlow AG, Jarrison A, Monteath A, Roach P, Balfe JW. Nosocomial transmission of tuberculosis (TB) associated with care of an infant with peritoneal TB. *Infect Control Hosp Epidemiol* 2000;21:222–3.
141. Jensen PA. Airborne *Mycobacterium* spp. In: Hurst CJ, Knudsen GR, McInerney MJ, Stetzenbach LD, Walter MV, eds. Manual of environmental microbiology. Washington, DC: American Society for Microbiology Press, 1997;676–81.
142. **(41)** Wells WF. Aerodynamics of droplet nuclei. In: Airborne contagion and air hygiene. Cambridge, MA: Harvard University Press, 1955;13–9.
143. White A. Relation between quantitative nasal cultures and dissemination of staphylococci. *J Lab Clin Med* 1961;58:273–7.
144. Huijsmans-Evers AG. Results of routine tests for the detection of dispersers of *Staphylococcus aureus*. *Arch Chir Neerl* 1978;30:141–50.
145. Boyce JM, Opal SM, Potter-Bynoe G, Medeiros AA. Spread of methicillin-resistant *Staphylococcus aureus* in a hospital after exposure to a healthcare worker with chronic sinusitis. *Clin Infect Dis* 1993;17:496–504.
146. Hambræus A, Sanderson HF. The control of ventilation of airborne bacterial transfer between hospital patients, and its assessment by means of a particle tracer. 3. Studies with an airborne-particle tracer in an isolation ward for burned patients. *J Hyg (Lond)* 1972;70:299–312.
147. Nakashima AK, Allen JR, Martone WJ, et al. Epidemic bullous impetigo in a nursery due to a nasal carrier of *Staphylococcus aureus*: role of epidemiology and control measures. *Infect Control* 1984;5:326–31.
148. Bethune DW, Blowers R, Parker M, Pask EA. Dispersal of *Staphylococcus aureus* by patients and surgical staff. *Lancet* 1965;1:480–3.
149. Sherertz RJ, Reagan DR, Hampton KD, et al. A cloud adult: the *Staphylococcus aureus* — virus interaction revisited. *Ann Intern Med* 1996;124:539–47.
150. Gryska PF, O'Dea AE. Postoperative streptococcal wound infection: the anatomy of an epidemic. *JAMA* 1970;213:1189–91.
151. Stamm WE, Feeley JC, Facklam RR. Wound infection due to group A *Streptococcus* traced to a vaginal carrier. *J Infect Dis* 1978;138:287–92.

152. Berkelman RL, Martin D, Graham DR. Streptococcal wound infection caused by a vaginal carrier. *JAMA* 1982;247:2680–2.
153. McIntyre DM. An epidemic of *Streptococcus pyrogenes* puerpural and postoperative sepsis with an unusual site — the anus. *Am J Obstet Gynecol* 1968;101:308–14.
154. Gaynes RP, Horan TC. Surveillance of nosocomial infections. In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;1285–318.
155. Wenzel RP, Veazey JM Jr, Townsend TR. Role of the inanimate environment in hospital-acquired infections. In: Cundy KR, Ball W, eds. *Infection control in healthcare facilities: microbiological surveillance*. Baltimore, MD: University Park Press, 1977;71–98.
156. Mortimer EA Jr, Wolinsky E, Gonzaga AJ, Rammelkamp CH Jr. Role of airborne transmission in staphylococcal infections. *Br Med J* 1966;5483:319–22.
157. Youngs ER, Roberts C, Kramer JM, Gilbert RJ. Dissemination of *Bacillus cereus* in a maternity unit. *J Infect* 1985;10:228–32.
158. Richardson AJ, Rothburn MM, Roberts C. Pseudo-outbreak of *Bacillus* species: related to fiberoptic bronchoscopy. *J Hosp Infect* 1986;7:208–10.
159. Bryce EA, Smith JA, Tweeddale M, Andruschak BJ, Maxwell MR. Dissemination of *Bacillus cereus* in an intensive care unit. *Infect Control Hosp Epidemiol* 1993;14:459–62.
160. Lie PY-F, Ke S-C, Chen S-L. Use of pulsed-field gel electrophoresis to investigate a pseudo-outbreak of *Bacillus cereus* in a pediatric unit. *J Clin Microbiol* 1997;35:1533–5.
161. McDonald LC, Walker M, Carson L, et al. Outbreak of *Acinetobacter* spp. bloodstream infections in a nursery associated with contaminated aerosols and air conditioners. *Pediatr Infect Dis J* 1998;17:716–22.
162. Leclair JM, Zaia JA, Levin MJ, Congdon RG, Goldmann DA. Airborne transmission of chickenpox in a hospital. *N Engl J Med* 1980;302:450–3.
163. Gustafson TL, Lavelly GB, Brawner ERJ, Hutcheson RHJ, Wright PF, Schaffner W. An outbreak of airborne nosocomial varicella. *Pediatrics* 1982;70:550–6.
164. Josephson A, Gombert ME. Airborne transmission of nosocomial varicella from localized zoster. *J Infect Dis* 1988;158:238–41.
165. Sawyer MJ, Chamberlin CJ, Wu YN, Aintablian N, Wallace MR. Detection of varicella-zoster virus DNA in air samples from hospital rooms. *J Infect Dis* 1994;169:91–4.
166. Menkhous NA, Lamphear B, Linnemann CC. Airborne transmission of varicella-zoster virus in hospitals. *Lancet* 1990;226:1315.
167. CDC. Prevention of varicella: recommendations of the advisory committee on immunization practices (ACIP). *MMWR* 1996;45(No. RR-11):1–36.
168. Davis RM, Orenstein WA, Frank JAJ, et al. Transmission of measles in medical settings: 1980 through 1984. *JAMA* 1986;255:1295–8.
169. Watkins NM, Smith RPJ, St Germain DL, Mackay DN. Measles infection in a hospital setting. *Am J Infect Control* 1987;15:201–6.
170. Revera ME, Mason WH, Ross LA, Wright HT Jr. Nosocomial measles infection in a pediatric hospital during a community-wide epidemic. *J Pediatr* 1991;119:183–6.
171. Atkinson WL, Markowitz LE, Adams NC, Seastrom GR. Transmission of measles in medical settings — United States, 1985–1989. *Am J Med* 1991;91(suppl):320S–4S.
172. Patriarca PA, Weber JA, Parker RA, et al. Efficacy of influenza vaccine in nursing homes: reduction in illness and complications during influenza A (H3N2) epidemics. *JAMA* 1985;253:1136–9.
173. Arden NH, Patriarca PA, Fasano MB, et al. The roles of vaccination and amantadine prophylaxis in controlling an outbreak of influenza A (H3N2) in a nursing home. *Arch Intern Med* 1988;148:865–8.
174. CDC. Influenza A outbreaks — Louisiana, August 1993. *MMWR* 1993;42:132–4.
175. Drinka PJ, Gravenstein S, Krause P, Schilling M, Miller BA, Shult P. Outbreaks of influenza A and B in a highly immunized nursing home population. *J Family Practice* 1997;45:509–14.
176. Schilling M, Povinelli L, Krause P, et al. Efficacy of zanamivir for chemoprophylaxis of nursing home influenza outbreaks. *Vaccine* 1998;16:1771–4.
177. Hall CB. Nosocomial viral infections: perennial weeds on pediatric wards. *Am J Med* 1981;70:670–6.
178. Whimbey E, Elting LS, Couch RB, et al. Influenza A virus infections among hospitalized adult bone marrow transplant recipients. *Bone Marrow Transpl* 1994;13:437–40.
179. Evans ME, Hall KL, Berry SE. Influenza control in acute care hospitals. *Am J Infect Control* 1997;25:357–62.

180. Munoz FM, Campbell JR, Atmar RL, et al. Influenza A virus outbreak in a neonatal intensive care unit. *Pediatr Infect Dis J* 1999;18:811–5.
181. Alford RH, Kasel JA, Gerone PJ, Knight V. Human influenza resulting from aerosol inhalation. *Proc Soc Exp Biol Med* 1966;122:800–4.
182. Moser MR, Bender TR, Margolis HS, Noble GR, Kendal AP, Ritter DG. An outbreak of influenza aboard a commercial airliner. *Am J Epidemiol* 1979;110:1–6.
183. Chanock RW, Kim HW, Vargosko AJ, et al. Respiratory syncytial virus 1: virus recovery and other observations during 1960 — outbreak of bronchiolitis, pneumonia, and other minor respiratory illness in children. *JAMA* 1961;176:647–53.
184. Gardner DS, Court SDM, Brocklebank JT, et al. Virus cross-infection in paediatric wards. *Br Med J* 1973;2:571–75.
185. Sawyer LA, Murphy JJ, Kaplan JE, et al. 25–30 nm virus particle associated with a hospital outbreak of acute gastroenteritis with evidence for airborne transmission. *Am J Epidemiol* 1988;127:1261–71.
186. Baxby D. Poxviruses. In: Belshe RB, ed. *Textbook of human virology*, 2nd ed. St. Louis, MO: Mosby Year Book, 1991;930–46.
187. Neff JM. Variola (smallpox) and monkeypox viruses. In: Mandell GL, Bennett JE, Dolin R. eds. *Principles and practice of infectious diseases*, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;1555–6.
188. Wehrle PF, Posch J, Richter KH, Henderson DA. An airborne outbreak of smallpox in a German hospital and its significance with respect to other recent outbreaks in Europe. *Bull WHO* 1970;43:669–79.
189. Hawkes N. Science in Europe: smallpox death in Britain challenges presumption of laboratory safety. *Science* 1979;203:855–6.
190. Eickhoff TC. Airborne nosocomial infection: a contemporary perspective. *Infect Control Hosp Epidemiol* 1994;15:663–72.
191. Nuzum EO, Rossi CA, Stephenson EH, LeDuc JW. Aerosol transmission of Hantaan and related viruses to laboratory rats. *Am J Trop Med Hyg* 1988;38:636–40.
192. CDC. Hantavirus infection — southwestern United States: Interim recommendations for risk reduction. *CDC. MMWR* 1993;42(No. RR-11):1–13.
193. Vitek CR, Breiman RF, Ksiazek TG, et al. Evidence against person-to-person transmission of hantavirus to health care workers. *Clin Infect Dis* 1996;22:824–6.
194. Wells RM, Young J, Williams RJ, et al. Hantavirus transmission in the United States. *Emerg Infect Dis* 1997;3:361–5.
195. Chaparro J, Vega J, Terry W, et al. Assessment of person-to-person transmission of hantavirus pulmonary syndrome in a Chilean hospital setting. *J Hosp Infect* 1998;40:281–5.
196. Nolte KB, Foucar K, Richmond JY. Hantaviral biosafety issues in the autopsy room and laboratory: Concerns and recommendations. *Hum Pathol* 1996;27:1253–4.
197. Stephenson EH, Larson EW, Dominik JW. Effect of environmental factors on aerosol-induced Lassa virus infection. *J Med Virol* 1984;14:295–303.
198. Monath TP. Lassa fever: review of epidemiology and epizootiology. *Bull World Health Organ* 1975;52:577–92.
199. Monath TP, Casals J. Diagnosis of Lassa fever and the isolation and management of patients. *Bull WHO* 1975;52:707–15.
200. Zweighaft RM, Fraser DW, Hattwick MA, et al. Lassa fever: response to an imported case. *N Engl J Med* 1977;297:803–7.
201. Cooper CB, Gransden WR, Webster M, et al. A case of Lassa fever: experience at St Thomas' hospital. *Br Med J (Clin Res Ed)* 1982;285:1003–5.
202. **(108)** Monath TP. Yellow fever: Victor, victoria? Conqueror, conquest? Epidemics and research in the last forty years and prospects for the future. *Am J Trop Med Hyg* 1991;45:1–43.
203. **(109)** CDC. Update: management of patients with suspected viral hemorrhagic fever — United States. *MMWR* 1995;44:475–9.
204. **(110)** Weber DJ, Rutala WA. Risks and prevention of nosocomial transmission rare zoonotic diseases. *Clin Infect Dis* 2001;32:446–56.
205. Decker MD, Schaffner W. Nosocomial diseases of healthcare workers spread by the airborne or contact routes (other than tuberculosis). In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;1101–26.

206. **(46)** Fridkin SK, Kremer FB, Bland LA, Padhye A, McNeil MM, Jarvis WR. *Acremonium kiliense* endophthalmitis that occurred after cataract extraction in an ambulatory surgical center and was traced to an environmental reservoir. *Clin Infect Dis* 1996;22:222–7.
207. Loeb M, Wilcox L, Thornley D, et al. *Bacillus* species pseudobacteremia following hospital construction. *Can J Infect Control* 1995;10:37–40.
208. Olle-Goig JE, Canela-Soler J. An outbreak of *Brucella melitensis* infection by airborne transmission among laboratory workers. *Am J Public Health* 1987;77:335–8.
209. Kiel FW, Khan MY. Brucellosis among hospital employees in Saudi Arabia. *Infect Control Hosp Epidemiol* 1993;14:268–72.
210. Staszkiwicz J, Lewis CM, Colville J, Zervos M, Band J. Outbreak of *Brucella melitensis* among microbiology laboratory workers in a community hospital. *J Clin Microbiol* 1991;20:287–90.
211. Fiori PL, Mastrandrea S, Rappelli P, Cappuccinelli P. *Brucella abortus* infection acquired in microbiology laboratories. *J Clin Microbiol* 2000;38:2005–6.
212. Spinelli JS, Ascher MS, Brooks DL, et al. Q fever crisis in San Francisco: controlling a sheep zoonosis in a lab animal facility. *Lab Anim* 1981;10:29–38.
213. **(Table 1)** American Conference of Governmental Industrial Hygienists (ACGIH). HVAC components, functions and malfunctions (topic 8-4). In: *Industrial ventilation: a Manual of recommended practice*, 24th ed. Cincinnati, OH : American Conference of Governmental Industrial Hygienists, Inc., 2001.
214. **(35)** American Society of Heating, Refrigerating and Air-conditioning Engineers, Inc. Ventilation for Indoor Air Quality. ASHRAE Standard 62-1999. Atlanta, GA: ASHRAE 1999;1–27.
215. **(133)** Burroughs HEB. Sick building syndrome: fact, fiction, or facility? In: Hansen W, ed. *A guide to managing indoor air quality in health care organizations*. Oakbrook Terrace, IL: Joint Commission on Accreditation of Health Care Organizations, 1997;3–13.
216. American Society of Heating, Refrigerating and Air-conditioning Engineers, Inc. Gravimetric and dust spot procedures for testing air cleaning devices used in general ventilation for removing particulate matter. ANSI/ASHRAE Standard 52–1–1999. Atlanta, GA: ASHRAE 1999;1–25.
217. Robinson TJ, Ouellet AE. Filters and filtration. *ASHRAE J* 1999;65–70.
218. Dryden GE, Dryden SR, Brown DG, Schatzle KC, Godzeski C. Performance of bacteria filters. *Respir Care* 1980;25:1127–35.
219. **(33)** Rutala WA, Jones SM, Worthington JM, Reist PC, Weber DJ. Efficacy of portable filtration units in reducing aerosolized particles in the size range of *Mycobacterium tuberculosis*. *Infect Control Hosp Epidemiol* 1995;16:391–8.
220. **(5)** U.S. Environmental Protection Agency, Office of Air and Radiation and U.S. Department of Health & Human Services, National Institute of Occupational Safety and Health. *Building air quality: a guide for building owners and facilities managers*. Washington, DC: USEPA, 1991. EPA/400/1-91/033, or NIOSH 91-114. Available at: www.cdc.gov/niosh/baqtoc.html
221. **(28)** Ko G, Burge HA, Muileberg M, Rudnick S, First M. Survival of mycobacteria on HEPA filter material. *J Am Biol Safety Assoc* 1998;3:65–78.
222. **(6)** Rao CY, Burge HA, Chang JCS. Review of quantitative standards and guidelines for fungi in indoor air. *J Air & Waste Manage Assoc* 1996;46:899–906.
223. Riley RL, Wells WF, Mills CC, Nyka W, McLean RL. Air hygiene in tuberculosis: quantitative studies of infectivity and control in a pilot ward. *Am Rev Tuberc* 1957;75:420–31.
224. Riley RL, Nardell EA. Cleaning the air: the theory and application of UV air disinfection. *Am Rev Respir Dis* 1989;139:1286–94.
225. Riley RL. Ultraviolet air disinfection for control of respiratory contagion. In: Kundsinn RB, ed. *Architectural design and indoor microbial pollution*. New York, NY: Oxford University Press, 1988;174–97.
226. Willmon TL, Hollaender A, Langmuir AD. Studies of the control of acute respiratory diseases among naval recruits. I. A review of a four-year experience with ultraviolet irradiation and dust suppressive measures, 1943 to 1947. *Am J Hyg* 1948;48:227–32.
227. Wells WF, Wells MW, Wilder TS. The environmental control of epidemic contagion. I. An epidemiologic study of radiant disinfection of air in day schools. *Am J Hyg* 1942;35:97–121.
228. Perkins JE, Bahlke AM, Silverman HF. Effect of ultra-violet irradiation of classrooms on spread of measles in large rural central schools. *Am J Public Health Nations Health* 1947;37:529–37.
229. Nagy R. Application and measurement of ultraviolet radiation. *Am Ind Hyg Assoc J* 1964;25:274–81.
230. Illuminating Engineering Society. *IES Lighting handbook*, 4th ed. New York, NY: Illuminating Engineering Society, 1966;25–7.

231. Riley RL. Indoor spread of respiratory infection by recirculation of air. *Bull Physiopathol Respir* 1979;15:699–705.
232. Menzies D, Pasztor J, Rand T, Bourbeau J. Germicidal ultraviolet irradiation in air conditioning systems: effect on office worker health and wellbeing — a pilot study. *Occup Environ Med* 1999;56:397–402.
233. Riley RL, Permutt S. Room air disinfection by ultraviolet irradiation of upper air: air mixing and germicidal effectiveness. *Arch Environ Health* 1971;22:208–19.
234. Nicas M, Miller SL. A multi-zone model evaluation of the efficacy of upper-room air ultraviolet germicidal irradiation. *Appl Occup Environ Hyg* 1999;14:317–28.
235. Kethley TW, Branch K. Ultraviolet lamps for room air disinfection: effect of sampling location and particle size of bacterial aerosol. *Arch Environ Health* 1972;25:205–14.
236. Riley RL, Knight M, Middlebrook G. Ultraviolet susceptibility of BCG and virulent tubercle bacilli. *Am Rev Respir Dis* 1976;113:413–8.
237. Collins FM. Relative susceptibility of acid-fast and non-acid-fast bacteria to ultraviolet light. *Appl Microbiol* 1971;21:411–3.
238. Riley RL, Permutt S, Kaufman JE. Convection, air mixing, and ultraviolet air disinfection in rooms. *Arch Environ Health* 1971;22:200–7.
239. Nardell EA. Fans, filters, or rays? Pros and cons of the current environmental tuberculosis control technologies. *Infect Control Hosp Epidemiol* 1993;14:681–5.
240. ECRI. Health devices evaluation of mobile high efficiency filter air cleaners (MHEFACs). *ECRI* 1997;26:367–88.
241. **(103)** American Society of Heating, Refrigerating, and Air-Conditioning Engineers. 1999 ASHRAE Handbook: heating, ventilating, and air-conditioning applications. Chapter 7: Health care facilities. Atlanta, GA: ASHRAE, 1999;7.1–7.13.
242. Elovitz KM. Understanding what humidity does and why. *ASHRAE J* 1999;April:84–90.
243. Orme I. Patient impact. In: Hansen W, ed. *A guide to managing indoor air quality in health care organizations*. Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997:43–52.
244. Gundermann KO. Spread of microorganisms by air-conditioning systems — especially in hospitals. *Ann NY Acad Sci* 1980;209–17.
245. Arundel AV, Sterling EM, Biggin JH, Sterling TD. Indirect health effects of relative humidity in indoor environments. *Environ Health Perspect* 1986;65:351–61.
246. U.S. Environmental Protection Agency. *Ventilation and air quality in offices*. Washington, DC: EPA Document #402-F-94-003, Revision: July 1990.
247. Hermans RD, Streifel AJ. Ventilation design. In: Bierbaum PJ, Lippman M, eds. *Workshop on engineering controls for preventing airborne infections in workers in health care and related facilities*. Cincinnati, OH: NIOSH and CDC, 1993;107–46.
248. Memarzadeh F, Jiang J. A methodology for minimizing risk from airborne organisms in hospital isolation rooms. *ASHRAE Trans* 2000;106:731–47.
249. **(11)** Hansen W. The need for an integrated indoor air quality program. In: Hansen W, ed. *A guide to managing indoor air quality in health care organizations*. Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997;xiii – xviii.
250. **(12)** Bartley J. Ventilation. In: Pfeiffer J, ed. *APIC Text of infection control and epidemiology*. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc (APIC), 2000;77.1–77.11.
251. Levine AS, Siegel SE, Schreiber AD, et al. Protected environments and prophylactic antibiotics. *N Engl J Med* 1973;288:477–483.
252. **(90)** Denning DW, Clemons KV, Hanson LH, Stevens DA. Restriction endonuclease analysis of total cellular DNA of *Aspergillus fumigatus* isolates of geographically and epidemiologically diverse origin. *J Infect Dis* 1990;162:1151–8.
253. Rhame FS. Prevention of nosocomial aspergillosis. *J Hosp Infect* 1991;18 (Suppl. A):466–72.
254. **(85)** Barnes RA, Rogers TR. Control of an outbreak of nosocomial aspergillosis by laminar air-flow isolation. *J Hosp Infect* 1989;14:89–94.
255. Roy M-C. The operating theater: a special environmental area. In: Wenzel RP, ed. *Prevention and control of nosocomial infections*, 3rd ed. Baltimore, MD: William & Wilkins, 1997;515–38.
256. Pavelchak N, DePersis RP, London M, et al. Identification of factors that disrupt negative air pressurization of respiratory isolation rooms. *Infect Control Hosp Epidemiol* 2000;21:191–5.

257. Anderson K. *Pseudomonas pyocyanea* disseminated from an air cooling apparatus. *Med J Austr* 1959;529–32.
258. Shaffer JG, McDade JJ. Airborne *Staphylococcus aureus*: a possible source in air control equipment. *Arch Environ Health* 1963;5:547–51.
259. Morey PR. Building-related illness with a focus on fungal issues. In: Hansen W, ed. A guide to managing indoor air quality in health care organizations. Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997;15–25.
260. Streifel AJ. Recognizing IAQ risk and implementing an IAQ program. In: Hansen W, ed. A guide to managing indoor air quality in health care organizations; Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997;75–91.
261. Morey PR. Appendix B. Fungal growth checklist. In: Hansen W. ed. A guide to managing indoor air quality in health care organizations. Oakbrook Terrace, IL: Joint Commission on Accreditation of Health Care Organizations Publications, 1997;129–35.
262. Brock DL, Jiankang J, Rinaldi MG, Wickes BL, Huycke MM. Outbreak of invasive *Aspergillus* infection in surgical patients, associated with a contaminated air-handling system. *Clin Inf Dis* 2003;37:786–93.
263. (31) National Air Duct Cleaners Association. General specifications for the cleaning of commercial HVAC systems. Washington, DC: NADCA, 2002. Publication No. NAD-06. Available at: www.nadca.com/standards/standards.asp
264. (32) U.S. Environmental Protection Agency. Use of disinfectants and sanitizers in heating, ventilation, air conditioning, and refrigeration systems [letter]. March 14, 2002. Available at: www.epa.gov/oppad001/hvac.htm
265. U.S. Environmental Protection Agency. Should you have the air ducts in your home cleaned? Washington, DC: EPA, 1997. EPA Document No. 402-K-97-002.
266. (159) Vujanovic V, Smoragiewicz W, Krzysztyniak K. Airborne fungal ecological niche determination as one of the possibilities for indirect mycotoxin risk assessment in indoor air. *Environ Toxicol* 2001;16:1–8.
267. Soules WJ. Airflow management techniques. *Clean Rooms* 1993;2:18–20.
268. Lawson CN. Commissioning hospitals for compliance. *ASHRAE Trans* 1993;99(2).
269. Wadowsky R, Benner S. Distribution of the genus *Aspergillus* in hospital room air conditioners. *Infect Control* 1987;8:516–8.
270. Streifel AJ. Aspergillosis and construction. In: Kundsin RB, ed. Architectural design and indoor microbial pollution. New York, NY: Oxford University Press, 1988;198–217.
271. Streifel AJ, Vesley D, Rhame FS. Occurrence of transient high levels of airborne fungal spores. Proceedings of the 6th Conference on Indoor Air Quality and Climate. Toronto, ON: 1990.
272. (73) Morey R, Williams C. Porous insulation in buildings: a potential source of microorganisms. Proceedings - Indoor Air '90, 5th International Conference. Toronto, ON: 1990;1–6.
273. (13) Bartley J. Construction and renovation. In: Pfeiffer J, ed. APIC Text of infection control and epidemiology. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc., 2000; 72.1–72.11.
274. (14) Harvey MA. Critical-care-unit design and furnishing: Impact on nosocomial infections. *Infect Control Hosp Epidemiol* 1998;19:597–601.
275. (15) National Association of Children's Hospitals and Related Institutions. Patient Care Focus Groups 1998. Assessing organizational readiness for infection control issues related to construction, renovation, and physical plant projects.
276. (50) Bartley JM. APIC State-of-the-art report: the role of infection control during construction in health care facilities. *Am J Infect Control* 2000;28:156–9.
277. (16) Carter CD, Barr BA. Infection control issues in construction and renovation. *Infect Control Hosp Epidemiol* 1997;18:587–96.
278. (47) Streifel AJ. Maintenance and engineering. In: Pfeiffer J, ed. APIC Text of Infection Control and Epidemiology. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc., 2000;76.1-76.8.
279. Kennedy V, Barnard B, Hackett B. Use of a risk matrix to determine the level of barrier protection during construction activities. Atlanta, GA : Peachtree Publications, 1997;27–8.
280. Morey PR. Building-related illness with a focus on fungal issues. In: Hansen W, ed. A guide to managing indoor air quality in health care organizations. Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997;15–25.

281. (67) Bartley J, ed. Infection control tool kit series – construction and renovation. Washington, DC: Association for Professionals in Infection Control and Epidemiology, 1999.
282. Bryce EA, Walker M, Scharf S, et al. An outbreak of cutaneous aspergillosis in a tertiary-care hospital. *Infect Control Hosp Epidemiol* 1996;17:170–2.
283. (62) Thio CL, Smith D, Merz WG, et al. Refinements of environmental assessment during an outbreak investigation of invasive aspergillosis in a leukemia and bone marrow transplant unit. *Infect Control Hosp Epidemiol* 2000;21:18–23.
284. (65) Kuehn TH, Gacek B, Yang CH, et al. Final report: ASHRAE 804-RP Phase I identification of contaminants, exposures effects, and control options for construction/renovation activities. Atlanta, GA:ASHRAE, Inc. 1995.
285. Kennedy HF, Michie JR, Richardson MD. Air sampling for *Aspergillus* spp. during building activity in a paediatric hospital ward. *J Hosp Infect* 1995;31:322–25.
286. Leenders ACAP, van Belkum A, Behrendt M, Luijendijk AD, Verbrugh HA. Density and molecular epidemiology of *Aspergillus* in air and relationship to outbreaks of *Aspergillus* infection. *J Clin Microbiol* 1999;37:1752–7.
287. Rath PM, Ansorg R. Value of environmental sampling and molecular typing of aspergilli to assess nosocomial sources of aspergillosis. *J Hosp Infect* 1997;37:47–53.
288. (71) Streifel AJ, Marshall JW. Parameters for ventilation controlled environments in hospitals. In: Design, Construction, and Operation of Healthy Buildings. Atlanta, GA: ASHRAE Press, 1998.
289. (348) Streifel AJ. Air cultures for fungi. In: Gilchrist M, ed. Clinical microbiology procedures handbook. Washington, DC: American Society for Microbiology Press, 1992;11.8.1–11.8.7.
290. American Conference of Governmental Industrial Hygienists (ACGIH). 2000 Threshold limit Values and biological exposure indices. Cincinnati, OH: American Conference of Governmental Industrial Hygienists, Inc., 2000;1–184.
291. U.S. Department of Labor, Occupational Safety and Health Administration. Air contaminants standard. 29 CFR 1910.1000, §1910.1000, Tables Z-1, Z-3. *Federal Register* 1993;58:35338–51.
292. (86) Leenders A, vanBelkum A, Janssen S, et al. Molecular epidemiology of apparent outbreaks of invasive *Aspergillus* in a hematology ward. *J Clin Microbiol* 1996;34:345–51.
293. (91) James MJ, Lasker BA, McNeil MM, Shelton M, Warnock DW, Reiss E. Use of a repetitive DNA probe to type clinical and environmental isolates of *Aspergillus flavus* from a cluster of cutaneous infections in a neonatal intensive care unit. *J Clin Microbiol* 2000;38:3612–8.
294. (92) Skladny H, Buchheidt D, Baust C, et al. Specific detection of *Aspergillus* species in blood and bronchoalveolar lavage samples of immunocompromised patients by two-step PCR. *J Clin Microbiol* 1999;37:3865–71.
295. (93) Symoens F, Bouchara J-P, Heinemann S, Nolard N. Molecular typing of *Aspergillus terreus* isolates by random amplification of polymorphic DNA. *J Hosp Infect* 2000;44:273–80.
296. (94) Diaz-Guerra TM, Mellado E, Cuenca-Estrella M, Gaztelurrutia L, Villate Navarro JI, Rodríguez Tudela JL. Genetic similarity among one *Aspergillus flavus* strain isolated from a patient who underwent heart surgery and two environmental strains obtained from the operating room. *J Clin Microbiol* 2000;38:2419–22.
297. Buttner MP, Stetzenbach LD. Monitoring airborne fungal spores in an experimental indoor environment to evaluate sampling methods and the effects of human activity on air sampling. *Appl Environ Microbiol* 1993;59:219–26.
298. Sayer WJ, Shean DB, Ghosseiri J. Estimation of airborne fungal flora by the Anderson sampler versus the gravity settling culture plate. *J Allerg* 1969;44:214–27.
299. Hay RJ, Clayton YM, Goodley JM. Fungal aerobiology: how, when and where? *J Hosp Infect* 1995;30(Suppl):S352–7.
300. Morris G, Kokki MH, Anderson K, Richardson MD. Sampling of *Aspergillus* spores in air. *J Hosp Infect* 2000;44:81–92.
301. Iwen PC, Davis JC, Reed EC, Winfield BA, Hinrichs SH. Airborne fungal spore monitoring in a protective environment during hospital construction and correlation with an outbreak of invasive aspergillosis. *Infect Control Hosp Epidemiol* 1994;15:303–6.
302. Pegues DA, Lasker BA, McNeil MM, Hamm PM, Lundal JL, Kubak BM. Cluster of cases of invasive aspergillosis in a transplant intensive care unit: evidence of person-to-person airborne transmission. *Clin Infect Dis* 2002;34:412–6.

303. Goodley JM, Clayton YM, Hay RJ. Environmental sampling for aspergilli during building construction on a hospital site. *J Hosp Infect* 1994;26:27–35.
304. (72) American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE). The HVAC commissioning process. Atlanta, GA: ASHRAE, 1996;1–48. ASHRAE Guideline 1.
305. (63) Mermel LA, Josephson SL, Giorgio CH, Dempsey J, Parenteau S. Association of Legionnaires' disease with construction: contamination of potable water? *Infect Control Hosp Epidemiol* 1995;16:76–81.
306. Loo VG, Bertrand C, Dixon C, et al. Control of construction-associated nosocomial aspergillosis in an antiquated hematology unit. *Infect Control Hosp Epidemiol* 1996;17:360–4.
307. (68) Ottney TC. Particle management for HVAC systems. *ASHRAE J* 1993;35:26–28, 30, 32, 34.
308. Rautiala S, Reponen T, Nevalainen A, et al. Control of exposure to airborne viable microorganisms during remediation of moldy buildings: report of three case studies. *Am Ind Hyg Assoc J* 1998;59:455–60.
309. (69) Finkelstein LE, Mendelson MH. Infection control challenges during hospital renovation. *Am J Nursing* 1997;97:60–1.
310. Hruszkewycz V, Ruben B, Hypes CM, Bostic GD, Staszkiwicz J, Band JD. A cluster of pseudofungemia associated with hospital renovation adjacent to the microbiology laboratory. *Infect Control Hosp Epidemiol* 1992;13:147–50.
311. Laurel VL, Meier PA, Astorga A, Dolan D, Brockett R, Rinaldi MG. Pseudoepidemic of *Aspergillus niger* infections traced to specimen contamination in the microbiology laboratory. *J Clin Microbiol* 1999;37:1612–6.
312. (66) Opal SM, Asp AA, Cannady PB Jr, Morse PL, Burton LJ, Hammer II PG. Efficacy of infection control measures during a nosocomial outbreak of disseminated aspergillosis associated with hospital construction. *J Infect Dis* 1986;153: 634–7.
313. Fitzpatrick F, Prout S, Gilleece A, Fenelon LE, Murphy OM. Nosocomial aspergillosis during building work — a multidisciplinary approach. *J Hosp Infect* 1999;42:170–1.
314. Garrett DO, Jochimsen E, Jarvis W. Invasive *Aspergillus* spp. infections in rheumatology patients. *J Rheumatol* 1999;26:146–9.
315. Larsson L, Larsson PF. Analysis of chemical markers as a means of characterizing airborne microorganisms in indoor environments: a case study. *Indoor Built Environ* 2001;10:232–7.
316. (99) Buckner CD, Clift RA, Sanders JE, et al. Protective environment for marrow transplant recipients: a prospective study. *Ann Intern Med* 1978;89:893–901.
317. (100) Murray WA, Streifel AJ, O'Dea TJ, Rhame FS. Ventilation for protection of immune compromised patients. *ASHRAE Trans* 1988;94:1185–91.
318. (101) Streifel AJ, Vesley D, Rhame FS, Murray B. Control of airborne fungal spores in a university hospital. *Environment International* 1989;12:441–4.
319. Perry S, Penland WZ. The portable laminar flow isolator: new unit for patient protection in a germ-free environment. In: *Recent Results in Cancer Research*. New York, NY: Springer-Verlag, 1970.
320. Hayden CS, Fischbach M, Johnston OE. A model for calculating air leakage in negative pressure isolation areas. Cincinnati, OH: DHHS, 1997. NIOSH Report ECTR 212-05c.
321. DeLuga GF. Differential airflow, pressure, have key relationship in pressurization. *Lab Design* 1997;2:6–7.
322. Rhame FS. Nosocomial aspergillosis: How much protection for which patients? *Infect Control Hosp Epidemiol* 1989;10:296–8.
323. Hofflin JM, Potasman I, Baldwin JC, Oyster PE, Stinson EB, Remington JS. Infectious complications in heart transplant recipients receiving cyclosporine and corticosteroids. *Ann Intern Med* 1987;106:209–16.
324. Schulman LL, Smith CR, Drusin R, Rose EA, Enson Y, Reemtsma K. Respiratory complications of cardiac transplantation. *Am J Med Sci* 1988;296:1–10.
325. Dummer JS, Ho M. Risk factors and approaches to infections in transplant recipients. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and practice of infectious diseases*, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;3126–35.
326. Dummer JS, Ho M. Infections in solid organ transplant recipients. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and practice of infectious diseases*, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;3148–58.
327. Walsh TR, Guttendorf J, Dummer S, et al. The value of protective isolation procedures in cardiac transplant recipients. *Ann Thorac Surg* 1989;47:539–45.
328. Streifel AJ. Health-care IAQ: guidance for infection control. *HPAC Heating/Piping/Air Cond Eng* 2000; Oct:28–30, 33, 34, 36.

329. (87) Yeager CC. Copper and zinc preservatives. In: Block SS, ed. Disinfection, sterilization, and preservation, 4th ed. Philadelphia, PA: Lea & Febiger, 1991;358–61.
330. Cookson ST, Jarvis WR. Prevention of nosocomial transmission of *Mycobacterium tuberculosis*. Infect Dis Clin North Am 1997;11:367–409.
331. (42) CDC. Nosocomial transmission of multidrug-resistant tuberculosis among HIV-infected persons: Florida and New York, 1988–1991. MMWR 1991;40:585–91.
332. (43) CDC. Outbreak of multidrug-resistant tuberculosis at a hospital — New York City, 1991. MMWR 1993;42:427–34.
333. (7) Beck-Sague CM, Dooley SW, Hutton MD, et al. Hospital outbreak of multidrug-resistant *Mycobacterium tuberculosis*. JAMA 1992;268:1280–6.
334. (17) Coronado VG, Beck-Sague CM, Hutton MD, et al. Transmission of multidrug-resistant *Mycobacterium tuberculosis* among persons with human immunodeficiency virus infection in an urban hospital: Epidemiologic and restriction fragment length polymorphism analysis. J Infect Dis 1993;168:1052–5.
335. (18) Coronado VG, Valway S, Finelli L, et al. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis* among intravenous drug users with human immunodeficiency virus infection [abstract]. In: Abstracts of the Third Annual Meeting of the Society for Hospital Epidemiology of America. Chicago, IL: Infect Control Hosp Epidemiol 1993;14:428.
336. (8) Dooley SW, Villarino ME, Lawrence M, et al. Tuberculosis in a hospital unit for patients infected with the human immunodeficiency virus (HIV): evidence of nosocomial transmission. JAMA 1992;267:2632–4.
337. (19) Edlin BR, Tokars JI, Grieco MH, et al. An outbreak of multidrug-resistant tuberculosis among hospitalized patients with the acquired immunodeficiency syndrome: epidemiologic studies and restriction fragment length polymorphism analysis. N Engl J Med 1992;326:1514–22.
338. (20) Fischl MA, Uttamchandani RB, Daikos GL, et al. An outbreak of tuberculosis caused by multiple-drug-resistant tubercle bacilli among patients with HIV infection. Ann Intern Med 1992;117:177–83.
339. (21) Ikeda ARM, Birkhead GS, DeFerdinando Jr GT, et al. Nosocomial tuberculosis: an outbreak of a strain resistant to seven drugs. Infect Control Hosp Epidemiol 1995;16:152–9.
340. (22) Jarvis WR. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*. Res Microbiol 1992;144:117–22.
341. (23) Jarvis WR. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*. Am J Infect Control 1995;23:146–51.
342. (24) Jereb JA, Klevens RM, Privett TD, et al. Tuberculosis in health care workers at a hospital with an outbreak of multidrug-resistant *Mycobacterium tuberculosis*. Arch Intern Med 1995;155:854–9.
343. (25) Moran GJ, McCabe F, Morgan MT, Talan DA. Delayed recognition and infection control for tuberculosis patients in the emergency department. Ann Emerg Med 1995;26:283–9.
344. (26) Pearson ML, Jereb JA, Frieden TR, et al. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*: a risk to hospitalized patients and health-care workers. Ann Intern Med 1992;117:191–6.
345. Tokars JI, Jarvis WR, Edlin BR, et al. Tuberculin skin testing of hospital employees during an outbreak of multidrug-resistant tuberculosis in human immunodeficiency virus (HIV) infected patients. Infect Control Hosp Epidemiol 1992;13:509–10.
346. Macher JM. The use of germicidal lamps to control tuberculosis in healthcare facilities. Infect Control Hosp Epidemiol 1993;14:723–9.
347. (129) U.S. Department of Labor, Occupational Safety and Health Administration. Respiratory Protection, 29 CFR 1910.139. Federal Register 1998;63:1152–300.
348. (105) Ehrenkranz NJ, Kicklighter JL. Tuberculosis outbreak in a general hospital: evidence for airborne spread of infection. Ann Intern Med 1972;77:377–82.
349. (106) Calder RA, Duclos P, Wilder MH, et al. *Mycobacterium tuberculosis* transmission in a health clinic. Bull Int Union Tuberc Lung Dis 1991;66:103–6.
350. (107) Jereb JA, Burwen DR, Dooley SW, et al. Nosocomial outbreak of tuberculosis in a renal transplant unit: application of a new technique for restriction fragment length polymorphism analysis of *Mycobacterium tuberculosis* isolates. J Infect Dis 1993;168:1219–24.
351. (127) Ayliffe GAJ. Role of the environment of the operating suite in surgical wound infection. Rev Infect Dis 1991;13(suppl):S800–S804.
352. (128) Choux M, Genitori L, Lang D, Lena G. Shunt implantation: reducing the incidence of shunt infection. J Neurosurg 1992;77:875–80.

353. Edmiston CE Jr, Sinski S, Seabrook GR, Simons D, Goheen MP. Airborne particulates in the OR environment. *AORN J* 1999;69:1169–72.
354. Duhaime AC, Bonner K, McGowan KL, Schut L, Sutton LN, Plotkin S. Distribution of bacteria in the operating room environment and its relation to ventricular shunt infections: a prospective study. *Childs Nerv Syst* 1991;7:211–4.
355. Everett WD, Kipp H. Epidemiologic observations of operating room infections resulting from variations in ventilation and temperature. *Am J Infect Control* 1991;19:277–82.
356. **(115)** Lidwell OM. Clean air at operation and subsequent sepsis in the joint. *Clin Orthop* 1986;211:91–02.
357. **(116)** Nichols RL. The operating room. In: Bennett JV, Brachman PS, eds. *Hospital infections*, 3rd ed. Boston, MA: Little, Brown and Company, 1992;461–73.
358. **(117)** Clark RP, Reed PJ, Seal DV, Stephenson ML. Ventilation conditions and air-borne bacteria and particles in operating theatres: proposed safe economies. *J Hyg (Lond)* 1985;95:325–35.
359. **(119)** Laufman H. The operating room. In: Bennett JV, Brachman PS, eds. *Hospital infections*, 2nd ed. Boston, MA/Toronto, ON: Little, Brown and Company, 1986;315–23.
360. Pittet D, Ducle G. Infectious risk factors related to operating rooms. *Infect Control Hosp Epidemiol* 1994;15:456–62.
361. Hambræus A. Aerobiology in the operating room — a review. *J Hosp Infect* 1988;11(suppl. A):68–76.
362. **(118)** Babb JR, Lynam P, Ayliffe GAJ. Risk of airborne transmission in an operating theater containing four ultraclean air units. *J Hosp Infect* 1995;31:159–68.
363. Velesco, E, Thuler LCS, Martins CAS, deCastroDias LM, Conalves VMSC. Risk factors for infectious complications after abdominal surgery for malignant disease. *Am J Infect Control* 1996;24:1–6.
364. **(120)** National Academy of Sciences, National Research Council, Division of Medical Sciences, Ad Hoc Committee on Trauma. Postoperative wound infections: the influence of ultraviolet irradiation of the operating room and of various other factors. *Ann Surg* 1964;160 (suppl.):1–192.
365. **(121)** Charnley J. A clean-air operating enclosure. *Br J Surg* 1964;51:202–5.
366. **(122)** Lidwell OM, Lowbury EJJ, Whyte W, Blowers R, Stanley SJ, Lowe D. Effect of ultraclean air in operating rooms on deep sepsis in the joint after total hip or knee replacement: a randomized study. *Br Med J* 1982;285:10–4.
367. **(123)** Hill C, Flamant R, Mazas F, Evrard J. Prophylactic cefazolin versus placebo in total hip replacement: report of a multicentre double-blind randomized trial. *Lancet* 1981;1:795–6.
368. **(124)** Ha'eri GB, Wiley AM. Total hip replacement in a laminar flow environment with special reference to deep infections. *Clin Orthop* 1980;148:163–8.
369. **(125)** Collins DK, Steinhaus K. Total hip replacement without deep infection in a standard operating room. *J Bone Joint Surg* 1976;58A:446–50.
370. **(126)** Taylor GD, Bannister GC, Leeming JP. Wound disinfection with ultraviolet radiation. *J Hosp Infect* 1995;30:85–93.
371. **(130)** Langevin PB, Rand KH, Layton AJ. The potential for dissemination of *Mycobacterium tuberculosis* through the anesthesia breathing circuit. *Chest* 1999;115:1107–14.
372. **(131)** U.S. Department of Labor, Occupational Safety and Health Administration. Occupational exposure to tuberculosis: proposed rule. (29 CFR 1910). *Federal Register* 1997;62:54159–209.
373. **(132)** Aranha-Creado H, Prince D, Greene K, Brandwein H. Removal of *Mycobacterium* species by breathing circuit filters. *Infect Control Hosp Epidemiol* 1997;18:252–254.
374. Anesthesiology Society of America. *Infection Control for Practice of Anesthesiology*. 1999. Available at: www.asahq.org/Profinfo/Infection/Infection_TOC.html
375. McCarthy JF. Risk factors for occupational exposures in healthcare professionals. In: Hansen W, ed. *A guide to managing indoor air quality in health care organizations*. Oakbrook Terrace IL: Joint Commission on Accreditation of Healthcare Organizations, 1997;27–41.
376. National Institute for Occupational Safety and Health. NIOSH Health Hazard Evaluation and Technical Assistance Report: HETA 85-126-1932;1988.
377. National Institute for Occupational Safety and Health. NIOSH Health Hazard Evaluation and Technical Assistance Report: HETA 88-101-2008;1990.
378. **(135)** National Institute for Occupational Safety and Health. Control of smoke from laser/electric surgical procedures. DHHS (NIOSH) Publication 96-128;1996. Available at: www.cdc.gov/niosh/hc11.html
379. Taravella MJ, Weinberg A, Blackburn P, May M. Do intact viral particles survive excimer laser ablation? *Arch Ophthalmol* 1997;115:1028–30.

380. Hagen KB, Kettering JD, Aprecio RM, et al. Lack of virus transmission by the excimer laser plume. *Am J Ophthalmol* 1997;124:206–11.
381. Kunachak S, Sithisarn P, Kulapaditharom B. Are laryngeal papilloma virus-infected cells viable in the plume derived from a continuous mode carbon dioxide laser, and are they infectious? A preliminary report on one laser mode. *J Laryng Otol* 1996;110:1031–3.
382. **(137)** Hughes PS, Hughes AP. Absence of human papillomavirus DNA in the plume of erbium:YAG laser-treated warts. *J Am Acad Dermatol* 1998;38:426–8.
383. Garden JM, O'Bannon K, Sheinitz LS, et al. Papillomavirus in the vapor of carbon dioxide laser treated verrucae. *JAMA* 1988;125:1199–202.
384. Sawchuck WS, Weber JP, Lowry DR, et al. Infectious papillomavirus in the vapour of warts treated with carbon dioxide laser or electrocoagulation: detection and protection. *J Am Acad Dermatol* 1989;21:41–9.
385. Baggish MS, Poiesz BJ, Joret D, et al. Presence of human immunodeficiency virus DNA in laser smoke. *Lasers Surg Med* 1991;11:197–203.
386. **(138)** Capizzi PJ, Clay RP, Battey MJ. Microbiologic activity in laser resurfacing plume and debris. *Lasers Surg Med* 1998;23:172–4.
387. McKinley IB Jr, Ludlow MO. Hazards of laser smoke during endodontic therapy. *J Endodont* 1994;20:558.
388. Favero MS, Bolyard EA. Microbiologic considerations: disinfection and sterilization strategies and the potential for airborne transmission of bloodborne pathogens. *Surg Clin North Am* 1995;75:1071–89.
389. **(136)** Association of periOperative Registered Nurses. Recommended practices for laser safety in practice settings. In: Standards, Recommended Practices and Guidelines. Denver CO; AORN;2003;301–5.
390. **(139)** ECRI. Surgical smoke evacuation systems. *Health Devices* 1997;26:132–72.
391. **(140)** ECRI. Update evaluation: Surgical smoke evacuation systems. *Health Devices* 1999;28:333–62.
392. **(141)** ECRI. Stationary surgical smoke evacuation systems. *Health Devices* 2001;30:73–86.
393. American National Standards Institute. ANSI National standard for safe use of lasers in health care facilities. ANSI Z136.3-1996.
394. Kaufman AF, McDade J, Patton C, et al. Pontiac fever: isolation of the etiologic agent (*Legionella pneumophila*) and demonstration of its mode of transmission. *Am J Epidemiol* 1981;114:337–47.
395. **(192)** Marston BJ, Lipman HB, Breiman RF. Surveillance for Legionnaires' disease: risk factors for morbidity and mortality related to infection with *Legionella*. *Arch Intern Med* 1994;154:2417–22.
396. **(Appendix; 4)** Hoge CW, Breiman RF. Advances in the epidemiology and control of *Legionella* infections. *Epidemiol Rev* 1991;13:329–40.
397. Breiman RF, Butler JC. Legionnaires' disease: clinical, epidemiological, and public health perspectives. *Semin Respir Infect* 1998;13:84–9.
398. Yu, VL. *Legionella pneumophila* (Legionnaires' disease). In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of infectious diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2424–35.
399. Muder RR. Other *Legionella* species. In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of infectious diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2435–41.
400. Yu VL. Could aspiration be the major mode of transmission for *Legionella*? *Am J Med* 1993;95:13–5.
401. Jimenez P, Torres A, Rodriguez-Roisin R, et al. Incidence and etiology of pneumonia acquired during mechanical ventilation. *Crit Care Med* 1989;17:882–5.
402. **(220)** Zuravleff JJ, Yu VL, Shonnard JW, Rihs JD, Best M. *Legionella pneumophila* contamination of a hospital humidifier: demonstration of aerosol transmission and subsequent subclinical infection in exposed guinea pigs. *Am Rev Respir Dis* 1983;128:657–61.
403. **(202)** Mastro TD, Fields BS, Breiman RF, Campbell J, Plikaytis BD, Spika JS. Nosocomial Legionnaires' disease and use of medication nebulizers. *J Infect Dis* 1991;163:667–70.
404. **(203)** Dondero TJ Jr, Rendtorff RC, Mallison GF, et al. An outbreak of Legionnaires' disease associated with a contaminated air-conditioning cooling tower. *N Engl J Med* 1980;302:365–70.
405. **(199)** Garbe PL, Davis BJ, Weisfield JS, et al. Nosocomial Legionnaires' disease: epidemiologic demonstration of cooling towers as a source. *JAMA* 1985;254:521–4.
406. **(204)** O'Mahony MC, Stanwell-Smith RE, Tillett HE, et al. The Stafford outbreak of Legionnaires' disease. *Epidemiol Infect* 1990;104:361–80.
407. **(205)** Breiman RF, Fields BS, Sanden G, Volmer L, Meier A, Spika J. An outbreak of Legionnaires' disease associated with shower use: possible role of amoebae. *JAMA* 1990;263:2924–6.

408. (200) Hanrahan JP, Morse DL, Scharf VB, et al. A community hospital outbreak of legionellosis: transmission by potable hot water. *Am J Epidemiol* 1987;125:639–9.
409. (206) Breiman RF, VanLoock FL, Sion JP, et al. Association of “sink bathing” and Legionnaires’ disease [abstract]. In: Abstracts of the 91st Meeting of the American Society for Microbiology, 1991.
410. (207) Struelens MJ, Maes N, Rost F, et al. Genotypic and phenotypic methods for the investigation of a nosocomial *Legionella pneumophila* outbreak and efficacy of control measures. *J Infect Dis* 1992;166:22–30.
411. Terranova W, Cohen ML, Fraser DW. Outbreak of Legionnaires’ disease diagnosed in 1977. *Lancet* 1978;2:122–4.
412. (219) Marrie TJ, Haldane D, MacDonald S, et al. Control of endemic nosocomial Legionnaires’ disease by using sterile potable water for high risk patients. *Epidemiol Infect* 1991;107:591–605.
413. Nechwatal R, Ehret W, Klatte OJ, et al. Nosocomial outbreak of legionellosis in a rehabilitation center: demonstration of potable water as a source. *Infection* 1993;21:235–40.
414. Hoebe CJP, Cluitmanans JJM, Wagenvoort JHT. Two fatal cases of nosocomial *Legionella pneumophila* pneumonia associated with a contaminated cold water supply. *Eur J Clin Microbiol Infect Dis* 1998;17:740–9.
415. Helms CM, Viner JP, Sturm RH, et al. Comparative features of pneumococcal, *Mycoplasma*, and Legionnaires’ disease pneumonias. *Ann Intern Med* 1979;90:543–7.
416. Yu V, Kroboth FJ, Shonnard J, Brown A, McDearman S, Magnussen M. Legionnaires’ disease: new clinical perspectives from a prospective pneumonia study. *Am J Med* 1982;73:357–61.
417. (194) Jimenez ML, Aspa J, Padilla B, et al. Fiberoptic bronchoscopic diagnosis of pulmonary disease in 151 HIV-infected patients with pneumonitis. *Eur J Clin Microbiol Infect Dis* 1991;10:491–6.
418. Lowry PW, Blankenship RJ, Gridley W, et al. A cluster of *Legionella* sternal wound infections due to postoperative topical exposure to contaminated tap water. *N Engl J Med* 1991;324:109–12.
419. Shah A, Check F, Baskin S. Legionnaires’ disease and acute renal failure: case report and review. *Clin Infect Dis* 1992;14:204–7.
420. Lowry PW, Tompkins LS. Nosocomial legionellosis: a review of pulmonary and extrapulmonary syndromes. *Am J Infect Control* 1993;21:21–7.
421. Schlanger G, Lutwick LI, Kurzman M, et al. Sinusitis caused by *L. pneumophila* in a patient with acquired immune deficiency syndrome. *Am J Med* 1984;77:957–60.
422. Tompkins LS, Roessler BJ, Redd SC, et al. *Legionella* prosthetic-valve endocarditis. *N Engl J Med* 1988;318:530–5.
423. (195) Bock BV, Kirby BD, Edelstein PH, et al. Legionnaires’ disease in renal transplant recipients. *Lancet* 1978;1:410–3.
424. (196) Kirby BD, Snyder KM, Meyer RD, Finegold SM. Legionnaires’ disease: report of 65 nosocomially acquired cases and review of the literature. *Medicine* 1980;59:188–205.
425. (197) Brady MT. Nosocomial Legionnaires’ disease in a children’s hospital. *J Pediatr* 1989;115:46–50.
426. Horie H, Kawakami H, Minoshima K, et al. Neonatal Legionnaires’ disease: histologic findings in an autopsied neonate. *Acta Pathol Jpn* 1992;42:427–31.
427. Roig J, Aguilar X, Ruiz J, et al. Comparative study of *Legionella pneumophila* and other nosocomial pneumonias. *Chest* 1991;99:344–50.
428. Redd SC, Schuster DM, Quan J, et al. Legionellosis cardiac transplant recipients: results of a nationwide survey. *J Infect Dis* 1988;158:651–3.
429. Seu P, Winston DJ, Olthoft KM, et al. Legionnaires’ disease in liver transplant recipients. *Infect Dis Clin Pract* 1993;2:109–13.
430. (215) Chow JW, Yu VL. *Legionella*: a major opportunistic pathogen in transplant recipients. *Semin Respir Infect* 1998;13:132–9.
431. (189) Kool JL, Fiore AE, Kioski CM, et al. More than ten years of unrecognized nosocomial transmission of Legionnaires’ disease among transplant patients. *Infect Control Hosp Epidemiol* 1998;19:898–904.
432. (190) Le Saux NM, Sekla L, McLeod J, et al. Epidemic of nosocomial Legionnaires’ disease in renal transplant recipients: a case-control and environmental study. *Can Med Assoc J* 1989;140:1047–53.
433. Berendt RF, Young HW, Allen RG, Knutsen GL. Dose-response of guinea pigs experimentally infected with aerosols of *Legionella pneumophila*. *J Infect Dis* 1980;141:186–92.
434. Marston BJ, Plouffe JF, File TM, et al. Incidence of community-acquired pneumonia requiring hospitalization — results of a population-based active surveillance study in Ohio. *Arch Intern Med* 1997;157:1709–18.

435. **(198)** Muder RR, Yu VL, McClure JK, Kroboth FJ, Kominos SD, Lumish RN. Nosocomial Legionnaires' disease uncovered in a prospective pneumonia study: implications for underdiagnosis. *JAMA* 1983;249:3184–8.
436. Brennen C, Vickers JP, Yu VL, Puntereri A, Yee YC. Discovery of occult *Legionella pneumonia* in a long-stay hospital: Results of prospective serologic survey. *Br Med J* 1987;295:306–7.
437. **(166)** Marrie TJ, MacDonald S, Clarke K, Haldane D. Nosocomial Legionnaires' disease: lessons from a four-year prospective study. *Am J Infect Control* 1991;19:79–85.
438. Stout JE, Yu VL. Current concepts: legionellosis. *N Engl J Med* 1997;337:682–7.
439. Vergis EN, Yu VL. Macrolides are ideal for empiric therapy of community-acquired pneumonia in the immunocompromised host. *Semin Respir Infect* 1998;13:322–8.
440. Sopena N, Sabria-Leal M, Pedro-Botet ML, et al. Comparative study of the clinical presentation of *Legionella pneumonia* and other community-acquired pneumonias. *Chest* 1998;113:1195–200.
441. **(217)** Hirani NA, MacFarlane JT. Impact of management guidelines on the outcome of severe community acquired pneumonia. *Thorax* 1997;52:17–21.
442. Lieberman D, Porath A, Schlaeffer F, Boldur L. *L. pneumophila* species community-acquired pneumonia: a review of 56 hospitalized patients. *Chest* 1996;109:1243–9.
443. Ewig S, Bauer T, Hasper E, et al. Value of routine microbial investigation in community-acquired pneumonia treated in a tertiary care center. *Respiration* 1996;63:164–9.
444. Marrie TJ, Peeling RW, Fine MJ, et al. Ambulatory patients with community-acquired pneumonia: the frequency of atypical agents and clinical course. *Am J Med* 1996;101:508–15.
445. Benin AI, Benson RF, Besser RE. Trends in Legionnaires' disease, 1980–1998: declining mortality and new patterns of diagnosis. *Clin Infect Dis* 2002;35:1039–46.
446. Fliermans CD, Cherry WB, Orrison LH, Smith SJ, Tison DL, Pope DH. Ecologic distribution of *Legionella pneumophila*. *Appl Environ Microbiol* 1981;41:9–16.
447. Morris GK, Patton CM, Feeley JC, et al. Isolation of the Legionnaires' disease bacterium from environmental samples. *Ann Intern Med* 1979;90:664–6.
448. Hsu SC, Martin R, Wentworth BB. Isolation of *Legionella* species from drinking water. *Appl Environ Microbiol* 1984;48:830–2.
449. Tison DL, Seidler RJ. *Legionella* incidence and density in potable drinking water. *Appl Environ Microbiol* 1983;45:337–9.
450. Parry MF, Stampleman L, Hutchinson JH, et al. Waterborne *Legionella bozemanii* and nosocomial pneumonia in immunosuppressed patients. *Ann Intern Med* 1985;103:205–10.
451. England AC, Fraser DW. Sporadic and epidemic nosocomial legionellosis in the United States: epidemiologic features. *Am J Med* 1981;70:707–11.
452. Cohen ML, Broome CV, Paris AL, et al. Fatal nosocomial Legionnaires' disease: clinical and epidemiological characteristics. *Ann Intern Med* 1979;90:611–3.
453. **(193)** Haley CE, Cohen ML, Halter J, Meyer RD. Nosocomial Legionnaires' disease: a continuing common-source epidemic at Wadsworth Medical Center. *Ann Intern Med* 1979;90:583–6.
454. Stout JE, Yu VL, Vickers RM, Shonnard J. Potable water supply as the hospital reservoir for Pittsburgh pneumonia agent. *Lancet* 1982;1:471–2.
455. **(201)** Arnow PM, Chou T, Weil D, Shapiro EN, Kretzschmar C. Nosocomial Legionnaires' disease caused by aerosolized tap water from respiratory devices. *J Infect Dis* 1982;146:460–7.
456. Farrell ID, Barker JE, Miles EP, Hutchinson JCP. A field study of the survival of *Legionella pneumophila* in a hospital hot-water system. *Epidemiol Infect* 1990;104:381–7.
457. Stout JE, Yu VL, Best MG. Ecology of *Legionella pneumophila* within water distribution systems. *Appl Environ Microbiol* 1985;49:221–8.
458. Sanden GN, Fields BS, Barbaree JM, et al. Viability of *Legionella pneumophila* in chlorine-free water at elevated temperatures. *Curr Microbiol* 1989;61–5.
459. Schulze-Röbbecke R, Rodder M, Exner M. Multiplication and killing temperatures of naturally occurring legionellae. *Zbl Bakt Hyg B* 1987;184:495–500.
460. Habicht W, Muller HE. Occurrence and parameters of frequency of *Legionella* in warm water systems of hospitals and hotels in Lower Saxony. *Zbl Bakt Hyg B* 1988;186:79–88.
461. Ciesielski CA, Blaser MJ, Wang WL. Role of stagnation and obstruction of water flow in isolation of *Legionella pneumophila* from hospital plumbing. *Appl Environ Microbiol* 1984;48:984–7.
462. Rowbotham TJ. Preliminary report on the pathogenicity of *Legionella pneumophila* for freshwater and soil amoebae. *J Clin Path* 1980;33:179–83.

463. Fields BS, Sanden GN, Barbaree JM, et al. Intracellular multiplication of *Legionella pneumophila* in amoebae isolated from hospital hot water tanks. *Curr Microbiol* 1989;18:131–7.
464. (142) Villarino ME, Stevens LE, Schable B, et al. Risk factors for epidemic *Xanthomonas maltophilia* infection/colonization in intensive care unit patients. *Infect Control Hosp Epidemiol* 1992;13:201–6.
465. (147) Burdge DR, Nakielna EM, Noble MA. Case-control and vector studies of nosocomial acquisition of *Pseudomonas cepacia* in adult patients with cystic fibrosis. *Infect Control Hosp Epidemiol* 1993;14:127–30.
466. Stephenson JR, Heard SR, Richards MA, Tabaqchali S. Gastrointestinal colonization and septicaemia with *Pseudomonas aeruginosa* due to contaminated thymol mouthwash in immunocompromised patients. *J Hosp Infect* 1985;6:369–78.
467. Kolmos HJ, Thusen B, Neilsen SV, Lohmann M, Kristoffersen K, Rosdahl VT. Outbreak of infection in a burns unit due to *Pseudomonas aeruginosa* originating from contaminated tubing used for irrigating patients. *J Hosp Infect* 1993;24:11–21.
468. Vanholder R, Vanhaecke E, Ringoir S. Waterborne *Pseudomonas* septicemia. *ASAIO Trans* 1990;36:M215–6.
469. Ehni WF, Reller LB, Ellison RT III. Bacteremia in granulocytopenic patients in a tertiary-care general hospital. *Rev Infect Dis* 1991;13:613–9.
470. Gallagher PG, Watanakunakorn C. *Pseudomonas* bacteremia in a community teaching hospital, 1980–1984. *Rev Infect Dis* 1989;11:846–52.
471. Centers for Disease Control. Nosocomial infection and pseudoinfection from contaminated endoscopes and bronchoscopes — Wisconsin and Missouri. *MMWR* 1991;40:675–8.
472. Kerr JR, Moore JE, Curran MD, et al. Investigation of a nosocomial outbreak of *Pseudomonas aeruginosa* pneumonia in an intensive care unit by random amplification of polymorphic DNA assay. *J Hosp Infect* 1995;30:125–31.
473. Brewer SC, Wunderink RG, Jones CB, Leeper KV. Ventilator-associated pneumonia due to *Pseudomonas aeruginosa*. *Chest* 1996;109:1019–22.
474. Rello J, Jubert P, Valles J, et al. Evaluation of outcome for intubated patients with pneumonia due to *Pseudomonas aeruginosa*. *Clin Infect Dis* 1996;23:973–8.
475. Henderson A, Kelly W, Wright M. Fulminant primary *Pseudomonas aeruginosa* pneumonia and septicaemia in previously well adults. *Intensive Care Med* 1992;18:430–2.
476. Torres A, Serra-Battles J, Ferrer A, et al. Severe community acquired pneumonia: epidemiology and prognostic factors. *Am Rev Respir Dis* 1991;144:312–8.
477. Pedersen SS, Koch C, Høiby N, Rosendal K. An epidemic spread of multiresistant *Pseudomonas aeruginosa* in a cystic fibrosis center. *J Antimicrob Chemother* 1986;17:505–6.
478. Kubesch P, Dörk T, Wulbrand U, et al. Genetic determinants of airways' colonization with *Pseudomonas aeruginosa* in cystic fibrosis. *Lancet* 1993;341:189–93.
479. Koch C, Høiby N. Pathogenesis of cystic fibrosis. *Lancet* 1993;341:1065–9.
480. Worlitzsch D, Wolz C, Botzenart K, et al. Molecular epidemiology of *Pseudomonas aeruginosa* – urinary tract infections in paraplegic patients. *Zentrabl Hyg Umweltmed* 1989;189:175–84.
481. Glenister H, Holton J, Teall A. Urinary tract pressure recording equipment as a source for infection. *J Hosp Infect* 1985;6:224–6.
482. Ferroni A., Nguyen L, Pron B, Quense G, Brusset MC, Berche P. Outbreak of nosocomial urinary tract infections due to *Pseudomonas aeruginosa* in a paediatric surgical unit associated with tap water contamination. *J Hosp infect* 1998;39:301–7.
483. Marrie TJ, Major H, Gurwith M, et al. Prolonged outbreak of nosocomial urinary tract infection with a single strain of *Pseudomonas aeruginosa*. *Can Med Assoc J* 1978;119:593–8.
484. Moore B, Forman A. An outbreak of urinary *Pseudomonas aeruginosa* infection acquired during urological operations. *Lancet* 1966;2:929–31.
485. Anderson RJ, Schafer LA, Olin DB, Eickhoff TC. Septicemia in renal transplant recipients. *Arch Surg* 1973;106:692–4.
486. Fang G, Brennen C, Wagener M, et al. Use of ciprofloxacin versus use of aminoglycosides for therapy of complicated urinary tract infection: prospective, randomized clinical and pharmacokinetic study. *Antimicrob Agents Chemother* 1991;35:1849–55.
487. Dorff GJ, Beimer NF, Rosenthal DR, Rytel MW. *Pseudomonas* septicemia: illustrated evolution of its skin lesions. *Arch Intern Med* 1971;128:591–5.
488. Teplitz C. Pathogenesis of *Pseudomonas vasculitis* and septic lesions. *Arch Pathol* 1965;80:297–307.

489. Roberts R, Tarpay MM, Marks MI, Nitschke R. Erysipelas-like lesions and hyperesthesia as manifestations of *Pseudomonas aeruginosa* sepsis. *JAMA* 1982;248:2156–7.
490. Duncan BW, Adzick NS, deLorimier AA, et al. Necrotizing fasciitis in childhood. *J Pediatr Surg* 1992;27:668–71.
491. McManus AT, Mason AD Jr, McManus WF, Pruitt BA Jr. Twenty-five year review of *Pseudomonas aeruginosa* bacteremia in a burn center. *Eur J Clin Microbiol* 1985;4:219–23.
492. Tredget EE, Shankowsky HA, Joffe AM, et al. Epidemiology of infections with *Pseudomonas aeruginosa* in burn patients: the role of hydrotherapy. *Clin Infect Dis* 1992;15:941–9.
493. Schlech WF III, Simosen N, Sumarah R, Martin RS. Nosocomial outbreak of *Pseudomonas aeruginosa* folliculitis associated with a physiotherapy pool. *Can Med Assoc J* 1986;134:909–13.
494. Fang G, Keys TF, Gentry LO, et al. Prosthetic valve endocarditis resulting from nosocomial bacteremia: a prospective, multicenter study. *Ann Intern Med* 1993;119:560–7.
495. Cohen PS, Maguire JH, Weinstein L. Infective endocarditis caused by gram-negative bacteria: a review of the literature, 1945–1977. *Prog Cardiovasc Dis* 1980;22:205–42.
496. Wise BL, Mathis JL, Jawetz E. Infections of the central nervous system due to *Pseudomonas aeruginosa*. *J Neurosurg* 1969;31:432–4.
497. Bray DA, Calcaterra TC. *Pseudomonas* meningitis complicating head and neck surgery. *Laryngoscope* 1976;86:1386–90.
498. Schein OD, Wasson PJ, Boruchoff SA, Kenyon KR. Microbial keratitis associated with contaminated ocular medications. *Am J Ophthalmol* 1988;105:361–5.
499. Procope JA. Delayed-onset *Pseudomonas* keratitis after radial keratotomy. *J Cataract Refract Surg* 1997;23:1271–2.
500. Sapico FL, Montgomerie JZ. Vertebral osteomyelitis in intravenous drug abusers: report of three cases and review of the literature. *Rev Infect Dis* 1980;2:196–206.
501. Tindel JR, Crowder JG. Septic arthritis due to *Pseudomonas aeruginosa*. *JAMA* 1971;218:559–61.
502. Martone WJ, Tablan OC, Jarvis WR. The epidemiology of nosocomial epidemic *Pseudomonas cepacia* infections. *Eur J Epidemiol* 1987;3:222–32.
503. Goldmann DA, Klinger JD. *Pseudomonas cepacia*: biology, mechanisms of virulence, epidemiology. *J Pediatr* 1986;108:806–12.
504. Widmer AF, Wenzel RP, Trilla A, et al. Outbreak of *Pseudomonas aeruginosa* infections in a surgical intensive care unit: probable transmission via hands of a health care worker. *Clin Infect Dis* 1993;16: 372–6.
505. Döring G, Hörz M, Ortelt J, et al. Molecular epidemiology of *Pseudomonas aeruginosa* in an intensive care unit. *Epidemiol Infect* 1993;110:427–36.
506. Hollyoak V, Allison D, Summers J. *Pseudomonas aeruginosa* wound infection associated with a nursing home whirlpool bath. *CDR Review* 1995;5:R100–2.
507. Grundmann H, Kropec A, Hartung D, Berner R, Daschner F. *Pseudomonas aeruginosa* in a neonatal intensive care unit: reservoirs and ecology of the nosocomial pathogen. *J Infect Dis* 1993;168:943–7.
508. Martino P, Venditti M, Papa G, Orefici G, Serra P. Water supply as a source of *Pseudomonas aeruginosa* in a hospital for hematological malignancies. *Bollettino dell Istituto Sieroterapico Milanese* 1985;64:109–14.
509. Ayliffe GAJ, Babb JR, Collins BJ, Lowbury EJ, Newsom SWB. *Pseudomonas aeruginosa* in hospital sinks. *Lancet* 1974;2:578–81.
510. Kluyver AJ. *Pseudomonas aureofaciens* nov. spec and its pigments. *J Bacteriol* 1956;72:406–11.
511. Romling U, Fiedler B, Bosshammer J, et al. Epidemiology of chronic *Pseudomonas aeruginosa* infections in cystic fibrosis. *J Infect Dis* 1994;170:1616–21.
512. Jones F, Bartlett CL. Infections associated with whirlpools and spas. *Soc Appl Bacteriol Symp Series* 1985;14:61S–6S.
513. Casewell MW, Slater NG, Cooper JE. Operating theater water-baths as a cause of *Pseudomonas* septicemia. *J Hosp Infect* 1981;2:237–47.
514. Rechsteiner J, Landheer JE, de Jong J, van Kregten E, Lindner JG. [Kidney lithotripter as a possible source of hospital infection]. *Nederlands Tijdschrift voor Geneeskunde* 1988;132:1849–59. (Dutch)
515. (308) Taplin D, Mertz PM. Flower vases in hospitals as reservoirs for pathogens. *Lancet* 1973;2: 1279–1281.
516. Kaiser AB. Humidifiers and *Pseudomonas* infections. *N Engl J Med* 1970;283:708.

517. Levin MH, Olson B, Nathan C, et al. *Pseudomonas* in the sinks in an intensive care unit: relation to patients. *J Clin Pathol* 1984;37:424–7.
518. Paszko-Kolva C, Yamamoto H, Shahamat M, Sawyer TK, Morris G, Colwell RR. Isolation of amoebae and *Pseudomonas* and *Legionella* spp. from eyewash stations. *Appl Environ Microbiol* 1991;57:163–7.
519. Struelens MJ, Rost F, Deplano A, et al. *Pseudomonas aeruginosa* and *Enterobacteriaceae* bacteremia after biliary endoscopy: an outbreak investigation using DNA macrorestriction analysis. *Am J Med* 1993;95:489–98.
520. Blanc DS, Parret T, Janin B, Raselli P, Francioli P. Nosocomial infections and pseudoinfections from contaminated bronchoscopes: two-year follow up using molecular markers. *Infect Control Hosp Epidemiol* 1997;18:134–6.
521. Boukadida J, de Montalembert M, Gaillard JL, et al. Outbreak of gut colonization by *Pseudomonas aeruginosa* in immunocompromised children undergoing total digestive decontamination: analysis by pulsed-field electrophoresis. *J Clin Microbiol* 1991;29:2068–71.
522. Grigis A, Goglio A, Parea M, Gnechi F, Minetti B, Barbui T. Nosocomial outbreak of severe *Pseudomonas aeruginosa* infections in haematological patients. *Eur J Epidemiol* 1993;9:390–5.
523. Gupta AK, Shashi S, Mohan M, Lamba IM, Gupta R. Epidemiology of *Pseudomonas aeruginosa* infections in a neonatal intensive care unit. *J Trop Pediatr* 1993;39:32–6.
524. Sader HS, Pignatari AC, Leme IL, et al. Epidemiologic typing of multiply drug-resistant *Pseudomonas aeruginosa* isolated from an outbreak in an intensive care unit. *Diagn Microbiol Infect Dis* 1993;17:13–8.
525. Krecmery V, Trupl J. Nosocomial outbreak of meropenem-resistant *Pseudomonas aeruginosa* infections in a cancer center. *J Hosp Infect* 1994;28:209–18.
526. Jumaa P, Chattopadhyay B. Outbreak of gentamicin, ciprofloxacin-resistant *Pseudomonas aeruginosa* in an intensive care unit, traced to contaminated quivers. *J Hosp Infect* 1994;28:209–18.
527. Carson LA, Favero MS, Bond WW, Petersen NJ. Morphological, biochemical, and growth characteristics of *Pseudomonas cepacia* from distilled water. *Appl Microbiol* 1973;25:476–83.
528. Bassett DC, Stokes KJ, Thomas WR. Wound infection with *Pseudomonas multivorans*: a waterborne contaminant of disinfectant solutions. *Lancet* 1970;1:1188–91.
529. Wishart MM, Riley TV. Infection with *Pseudomonas maltophilia*: hospital outbreak due to contaminated disinfectant. *Med J Aust* 1976;2:710–2.
530. Conly JM, Klass L, Larson L. *Pseudomonas cepacia* colonization and infection in intensive care units. *Can Med Assoc J* 1986;134:363–6.
531. Bosshammer J, Fielder B, Gudowis P, von der Hardt H, Romling U, Tummeler B. Comparative hygienic surveillance of contamination with pseudomonads in a cystic fibrosis ward over a 4-year period. *J Hosp Infect* 1995;31:261–74.
532. Hutchinson GR, Parker S, Pryor JA, et al. Home-use nebulizers: a potential primary source of *B. cepacia* and other colistin-resistant, gram-negative bacteria in patients with cystic fibrosis. *J Clin Microbiol* 1996;34:584–7.
533. Pegues DA, Carson LA, Anderson RL, et al. Outbreak of *Pseudomonas cepacia* bacteremia in oncology patients. *Clin Infect Dis* 1993;16:407–11.
534. CDC. Nosocomial *Burkholderia cepacia* infection and colonization with intrinsically contaminated mouthwash — Arizona, 1998. *MMWR* 1998;47:926–8.
535. Berthelot P, Grattard F, Mahul P, et al. Ventilator temperature sensors: an unusual source of *Pseudomonas cepacia* in nosocomial infection. *J Hosp Infect* 1993;25:33–43.
536. Khardori N, Elting L, Wong E, et al. Nosocomial infections due to *Xanthomonas maltophilia* (*Pseudomonas maltophilia*) in patients with cancer. *Rev Infect Dis* 1990;12:997–1003.
537. Oie S, Oomaki M, Yorioka K, et al. Microbial contamination of “sterile water” used in Japanese hospitals. *J Hosp Infect* 1998;38:61–5.
538. Crane LR, Tagle LC, Palutke WA. Outbreak of *Pseudomonas paucimobilis* in an intensive care facility. *JAMA* 1981;246:985–7.
539. Lemaitre D, Elaichouni A, Hundhausen M, et al. Tracheal colonization with *Sphingomonas paucimobilis* in mechanically-ventilated neonates due to contaminated ventilator temperature probes. *J Hosp Infect* 1996;32:199–206.
540. Maki DG, Klein BS, McCormick RD, et al. Nosocomial *Pseudomonas pickettii* bacteremias traced to narcotic tampering. A case for selective drug screening of health care personnel. *JAMA* 1991;265:981–6.
541. Maroye P, Doermann HP, Rogues AM, Gachie JP, Mégraud F. Investigation of an outbreak of *Ralstonia pickettii* in a paediatric hospital by RAPD. *J Hosp Infection* 2000;44:267–72.

542. McNeil MM, Solomon SL, Anderson RL, et al. Nosocomial *Pseudomonas pickettii* colonization associated with a contaminated respiratory therapy solution in a special care nursery. *J Clin Microbiol* 1985;22:903–7.
543. Lamka KG, LeChevallier MW, Seidler RJ. Bacterial contamination of drinking water supplies in a modern rural neighborhood. *Appl Environ Microbiol* 1980;39:734–8.
544. Nakashima AK, McCarthy MA, Martone WJ, Anderson RL. Epidemic septic arthritis caused by *Serratia marcescens* and associated with a benzalkonium chloride antiseptic. *J Clin Microbiol* 1987;25:1014–8.
545. Nakashima AK, Highsmith AK, Martone WJ. Survival of *Serratia marcescens* in benzalkonium chloride and in multiple-dose medication vials: relationship to epidemic septic arthritis. *J Clin Microbiol* 1987;25:1019–21.
546. Bosi C, Davin-Regli A, Charrel R, Rocca B, Monnet D, Bollet C. *Serratia marcescens* nosocomial outbreak due to contamination of hexetidine solution. *J Hosp Infect* 1996;33:217–24.
547. Ehrenkranz NJ, Bolyard EA, Wiener M, Cleary TJ. Antibiotic-sensitive *Serratia marcescens* infections complicating cardiopulmonary operations: contaminated disinfectant as a reservoir. *Lancet* 1980;2:1289–92.
548. Cimolai N, Trombley C, Wensley D, LeBlanc J. Heterogeneous *Serratia marcescens* genotypes from a nosocomial pediatric outbreak. *Chest* 1997;111:194–7.
549. Hartstein AI, Rashad AL, Liebler JM, et al. Multiple intensive care unit outbreaks of *Acinetobacter calcoaceticus* subspecies anitratus respiratory infection and colonization associated with contaminated, reusable ventilator circuits and resuscitation bags. *Am J Med* 1988;85:624–31.
550. Stone JW, Das BC. Investigation of an outbreak of infection with *Acinetobacter calcoaceticus* in a special care baby unit. *J Hosp Infect* 1986;7:42–8.
551. Vandebroucke-Grauls CMJE, Kerver AJH, Rommes JH, Jansen R, den Dekker C, Verhoef J. Endemic *Acinetobacter anitratus* in a surgical intensive care unit: mechanical ventilators as reservoir. *Eur J Clin Microbiol Infect Dis* 1988;7:485–9.
552. Cefai C, Richards J, Gould FK, McPeake P. An outbreak of *Acinetobacter* respiratory infection resulting from incomplete disinfection of ventilatory equipment. *J Hosp Infect* 1990;15:177–82.
553. Gervich DH, Grout CS. An outbreak of nosocomial *Acinetobacter* infections from humidifiers. *Am J Infect Control* 1985;13:210–5.
554. Castle M, Tenney JH, Weinstein MP, Eickhoff TC. Outbreak of a multiply resistant *Acinetobacter* in a surgical intensive care unit. *Heart Lung* 1978;7:641–4.
555. Smith PW, Massanari RM. Room humidifiers as a source of *Acinetobacter* infections. *JAMA* 1977;237:795–7.
556. Snyderman DR, Maloy MF, Brock SM, Lyons RW, Rubin SJ. Pseudobacteremia: false-positive blood cultures from mist tent contamination. *Am J Epidemiol* 1977;106:154–9.
557. Rosenthal SL. Sources of *Pseudomonas* and *Acinetobacter* species found in human culture materials. *Am J Clin Pathol* 1974;62:807–11.
558. Allen KD, Green HT. Hospital outbreak of multi-resistant *Acinetobacter anitratus*: an airborne mode of spread. *J Hosp Infect* 1987;9:169–75.
559. Crombach WHJ, Dijkshoorn L, van Noort-Klaassen M, Niessen J, van Knippenbert-Gordebeke G. Control of an epidemic spread of multi-resistant *Acinetobacter calcoaceticus* in a hospital. *Intensive Care Med* 1989;15:166–170.
560. Catalano M, Quelle LS, Jeric PE, Di Martino A, Maimone SM. Survival of *Acinetobacter baumannii* on bed rails during an outbreak and during sporadic cases. *J Hosp Infect* 1999;42:27–35.
561. D'Agata EMC, Venkataraman L, DeGirolami P, Samore M. Molecular epidemiology of ceftazidime-resistant gram-negative bacilli on inanimate surfaces and their role in cross-transmission during non-outbreak periods. *J Clin Microbiol* 1999;37:3065–7.
562. Jawad A, Snelling AM, Heritage J, Hawkey PM. Exceptional desiccation tolerance of *Acinetobacter radioresistens*. *J Hosp Infect* 1998;39:235–40.
563. Jawad A, Seifert H, Snelling AM, Heritage J, Hawkey PM. Survival of *Acinetobacter baumannii* on dry surfaces: comparison of outbreak and sporadic isolates. *J Clin Microbiol* 1998;36:1938–41.
564. Getschell-White, SI, Donowitz LG, Groschel DHM. The inanimate environment of an intensive care unit as a potential source of nosocomial bacteria: evidence for long survival of *Acinetobacter calcoaceticus*. *Infect Control Hosp Epidemiol* 1989;10:402–6.
565. Loiwal V, Kumar A, Gupta P, Gomber S, Ramchandran VG. *Enterobacter aerogenes* outbreak in a neonatal intensive care unit. *Pediatr Int* 1999;41:157–61.

566. Matsaniotis NS, Syriopoulou VP, Theodoridou MC, Tzanetou KG, Mostrou GI. *Enterobacter* sepsis in infants and children due to contaminated intravenous fluids. *Infect Control* 1984;5:471-7.
567. Zembrzuska-Sadlowska E. The dangers of infections of the hospitalized patients with the microorganisms present in preparations and in the hospital environment. *Acta Pol Pharm* 1995;52:173-8.
568. Felts SK, Schaffner W, Melly MA, Koenig MG. Sepsis caused by contaminated intravenous fluids. *Ann Intern Med* 1972;77:881-90.
569. Modi N, Damjanovic V, Cooke RW. Outbreak of cephalosporin resistant *Enterobacter cloacae* infection in a neonatal intensive care unit. *Arch Dis Child* 1987;62:148-51.
570. Graham DR, Wu E, Highsmith AK, Ginsburg ML. An outbreak of pseudobacteremia caused by *Enterobacter cloacae* from a phlebotomist's vial of thrombin. *Ann Intern Med* 1981;95:585-8.
571. Andersen BM, Sorlie D, Hotvedt R, et al. Multiply beta-lactam-resistant *Enterobacter cloacae* infections linked to the environmental flora in a unit for cardiothoracic and vascular surgery. *Scand J Infect Dis* 1989;21:181-91.
572. Wisplinghoff H, Perbix W, Seifert H. Risk factors for nosocomial bloodstream infections due to *Acinetobacter baumannii*: a case-control study of adult burn patients. *Clin Infect Dis* 1999;28:59-66.
573. Crowe M, Towner KJ, Humphreys H. Clinical and epidemiological features of an outbreak of *Acinetobacter* infection in an intensive therapy unit. *J Med Microbiol* 1995;43:55-62.
574. National Nosocomial Infections Surveillance (NNIS) Report: Data summary from October 1986-April 1996, issued May 1996. *Am J Infect Control* 1996;24:380-8.
575. Bergogne-Bérézin E, Joly-Guillou ML. Hospital infection with *Acinetobacter* spp.: an increasing problem. *J Hosp Infect* 1991;18 (suppl A):250-5.
576. Fagon JY, Chastre J, Hance AJ, Montravers P, Novara A, Gibert C. Nosocomial pneumonia in ventilated patients: a cohort study evaluating attributable mortality and hospital stay. *Am J Med* 1993;94: 281-8.
577. (143) Seifert H, Strate A, Pulverer G. Nosocomial bacteremia due to *Acinetobacter baumannii*: clinical features, epidemiology, and predictors of mortality. *Medicine* 1995;74:340-9.
578. Cisneros JM, Reyes MJ, Pachón J, et al. Bacteremia due to *Acinetobacter baumannii*: epidemiology, clinical findings, and prognostic features. *Clin Infect Dis* 1996;22:1026-32.
579. Schaberg DR, Culver DH, Gaynes RP. Major trends in the microbial ecology of nosocomial infections. *Am J Med* 1991;91(suppl 3B):72S-5S.
580. Wang CC, Chu ML, Ho LJ, Hwang RC. Analysis of plasmid pattern in pediatric intensive care outbreaks of nosocomial infection due to *Enterobacter cloacae*. *J Hosp Infect* 1991;19:33-40.
581. Acolet D, Ahmet Z, Houang E, Hurley R, Kaufman ME. *Enterobacter cloacae* in a neonatal intensive care unit: account of an outbreak and its relationship to use of third generation cephalosporins. *J Hosp Infect* 1994;28:273-86.
582. Mayhall CG, Lamb VA, Gayle WE Jr, Haynes BW Jr. *Enterobacter cloacae* septicemia in a burn center: epidemiology and control of an outbreak. *J Infect Dis* 1979;139:166-71.
583. John JF Jr, Sharbaugh RJ, Bannister ER. *Enterobacter cloacae*: bacteremia, epidemiology, and antibiotic resistance. *Rev Infect Dis* 1982;4:13-28.
584. McDonald C, Banerjee SN, Jarvis WR, NNIS. Seasonal variation of *Acinetobacter* infections: 1987-1996. *Clin Infect Dis* 1999;29:1133-7.
585. Beck-Sague CM, Jarvis WR, Brook JH, et al. Epidemic bacteremia due to *Acinetobacter baumannii* in five intensive care units. *Am J Epidemiol* 1990;132:723-33.
586. (144) Yu VL. *Serratia marcescens*: historical perspective and clinical review. *N Engl J Med* 1979;300:887-93.
587. Wenger PN, Tokars JI, Brennan P, et al. An outbreak of *Enterobacter hormaechei* infection and colonization in an intensive care nursery. *Clin Infect Dis* 1997;24:1243-4.
588. Buxton AE, Anderson RL, Wedegar D, Atlas E. Nosocomial respiratory tract infection and colonization with *Acinetobacter calcoaceticus*. *Am J Med* 1978;65:507-13.
589. French GL, Casewell MW, Roncoroni AJ, Knight S, Philipps I. A hospital outbreak of antibiotic-resistant *Acinetobacter anitratus*: epidemiology and control. *J Hosp Infect* 1980;1:125-31.
590. Guenter SH, Hendley JO, Wenzel RP. Gram-negative bacilli as nontransient flora on the hands of hospital personnel. *J Clin Microbiol* 1987;25:488-90.
591. Dreyfuss D, Djedaini K, Weber P, et al. Prospective study of nosocomial pneumonia and of patient and circuit colonization during mechanical ventilation with circuit changes every 48 hours versus no change. *Am Rev Respir Dis* 1991;143:738-43.

592. (145) Go SE, Urban C, Burns J, et al. Clinical and molecular epidemiology of *Acinetobacter* infections sensitive only to polymixin B and sublactam. *Lancet* 1994;344:1329–32.
593. Musa EK, Desai N, Casewell MW. The survival of *Acinetobacter calcoaceticus* inoculated on fingertips and on formica. *J Hosp Infect* 1990;15:219–27.
594. Jawad A, Heritage J, Snelling AM, Gascoyne-Binzi DM, Hawkey PM. Influence of relative humidity and suspending menstrua on survival of *Acinetobacter* spp. on dry surfaces. *J Clin Microbiol* 1996;34:2881–7.
595. Mulin B, Talon D, Viel JF, et al. Risk factors for nosocomial colonization with multiresistant *Acinetobacter baumannii*. *Eur J Clin Microbiol Infect Dis* 1995;14:569–76.
596. O'Brien RJ. The epidemiology of nontuberculous mycobacterial disease. *Clin Chest Med* 1989;10:407–18.
597. Böttger EC, Teske A, Kirschner P, et al. Disseminated "*Mycobacterium genavense*" infection in patients with AIDS. *Lancet* 1992;340:76–80.
598. Wallace RJ Jr, Brown BA, Griffith DE. Nosocomial outbreaks/pseudo-outbreaks caused by nontuberculous mycobacteria. *Ann Rev Microbiol* 1998;52:453–90.
599. Chapman JS, Dewlett HJ, Potts WE. Cutaneous reactions to unclassified mycobacterial antigens: a study of children in household contact with patients who excrete unclassified mycobacteria. *Am Rev Respir Dis* 1962;86:547–52.
600. (245) Crow HE, Corpe RF, Smith CE. Is serious pulmonary disease caused by nonphotochromogenic ("atypical") acid-fast mycobacteria communicable? *Dis Chest* 1961;39:372–81.
601. Kuritsky JM, Bullen MG, Broome CV, Silcox VA, Good RC, Wallace, RJ Jr. Sternal wound infections and endocarditis due to organisms of the *Mycobacterium fortuitum* complex. *Ann Intern Med* 1983;98:938–9.
602. Laussucq S, Baltch AL, Smith RP, et al. Nosocomial *Mycobacterium fortuitum* colonization from a contaminated ice machine. *Am Rev Respir Dis* 1988;138:891–4.
603. Panwalker AP, Fuhse E. Nosocomial *Mycobacterium gordonae* pseudo-infection from contaminated ice machines. *Infect Control* 1986;7:67–70.
604. Wallace RJ Jr, Musser JM, Hull SI, et al. Diversity and sources of rapidly growing mycobacteria associated with infections following cardiac surgery. *J Infect Dis* 1989;159:708–16.
605. Burns DN, Wallace RJ Jr, Schultz ME, et al. Nosocomial outbreak of respiratory tract colonization with *Mycobacterium fortuitum*: demonstration of the usefulness of pulsed-field gel electrophoresis in an epidemiologic investigation. *Am Rev Respir Dis* 1991;144:1153–9.
606. Lessing MPA, Walker MM. Fatal pulmonary infection due to *Mycobacterium fortuitum*. *J. Clin Pathol* 1993;46:271–2.
607. (149) Hoy J, Rolston K, Hopfer RL. Pseudoepidemic of *Mycobacterium fortuitum* in bone marrow cultures. *Am J Infect Control* 1987;15:268–71.
608. Lockwood WW, Friedman C, Bus N, Pierson C, Gaynes R. An outbreak of *Mycobacterium terrae* in clinical specimens associated with a hospital potable water supply. *Am Rev Respir Dis* 1989;140:1614–7.
609. Sniadack DH, Ostroff SM, Karlix MA, et al. A nosocomial pseudo-outbreak of *Mycobacterium xenopi* due to a contaminated water supply: lessons in prevention. *Infect Control Hosp Epidemiol* 1993;14:636–41.
610. (148) Cox R, deBorja K, Bach MC. A pseudo-outbreak of *Mycobacterium chelonae* infections related to bronchoscopy. *Infect Control Hosp Epidemiol* 1997;18:136–7.
611. (150) Stine TM, Harris AA, Levin S, Rivera N, Kaplan, RL. A pseudoepidemic due to atypical mycobacteria in a hospital water supply. *JAMA* 1987;258:809–11.
612. (151) Bennett SN, Peterson DE, Johnson DR, Hall WN, Robinson-Dunn B, Dietrich S. Bronchoscopy-associated *Mycobacterium xenopi* pseudo-infections. *Am J Respir Crit Care Med* 1994;150:245–50.
613. Chadha R, Grover M, Sharma A, et al. An outbreak of post-surgical wound infections due to *Mycobacterium abscessus*. *Pediatr Surg Int* 1998;13:406–10.
614. Von Reyn CF, Maslow JN, Barber TW, Falkinham JO III, Arbeit RD. Persistent colonization of potable water as a source of *Mycobacterium avium* infection in AIDS. *Lancet* 1994;343:1137–41.
615. du Moulin GC, Stottmeier KD, Pelletier PA, Tsang AY, Hedley-Whyte J. Concentration of *Mycobacterium avium* by hospital hot water systems. *JAMA* 1988;260:1599–601.
616. Peters M, Müller C, Rüscher-Gerdes S, et al. Isolation of atypical mycobacteria from tap water in hospitals and homes: Is this a possible source of disseminated MAC infection in AIDS patients? *J Infect* 1995;31:39–44.
617. Soto LE, Bobadilla M, Villalobos Y, et al. Post-surgical nasal cellulitis outbreak due to *Mycobacterium chelonae*. *J Hosp Infect* 1991;19:99–106.
618. Wenger JD, Spika JS, Smithwick RW, et al. Outbreak of *Mycobacterium chelonae* infection associated with use of jet injectors. *JAMA* 1990;264:373–6.

619. Safranek TJ, Jarvis WR, Carson LA, et al. *Mycobacterium chelonae* wound infections after plastic surgery employing contaminated gentian violet skin-marking solution. *N Eng J Med* 1987;317:197–201.
620. Gremillion DH, Mursch SB, Lerner CJ. Injection site abscesses caused by *Mycobacterium chelonae*. *Infect Control* 1983;4:25–8.
621. Begg N, O'Mahoney M, Penny P, Richardson AE. *Mycobacterium chelonae* associated with a hospital hydrotherapy pool. *Community Med* 1986;8:348–50.
622. Aubuchon C, Hill JJ Jr, Graham DR. Atypical mycobacterial infection of soft tissue associated with use of a hot tub. A case report. *J Bone Joint Surg* 1986;68-A:766–8.
623. Kirk J, Kaminski GW. *Mycobacterium marinum* infection. *Aust J Dermatol* 1976;17:111–6.
624. Ross BC, Johnson PDR, Oppedisano F, et al. Detection of *Mycobacterium ulcerans* in environmental samples during an outbreak of ulcerative disease. *Appl Environ Microbiol* 1997;63:4135–8.
625. Tokars JI, McNeil MM, Tablan OC, et al. *Mycobacterium gordonae* pseudoinfection associated with a contaminated antimicrobial solution. *J Clin Microbiol* 1990;28:2765–9.
626. Arnow PM, Bakir M, Thompson K, Bova JL. Endemic contamination of clinical specimens by *Mycobacterium gordonae*. *Clin Infect Dis* 2000;31:472–6.
627. Wright EP, Collins CH, Yates MD. *Mycobacterium xenopi* and *Mycobacterium kansasii* in a hospital water supply. *J Hosp Infect* 1985;6:175–8.
628. du Moulin GC, Stottmeier KD. Waterborne mycobacteria: an increasing threat to health. *ASM News* 1986;10:525–9.
629. Engel HWB, Berwald LG. The occurrence of *Mycobacterium kansasii* in tapwater. *Tubercle* 1980;61:21–6.
630. Kubalek I, Mysak J. The prevalence of environmental mycobacteria in drinking water supply systems in a demarcated region in Czech Republic in the period 1984–1989. *Eur J Epidemiol* 1996;12:471–4.
631. Fox C, Smith F, Brogan O, et al. Non-tuberculous mycobacteria in a hospital's piped water supply. *J Hosp Infect* 1992;21:152–4.
632. Aronson T, Holtzman A, Glover N, et al. Comparison of large restriction fragments of *Mycobacterium avium* isolates recovered from AIDS and non-AIDS patients with those of isolates from potable water. *J Clin Microbiol* 1999;37:1008–12.
633. Carson LA, Bland LA, Cusick LB, et al. Prevalence of nontuberculous mycobacteria in water supplies of hemodialysis centers. *Appl Environ Microbiol* 1988;54:3122–5.
634. Carson LA, Petersen NJ, Favero MS, Aguero SM. Growth characteristics of atypical mycobacteria in water and their comparative resistance to disinfectants. *Appl Environ Microbiol* 1978;36:839–46.
635. Taylor RH, Falkinham III JO, Norton CD, LeChevallier MW. Chlorine, chloramine, chlorine dioxide, and ozone susceptibility of *Mycobacterium avium*. *Appl Environ Microbiol* 2000;66:1702–5.
636. Schulze-Röbbecke R, Fischeder R. Mycobacteria in biofilms. *Zbl Hyg* 1989;188:385–90.
637. Schulze-Röbbecke R, Feldmann C, Fischeder R, Janning B, Exner M, Wahl G. Dental units: an environmental study of sources of potentially pathogenic mycobacteria. *Tubercle Lung Dis* 1995;76:318–23.
638. Meisel JL, Perera DR, Meligro C, Rublin CE. Overwhelming watery diarrhea associated with *Cryptosporidium* in an immunosuppressed patient. *Gastroenterology* 1976;70:1156–60.
639. Nime FA, Page DL, Holscher MA, Yardley JH. Acute enterocolitis in a human being infected with the protozoan *Cryptosporidium*. *Gastroenterology* 1976;70:592–8.
640. Goldstein ST, Juranek DD, Ravenholt O, et al. Cryptosporidiosis: an outbreak associated with drinking water despite state-of-the-art treatment. *Ann Intern Med* 1996;124:459–68.
641. Rose JB. Enteric waterborne protozoa: hazard and exposure assessment. In: Craun GF, ed. *Safety of water disinfection: balancing chemical and microbial risks*. Washington, DC: ILSI Press, 1993;115–26.
642. (157) Juranek DD, Addiss D, Bartlett ME, et al. Cryptosporidiosis and public health: workshop report. *J AWWA* 1995;87:69–80.
643. DuPont HL, Chappell CL, Sterling CR, Okhuysen PC, Rose JB, Jakubowski W. The infectivity of *Cryptosporidium parvum* in healthy volunteers. *N Engl J Med* 1995;332:855–9.
644. Okhuysen PC, Chappell CL, Crabb JH, Sterling CR, DuPont HL. Virulence of three distinct *Cryptosporidium parvum* isolates for healthy adults. *J Infect Dis* 1999;180:1275–81.
645. Chappell CL, Okhuysen PC, Sterling CR, Wang C, Jakubowski W, DuPont HL. Infectivity of *Cryptosporidium parvum* in healthy adults with pre-existing anti-*C. parvum* serum immunoglobulin G. *Am J Trop Med* 1999;60:157–64.

646. Meinhardt PL, Casemore DP, Miller KB. Epidemiologic aspects of human cryptosporidiosis and the role of waterborne transmission. *Epidemiol Rev* 1996;18:118–36.
647. Rose JB. Occurrence and significance of *Cryptosporidium* in water. *JAWWA* 1988;80:53–8.
648. Rose JB, Gerba CP, Jakubowski W. Survey of potable water supplies for *Cryptosporidium* and *Giardia*. *Environ Sci Technol* 1991;25:1393–400.
649. LeChevallier MW, Norton WD, Lee RG. *Giardia* and *Cryptosporidium* spp. in filtered drinking water supplies. *Appl Environ Microbiol* 1991;57:2617–21.
650. Mackenzie WR, Hoxie NJ, Proctor ME, et al. A massive outbreak in Milwaukee of *Cryptosporidium* infection transmitted through the public drinking water supply. *N Engl J Med* 1994;331:161–7.
651. Atherton F, Newman CP, Casemore DP. An outbreak of waterborne cryptosporidiosis associated with a public water supply in the UK. *Epidemiol Infect* 1995;115:123–31.
652. Hayes EB, Matte TD, O'Brien TR, et al. Large community outbreak of cryptosporidiosis due to contamination of a filtered public water supply. *N Engl J Med* 1989;320:1372–5.
653. Neill MA, Rice SK, Ahmad NV, Flanigan TP. Cryptosporidiosis: an unrecognized cause of diarrhea in elderly hospitalized patients. *Clin Infect Dis* 1996;22:168–70.
654. Rutala WA, Weber DJ. Water as a reservoir of nosocomial pathogens. *Infect Control Hosp Epidemiol* 1997;18:609–16.
655. Chadwick P. The epidemiological significance of *Pseudomonas aeruginosa* in hospital sinks. *Can J Public Health* 1976;67:323–8.
656. Cordes LG, Wiesenthal AM, Gorman GW, et al. Isolation of *Legionella pneumophila* from hospital shower heads. *Ann Intern Med* 1981;94:195–7.
657. Bollin GE, Plouffe JF, Para MF, Hackman B. Aerosols containing *Legionella pneumophila* generated by shower heads and hot-water faucets. *Appl Environ Microbiol* 1985;50:1128–31.
658. Weber DJ, Rutala WA, Blanchet CN, Jordan M, Gergen MF. Faucet aerators: a source of patient colonization with *Stenotrophomonas maltophilia*. *Am J Infect Control* 1999;27:59–63.
659. Kappstein I, Grundmann H, Hauer T, Niemeyer C. Aerators as a reservoir of *Acinetobacter junii*: an outbreak of bacteraemia in paediatric oncology patients. *J Hosp Infect* 2000;44:27–30.
660. Dennis PJL, Wright AE, Rutter DA, Death JE, Jones BPC. *Legionella pneumophila* in aerosols from shower baths. *J Hyg (Camb)* 1984;93:349–53.
661. (153) American Society of Heating, Refrigerating, and Air-Conditioning Engineers. ASHRAE Guideline 12-2000: minimizing the risk of legionellosis associated with building water systems. Atlanta, GA: ASHRAE, Inc., 2000;1–16.
662. Newsom SWB. Microbiology of hospital toilets. *Lancet* 1972;2:700–3.
663. Gerba CP, Wallis C, Melnick JL. Microbiological hazards of household toilets: droplet production and the fate of residual organisms. *Appl Microbiol* 1975;30:229–37.
664. (152) Hlady WG, Mullen RC, Mintz CS, Shelton BG, Hopkins RS, Daikos GL. Outbreak of Legionnaires' disease linked to a decorative fountain by molecular epidemiology. *Am J Epidemiol* 1993;138:555–62.
665. Rees JC, Allen KD. Holy water — A risk factor for hospital-acquired infection. *J Hosp Infect* 1996;32:51–5.
666. (226) Favero MS, Petersen NJ, Boyer KM, Carson LA, Bond WW. Microbial contamination of renal dialysis systems and associated risks. *ASAIO Trans* 1974;20:175–83.
667. (227) Favero MS, Petersen NJ, Carson LA, Bond WW, Hindman SH. Gram-negative bacteria in hemodialysis systems. *Health Lab Sci* 1975;12:321–34.
668. (228) Favero MS, Petersen NJ. Microbiologic guidelines for hemodialysis systems. *Dialysis Transplant* 1979;6:34–6.
669. Griffiths PA, Babb JR, Bradley CR, Fraise AP. Glutaraldehyde-resistant *Mycobacterium chelonae* from endoscope washer disinfectors. *J Appl Microbiol* 1997;82:519–26.
670. Phillips G, McEwan H, Butler J. Quality of water in washer-disinfectors. *J Hosp Infect* 1995;31:152–4.
671. (259) Cooke RPD, Whymant-Morris A, Umasankar RS, Goddard SV. Bacteria-free water for automatic washer-disinfectors: an impossible dream? *J Hosp Infect* 1998;48:63–5.
672. Humphreys H, Lee JV. Water quality for endoscopy washer-disinfectors. *J Hosp Infect* 1999;42:76–8.
673. Muscarella LF. Are all sterilization processes alike? *AORN J* 1998;67:966–70, 973–6.
674. U.S. Food and Drug Administration. MAUDE database. Available at: www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfMAUDE/search.CFM

675. Alvarado CJ, Stolz SM, Maki DG. Nosocomial infections from contaminated endoscopes: a flawed automated endoscope washer — an investigation using molecular epidemiology. *Am J Med* 1991;91 (Suppl 3b):272–80.
676. Abrutyn E, Goodhart GL, Roos K, Anderson R, Buxton A. *Acinetobacter calcoaceticus* outbreak associated with peritoneal dialysis. *Am J Epidemiol* 1978;107:328–35.
677. Mader JT, Reinartz JA. Peritonitis during peritoneal dialysis — the role of the preheating water bath. *J Chronic Dis* 1978;31:635–41.
678. Kosatsky T, Kleeman J. Superficial and systemic illness related to a hot tub. *Am J Med* 1985;79:10–2.
679. McGuckin MB, Thorpe RJ, Abrutyn E. Hydrotherapy: An outbreak of *Pseudomonas aeruginosa* wound infections related to Hubbard tank treatments. *Arch Phys Med Rehabil* 1981;62:283–5.
680. (243) Koepke GH, Christopher RP. Contamination of whirlpool baths during treatment of infected wounds. *Arch Phys Med Rehabil* 1965;46:261–3.
681. Miller JK, LaForest NT, Hedberg M, Chapman V. Surveillance and control of Hubbard tank bacterial contaminants. *Phys Ther* 1972;50:1482–6.
682. Nelson RM, Reed JR, Kenton DM. Microbiological evaluation of decontamination procedures for hydrotherapy tanks. *Phys Ther* 1972;52:919–23.
683. Page CF. The whirlpool bath and cross-infection. *Arch Phys Med Rehabil* 1954;35:97–8.
684. Newsom SWB. Hospital infection from contaminated ice. *Lancet* 1968;2:620–2.
685. Ravn P, Lundgren JD, Kjaeldgaard P, et al. Nosocomial outbreak of cryptosporidiosis in AIDS patients. *Br Med J* 1991;302:277–80.
686. Bangsberg JM, Uldum S, Jensen JS, Bruun BG. Nosocomial legionellosis in three heart-lung transplant patients: case reports and environmental observations. *Eur J Clin Microbiol Infect Dis* 1995;14:99–104.
687. (246) Stout JE, Yu VL, Muraca P. Isolation of *Legionella pneumophila* from the cold water of hospital ice machines: implications for origin and transmission of the organism. *Infect Control* 1985;6:141–6.
688. Cross DF, Benchimol A, Dimond EG. The faucet aerator — a source of *Pseudomonas* infection. *N Engl J Med* 1966;274:1430–1.
689. *Chryseobacterium (Flavobacterium) meningosepticum* outbreak associated with colonization of water taps in a neonatal intensive care unit. *J Hosp Infect* 2001;47:188–92.
690. Brown DG, Baublis J. Reservoirs of *Pseudomonas* in an intensive care unit for newborn infants: Mechanisms of control. *J Pediatr* 1977;90:453–7.
691. Perryman FA, Flournoy DJ. Prevalence of gentamicin- and amikacin-resistant bacteria in sink drains. *J Clin Microbiol* 1980;12:79–83.
692. Doring G, Horz M, Ortelt J, Grupp H, Wolz C. Molecular epidemiology of *Pseudomonas aeruginosa* in an intensive care unit. *Epidemiol Infect* 1993;110:427–36.
693. Teres D, Schweers P, Bushnell LS, Hedley-Whyte J, Feingold DS. Sources of *Pseudomonas aeruginosa* infection in a respiratory/surgical intensive care unit. *Lancet* 1973;1:415–7.
694. Barbeau J, Tanguay R, Faucher E, et al. Multiparametric analysis of waterline contamination in dental units. *Appl Environ Microbiol* 1996;62:3954–9.
695. Atlas RM, Williams JF, Huntington MK. *Legionella* contamination of dental-unit waters. *Appl Environ Microbiol* 1995;61:1208–13.
696. Fayle SA, Pollard MA. Decontamination of dental unit water systems: a review of current recommendations. *Br Dent J* 1996;181:369–72.
697. Pien FD, Bruce AE. Nosocomial *Ewingella americana* bacteremia in an intensive care unit. *Arch Intern Med* 1986;146:111–2.
698. Stiles GM, Singh L, Imazaki G, Stiles QR. Thermodilution cardiac output studies as a cause of prosthetic valve bacterial endocarditis. *J Thorac Cardiovasc Surg* 1984;88:1035–7.
699. Tyndall RL, Lyle MM, Ironside KS. The presence of free-living amoebae in portable and stationary eye wash stations. *Am Ind Hyg Assoc J* 1987;48:933–4.
700. Bowman EK, Vass AA, Mackowski R, Owen BA, Tyndall RL. Quantitation of free-living amoebae and bacterial populations in eyewash stations relative to flushing frequency. *Am Ind Hyg Assoc J* 1996;57:626–33.
701. Siegman-Igra Y, Shalem A, Berger SA, Livio S, Michaeli D. Should potted plants be removed from hospital wards? *J Hosp Infect* 1986;7:82–5.
702. (309) Kates SG, McGinley KJ, Larson EL, Leyden JJ. Indigenous multiresistant bacteria from flowers in hospital and nonhospital environments. *Am J Infect Control* 1991;19:156–61.

703. Zanetti F, Stampi S, De L, et al. Water characteristics associated with the occurrence of *Legionella pneumophila* in dental units. *Eur J Oral Sci* 2000;108:22–8.
704. Peel MM, Calwell JM, Christopher PJ, Harkness JL, Rouch GJ. *Legionella pneumophila* and water temperatures in Australian hospitals. *Aust NZ J Med* 1985;15:38–41.
705. Groothuis DG, Veenendaal HR, Dijkstra HL. Influence of temperature on the number of *Legionella pneumophila* in hot water systems. *J Appl Bacteriol* 1985;59:529–36.
706. Plouffe JF, Webster LR, Hackman B. Relationship between colonization of a hospital building with *Legionella pneumophila* and hot water temperatures. *Appl Environ Microbiol* 1983;46:769–70.
707. (112) Alary Ma, Joly JR. Factors contributing to the contamination of hospital water distribution systems by Legionellae. *J Infect Dis* 1992;165:565–9.
708. U.K. Health & Safety Executive. The control of legionellosis in hot and cold water systems. Supplement to the control of Legionellosis, including Legionnaires' disease. London: Health & Safety Executive Office, 1998;1–4.
709. (167) Marrie TJ, Haldane D, Bezanson G, Peppard R. Each water outlet is a unique ecologic niche for *Legionella pneumophila*. *Epidemiol Infect* 1992;108:261–70.
710. (154) Snyder MB, Siwicki M, Wireman J, et al. Reduction of *Legionella pneumophila* through heat flushing followed by continuous supplemental chlorination of hospital hot water. *J Infect Dis* 1990;162:127–32.
711. (155) Ezzeddine H, Van Ossel C, Delmee M, Wauters G. *Legionella* spp. in a hospital hot water system: effect of control measures. *J Hosp Infect* 1989;13:121–31.
712. Reichert M. Automatic washers/disinfectors for flexible endoscopes. *Infect Control Hosp Epidemiol* 1991;12:497–9.
713. (45) Joint Commission on Accreditation of Healthcare Organizations. Hospital accreditation standards, 2001: environment of care. Oakbrook Terrace, IL: JCAHO Press, 2001;193–220.
714. (161) Best M, Yu VL, Stout J, Goetz A, Muder RR, Taylor F. *Legionellaceae* in the hospital water supply: epidemiologic link with disease and evaluation of a method for control of nosocomial Legionnaires' disease and Pittsburgh pneumonia. *Lancet* 1983;2:307–10.
715. CDC. Emergency Response Planning and Coordination. Available at: www.cdc.gov/nceh/emergency/emergency.htm
716. McGlown KJ, Fottler MD. The impact of flooding on the delivery of hospital services in the southeastern United States. *Health Care Manage Rev* 1996;21:55–71.
717. Fisher, HL. Emergency evacuation of the Denver Veteran's Administration Medical Center. *Milit Med* 1986;151:154–61.
718. Peters, MS. Hospitals respond to water loss during the midwest floods of 1993: preparedness and improvisation. *J Emerg Med* 1996;14:345–50.
719. (156) Joint Commission on Accreditation of Healthcare Organizations. Comprehensive Accreditation manual for hospitals: the official handbook (CAH00SJ). Oakbrook Terrace, IL: JCAHO Press, 2000.
720. Hargreaves J, Shireley L, Hansen S, et al. Bacterial contamination associated with electronic faucets: a new risk for healthcare facilities. *Infect Control Hosp Epidemiol* 2001;22:202–5.
721. (158) CDC. National surveillance of dialysis-associated diseases in the United States, 1997. Atlanta, GA: Public Health Service, U.S. Department of Health and Human Services, 1998.
722. Stout JE, Best ME, Yu VL. Susceptibility of members of the family *Legionellaceae* to thermal stress: implications for heat eradication methods in water distribution systems. *Appl Environ Microbiol* 1986;52:396–9.
723. Bornstein N, Vieilly C, Nowiki M, Paucod JC, Fleurette J. Epidemiological evidence of legionellosis transmission through domestic hot water supply systems and possibilities of control. *Isr J Med Sci* 1986;13:39–40.
724. (162) Meenhorst PL, Reingold AL, Groothuis DG, et al. Water-related nosocomial pneumonia caused by *Legionella pneumophila* serogroups 1 and 10. *J Infect Dis* 1985;152:356–64.
725. (216) Mandel AS, Sprauer MA, Sniadack DH, Ostroff SM. State regulation in hospital water temperature. *Infect Control Hosp Epidemiol* 1993;14:642–5.
726. (168) Department of Health. The control of *Legionella* in health care premises: a code of practice. London: HMSO, 1991.
727. (169) Helms CM, Massanari RM, Wenzel RP, et al. Legionnaires' disease associated with a hospital water system: a five-year progress report on continuous hyperchlorination. *JAMA* 1988;259:2423–7.

728. (170) Edelstein PH, Whittaker RE, Kreiling RL, Howell, CL. Efficacy of ozone in eradication of *Legionella pneumophila* from hospital plumbing fixtures. *Appl Environ Microbiol* 1982;44:1330–4.
729. (171) Muraca P, Stout JE, Yu, VL. Comparative assessment of chlorine, heat, ozone, and UV light for killing *Legionella pneumophila* within a model plumbing system. *Appl Environ Microbiol* 1987;53:447–53.
730. (172) Domingue EL, Tyndall RL, Mayberry WR, Pancorbo OC. Effects of three oxidizing biocides on *Legionella pneumophila* serogroup 1. *Appl Environ Microbiol* 1988;54:741–7.
731. (173) Landeen LK, Yahya MT, Gerba CP. Efficacy of copper and silver ions and reduced levels of free chlorine in inactivation of *Legionella pneumophila*. *Appl Environ Microbiol* 1989;55:3045–50.
732. (174) Matulonis U, Rosenfeld CS, Shaddock RK. Prevention of *Legionella* infections in bone marrow transplant unit: multifaceted approach to decontamination of a water system. *Infect Control Hosp Epidemiol* 1993;14:571–83.
733. (175) Liu Z, Stout JE, Tedesco L, et al. Controlled evaluation of copper-silver ionization in eradicating *Legionella pneumophila* from a hospital water distribution system. *J Infect Dis* 1994;169:919–22.
734. (176) Margolin AB. Control of microorganisms in source water and drinking water. In: Hurst CJ, Knudsen GR, McInerney MJ, Stetzenback LD, Walter MV, eds. *Manual of environmental microbiology*. Washington, DC: American Society for Microbiology Press, 1997;195–202.
735. (177) Freije MR. *Legionella* control in health care facilities: a guide for minimizing risk. HC Information Resources, Inc. 1996;65–75.
736. (178) Yu-sen E, Lin R, Vidic D, Stout JE, Yu VL. *Legionella* in water distribution systems. *J AWWA* 1998;90:112–21.
737. (179) Biurrun A, Caballero L, Pelaz C, Leon E, Gago A. Treatment of a *Legionella pneumophila*-colonized water distribution system using copper-silver ionization and continuous chlorination. *Infect Control Hosp Epidemiol* 1999;20:426–8.
738. (180) Goetz A, Yu VL. Copper-silver ionization: cautious optimism for *Legionella* disinfection and implications for environmental culturing. *Am J Infect Control* 1997;25:449–51.
739. (181) Stout JE, Lin YS, Goetz AM, Muder RR. Controlling *Legionella* in hospital water systems: experience with the superheat-and-flush method and copper-silver ionization. *Infect Control Hosp Epidemiol* 1998;19:911–4.
740. (182) Walker JT, Mackerness CW, Mallon D, Makin T, Williets T, Keevil CW. Control of *Legionella pneumophila* in a hospital water system by chlorine dioxide. *J Ind Microbiol* 1995;15:384–90.
741. (183) Hambidge A. Reviewing efficacy of alternative water treatment techniques. *Health Estate* 2001;55:23–5.
742. (184) Rohr U, Senger M, Selenka F, Turley R, Wilhelm M. Four years of experience with silver-copper ionization for control of *Legionella* in a German university hospital hot water plumbing system. *Clin Infect Dis* 1999;29:1507–11.
743. (185) Cunliffe DA. Inactivation of *Legionella pneumophila* by monochloramine. *J Appl Bacteriol* 1990;68:453–9.
744. (186) Kirmeyer GJ, Foust GW, Pierson GL, Simmler JJ, LeChevalier MW. Optimizing chloramine treatment. Denver, CO: American Water Works Research Foundation, 1993.
745. (187) Kool JL, Carpenter JC, Fields BS. Effect of monochloramine disinfection of municipal drinking water on risk of nosocomial Legionnaires' disease. *Lancet* 1999;353:272–7.
746. (188) Kool JL, Bergmire-Sweat D, Butler JC, et al. Hospital characteristics associated with colonization of water systems by *Legionella* and risk of nosocomial Legionnaires' disease: a cohort study of 15 hospitals. *Infect Control Hosp Epidemiol* 1999;20:798–805.
747. (213) Yu VL, Beam TR Jr, Lumish RM, et al. Routine culturing for *Legionella* in the hospital environment may be a good idea: a three-hospital prospective study. *Am J Med Sci* 1987;294:97–9.
748. Allegheny County Health Department. Approaches to prevention and control of *Legionella* infection in Allegheny County health care facilities. Pittsburgh, PA: Allegheny County Health Department, 1997;1–13.
749. Goetz AM, Stout JE, Jacobs SL, et al. Nosocomial Legionnaires' disease discovered in community hospitals following cultures of the water system: seek and ye shall find. *Am J Infect Control* 1998;26:8–11.
750. Maryland Department of Health and Mental Hygiene. Report of the Maryland Scientific Working Group to study *Legionella* in the water systems in healthcare institutions. Available at: www.dhmd.state.md.us/html/legionella.htm
751. Yu VL. Nosocomial legionellosis: Current epidemiologic issues. In: Remington JS, Swartz MN, eds. *Current clinical topics in infectious diseases*. New York, NY: McGraw-Hill, 1986;239–53.

752. Vickers RM, Yu VL, Hanna SS. Determinants of *Legionella pneumophila* contamination of water distribution systems: 15-hospital prospective study. *Infect Control* 1987;8:357–63.
753. **(214)** Tobin JO, Swann RA, Bartlett CLR. Isolation of *Legionella pneumophila* from water systems: methods and preliminary results. *Br Med J* 1981;282:515–7.
754. Marrie TJ, Bezanson G, Fox J, Kuehn R, Haldane D, Birbridge S. Dynamics of *Legionella pneumophila* in the potable water of one floor of a hospital. In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;238–40.
755. Plouffe JF, Para MF, Maher WE, Hackman B, Webster L. Subtypes of *Legionella pneumophila* serogroup 1 associated with different attack rates. *Lancet* 1983;2:649–50.
756. Fraser DW. Sources of legionellosis. In: Thornsberry C, Balows A, Feeley JC, Jakubowski W, eds. *Legionella: Proceedings of the 2nd International Symposium*. Washington, DC: American Society for Microbiology Press, 1994:277–80.
757. Dourmon E, Bibb WF, Rajagopalan P, Desplaces N, McKinney RM. Monoclonal antibody reactivity as a virulence marker for *Legionella pneumophila* serogroup 1 strain. *J Infect Dis* 1992;165:569–73.
758. Brundrett GW. Guides on avoiding Legionnaires' disease. In: *Legionella and building services*. Oxford, UK: Butterworth Heineman, 1992;346–73.
759. **(191)** Kugler JW, Armitage JO, Helms CM, et al. Nosocomial Legionnaires' disease: occurrence in recipients of bone marrow transplants. *Am J Med* 1983;74:281–8.
760. Lepine LA, Jernigan DB, Butler JC, et al. A recurrent outbreak of nosocomial Legionnaires' disease detected by urinary antigen testing: evidence for long-term colonization of a hospital plumbing system. *Infect Control Hosp Epidemiol* 1998;19:905–10.
761. Barbaree JM. Selecting a subtyping technique for use in investigations of legionellosis epidemics. In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;169–72.
762. Joly JR, McKinney RM, Tobin JO, Bibb WF, Watkins ID, Ramsay D. Development of a standardized subgrouping scheme for *Legionella pneumophila* serogroup 1 using monoclonal antibodies. *J Clin Microbiol* 1986;23:768–71.
763. **(209)** Schoonmaker D, Helmberger T, Birkhead G. Comparison of ribotyping and restriction enzyme analysis using pulsed-field gel electrophoresis for distinguishing *Legionella pneumophila* isolates obtained during a nosocomial outbreak. *J Clin Microbiol* 1992;30:1491–8.
764. **(163)** Johnston JM, Latham RH, Meier FA, et al. Nosocomial outbreak of Legionnaires' disease: molecular epidemiology and disease control measures. *Infect Control* 1987;8:53–8.
765. Best MG, Goetz A, Yu VL. Heat eradication measures for control of nosocomial Legionnaires' disease: Implementation, education, and cost analysis. *Infect Control* 1984;12:26–30.
766. **(164)** Muraca PW, Yu VL, Goetz A. Disinfection of water distribution systems for *Legionella*: a review of application procedures and methodologies. *Infect Control Hosp Epidemiol* 1990;11:79–88.
767. **(210)** Knirsch CA, Jakob K, Schoonmaker D, et al. An outbreak of *Legionella micdadei* pneumonia in transplant patients: evaluation, molecular epidemiology, and control. *Am J Med* 2000;108:290–5.
768. **(211)** CDC. Sustained transmission of nosocomial Legionnaires' Disease — Arizona and Ohio. *MMWR* 1997;46:416–21.
769. **(218)** Patterson WJ, Hay J, Seal DV, McLuckie JC. Colonization of transplant unit water supplies with *Legionella* and protozoa: precautions required to reduce the risk of legionellosis. *J Hosp Infect* 1997;37:7–17.
770. U.S. Department of Labor, Occupational Safety and Health Administration. OSHA technical manual, Section III, Chapter 7. Legionellosis. Available at: www.osha-slc.gov/dts/osta/otm/otm_iii/otm_iii_7.html
771. Rudnick JR, Beck-Sague CM, Anderson RL, Schable B, Miller JM, Jarvis WR. Gram-negative bacteremia in open-heart surgery patients traced to probable tap-water contamination of pressure-monitoring equipment. *Infect Control Hosp Epidemiol* 1996;17:281–5.
772. Miller RP. Cooling towers and evaporative condensers. *Ann Intern Med* 1979;90:667–70.
773. Butler JC, Breiman RF. Legionellosis. In: Evans AS, Brachman PS, eds. *Bacterial infections of humans*, 3rd ed. New York, NY: Plenum Medical, 1998;355–76.
774. Witherell LE, Novick LF, Stone KM, et al. *Legionella* in cooling towers. *J Environ Health* 1986;49:134–9.
775. Cordes LG, Fraser DW, Skaliy P, et al. Legionnaires' disease outbreak at an Atlanta, Georgia country club: evidence for spread from an evaporative condenser. *Am J Epidemiol* 1980;111:425–31.

776. Kaufmann AF, McDade JE, Patton CM, et al. Pontiac fever: isolation of the etiologic agent (*Legionella pneumophila*) and demonstration of its mode of transmission. *Am J Epidemiol* 1981;114:337–47.
777. Morton S, Bartlett CLR, Bibby LF, Hutchinson DM, Dyer JV, Dennis PJ. Outbreak of Legionnaires' disease from a cooling water system in a power station. *Br J Indust Med* 1986;43:630–5.
778. Friedman S, Spitalny K, Barbaree J, Faur Y, McKinney R. Pontiac fever outbreak associated with a cooling tower. *Am J Public Health* 1987;77:568–72.
779. Addiss DG, Davis JP, LaVenture M, Wand PJ, Hutchinson MA, McKinney RM. Community-acquired Legionnaires' disease associated with a cooling tower: evidence for longer-distance transport of *Legionella pneumophila*. *Am J Epidemiol* 1989;130:557–68.
780. Keller DW, Hajjeh R, DeMaria A Jr, et al. Community outbreak of Legionnaires' disease: an investigation confirming the potential for cooling towers to transmit *Legionella* species. *Clin Inf Dis* 1996;22:257–61.
781. Pastoris MC, Ciceroni L, Lo Monaco R, et al. Molecular epidemiology of an outbreak of Legionnaires' disease associated with a cooling tower in Genova-Sestri Ponente, Italy. *Eur J Clin Microbiol Infect Dis* 1997;16:883–92.
782. Brown CM, Nuorti PJ, Breiman RF, et al. A community outbreak of Legionnaires' disease linked to hospital cooling towers: an epidemiological method to calculate dose of exposure. *Inter J Epidemiol* 1999;28:353–9.
783. Broadbent CR. *Legionella* in cooling towers: Practical research, design, treatment, and control guidelines. In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;217–22.
784. (222) Bhopal RS, Barr G. Maintenance of cooling towers following two outbreaks of Legionnaires' disease in a city. *Epidemiol Infect* 1990;104:29–38.
785. CDC. Suggested health and safety guidelines for public spas and hot tubs. Atlanta, GA: Centers for Disease Control, 1985. Publication No. 99–960.
786. (221) World Health Organization. Environmental aspects of the control of Legionellosis, 14th ed. Copenhagen, Denmark: World Health Organization, 1986. Schriftenr Ver Wasser Boden Lufthyg 1993;91:249–52. (German)
787. (223) World Health Organization. Epidemiology, prevention, and control of legionellosis: memorandum from a WHO meeting. *Bull WHO* 1990;68:155–64.
788. Association for the Advancement of Medical Instrumentation. American National Standard Hemodialysis Systems ANSI/AAMI RD5-1981, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 1982.
789. (229) Association for the Advancement of Medical Instrumentation. American National Standard Hemodialysis Systems ANSI/AAMI RD5-1992, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 1993.
790. Association for the Advancement of Medical Instrumentation. Reuse of hemodialyzers ROH-1986, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 1986.
791. (230) Association for the Advancement of Medical Instrumentation. American National Standard Reuse of hemodialyzers ANSI/AAMI RD47-1993, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 1993.
792. (236) Association for the Advancement of Medical Instrumentation. Water treatment equipment for hemodialysis applications. ANSI/AAMI RD62-2001, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 2001.
793. Tokars JI, Miller ER, Alter MJ, Arduino MJ. National surveillance of dialysis associated diseases in the United States, 1997. *Semin Dialysis* 2000;13:75–85.
794. Hindman SH, Carson LA, Petersen NJ, et al. Pyrogenic reactions during hemodialysis caused by extramural endotoxin. *Lancet* 1975;2:732–4.
795. Stamm JE, Engelhard WE, Parson JE. Microbiological study of water softener resins. *Appl Microbiol* 1969;18:376–86.
796. Alter MJ, Favero MS, Miller JK, Coleman BJ, Bland LA. National surveillance of dialysis-associated diseases in the United States, 1988. *ASAIO Trans* 1990;36:107–18.
797. Tokars JI, Alter MJ, Favero MS, Moyer LA, Bland LA. National surveillance of dialysis-associated diseases in the United States, 1990. *ASAIO J* 1993;39:71–80.
798. Tokars JI, Alter MJ, Favero MS, Moyer LA, Bland LA. National surveillance of dialysis-associated diseases in the United States, 1991. *ASAIO J* 1993;39:966–75.

799. Tokars JI, Alter MJ, Favero MS, Moyer LA, Bland LA. National surveillance of dialysis-associated diseases in the United States, 1993. *ASAIO J* 1996;42:219–29.
800. **(231)** Petersen NJ, Boyer KM, Carson LA, Favero MS. Pyrogenic reactions from inadequate disinfection of a dialysis unit distribution system. *Dialysis Transpl* 1978;7:52–7.
801. Gazenfeldt-Gazit E, Elaihou HE. Endotoxin antibodies in patients on maintenance hemodialysis. *Israel J Med Sci* 1969;5:1032–6.
802. Laude-Sharp M, Canoff M, Simard L, Pusineri C, Kazatchkine M, Haeffner-Cavaillon N. Induction of IL-1 during hemodialysis: transmembrane passage of intact endotoxin (LPS). *Kidney Int* 1990;38:1089–94.
803. Arduino MJ, Bland LA, McAllister SK, Favero MS. The effects of endotoxin contaminated dialysate and polysulfone or cellulosic membranes on the release of TNF α during simulated dialysis. *Artif Organs* 1995;19:880–6.
804. Greisman SE, Hornick RB. Comparative pyrogenic reactivity of rabbit and man to bacterial endotoxin. *Proc Soc Exp Biol Med* 1969;131:1154–8.
805. Weary ME, Donohue G, Pearson FC, Story K. Relative potencies of four reference endotoxin standards as measured by the *Limulus* amoebocyte lysate and USP rabbit pyrogen tests. *Appl Environ Microbiol* 1980;40:1148–51.
806. **(239)** Bland LA, Ridgeway MR, Aguerro SM, Carson LA, Favero MS. Potential bacteriologic and endotoxin hazards associated with liquid bicarbonate concentrate. *ASAIO Trans* 1987;33:542–5.
807. **(232)** Dawids SG, Vejlsgaard R. Bacteriological and clinical evaluation of different dialysate delivery systems. *Acta Med Scand* 1976;199:151–5.
808. Favero MS, Alter MJ, Tokars JI, Arduino MJ. Dialysis-associated infections and their control. In: Bennett JV, Brachman PS, eds. *Hospital infections* 4th ed. Philadelphia, PA: Lippincott-Raven, 1998;357–80.
809. **233** Kidd EE. Bacterial contamination of dialyzing fluid of artificial kidney. *Br Med J* 1964;1:880–2.
810. Jones DM, Tobin BM, Harlow GR, et al. Bacteriological studies of the modified kiil dialyzer. *Br Med J* 1970;3:135–7.
811. **(240)** Raji L, Shapiro FL, Michael AF. Endotoxemia in febrile reactions during hemodialysis. *Kidney Int* 1973;4:57–60.
812. Vanholder R, Van Haecke E, Veys N, et al. Endotoxin transfer through dialysis membranes: small versus large-pore membranes. *Nephrol Dial Transplant* 1992;7:333–9.
813. Evans RC, Holmes CJ. In vitro study of the transfer of cytokine-inducing substances across selected high-flux hemodialysis membranes. *Blood Purif* 1991;9:92–101.
814. Lonnemann G, Behme TC, Lenzer B, et al. Permeability of dialyzer membranes to TNF α -inducing substances derived from water bacteria. *Kidney Int* 1992;42:61–8.
815. Ureña P, Herbelin A, Zingraff J, et al. Permeability of cellulosic and non-cellulosic membranes to endotoxin subunits and cytokine production during in-vitro hemodialysis. *Nephrol Dial Transplant*. 1992;7:16–28.
816. **(241)** Bommer J, Becker KP, Urbaschek R. Potential transfer of endotoxin across high-flux polysulfone membranes. *J Am Soc Nephrol* 1996;7:883–8.
817. Yamagami S, Adachi T, Sugimura, T, et al. Detection of endotoxin antibody in long-term dialysis patients. *Int J Artif Organs* 1990;13:205–10.
818. Arduino MJ. CDC investigations of noninfectious outbreaks of adverse events in hemodialysis facilities, 1979–1999. *Semin Dialysis* 2000;13:86–91.
819. Roth V, Jarvis WR. Outbreaks of infection and/or pyrogenic reactions in dialysis patients. *Semin Dialysis* 2000;13:92–100.
820. Gordon SM, Tipple MME, Bland LA, Jarvis WR. Pyrogenic reactions associated with reuse of disposable hollow-fiber hemodialyzers. *JAMA* 1988;260:2077–81.
821. Alter MJ, Tokars JI, Arduino MJ. Nosocomial infections in hemodialysis units — strategies for control. In: Owen WF, Periera BJG, Sayegh MH, eds. *Dialysis and transplantation: a companion to Brenner and Rector's "The Kidney."* Orlando, FL: WB Saunders Company, 1999;337–57.
822. Bernick JJ, Port FK, Favero MS, Brown DG. Bacterial and endotoxin permeability of hemodialysis membranes. *Kidney Int* 1979;16:491–6.
823. Bommer J, Becker KP, Urbaschek R, Ritz E, Urbaschek B. No evidence for endotoxin transfer across high flux polysulfone membranes. *Clin Nephrol* 1987;27:278–82.
824. Schindler R, Lonnemann G, Schaeffer J, et al. The effect of ultrafiltered dialysate on the cellular content of interleukin-1 receptor antagonist in patients on chronic hemodialysis. *Nephron* 1994;68:229–33.

825. Akrum RAE, Frolich M, Gerritsen AF, et al. Improvement of chronic inflammatory state in hemodialysis patients by the use of ultrapure water for dialysate. *J Am Soc Nephrol* 1997;8:226A.
826. Quellhorst E. Methods of Hemodialysis. *Nieren U Hochdruck* 1998;27:35–41.
827. Baz M, Durand C, Ragon A, et al. Using ultrapure water in hemodialysis delays carpal tunnel syndrome. *Int J Artif Organs* 1991;14:681–5.
828. Schwalbe S, Holzhauser M, Schaeffer J, et al. β 2-Microglobulin associated amyloidosis: a vanishing complication of long-term hemodialysis? *Kidney Int* 1997;52:1077–83.
829. (242) Arduino MJ, Favero MS. Microbiologic aspects of hemodialysis: water quality for hemodialysis. AAMI Monograph WQD-1998. Arlington, VA: Association for the Advancement of Medical Instrumentation, 1998.
830. Leyboldt JK, Schmidt B, Gurland, HJ. Measurement of backfiltration rates during hemodialysis with highly permeable membranes. *Blood Purif* 1991;9:74–84.
831. Carson LA, Bland LA, Cusick LB, Collin S, Favero MS, Bolan G. Factors affecting endotoxin levels in fluids associated with hemodialysis procedures. In: Novitsky TJ, Watson SW, eds. Detection of bacterial endotoxins with the Limulus Amoebocyte Lysate Test. New York, NY: Alan R. Liss, 1987;223–4.
832. Anderson RL, Holland BW, Carr JK, Bond WW, Favero MS. Effect of disinfectants on pseudomonads colonized on the interior surface of PVC pipes. *Am J Public Health* 1990;80:17–21.
833. Bland LA, Favero MS. Microbial contamination control strategies for hemodialysis. JCAHO Plant Tech Manage Series 1989;3:30–6.
834. (237) Bland LA. Microbiological and endotoxin assays of hemodialysis fluids. *Adv Renal Replacement Ther* 1995;2:70–9.
835. (238) Arduino MJ, Bland LA, Aguero SM, Carson LA, Ridgeway M, Favero MS. Comparison of microbiologic assay methods for hemodialysis fluids. *J Clin Microbiol* 1991;29:592–4.
836. Association for the Advancement of Medical Instrumentation. American national standard water treatment equipment for hemodialysis applications. ANSI/AAMI RD62-1999. Arlington, VA: Association for the Advancement of Medical Instrumentation, 1999.
837. Arduino MJ. How should dialyzers be reprocessed? *Semin Dialysis* 1998;11:282–4.
838. Jochimsen EM, Frenette C, Delorme M, et al. A cluster of bloodstream infections and pyrogenic reactions among hemodialysis patients traced to dialysis machine waste-handling option units. *Am J Nephrol* 1998;18:485–9.
839. Wang SA, Levine RB, Carson LA, et al. An outbreak of gram-negative bacteremia in hemodialysis patients traced to hemodialysis machine waste drain ports. *Infect Control Hosp Epidemiol* 1999;20:746–51.
840. National Institutes of Health. U.S. Renal Diseases Survey: 1999 Annual Data Report. Bethesda, MD: National Institute of Diabetes, Digestive and Kidney Diseases, Division of Kidney, Urologic, and Hematologic Diseases, 1999.
841. Monsen T, Olofson C, Ronnmark M, Wistrom J. Clonal spread of staphylococci among patients with peritonitis associated with continuous ambulatory peritoneal dialysis. *ASAIO J* 2000;57:613–8.
842. Band JD, Ward JI, Fraser DW, et al. Peritonitis due to a *Mycobacterium chelonae*-like organism associates with intermittent chronic peritoneal dialysis. *J Infect Dis* 1982;145:9–17.
843. Monsen T, Crabtree JH, Siddiqui RA, et al. Dialysis catheter infection related peritonitis: incidence and time dependent risk. *ASAIO J* 1999;45:574–80.
844. Vera G, Lew SQ. *Mycobacterium fortuitum* peritonitis in two patients receiving continuous ambulatory peritoneal dialysis. *Am J Nephrol* 1999;19:586–9.
845. Soriano F, Rodriguez-Tudela JL, Gomez-Garces JL, Velo M. Two possibly related cases of *Mycobacterium fortuitum* peritonitis in continuous ambulatory peritoneal dialysis. *Eur J Clin Microbiol* 1989;8:895–7.
846. Szeto CC, Li PK, Leung CB, Yu AW, Lui SF, Lai NK. *Xanthomonas maltophilia* peritonitis in uremic patients receiving ambulatory peritoneal dialysis. *Am J Kidney Dis* 1997;29:991–5.
847. Panlilio AL, Beck-Sague CM, Siegel JD, et al. Infections and pseudoinfections due to povidone-iodine solution contaminated with *Pseudomonas cepacia*. *Clin Infect Dis* 1992;14:1078–83.
848. Riebel W, Frantz N, Adelstein D, Spanguolo PJ. *Corynebacterium JK*: a cause of nosocomial device-related infection. *Rev Infect Dis* 1986;8:42–9.
849. Radix AE, Bieluch VM, Graeber CW. Peritonitis caused by *Monilia sitophila* in a patient undergoing peritoneal dialysis. *Int J Artif Organs* 1996;19:218–20.
850. Banerjee S, Marwaha RK, Bajwa RP. Fungal peritonitis complicating peritoneal dialysis. *Indian Pediatr* 1995;32:693–7.

851. Bergeson E, Denis R, Cartier P. Peritoneal dialysis: peritonitis and catheter infections. *Annales de Chirurgie* 1996;50:606–12. (French)
852. Troidle L, Kliger AS, Goldie SJ, et al. Continuous peritoneal dialysis-associated peritonitis of nosocomial origin. *Perit Dialysis International* 1996;16:505–10.
853. Smith CA. Reduced incidence of peritonitis by utilizing “flush before fill” in APD. *Adv Perit Dialysis* 1997;13:224–6.
854. Valeri A, Radhakrishnan J, Vernocchi L, Carmichael LD, Stern L. The epidemiology of peritonitis in acute peritoneal dialysis: a comparison between open- and closed drainage systems. *Am J Kidney Dis* 1993;21:300–9.
855. Stamm WE, Colelle JJ, Anderson RL, Dixon RE. Indwelling arterial catheters as a source of nosocomial bacteremia: an outbreak caused by *Flavobacterium* species. *N Engl J Med* 1975;292:1099–102.
856. Schimpff SC. Gram negative bacteremia. *Support Care Cancer* 1993;1:5–18.
857. Graman PS, Quinlan GA, Rank JA. Nosocomial legionellosis traced to contaminated ice. *Infect Control Hosp Epidemiol* 1997;18:637–40.
858. Gahrn-Hansen B, Uldum SA, Schmidt J, Nielsen B, Birkeland SA, Jorgensen KA. [Nosocomial *Legionella pneumophila* infection in a nephrology department]. *Ugeskrift for Laeger* 1995;157:590–4. (German)
859. Wilson IG, Hogg GM, Barr JG. Microbiological quality of ice in hospital and community. *J Hosp Infect* 1997;36:171–80.
860. Spencer RC. The emergence of epidemic, multiple-antibiotic-resistant *Stenotrophomonas (Xanthomonas) maltophilia* and *Burkholderia (Pseudomonas) cepacia*. *J Hosp Infect* 1995;30(suppl):453–64.
861. (248) Cannon RO, Poliner JR, Hirschhorn RB, et al. A multistate outbreak of Norwalk virus gastroenteritis associated with consumption of commercial ice. *J Infect Dis* 1991;164:860–3.
862. (249) Khan AS, Moe CL, Glass RI, et al. Norwalk virus-associated gastroenteritis traced to ice consumption aboard a cruise ship in Hawaii: comparison and application of molecular method-based assays. *J Clin Microbiol* 1994;32:318–22.
863. (244) CDC. Outbreak of viral gastroenteritis — Pennsylvania and Delaware. *MMWR* 1987;36:709–11.
864. Quick R, Paugh K, Addiss D, Kobayashi J, Baron R. Restaurant-associated outbreak of giardiasis. *J Infect Dis* 1992;166:673–6.
865. Hedberg CW, White KE, Johnson JA, et al. An outbreak of *Salmonella enteritidis* infection at a fast food restaurant: implications for foodhandler-associated transmission. *J Infect Dis* 1991;164:1135–40.
866. Burnett IA, Weeks GR, Harris DM. A hospital study of ice-making machines: their bacteriology, design, usage, and upkeep. *J Hosp Infect* 1994;28:305–13.
867. Petersen NJ. Don't culture the ice machines. *Hosp Infect Control* 1982;9:8–9.
868. CDC. Sanitary care and maintenance of ice chests and ice machines. Atlanta, GA: CDC, 1979. Publication No. 00-2384.
869. (247) Manangan LP, Anderson RL, Arduino MJ, Bond WW. Sanitary care and maintenance of ice-storage chests and ice-making machines in healthcare facilities. *Am J Infect Control* 1998;26:111–2.
870. Anonymous. Ice as a source of infection. *CDR Weekly* 1993;3:241.
871. Cardaney CR, Rodeheaver GT, Horowitz JH, Kenney JG, Edlich RF. Influence of hydrotherapy and antiseptic agents on burn wound bacteria contamination. *J Burn Care Rehab* 1985;6:230–2.
872. Gruber RP, Laub DR, Vistnes LM. The effect of hydrotherapy on the clinical course and pH of experimental cutaneous chemical burns. *Plastic Reconstruct Surg* 1975;55:200–4.
873. Mansell RE, Borchardt KA. Disinfecting hydrotherapy equipment. *Arch Phys Med Rehabil* 1974;55:318–20.
874. Hall J, Skevington SM, Maddison PH, Chapman K. A randomized and controlled trial of hydrotherapy in rheumatoid arthritis. *Arthritis Care Res* 1996;9:206–15.
875. Gross A, Cutright DE, Bhaskar SN. Effectiveness of pulsating water jet lavage in treatment of contaminated crush injuries. *Am J Surgery* 1972;124:373–7.
876. Rodeheaver GT, Paltry D, Thacker JG, Edgerton MT, Edlich RF. Wound cleansing by high pressure irrigation. *Surg Gynecol Obstetr* 1975;141:357–62.
877. Saxe A, Goldestein E, Dixon S, Ostrup R. Pulsatile lavage in the management of postoperative wound infections. *Am Surgeon* 1980;46:391–7.
878. Weller K. In search of efficacy and efficiency: an alternative to conventional wound cleansing modalities. *Ostomy/Wound Manage* 1991;37:23–8.
879. Solomon SL. Host factors in whirlpool-associated *Pseudomonas aeruginosa* skin disease. *Infect Control* 1985;6:402–6.

880. Hicks CB, Chulay JD. Bacteremic *Citrobacter freundii* cellulitis associated with tub immersion in a patient with the nephrotic syndrome. *Mil Med* 1988;153:400–1.
881. Mayhall CG, Lamb VA, Gayle WE, Haynes BW. *Enterobacter cloacae* septicemia in a burn center: epidemiology and control of an outbreak. *J Infect Dis* 1979;139:166–71.
882. Marrie TJ, Gass RSR, Yates L. *Legionella pneumophila* in a physiotherapy pool. *Eur J Clin Microbiol* 1987;6:212–3.
883. Havelaar AH, Berwald LG, Groothuis DG, Baas JG. Mycobacteria in semi-public swimming pools and whirlpools. *Ztb Bakteriol Mikrobiol Hyg [B]* 1985;180:505–14.
884. Favero MS. Whirlpool spa-associated infections: are we really in hot water? *Am J Public Health* 1984;74:653–5.
885. Ratnam S, Hogan K, March SB, Butler RW. Whirlpool-associated folliculitis caused by *Pseudomonas aeruginosa*: report of an outbreak and review. *J Clin Microbiol* 1986;23:655–9.
886. Stone HH, Kolb LD. The evolution and spread of gentamicin-resistant *Pseudomonas*. *J Trauma* 1971;11:586–9.
887. Richard P, LeFlock R, Chamoux C, Pannier M, Espaze E, Richet H. *Pseudomonas aeruginosa* outbreak in a burn unit: role of antimicrobials in the emergence of multiply resistant strains. *J Infect Dis* 1994;170:377–83.
888. Berrouane YF, McNutt L-A, Buschelman BJ, et al. Outbreak of severe *Pseudomonas aeruginosa* infections caused by a contaminated drain in a whirlpool bathtub. *Clin Infect Dis* 2000;31:1331–7.
889. (250) Schmidt OW, Cooney MK, Foy HM. Adeno-associated virus in adenovirus type 3 conjunctivitis. *Infect Immun* 1975;11:1362–70.
890. DeJonckheere JF. Hospital hydrotherapy pools treated with ultraviolet light: bad bacteriological quality and presence of thermophilic *Naegleria*. *J Hyg (Lond)* 1982;88:205–14.
891. American Physical Therapy Association. Hydrotherapy/therapeutic pool infection control guidelines. Alexandria, VA: APTA, 1995;112.
892. CDC. Disinfection of hydrotherapy pools and tanks. Atlanta, GA: Centers for Disease Control, Public Health Service, U.S. Department of Health and Human Services, 1974. Publication No. HHS 00-2383.
893. Price D, Ahearn DG. Incidence and persistence of *Pseudomonas aeruginosa* in whirlpools. *J Clin Microbiol* 1988;26:1650–4.
894. (252) White CG. Chemistry of chlorination. In: Handbook of chlorination and alternative disinfectants, 3rd ed. New York, NY: Van Nostrand Reinhold, 1992;184–249.
895. Mayhall CG. Burn patients. In: Pfeiffer J, ed. APIC Text of infection control and epidemiology. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc (APIC), 2000;32.1–32.8.
896. Smith RF, Blasi D, Dayton SL, Chipps DD. Effects of sodium hypochlorite on the microbial flora of burns and normal skin. *J Trauma* 1974;14:938–44.
897. Cardany CR, Rodeheaver GT, Horowitz JH, Kenney JG, Edlich RF. Influence of hydrotherapy and antiseptic agents on burn wound bacterial contamination. *J Burn Care Rehabil* 1985;6:230–2.
898. Steve L, Goodhart P, Alexander J. Hydrotherapy burn treatment: use of chloramine-T against resistant microorganisms. *Arch Phys Med Rehabil* 1979;60:301–3.
899. Golland A. Basic hydrotherapy. *Physiotherapy* 1981;67:258–62.
900. Edlich RF, Becker DG, Phung D, McClelland WA, Day SG. Water treatment of hydrotherapy exercise pools. *J Burn Care Rehabil* 1988;9:9510–5.
901. Penny PT. Hydrotherapy pools of the future — the avoidance of health problems. *J Hosp Infect* 1991;18:535–42.
902. CDC. Swimming pools: safety and disease control through proper design and operation. Atlanta, GA: U.S. Department of Health and Human Services, 1976. Publication No. HHS No. 88–8319.
903. Linneman CC Jr. Nosocomial infections associated with physical therapy, including hydrotherapy. In: Mayhall CG, ed. Hospital epidemiology and infection control, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;931–6.
904. Aspinall ST, Graham R. Two sources of contamination of a hydrotherapy pool by environmental organisms. *J Hosp Infect* 1989;14:285–92.
905. (251) McCandlish R, Renfrew M. Immersion in water during labor and birth: the need for evaluation. *Birth* 1993;20:79–85.
906. Hawkins S. Water vs conventional births: Infection rates compared. *Nursing Times* 1995;91:38–40.

907. Vochem M, Vogt M, Doring G. Sepsis in a newborn due to *Pseudomonas aeruginosa* from a contaminated tub bath. *N Engl J Med* 2001;345:378–9.
908. Eriksson M, Ladfors L, Mattsson LA, Fall O. Warm tub bath during labor: a study of 1385 women with prelabor rupture of the membranes after 34 weeks of gestation. *Acta Obstet Gynaecol Scand* 1996;75:642–4.
909. Rush J, Burlock S, Lambert K, Loosley-Millman M, Hutchinson B, Enkin M. The effects of whirlpool baths in labor: a randomized, controlled trial. *Birth* 1996;23:136–3.
910. Davis BJ. Whirlpool operation and the prevention of infection. *Infect Control* 1985;6:394–7.
911. (253) Muscarella LF. Automatic flexible endoscope reprocessors. *Gastrointest Endosc Clin N Am* 2000;10:245–57.
912. (254) Muscarella LF. Anticipated reliability of liquid chemical sterilants [letter]. *Am J Infect Control* 1998;26:155–6.
913. (255) Muscarella LF. Dejà vu... all over again? The importance of instrument drying [letter]. *Infect Control Hosp Epidemiol* 2000;21:628–9.
914. (256) Gubler JGH, Salfinger M, von Graevenitz A. Pseudoepidemic of nontuberculous mycobacteria due to a contaminated bronchoscope cleaning machine: report of an outbreak and review of the literature. *Chest* 1992;101:1245–9.
915. (257) Fraser VJ, Jones M, Murray PR, Medoff G, Zhang Y, Wallace RJ Jr. Contamination of flexible fiberoptic bronchoscopes with *Mycobacterium chelonae* linked to an automated bronchoscope disinfection machine. *Am Rev Respir Dis* 1992;145:853–5.
916. Maloney S, Welbel S, Daves B, et al. *Mycobacterium abscessus* pseudoinfection traced to an automated endoscope washer: utility of epidemiologic and laboratory investigation. *J Infect Dis* 1994;169:1166–9.
917. Merighi A, Contato E, Scagliarini R, et al. Quality improvement in gastrointestinal endoscopy: microbiologic surveillance of disinfection. *Gastrointest Endosc* 1996;43:457–62.
918. (258) Muscarella LF. Application of environmental sampling to flexible endoscope reprocessing: the importance of monitoring the rinse water. *Infect Control Hosp Epidemiol* 2002;23:285–9.
919. Mitchell DH, Hicks LJ, Chiew R, Montanaro JC, Chen SC. Pseudoepidemic of *Legionella pneumophila* serogroup 6 associated with contaminated bronchoscopes. *J Hosp Infect* 1997;37:19–23.
920. Ido K, Ishino Y, Ota Y, et al. Deficiencies of automatic endoscopic reprocessors: a method to achieve high-grade disinfection of endoscopes. *Gastrointest Endosc* 1996;44:583–6.
921. (260) Allen JJ, Allen MO, Olsen MM, et al. *Pseudomonas* infection of the biliary system resulting from the use of a contaminated endoscope. *Gastroenterology* 1987;92:759–63.
922. Agerton T, Valway S, Gore B, et al. Transmission of a highly drug-resistant strain (Strain W-1) of *Mycobacterium tuberculosis*: community outbreak and nosocomial transmission via a contaminated bronchoscope. *JAMA* 1997;278:1073–7.
923. (261) Michele TM, Cronin WA, Graham NMH, et al. Transmission of *Mycobacterium tuberculosis* by a fiberoptic bronchoscope: identification by DNA fingerprinting. *JAMA* 1997;278:1093–5.
924. Bronowicki J-P, Venard V, Botte C, et al. Patient-to-patient transmission of hepatitis C virus during colonoscopy. *N Engl J Med* 1997;337:237–40.
925. (262) U.S. Food and Drug Administration, CDC. FDA and CDC Public health advisory: infection from endoscopes inadequately reprocessed by an automated endoscope reprocessing system. September 10, 1999. Available at: www.fda.gov/cdrh/safety.html
926. Rey JF. Endoscopic disinfection. A worldwide problem. *J Clin Gastroenterol* 1999;28:291–7.
927. Wang HC, Liaw YS, Yang PC, Kuo SH, Luh KT. A pseudoepidemic of *Mycobacterium chelonae* infection caused by contamination of a fiberoptic bronchoscope suction channel. *Eur Respir J* 1995;8:1259–62.
928. (263) Alvarado CJ, Reichelderfer M. APIC guideline for infection prevention and control in flexible endoscopy. *Am J Infect Control* 2000;28:138–55.
929. Van Klingeren B, Pullen W. Glutaraldehyde resistant mycobacteria from endoscope washers. *J Hosp Infect* 1993;25:147–9.
930. Flournoy DJ, Petrone RL, Voth DW. A pseudo-outbreak of *Methylobacterium mesophilica* isolated from patients undergoing bronchoscopy. *Eur J Clin Microbiol Infect Dis* 1992;11:240–3.
931. Reeves DS, Brown NM. Mycobacterial contamination of fiberoptic bronchoscopes. *J Hosp Infect* 1995;30(suppl):S531–S536.
932. Kelstrup J, Funder-Nielsen T, Theilade J. Microbial aggregate contamination of water lines in dental equipment and its control. *Acta Path Scand* 1977;85:177–83.

933. Challacombe SJ, Fernandes LL. Detecting *Legionella pneumophila* in water systems: a comparison of various dental units. *J Am Dent Assoc* 1995;126:603–8.
934. Singh R, Stine OC, Smith DL, Spitznagel JK Jr, Labib ME, Williams HN. Microbial diversity of biofilms in dental unit water systems. *Appl Environ Microbiol* 2003;69:3412–20.
935. (264) CDC. Statement from the Centers for Disease Control and Prevention (CDC) regarding biofilm and dental unit water quality. Atlanta, GA: U.S. Public Health Service, Department of Health and Human Services, 1999. Available at: www.cdc.gov/nccdphp/oh/ic-fs-biofilm.htm
936. (265) CDC. Recommended infection control practices for dentistry, 1993. *MMWR* 1993;42(No. RR-8):1–12.
937. (268) Bagga BS, Murphy RA, Anderson AW, Punwani I. Contamination of dental unit cooling water with oral microorganisms and its prevention. *J Am Dent Assoc* 1984;109:712–6.
938. Scheid RC, Rosen S, Beck FM. Reduction of CFUs in high-speed handpiece water lines over time. *Clin Prev Dent* 1990;12:9–12.
939. Williams JF, Johnston AM, Johnson B, Huntington MK, Mackenzie CD. Microbial contamination of dental unit waterlines: prevalence, intensity, and microbiological characteristics. *J Am Dent Assoc* 1993;124:59–65.
940. Santiago JI, Huntington MK, Johnston AM, Quinn RS, Williams JF. Microbial contamination of dental unit waterlines: short- and long-term effects of flushing. *Gen Dent* 1994;42:528–35.
941. Williams HN, Johnson A, Kelley JI, et al. Bacterial contamination of the water supply in newly installed dental units. *Quintessence Int* 1995;26:331–7.
942. CDC. Guideline for infection control in dental health-care settings. *MMWR* 2003;52:in press.
943. (266) Office of Safety and Asepsis Procedures Research Foundation. Position paper on dental unit waterlines. Annapolis, MD: OSAPRF, 2000. Available at: www.osap.org/issues/pages/water/duwl.htm
944. (267) U.S. Environmental Protection Agency. National primary drinking water regulations, 40 CFR 1, Part 141, Subpart G;1999. At: www.epa.gov/safewater/mcl.html
945. (344) Eaton AD, Clesceri LS, Greenberg AE, eds. Standard methods for the examination of water and wastewater, 20th ed. Washington, DC: American Public Health Association, 1998;9–1 through 9–41.
946. (269) Shearer BG. Biofilm and the dental office. *J Am Dent Assoc* 1996;127:181–9.
947. Maki DG, Alvarado CJ, Hassemer CA, Zilz MA. Relation of the inanimate hospital environment to endemic nosocomial infection. *N Engl J Med* 1982;307:1562–6.
948. Danforth D, Nicolle LE, Hume K, Alfieri N, Sims H. Nosocomial infections on nursing units with floors cleaned with a disinfectant compared with detergent. *J Hosp Infect* 1987;10:229–35.
949. Spaulding EH. Role of chemical disinfection in the prevention of nosocomial infections. In: Brachman PS, Eickhoff TC, eds. *Proceedings of the International Conference on Nosocomial Infections, 1970*. Chicago, IL: American Hospital Association, 1971;247–54.
950. Spaulding EH. Chemical disinfection and antisepsis in the hospital. *J Hosp Res* 1972;9:5–31.
951. (273) Favero MS, Bond WW. Chemical disinfection of medical and surgical materials. In: Block SS, ed. *Disinfection, sterilization, and preservation, 5th ed.* Philadelphia, PA: Lippincott Williams & Wilkins, 2001;881–917.
952. (274) Rutala WA. APIC guideline for selection and use of disinfectants. *Am J Infect Control* 1996;24:313–42.
953. Agolini G, Russo A, Clementi M. Effect of phenolic and chlorine disinfectants on hepatitis C virus binding and infectivity. *Am J Infect Control* 1999;27:236–9.
954. (279) Favero MS, Bond WW. Sterilization, disinfection, and antisepsis in the hospital. In: Balows A, Hausler WJ Jr, Herrmann KL, Isenberg HD, Shadomy HJ, eds. *Manual of clinical microbiology, 5th ed.* Washington, DC: American Society for Microbiology, 1991;183–200.
955. Nyström B. Bioburden of non-disposable surgical instruments and operating room textiles. In: Gaughran ERL, Morrissey RF, eds. *Sterilization of medical products, Vol II*. Montreal, Québec: Multiscience Publications Ltd., 1981;156–63.
956. Nyström B. Disinfection of surgical instruments. *J Hosp Infect* 1981;2:3636–8.
957. Rutala WA, Weber DJ. FDA labeling requirements for disinfection of endoscopes: a counterpoint. *Infect Control Hosp Epidemiol* 1995;16:231–5.
958. Parker HH IV, Johnson RD. Effectiveness of ethylene oxide for sterilization of dental handpieces. *J Dent* 1995;1:1–3.

959. Alfa MJ, DeGagne P, Olson N, Puchalski T. Comparison of ion plasma, vaporized hydrogen peroxide, and 100% ethylene oxide sterilizers to the 12/88 ethylene oxide gas sterilizer. *Infect Control Hosp Epidemiol* 1996;17:92–100.
960. Rutala WA, Gergen MF, Jones JF, Weber DJ. Levels of microbial contamination on surgical instruments. *Am J Infect Control* 1998;26:143–5.
961. (275) Stingeni L, Lapomarda V, Lisi P. Occupational hand dermatitis in hospital environments. *Contact Dermatitis* 1995;33:172–6.
962. (276) Ashdown BC, Stricof DD, May ML, Sherman SJ, Carmody RF. Hydrogen peroxide poisoning causing brain infarction: neuroimaging findings. *AM J Roentgenol* 1998;170:1653–5.
963. (277) Busch A, Werner E. [Animal tolerance to peracetic acid. 1. Experimental results following the application of peracetic acid solutions on the skin of pigs]. *Monatshefte für Veterinaermedizin* 1974;29:494–8. (German)
964. (278) U.S. Food and Drug Administration. Medical devices: adequate directions for use. 21 CFR Part 801.5, 807.87.e.
965. U.S. Food and Drug Administration (FDA) and U.S. Environmental Protection Agency (EPA). Memorandum of understanding between the Food and Drug Administration, Public Health Service, Department of Health and Human Services, and the Environmental Protection Agency: Notice regarding matters of mutual responsibility — regulation of liquid chemical germicides intended for use on medical devices, 1993. Available from FDA, Center for Devices and Radiological Health (CDRH), Office of Health and Industry Programs, Division of Small Manufacturers Assistance, Rockville MD 20850, or EPA, Registration Division, Antimicrobial Program Branch, 401 M St., SW, Washington DC 20460.
966. U.S. Food and Drug Administration (FDA). Interim measures for the registration of antimicrobial products/liquid chemical germicides with medical device use claims under the memorandum of understanding between EPA and FDA, 1994. CDRH Facts on Demand, Shelf #851, p. 14; June 30, 1994. Available from FDA, CDRH, Office of Health and Industry Programs, Division of Small Manufacturers Assistance, Rockville MD 20850.
967. (293) U.S. Department of Labor, Occupational Safety and Health Administration. Occupational Exposure to Bloodborne Pathogens: final rule (29 CFR 1910.1030). *Federal Register* 1991;56:64004–182.
968. Collins BJ. The hospital environment: how clean should a hospital be? *J Hosp Infect* 1988;11 (Suppl A): 53–6.
969. Van den Berg RWA, Claahsen HL, Niessen M, Muytjens HL, Liem K, Voss A. *Enterobacter cloacae* outbreak in the NICU related to disinfected thermometers. *J Hosp Infect* 2000;45:29–34.
970. Spaulding EH. Alcohol as a surgical disinfectant. *AORN J* 1964;2:67–71.
971. (282) Ayliffe GAJ, Collins BJ, Lowbury EJJ, Babb JR, Lilly HA. Ward floors and other surfaces as reservoirs of hospital infection. *J Hyg (Camb)* 1967;65:515–37.
972. (283) Dancer SJ. Mopping up hospital infection. *J Hosp Infect* 1999;43:85–100.
973. Gable TS. Bactericidal effectiveness of floor cleaning methods in a hospital environment. *Hospitals JAHA* 1966;40:107–11.
974. (271) U.S. Environmental Protection Agency. Federal Insecticide, Fungicide, and Rodenticide Act. 7 USC 6 § 136 et seq.; 1972. Available at: www4.law.cornell.edu/uscode/7/ch6schII.html
975. Petersen NJ, Marshall JH, Collins DE. Why wash walls in hospital isolation rooms? *Health Lab Sci* 1973;10:23–7.
976. (285) Mallison GF. Decontamination, disinfection, and sterilization. *Nurs Clin North Am* 1980;15:757–67.
977. Ayliffe GAJ, Collins BJ, Lowbury EJJ. Cleaning and disinfection of hospital floors. *Br Med J* 1966;2:442–5.
978. Vesley D, Pryor AK, Walter WG, Shaffer JG. A cooperative microbiological evaluation of floor-cleaning procedures in hospital patient rooms. *Health Lab Sci* 1970;7:256–64.
979. Daschner J, Rabbenstein G, Langmaack H. [Flächenkontamination zur Verhütung und Bekämpfung von Drakenhausinfektionen.] *Deutsche Medizinische Wochenschrift* 1980;10:325–9. (German)
980. Dharan S, Mourouga P, Copin P, Bessmer G, Tschanz B, Pittet D. Routine disinfection of patients' environmental surfaces: Myth or reality? *J Hosp Infect* 1999;42:113–7.
981. Palmer PH, Yeoman DM. A study to assess the value of disinfectants when washing ward floors. *Med J Australia* 1972;2:1237–9.
982. (284) Schmidt EA, Coleman DL, Mallison GF. Improved system for floor cleaning in health care facilities. *Appl Environ Microbiol* 1984;47:942–6.

983. (272) Mallison GF. Hospital disinfectants for housekeeping: floors and tables. *Infect Control* 1984;5:537.
984. Vesley D, Klapes NA, Benzow K, Le CT. Microbiological evaluation of wet and dry floor sanitization systems in hospital patient rooms. *Appl Environ Microbiol* 1987;53:1042–5.
985. Werry C, Lawrence JM, Sanderson PJ. Contamination of detergent cleaning solutions during hospital cleaning. *J Hosp Infect* 1988;11:44–9.
986. (280) Chou T. Environmental services. In: APIC Text of Infection Control and Epidemiology. Pfeiffer J, ed. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc.;2000;73.1–73.8.
987. (281) Rutala WA, Weber D. General information on cleaning, disinfection, and sterilization. In: Pfeiffer J, ed. APIC Text of infection control and epidemiology. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc (APIC);2000:55.1–55.6.
988. (286) Walter CW, Kundsinn RB. The floor as a reservoir of hospital infections. *Surg Gynec Obstet* 1960;111:412–22.
989. (287) Scott E, Bloomfield SF. The survival and transfer of microbial contamination via cloths, hands and utensils. *J Appl Bacteriol* 1990;68:271–8.
990. (288) Scott E, Bloomfield SF. Investigations of the effectiveness of detergent washing, drying and chemical disinfection on contamination of cleaning cloths. *J Appl Bacteriol* 1990;68:279–83.
991. Givan KF, Black BL, Williams PF. Multiplication of *Pseudomonas* species in phenolic germicidal detergent solution. *Can J Pub Health* 1971;62:72.
992. Thomas MEM, Piper E, Maurer IM. Contamination of an operating theater by gram-negative bacteria: examination of water supplies, cleaning methods, and wound infections. *J Hyg (Camb)* 1972;70:63–73.
993. Medcraft JW, Hawkins JM, Fletcher BN, Dadswell JV. Potential hazard from spray cleaning of floors in hospital wards. *J Hosp Infect* 1987;9:151–7.
994. (289) Brown DG, Schaltzle K, Gable T. The hospital vacuum cleaner: mechanism for redistributing microbial contaminants. *J Environ Health* 1980;42:192–6.
995. (290) Wysowski DK, Flynt JW, Goldfield M, et al. Epidemic hyperbilirubinemia and use of a phenolic disinfectant detergent. *Pediatrics* 1978;61:165–70.
996. (291) Doan HM, Keith L, Shennan AT. Phenol and neonatal jaundice. *Pediatrics* 1979;64:324–5.
997. (292) American Academy of Pediatrics, American College of Obstetricians and Gynecologists. Infection control. In: Guidelines for perinatal care, 4th ed. Evanston, IL: AAP, ACOG, 1997;269–74.
998. (294) Spire B, Montagnier L, Barré-Sinoussi F, Chermann JC. Inactivation of lymphadenopathy associated virus by chemical disinfectants. *Lancet* 1984;2:899–901.
999. (295) Martin LS, McDougal JS, Loskoski SL. Disinfection and inactivation of the human T lymphotropic virus type-III/lymphadenopathy-associated virus. *J Infect Dis* 1985;152:400–3.
1000. (296) Hanson PJ, Gor D, Jeffries DJ, Collins JV. Chemical inactivation of HIV on surfaces. *Br Med J* 1989;298:862–4.
1001. (297) Bloomfield SF, Smith-Burchnell CA, Dalglish AG. Evaluation of hypochlorite-releasing disinfectants against the human immunodeficiency virus (HIV). *J Hosp Infect* 1990;15:273–8.
1002. (298) Druce JD, Jardine D, Locarnini SA, Birch CJ. Susceptibility of HIV to inactivation by disinfectants and ultraviolet light. *J Hosp Infect* 1995;30:167–80.
1003. (299) Van Bueren J, Simpson RA, Salman H, Farrelly HD, Cookson BD. Inactivation of HIV-1 by chemical disinfectants: sodium hypochlorite. *Epidemiol Infect* 1995;115:567–79.
1004. (300) Prince DL, Prince HN, Thraehart O, et al. Methodological approaches to disinfection of human hepatitis viruses. *J Clin Microbiol* 1993;31:3296–304.
1005. Tabor E, Gerety RJ. A survey of formalin inactivation of hepatitis A virus, hepatitis B virus, and a non-A, non-B hepatitis agent. In: Second International Max von Pettenkofer Symposium on Viral Hepatitis. Munich, Germany: October 1982.
1006. Thraehart O, Kuwert EK, Scheiermann N, et al. Comparison of the morphological alteration and disintegration test (MADT) and the chimpanzee infectivity test for determination of hepatitis B virucidal activity of chemical disinfectants. *Zentralbl Bakteriol Mikrobiol Hyg (B)* 1982;176:472–84.
1007. (303) U.S. Department of Labor, Occupational Safety and Health Administration. EPA-registered disinfectants for HIV/HBV. [Memorandum 2/28/97]. Available at: www.osha-slc.gov/OshDoc/Interp_data/I19970228C.html
1008. U.S. Environmental Protection Agency. Lists A, B, C, D, E, and F: EPA registered disinfectants, sanitizers and sterilants. Available at: www.epa.gov/oppad001/chemregindex.htm

1009. U.S. Environmental Protection Agency. Protocols for testing the efficacy of disinfectants against hepatitis B. *Federal Register* 2000;65:51828–30.
1010. (301) CDC. Recommendations for prevention of HIV transmission in health-care settings. *MMWR* 1987;36(No.2S):1S–18S.
1011. (304) Weber DJ, Barbee SL, Sobsey MD, Rutala WA. The effect of blood on the antiviral activity of sodium hypochlorite, a phenolic, and a quaternary ammonium compound. *Infect Control Hosp Epidemiol* 1999;20:821–7.
1012. (302) Sattar SA, Springthorpe VS. Survival and disinfectant inactivation of the human immunodeficiency virus: a critical review. *Rev Infect Dis* 1991;13:430–47.
1013. (400) CDC, National Institutes of Health. Biosafety in microbiological and biomedical laboratories, 4th ed. Washington, DC: U.S. Government Printing Office, 1999.
1014. Lee R. The advantages of carpets in mental hospitals. *Ment Hosp* 1965;16:324–5.
1015. Simmons D, Reizenstein J, Grant M. Considering carpets in hospital use. *Dimensions* 1982;June:18–21.
1016. Willmott M. The effect of a vinyl floor surface and a carpeted floor surface upon walking in elderly hospital in-patients. *Age Ageing* 1986;15:119–20.
1017. Shaffer J, Key I. The microbiological effects of carpeting on the hospital environment. *Hospitals JAHA* 1966;40:126–39.
1018. Walter W, Stober A. Quantitative and qualitative microbial studies of hospital carpets. *J Environ Health* 1967;30:293–300.
1019. Anderson RL. Biological evaluation of carpeting. *Appl Microbiol* 1969;18:180–7.
1020. Lanese RR, Keller MD, Macpherson CR, Covey RC. A study of microflora on tiled and carpeted surfaces in a hospital nursery. *Am J Public Health* 1973;63:174–8.
1021. Bonde GJ. Bacterial flora of synthetic carpets in hospitals. *Health Lab Sci* 1973;10:308–18.
1022. Rylander R, Myrback K, Verner-Carlson B, Ohrstrom M. Bacteriological investigation of wall-to-wall carpeting. *Am J Public Health* 1974;64:163–8.
1023. (305) Suzuki A, Namba Y, Matsuura M, Horisawa A. Bacterial contamination of floors and other surfaces in operating rooms: a five-year survey. *J Hyg (Camb)* 1984;93:559–66.
1024. Skoutelis AT, Westenfelder GO, Beckerdite M, Phair JP. Hospital carpeting and epidemiology of *Clostridium difficile*. *Am J Infect Control* 1993;22:212–7.
1025. Anderson RL, Mackel DC, Stoler BS, Mallison GF. Carpeting in hospitals: an epidemiological evaluation. *J Clin Microbiol* 1982;15:408–15.
1026. (160) Vesper S, Dearborn DG, Yike I, et al. Evaluation of *Stachybotrys chartarum* in the house of an infant with pulmonary hemorrhage: quantitative assessment before, during, and after remediation. *J Urban Health* 2000;77:68–85.
1027. Bakker PGH, Faogali JL. The effect of carpet on the number of microbes in the hospital environment. *N Zeal Med J* 1977;85:88–92.
1028. (306) Richet H, McNeil M, Pewters W, et al. *Aspergillus flavus* in a bone marrow transplant unit (BMTU): Pseudofungemia traced to hallway carpeting [abstract]. In: Abstracts of the 89th Annual Meeting of the American Society for Microbiology, 1989.
1029. CDC. Respiratory illness associated with carpet cleaning at a hospital clinic — Virginia. *MMWR* 1983;32:378,383–4.
1030. Maley MP. Bacterial threats to new hospitals. *Lancet* 1997;350:223–4.
1031. U.S. Environmental Protection Agency. Office of Toxic Substances. Efficacy data requirements. Supplemental recommendations DIS/TSS-2;1/25/79.
1032. (307) U.S. Department of Labor, Occupational Safety and Health Administration. OSHA Standards Interpretation and Compliance Letters;6/10/94: Decontamination of a plush carpet surface after a spill. Available at: www.osha-slc.gov/OshDoc/Interp_data/I19940610.html
1033. Richards K. New York City: special suites — Memorial Sloan Kettering. *Interiors* 1998;157:56–9.
1034. Noskin BA, Bednarz P, Suriano T, Reiner S, Peterson LR. Persistent contamination of fabric-covered furniture by vancomycin-resistant enterococci: implications for upholstery selection in hospitals. *Am J Infect Control* 2000;28:311–3.
1035. Sanderson PJ, Alshafi KM. Environmental contamination by organisms causing urinary tract infection. *J Hosp Infect* 1995;29: 301–3.
1036. Babe KS Jr, Arlian LG, Confer PD, Kim R. House dust mite (*Dermatophagoides farinae* and *Dermatophagoides pteronyssinus*) prevalence in the rooms and hallways of a tertiary care hospital. *J Allergy Clin Immunol* 1995;95:801–5.

1037. Custovic A, Fletcher A, Pickering CAC, et al. Domestic allergens in public places III: house dust mite, cat, dog, and cockroach allergens in British hospitals. *Clin Exper Allergy* 1998;28:53–9.
1038. Ansorg R, Thomssen R, Stubbe P. *Erwinia* species causing fatal septicemia in a newborn. *Med Microbiol Immunol (Berl)* 1974;159:161–70.
1039. Trust TJ, Bartlett KH. Isolation of *Pseudomonas aeruginosa* and other bacterial species from ornamental aquarium plants. *Appl Environ Microbiol* 1976;31:992–4.
1040. (310) Bartzokas CA, Holley MP, Sharp CA. Bacteria in flower vase water: incidence and significance in general ward practice. *Br J Surg* 1975;62:295–7.
1041. Watson AG, Koons CE. *Pseudomonas* on the chrysanthemums. *Lancet* 1973;2:91.
1042. (311) Siegman-Igra Y, Shalem A, Berger SA, Livio S, Michaeli D. Should potted plants be removed from hospital wards? *J Hosp Infect* 1986;7:82–5.
1043. Rosenzweig AL. Contaminated flower vases. *Lancet* 1973;2:568–9.
1044. Johansen KS, Laursen H, Wilhjelmsen BJ. Flower vases as reservoirs of pathogens. *Lancet* 1974;1:359.
1045. Rogues AM, Quesnel C, Revel P, Saric J, Gachie JP. Potted plants as a potential reservoir of *Fusarium* species. *J Hosp Infect* 1997;35:163–4.
1046. (312) Lass-Flörl C, Rath P, Niederwieser D, et al. *Aspergillus terreus* infections in haematological malignancies: molecular epidemiology suggests association with in-hospital plants. *J Hosp Infect* 2000;46:31–5.
1047. Levine OS, Levine MM. Houseflies (*Musca domestica*) as mechanical vectors of shigellosis. *Rev Infect Dis* 1991;13:688–96.
1048. Šrámová H, Daniel M, Absolonová V, Dědičová D, Jedličková Z, Lhotová H, Petráš P, Subertová V. Epidemiological role of arthropods detectable in health facilities. *J Hosp Infect* 1992;20:281–92.
1049. Tan SW, Yap KL, Lee HL. Mechanical transport of rotavirus by the legs and wings of *Musca domestica* (Diptera: *Muscidae*). *J Med Entomol* 1997;34:527–31.
1050. (313) Burgess NR. Hospital design and cockroach control. *Trans R Soc Trop Med Hyg* 1984;78:293–4.
1051. Ash N, Greenberg B. Vector potential for the German cockroach (Diptera: *Blattellidae*) in dissemination of *Salmonella enteritidis* serotype typhimurium. *J Med Entomol* 1980;17:417–23.
1052. Fotedar R, Banerjee U. Nosocomial fungal infections — study of the possible role of cockroaches (*Blattella germanica*) as vectors. *Acta Trop* 1992;50:339–43.
1053. Rosef O, Kapperud G. House flies (*Musca domestica*) as possible vectors of *Campylobacter fetus* subsp. jejuni. *Appl Environ Microbiol* 1983;45:381–3.
1054. Forsey T, Darougar S. Transmission of chlamydiae by the housefly. *Br J Ophthalmol* 1981;65:147–50.
1055. Grübel P, Hoffman JS, Chong FK, Burstein NA, Mepani C, Cave DR. Vector potential of houseflies (*Musca domestica*) for *Helicobacter pylori*. *J Clin Microbiol* 1997;35:1300–3.
1056. Oothuman P, Jeffery J, Aziz, AHA, Bakar EA, Jegathesan M. Bacterial pathogens isolated from cockroaches trapped from pediatric wards in peninsular Malaysia. *Trans R Soc Trop Med Hyg* 1989;83:133–5.
1057. Beatson SH. Pharaoh's ants as pathogen vectors in hospitals. *Lancet* 1972;1:425–7.
1058. LeGuyader A, Rivault C, Chaperon J. Microbial organisms carried by brown-banded cockroaches in relation to their spatial distribution in a hospital. *Epidemiol Infect* 1989;102:485–92.
1059. Fotedar R, Banerjee U, Shrinivas, Verma A. Cockroaches (*Blattella germanica*) as carriers of microorganisms of medical importance in hospitals. *Epidemiol Infect* 1991;107:181–7.
1060. Fotedar R, Banerjee U, Singh S, Shrinivas, Verma AK. The housefly (*Musca domestica*) as a carrier of pathogenic microorganisms in a hospital environment. *J Hosp Infect* 1992;20:209–15.
1061. Fotedar R, Shrinivas, Banerjee U, Samantray JC, Nayar E, Verma A. Nosocomial infections: cockroaches as possible vectors of drug-resistant *Klebsiella*. *J Hosp Infect* 1991;18:155–9.
1062. Devi SJN, Murray CJ. Cockroaches (*Blatta* and *Periplaneta* species) as reservoirs of drug-resistant salmonellas. *Epidemiol Infect* 1991;107:357–64.
1063. Cotton MF, Wasserman E, Pieper CH, et al. Invasive disease due to extended spectrum beta-lactamase-producing *Klebsiella pneumoniae* in a neonatal unit: the possible role of cockroaches. *J Hosp Infect* 2000;44:13–7.
1064. Baker LF. Pests in hospitals. *J Hosp Infect* 1981;2:5–9.
1065. (316) Allen BW. Excretion of viable tubercle bacilli by *Blatta orientalis* (the oriental cockroach) following ingestion of heat-fixed sputum smears: a laboratory investigation. *Trans R Soc Trop Med Hyg* 1987;81:98–9.

1066. (317) Laszlo A. Technical guide: sputum examination for tuberculosis by direct microscopy in low income countries, 5th ed. Paris, France: International Union Against Tuberculosis and Lung Disease, 2000. Available at: www.iatld.org/html/body_guides.htm
1067. Cohen D, Green M, Block C, et al. Reduction of transmission of shigellosis by control of houseflies (*Musca domestica*). *Lancet* 1991;337:993–7.
1068. Daniel M, Šrámová H, Zálabská E. *Lucilia sericata* (Diptera: Calliphoridae) causing hospital-acquired myiasis of a traumatic wound. *J Hosp Infect* 1994;28:149–52.
1069. Jacobson JA, Kolts RL, Conti M, Burke JP. Hospital-acquired myiasis. *Infect Control* 1980;1:319–20.
1070. Mielke U. Nosocomial myiasis. *J Hosp Infect* 1997;37:1–5.
1071. Sherman RA. Wound myiasis in urban and suburban United States. *Arch Intern Med* 2000;160:2004–14.
1072. (314) Lukin LG. Human cutaneous myiasis in Brisbane: a prospective study. *Med J Aust* 1989;150:237–40.
1073. Watkins M, Wyatt T. A ticklish problem: pest infestation in hospitals. *Prof Nurse* 1989;4:389–92.
1074. Schoninger S. Pest control and extermination in health care facilities. *Prof Sanit Manage* 1978;9:24–7.
1075. (315) Bruesch J. Institutional pest management: current trends. *Exec Housekeep Today* 1994;15:6–12.
1076. Tenover FC. VRSA, VISA, GISA: the dilemma behind the name game. *Clin Microbiol Newsletter* 2000;22:49–53.
1077. CDC. National Nosocomial Infections Surveillance System (NNIS): Semiannual report. December 1999. Available at: www.cdc.gov/ncidod/hip/SURVEILL/NNIS.HTM
1078. Hartstein AI, Mulligan ME. Methicillin-resistant *Staphylococcus aureus*. In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;347–64.
1079. Walsh TJ, Vlahov D, Hansen SL, et al. Prospective microbiologic surveillance in control of nosocomial methicillin-resistant *Staphylococcus aureus*. *Infect Control* 1987;8:7–14.
1080. Walsh TJ, Auger F, Tatem BA, Hansen SL, Standford HJ. Novobiocin and rifampin in combination against methicillin-resistant *Staphylococcus aureus*: an in vitro comparison with vancomycin plus rifampin. *J Antimicrob Chemother* 1986;17:75–82.
1081. McNeil MM, Solomon SL. The epidemiology of MRSA. *Antimicrobiol Newsl* 1985;2:49–56.
1082. Oie S, Kamiya A. Survival of methicillin-resistant *Staphylococcus aureus* (MRSA) on naturally contaminated dry mops. *J Hosp Infect* 1996;34:145–9.
1083. Arnow PM, Allyn PA, Nichols EM, Hill DL, Pezzlo M, Bartlett RH. Control of methicillin-resistant *Staphylococcus aureus* in a burn unit: role of nurse staffing. *J Trauma* 1982;22:954–9.
1084. (329) Karanfil LV, Murphy M, Josephson A, et al. A cluster of vancomycin-resistant *Enterococcus faecium* in an intensive care unit. *Infect Control Hosp Epidemiol* 1992;13:195–200.
1085. Handwerker S, Raucher B, Altarac D, et al. Nosocomial outbreak due to *Enterococcus faecium* highly resistant to vancomycin, penicillin, and gentamicin. *Clin Infect Dis* 1993;16:750–5.
1086. Boyle JF, Soumakis SA, Rendo A, et al. Epidemiologic analysis and genotypic characterization of a nosocomial outbreak of vancomycin-resistant enterococci. *J Clin Microbiol* 1993;31:1280–5.
1087. (330) Boyce JM, Opal SM, Chow JW, et al. Outbreak of multidrug-resistant *Enterococcus faecium* with transferable vanB class vancomycin resistance. *J Clin Microbiol* 1994;32:1148–53.
1088. (331) Rhinehart E, Smith NE, Wennerstein C, et al. Rapid dissemination of beta-lactamase-producing, aminoglycoside-resistant *Enterococcus faecalis* among patients and staff on an infant-toddler surgical ward. *N Engl J Med* 1990;323:1814–8.
1089. Crossley K, Landesman B, Zaske D. An outbreak of infections caused by strains of *Staphylococcus aureus* resistant to methicillin and aminoglycosides. II. epidemiologic studies. *J Infect Dis* 1979;139:280–7.
1090. Peacock JE Jr, Marsik FJ, Wenzel RP. Methicillin-resistant *Staphylococcus aureus*: introduction and spread within a hospital. *Ann Intern Med* 1980;93:526–32.
1091. Walsh TJ, Hansen SL, Tatem BA, Auger F, Standford HJ. Activity of novobiocin against methicillin-resistant *Staphylococcus aureus*. *J Antimicrob Chemother* 1985;15:435–40.
1092. (332) Livornese LL Jr, Sias S, Samel C, et al. Hospital-acquired infection with vancomycin-resistant *Enterococcus faecium* transmitted by electronic thermometers. *Ann Intern Med* 1992;117:112–6.
1093. Gould FK, Freeman R. Nosocomial infection with microsphere beds. *Lancet* 1993;342:241–2.

1094. Morris JG, Shay DK, Hebden JN, et al. *Enterococci* resistant to multiple antimicrobial agents, including vancomycin: establishment of endemicity in a university medical center. *Ann Intern Med* 1995;123:250–9.
1095. Edmond MB, Ober JS, Weinbaum DL, et al. Vancomycin-resistant *Enterococcus faecium* bacteremia: risk factors for infection. *Clin Infect Dis* 1995;20:1126–33.
1096. (333) Zervos MJ, Kauffman CA, Therasse PM, Bergman AG, Mikesell TS, Schaberg DR. Nosocomial infection by gentamicin-resistant *Streptococcus faecalis*: an epidemiologic study. *Ann Intern Med* 1987;106:687–91.
1097. Zervos MJ, Dembinski S, Mikesell T, Schaberg DR. High-level resistance to gentamicin in *Streptococcus faecalis*: risk factors and evidence for exogenous acquisition of infection. *J Infect Dis* 1986;153: 1075–83.
1098. Bonilla HF, Zervos MA, Lyons MJ, et al. Colonization with vancomycin-resistant *Enterococcus faecium*: comparison of a long-term-care unit with an acute-care hospital. *Infect Control Hosp Epidemiol* 1997;18:333–9.
1099. Bonilla HF, Zervos MJ, Kauffman CA. Long-term survival of vancomycin-resistant *Enterococcus faecium* on a contaminated surface. *Infect Control Hosp Epidemiol* 1996;17:770–1.
1100. Boyce JM, Bermel LA, Zervos MJ, et al. Controlling vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 1995;16:634–7.
1101. Boyce JM, Potter-Bynoe G, Chenevert C, King T. Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. *Infect Control Hosp Epidemiol* 1997;18:622–7.
1102. (328) Layton MC, Perez M, Heald P, Patterson JE. An outbreak of mupirocin-resistant *Staphylococcus aureus* on a dermatology ward associated with an environmental reservoir. *Infect Control Hosp Epidemiol* 1993;14:369–75.
1103. Collins SM, Hacek DM, Degen LA, Wright MP, Noskin GA, Peterson LR. Contamination of the clinical microbiology laboratory with vancomycin-resistant enterococci and multidrug-resistant *Enterobacteriaceae*: implications for hospital and laboratory workers. *J Clin Microbiol* 2001;39:3772–4.
1104. Bonten MJM, Hayden MK, Nathan C, et al. Epidemiology of colonisation of patients and environment with vancomycin-resistant enterococci. *Lancet* 1996;348:1615–9.
1105. Wendt C, Wiesenthal B, Dietz E, Rüden H. Survival of vancomycin-resistant and vancomycin-susceptible enterococci on dry surfaces. *J Clin Microbiol* 1998;36:3734–46.
1106. (323) Bradley CR, Fraiese AP. Heat and chemical resistance of enterococci. *J Hosp Infect* 1996;34:191–6.
1107. (324) Anderson RL, Carr JH, Bond WW, Favero MS. Susceptibility of vancomycin-resistant enterococci to environmental disinfectants. *Infect Control Hosp Epidemiol* 1997;18:195–9.
1108. (325) Saurina G, Landman D, Quale JM. Activity of disinfectants against vancomycin-resistant *Enterococcus faecium*. *Infect Control Hosp Epidemiol* 1997;18:345–7.
1109. (326) Rutala WA, Stiegel MM, Sarubbi FA, Weber DJ. Susceptibility of antibiotic-susceptible and antibiotic-resistant hospital bacteria to disinfectants. *Infect Control Hosp Epidemiol* 1997;18:417–21.
1110. (327) Schulster LM, Anderson RL. Susceptibility of glycopeptide-intermediate resistant *Staphylococcus aureus* (GISA) to surface disinfectants, hand washing chemicals, and a skin antiseptic [abstract Y-3]. In: Abstracts of the 98th General Meeting, American Society for Microbiology. 1998.
1111. Armstrong-Evans M, Litt M, McArthur MA, et al. Control of transmission of vancomycin-resistant *Enterococcus faecium* in a long-term-care facility. *Infect Control Hosp Epidemiol* 1999;20:312–17.
1112. Global Consensus Conference: Final Recommendations. Global Consensus Conference on Infection Control Issues Related to Antimicrobial Resistance. *Am J Infect Control* 1999;27:503–13.
1113. Freeman R, Kearns AM, Lightfoot NF. Heat resistance of nosocomial enterococci. *Lancet* 1994;345:64–5.
1114. CDC. *Staphylococcus aureus* resistant to vancomycin — United States. *MMWR* 2002;51:565–7.
1115. CDC. Vancomycin resistant *Staphylococcus aureus* — Pennsylvania, 2002. *MMWR* 2002;51:902.
1116. (320) Weber DJ, Rutala WA. Role of environmental contamination in the transmission of vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 1997;18:306–9.
1117. (321) Lai KK, Kelley AL, Melvin ZS, Belliveau PP, Fontecchio SA. Failure to eradicate vancomycin-resistant enterococci in a university hospital and the cost of barrier precautions. *Infect Control Hosp Epidemiol* 1998;19:647–2.

1118. (322) Byers KE, Durbin LJ, Simonton BM, Anglim AM, Adal KA, Farr BM. Disinfection of hospital rooms contaminated with vancomycin-resistant *Enterococcus faecium*. *Infect Control Hosp Epidemiol* 1998;19:261–4.
1119. Siegel DL, Edelstein PH, Nachamkin I. Inappropriate testing for diarrheal diseases in the hospital. *JAMA* 1990;263:979–82.
1120. Yannelli B, Gurevich I, Schoch PE, Cunha BA. Yield of stool cultures, ova and parasite tests, and *Clostridium difficile* determination in nosocomial diarrhea. *Am J Infect Control* 1988;16:246–9.
1121. Gerding DN, Olson MM, Peterson LR, et al. *Clostridium difficile*-associated diarrhea and colitis in adults: a prospective case-controlled epidemiologic study. *Arch Intern Med* 1986;146:95–100.
1122. Svenungsson B, Burman LG, Jalakas-Pörnnull K, Lagergren Å, Struwe J, Åkerlund T. Epidemiology and molecular characterization of *Clostridium difficile* strains from patients with diarrhea: low disease incidence and evidence of limited cross-infection in a Swedish teaching hospital. *J Clin Microbiol* 2003;41:4031–7.
1123. Barlett JG. Antibiotic-associated colitis. *Dis Mon* 1984;30:1–55.
1124. Pierce PF Jr, Wilson R, Silva J Jr, et al. Antibiotic-associated pseudomembranous colitis: an epidemiologic investigation of a cluster of cases. *J Infect Dis* 1982;145:269–74.
1125. Aronsson B, Möllby, Nord C-E. Antimicrobial agents and *Clostridium difficile* in acute enteric disease: epidemiologic data from Sweden, 1980–1982. *J Infect Dis* 1985;151:476–81.
1126. Thibault A, Miller MA, Gaese C. Risk factors for the development of *Clostridium difficile*-associated diarrhea during a hospital outbreak. *Infect Control Hosp Epidemiol* 1991;12:345–8.
1127. McFarland LV, Surawicz CM, Stamm WE. Risk factors for *Clostridium difficile* carriage and *Clostridium difficile*-associated diarrhea in a cohort of hospitalized patients. *J Infect Dis* 1990;162:678–84.
1128. Zadik PM, Moore AP. Antimicrobial associations of an outbreak of diarrhoea due to *Clostridium difficile*. *J Hosp Infect* 1998;39:189–93.
1129. Johnson S, Homann SR, Bettin KM, et al. Treatment of asymptomatic *Clostridium difficile* carriers (fecal excretors) with vancomycin or metronidazole: a randomized, placebo controlled trial. *Ann Intern Med* 1992;117:297–302.
1130. (319) Gerding DN, Johnson S, Peterson LR, Mulligan ME, Silva J Jr. *Clostridium difficile*-associated diarrhea and colitis. *Infect Control Hosp Epidemiol* 1995;16:459–77.
1131. Titov L, Lebedkova N, Shabanov A, Tang YJ, Cohen SH, Silva J Jr. Isolation and molecular characterization of *Clostridium difficile* strains from patients and the hospital environment in Belarus. *J Clin Microbiol* 2000;38:1200–2.
1132. Mulligan ME, Rolfe RD, Finegold SM, George WL. Contamination of a hospital environment by *Clostridium difficile*. *Curr Microbiol* 1979;3:173–5.
1133. Fekety R, Kim KH, Brown D, Batts DH, Cudmore M, Silva J Jr. Epidemiology of antibiotic-associated colitis: isolation of *Clostridium difficile* from the hospital environment. *Am J Med* 1981;70:906–8.
1134. Malamou-Ladas H, Farrell SO, Nash JO, Tabaqchali S. Isolation of *Clostridium difficile* from patients and the environment of hospital wards. *J Clin Pathol* 1983;6:88–92.
1135. Kaatz GW, Gitlin SD, Schaberg DR, et al. Acquisition of *Clostridium difficile* from the hospital environment. *Am J Epidemiol* 1988;127:1289–94.
1136. Cohen SH, Tang YJ, Muenzer J, Gumerlock PH, Silva J Jr. Isolation of various genotypes of *Clostridium difficile* from patients and the environment in an oncology ward. *J Infect Dis* 1997;889–93.
1137. Savage AM. Nosocomial spread of *Clostridium difficile*. *Infect Control* 1983;4:31–3.
1138. Brooks SE, Veal RO, Kramer M, Dore L, Schupf N, Adachi M. Reduction in the incidence of *Clostridium difficile*-associated diarrhea in an acute care hospital and a skilled nursing facility following replacement of electronic thermometers with single-use disposables. *Infect Control Hosp Epidemiol* 1992;13:98–103.
1139. Johnson S, Gerding DN, Olson MM, et al. Prospective, controlled study of vinyl glove use to interrupt *Clostridium difficile* nosocomial transmission. *Am J Med* 1990;88:137–40.
1140. McFarland LV, Mulligan ME, Kwok RYY, Stamm WE. Nosocomial acquisition of *Clostridium difficile* infection. *New Engl J Med* 1989;320:204–10.
1141. Mayfield JL, Leet T, Miller J, Mundy LM. Environmental control to reduce transmission of *Clostridium difficile*. *Clin Inf Dis* 2000;31:995–1000.

1142. Wilcox MH, Fawley WN, Wigglesworth N, Parnell P, Verity P, Freeman J. Comparison of the effect of detergent versus hypochlorite cleaning on environmental contamination and incidence of *Clostridium difficile* infection. *J Hosp Infect* 2003;54:109–14.
1143. (334) Worsley MA. Infection control and prevention of *Clostridium difficile* infection. *J Antimicrobial Chemother* 1998;41 (Suppl. C):59–66.
1144. von Rheinbaben F, Schünemann S, Groß T, Wolff MH. Transmission of viruses via contact in a household setting: experiments using bacteriophage ϕ X174 as a model virus. *J Hosp Infect* 2000;46:61–6.
1145. Hall CB, Douglas G Jr, Gelman JM. Possible transmission by fomites of respiratory syncytial virus. *J Infect Dis* 1980;141:98–102.
1146. Brady MT, Evans J, Cuartas J. Survival and disinfection of parainfluenza viruses on environmental surfaces. *Am J Infect Control* 1990;18:18–23.
1147. Hendley JO, Wenzel RP, Gwaltney JM Jr. Transmission of rhinovirus colds by self-inoculation. *N Engl J Med* 1973;288:1361–4.
1148. Butz AM, Fosarelli P, Dick J, Cusack T, Yolken R. Prevalence of rotavirus on high-risk fomites in day-care facilities. *Pediatrics* 1993;92:202–5.
1149. Wilde J, Van R, Pickering LK, Eiden J, Yolken R. Detection of rotaviruses in the day care environment — detection by reverse transcriptase polymerase chain reaction. *J Infect Dis* 1992;166:507–11.
1150. Chapin M, Yatabe J, Cherry JD. An outbreak of rotavirus gastroenteritis on a pediatric unit. *Am J Infect Control* 1983;11:88–91.
1151. Appleton H, Higgins PG. Viruses and gastroenteritis in infants. *Lancet* 1975;i:1297.
1152. Abad FX, Villena C, Guix S, Caballero S, Pintó RM, Bosch A. Potential role of fomites in the vehicular transmission of human astroviruses. *Appl Environ Microbiol* 2001;67:3904–7.
1153. Chadwick PR, Beards G, Brown D, et al. Management of hospital outbreaks of gastro-enteritis due to small round structured viruses. Report of the Public Health Laboratory Service, Viral Gastroenteritis Working Group. *J Hosp Infect* 2000;45:1–10.
1154. Spender QW, Lewis D, Price EH. Norwalk-like viruses: study of an outbreak. *Arch Dis Child* 1986;61:142–7.
1155. Storr J, Rice S, Phillips AD, Price E, Walker Smith JA. Clinical associations of Norwalk-like virus in the stools of children. *J Pediatr Gastroenterol Nutr* 1986;5:576–80.
1156. Russo PL, Spelman DW, Harrington GA, et al. Hospital outbreak of Norwalk-like virus. *Infect Control Hosp Epidemiol* 1997;17:1374–8.
1157. Springthorpe VS, Grenier JL, Lloyd-Evans N, Sattar SA. Chemical disinfection of human rotaviruses: efficacy of commercially-available products in suspension tests. *J Hyg (Camb)* 1986;97:139–61.
1158. (335) Lloyd-Evans N, Springthorpe VS, Sattar SA. Chemical disinfection of human rotavirus-contaminated inanimate surfaces. *J Hyg (Camb)* 1986;97:163–73.
1159. CDC. Interim recommendations for cleaning and disinfection of the SARS patient environment. At: www.cdc.gov/ncidod/sars/cleaningpatientenviro.htm
1160. Brown P, Gajdusek DC. The human spongiform encephalopathies: Kuru, Creutzfeldt-Jakob disease, and Gerstmann-Sträussler-Scheinker syndrome. *Curr Top Microbiol Immunol* 1991;172:1–20.
1161. Will RG. Epidemiology of Creutzfeldt-Jakob disease. *Br Med Bull* 1993;49:960–70.
1162. Holman RC, Khan AS, Belay ED, Schonberger LB. Creutzfeldt-Jakob disease in the United States, 1979–1994: using national mortality data to assess the possible occurrence of variant cases. *Emerg Infect Dis* 1996;2:333–7.
1163. Will RG, Ironside JW, Zeidler M, et al. A new variant of Creutzfeldt-Jakob disease in the U.K. *Lancet* 1996;347:921–5.
1164. Lasmézas CI, Deslys JP, Demaimay R, et al. BSE transmission to macaques. *Nature* 1996;381:743–4.
1165. Collinge J, Sidle KCL, Heads J, Ironside J, Hill AF. Molecular analysis of prion strain variation and the aetiology of “new variant” CJD. *Nature* 1996;383:685–90.
1166. Bruce ME, Will RG, Ironside JW, et al. Transmission to mice indicates that “new variant” CJD is caused by the BSE agent. *Nature* 1997;389:498–501.
1167. Prusiner SB. Biology and genetics of prion diseases. *Ann Rev Microbiol* 1994;48:655–86.
1168. Prusiner SB. Human prion diseases. In: Zuckerman AJ, Banatvala JE, Pattison JR, eds. Principles and practice of clinical virology, 3rd ed. Chichester, UK: John Wiley & Sons, 1995;703–29.
1169. Prusiner SB. Prions. *Proc Natl Acad Sci USA* 1998;95:13363–83.

1170. (337) Kimberlin RH, Walker CA, Millson GC, et al. Disinfection studies with two strains of mouse-passaged scrapie agent. Guidelines for Creutzfeldt-Jakob and related agents. *J Neurol Sci* 1983;59:349–55.
1171. Sklaviadis TK, Manuelidis L, Manuelidis EE. Physical properties of the Creutzfeldt-Jakob disease agent. *J Virol* 1989;63:1212–22.
1172. Brown P, Gajdusek DC, Gibbs CJ Jr, Asher DM. Potential epidemic of Creutzfeldt-Jakob disease from human growth hormone therapy. *N Engl J Med* 1985;12:728–33.
1173. Brown P, Preece MA, Will RG. “Friendly fire” in medicine: hormones, homografts and Creutzfeldt-Jakob disease. *Lancet* 1992;340:24–7. Brown P, Preece MA, Will RG. “Friendly fire” in medicine: hormones, homografts and Creutzfeldt-Jakob disease. *Lancet* 1992;340:24–7.
1174. Frasier D, Foley TP Jr. Creutzfeldt-Jakob disease in recipients of pituitary hormones. *J Clin Endocrinol Metabol* 1994;78:1277–9.
1175. Centers for Disease Control. Epidemiologic notes and reports: rapidly progressive dementia in a patient who received a cadaveric dura mater graft. *MMWR* 1987;36:49–50, 55.
1176. Centers for Disease Control. Epidemiologic notes and reports update: Creutzfeldt-Jakob disease in a patient receiving cadaveric dura mater graft. *MMWR* 1987;36:324–5.
1177. Centers for Disease Control. Epidemiologic notes and reports update: Creutzfeldt-Jakob disease in a second patient who received a cadaveric dura mater graft. *MMWR* 1989;38:37–8, 43.
1178. CDC. Creutzfeldt-Jakob disease in patients who received a cadaveric dura mater graft — Spain, 1985–1992. *MMWR* 1993;42:560–3.
1179. Martinez-Lage JF, Poza M, Sola J, et al. Accidental transmission of Creutzfeldt-Jakob disease by dural cadaveric grafts. *J Neurol Neurosurg Psychiatry* 1994;57:1091–4.
1180. CDC. Creutzfeldt-Jakob disease associated with cadaveric dura mater grafts — Japan, January 1979–May 1997. *MMWR* 1997;46:1066–9.
1181. Lang CLG, Heckmann JG, Neundörfer B. Creutzfeldt-Jakob disease via dural and corneal transplants. *J Neurol Sci* 1998;160:128–39.
1182. Nevin S, McMenemey WH, Behrman S, Jones DP. Subacute spongiform encephalopathy — a subacute form of encephalopathy attributable to vascular dysfunction (spongiform cerebral atrophy). *Brain* 1960;83:519–69.
1183. Bernoulli C, Siegfried J, Baumgartner G, et al. Danger of accidental person-to-person transmission of Creutzfeldt-Jakob disease by surgery. *Lancet* 1977;1:478–9.
1184. Will RG, Matthews WB. Evidence for case-to-case transmission of Creutzfeldt-Jakob disease. *J Neurol Neurosurg Psychiatry* 1982;45:235–8.
1185. El Hachimi KH, Chaunu M-P, Cervenakova L, Brown P, Foncin J-F. Putative neurosurgical transmission of Creutzfeldt-Jakob disease with analysis of donor and recipient: agent strains. *Comp Rendus Acad Sci Iii: Science de la vie* 1997;320:319–28.
1186. Brown P, Gibbs CJ, Amyx HL, et al. Chemical disinfection of Creutzfeldt-Jakob disease virus. *N Engl J Med* 1982;306:1279–82.
1187. Brown P, Rohwer RG, Gajdusek DC. Newer data on the inactivation of scrapie virus or Creutzfeldt-Jakob disease virus in brain tissue. *J Infect Dis* 1986;153:1145–8.
1188. (338) Rosenberg RN, White CL, Brown P, Gajdusek DC, Volpe JJ, Dyck PJ. Precautions in handling tissues, fluids, and other contaminated materials from patients with documented or suspected Creutzfeldt-Jakob disease. *Ann Neurol* 1986;19:75–7.
1189. Taylor DM. Resistance of the ME7 scrapie agent to peracetic acid. *Vet Microbiol* 1991;27:19–24.
1190. Taguchi F, Tamai Y, Uchida K, et al. Proposal for a procedure for complete inactivation of the Creutzfeldt-Jakob disease agent. *Arch Virol* 1991;119:297–301.
1191. (339) Taylor D. Inactivation of the unconventional agents of scrapie, bovine spongiform encephalopathy, and Creutzfeldt-Jakob disease. *J Hosp Infect* 1991;18 (Suppl A):141–6.
1192. Favero MS. Current issues in hospital hygiene and sterilization technology. *J Infect Control (Asia Pacific Edition)* 1998;1:8–10.
1193. Ricketts MN, Cashman NR, Stratton EE, El Saadany S. Is Creutzfeldt-Jakob disease transmitted in blood? *Emerg Infect Dis* 1997;3:155–63.
1194. Will RG, Kimberlin RH. Creutzfeldt-Jakob disease and the risk from blood or blood products. *Vox Sang* 1998;75:178–80.
1195. Evatt B, Austin H, Barnhart E, et al. Surveillance for Creutzfeldt-Jakob disease among persons with hemophilia. *Transfusion* 1998;38:817–20.

1196. Patry D, Curry B, Easton D, Mastrianni JA, Hogan DB. Creutzfeldt-Jakob disease (CJD) after blood product transfusion from a donor with CJD. *Neurology* 1998;50:1872-3.
1197. (340) Budka H, Aguzzi A, Brown P, et al. Tissue handling in suspected Creutzfeldt-Jakob disease (CJD) and other human spongiform encephalopathies (prion diseases). *Brain Pathol* 1995;5:319-22.
1198. (341) Ironside JW, Bell JE. The "high-risk" neuropathological autopsy in AIDS and Creutzfeldt-Jakob disease: principles and practice. *Neuropathol Appl Neurobiol* 1996;22:388-93.
1199. (336) Rutala WA, Weber DJ. Creutzfeldt-Jakob disease: recommendations for disinfection and sterilization. *Clin Infect Dis* 2001;32:1348-56.
1200. Joint Commission for the Accreditation of Healthcare Organizations. Exposure to Creutzfeldt-Jakob Disease. Sentinel Alert, Issue 20; June 2001. Available at: www.jcaho.org/edu_pub/sealert/sea20.html
1201. (342) World Health Organization. WHO Infection control guideline for transmissible spongiform encephalopathies: report of a WHO consultation. Geneva, Switzerland: WHO, 1999. Available at: www.who.int/emc-documents/tse/whocdscsraph2003c.html
1202. Litsky BY. Results of bacteriological surveys highlight problem areas in hospitals. *Hospital Management* 1966;101:82-8.
1203. Eickhoff TC. Microbiologic sampling. *Hospitals* 1970;44:86-7.
1204. American Hospital Association Committee on Infections Within the Hospitals. Statement on microbiologic sampling in the hospital 1974;48:125-6.
1205. Rafferty KM, Pancoast SJ. Brief report: bacteriological sampling of telephones and other hospital staff-hand contact objects. *Infect Control* 1984;5:533-5.
1206. Haley RW, Shachtman RS. The emergence of infection control programs in U.S. hospitals: an assessment, 1976. *Am J Epidemiol* 1980;111:574-91.
1207. Mallison GF, Haley RW. Microbiologic sampling of the inanimate environment in U.S. hospitals, 1976-1977. *Am J Med* 1981;70:941-6.
1208. Gröschel DHM. Air sampling in hospitals. *Ann NY Acad Sci* 1980;353:230-40.
1209. (208, Appendix; 6) Barbaree JM, Gorman GW, Martin WT, Fields BS, Morrill WE. Protocol for sampling environmental sites for legionellae. *Appl Environ Microbiol* 1987;53:1454-8.
1210. Eickhoff TC. Microbiologic sampling of the hospital environment. *Health Lab Sci* 1974;11:73-5.
1211. Isenberg HD. Significance of environmental microbiology in nosocomial infections and the care of hospitalized patients. In: Lorian V, ed. Significance of medical microbiology in the care of patients. Baltimore, MD: Williams & Wilkins, 1977;220-34.
1212. McGowan JE Jr, Weinstein RA. The role of the laboratory in control of nosocomial infection. In: Bennett JV, Brachman PS, eds. *Hospital infections*, 4th ed. Philadelphia, PA: Lippincott Raven, 1998;143-64.
1213. Turner AG, Wilkins JR, Craddock JG. Bacterial aerosolization from an ultrasonic cleaner. *J Clin Microbiol* 1975;1:289-93.
1214. (343) Bond WW, Schulster LM. Microbiological culturing of environmental and medical-device surfaces. In: Isenberg HD, Miller JM, Bell M, eds. *Clinical microbiology procedures handbook*, section 11. Washington, DC: American Society for Microbiology Press, 2004 (in press).
1215. Cole EC, Cook CE. Characterization of infectious aerosols in health care facilities: an aid to effective engineering controls and preventive strategies. *Am J Infect Control* 1998;26:452-64.
1216. Nevalainen A, Willeke K, Liebhaber F, Pastuszka J, Burge H, Henningson E. Bioaerosol sampling. In: Willeke K, Baron PA, eds. *Aerosol management*. New York, NY: Van Nostrand Reinhold, 1993;471-92.
1217. Cox CS. *The aerobiological pathway of microorganisms*. Chichester UK: John Wiley & Sons, 1987.
1218. (349) Wolf HW, Skaliy P, Hall LB, et al. Sampling microbiological aerosols. Public Health Service publication No. 686. Government Printing Office, Washington, DC: 1964.
1219. Zeterberg JM. A review of respiratory virology and the spread of virulent and possible antigenic viruses via air conditioning systems. *Ann Allergy* 1973;31:228-34.
1220. Randall CW, Ledbetter JO. Bacterial air pollution from activated sludge units. *Am Ind Hyg Assoc J* 1966;Nov/Dec:506-19.
1221. Salem H, Gardner DE. Health aspects of bioaerosols. In: Lighthart B, Mohr AJ, eds. *Atmospheric microbial aerosols, theory and applications*. New York, NY: Chapman and Hall, 1985;304-30.
1222. Sattar SA, Ijaz MK. Spread of viral infections by aerosols. *Crit Rev Environ Control* 1987;17:89-131.
1223. (345) Buttner MP, Willeke K, Grinshpun SA. Sampling and analysis of airborne microorganisms. In: Hurst CJ, Knudsen GR, McInerney MJ, Stetzenbach LD, Walter MV, eds. *Manual of environmental microbiology*. Washington, DC: American Society for Microbiology Press, 1997;629-40.

1224. (346) Jensen PA, Schafer MP. Sampling and characterization of bioaerosols. In: NIOSH Manual of Analytical Methods; Cincinnati OH; CDC; 1998: p. 82–112. Available at: www.cdc.gov/niosh/nmam/pdfs/chapter-j.pdf
1225. Jolley AE. The value of surveillance cultures on neonatal intensive care units. *J Hosp Infect* 1993;25:153–9.
1226. Hardy KA, McGowan KL, Fisher MC, Schidlow DV. *Pseudomonas cepacia* in the hospital setting: lack of transmission between cystic fibrosis patients. *J Pediatr* 1986;109:51–4.
1227. Hambræus A, Lagerqvist-Widh A, Zettersten U, Engberg S, Sedin G, Sjöberg L. Spread of *Klebsiella* in a neonatal ward. *Scand J Infect Dis* 1991;23:189–94.
1228. Humphreys H, Peckham D, Patel P, Knox A. Airborne dissemination of *Burkholderia (Pseudomonas) cepacia* from adult patients with cystic fibrosis. *Thorax* 1994;49:1157–9.
1229. Pankhurst CL, Harrison VE, Philpott-Howard J. Evaluation of contamination of the dentist and dental surgery environment with *Burkholderia (Pseudomonas) cepacia* during treatment of children with cystic fibrosis. *Int J Paediatr Dent* 1995;5:243–7.
1230. Weber DO, Gooch JJ, Wood WR, Britt EM, Kraft RO. Influence of operating room surface contamination on surgical wounds: a prospective study. *Arch Surg* 1976;111:484–8.
1231. Pfeiffer EH, Wittig JR, Dunkelberg H, Werner HP. Hygienic and bacteriological comparative studies in 50 hospitals. V. Bacterial contamination of hospital surfaces. *Zentralbl Bakteriol [B]* 1978;167:11–21. (German)
1232. Sattar SA, Lloyd-Evans N, Springthorpe VS. Institutional outbreaks of rotavirus diarrhea: potential role of fomites and environmental surfaces as vehicles for virus transmission. *J Hyg (Camb)* 1986;96:277–89.
1233. Smith SM, Eng RH, Padberg FT Jr. Survival of nosocomial pathogenic bacteria at ambient temperature. *J Med* 1996;27:293–302.
1234. Craythorn JM, Barbour AG, Matsen JM, Britt MR, Garibaldi RA. Membrane filter contact technique for bacteriological sampling of moist surfaces. *J Clin Microbiol* 1980;12:250–5.
1235. Scott E, Bloomfield SF, Barlow CG. A comparison of contact plate and calcium alginate swab techniques for quantitative assessment of bacteriological contamination of environmental surfaces. *J Appl Bacteriol* 1984;56:317–20.
1236. Poletti L, Pasquarella C, Pitzurra M, Savino A. Comparative efficiency of nitrocellulose membranes versus RODAC plates in microbial sampling on surfaces. *J Hosp Infect* 1999;41:195–201.
1237. Russell AD. Factors influencing the efficacy of antimicrobial agents. In: Russell AD, Hugo WB, Ayliffe GAJ, eds. Principles and practices of disinfection, preservation and sterilization. Oxford, UK: Blackwell Science, 1999;95–123.
1238. (347) International Organization for Standardization (ISO). Sterilization of medical devices — microbiological methods, Part 1. ISO Standard 11737-1. Paramus, NJ: International Organization for Standardization, 1995.
1239. Favero MS, Gabis DA, Vesley D. Environmental monitoring procedures. In: Speck ML, ed. Compendium of methods for the microbiological examination of foods, 2nd ed. Washington, DC: American Public Health Association; 1984;47–61.
1240. Favero MS, Bond WW, Petersen NJ, Berquist KR, Maynard JE. Detection methods for study of the stability of hepatitis B antigen on surfaces. *J Infect Dis* 1974;129:210–2.
1241. Favero MS, McDade JJ, Robertsen JA, Hoffmann RK, Edwards RW. Microbiological sampling of surfaces. *J Appl Bacteriol* 1968;31:336–43.
1242. Petersen NJ, Collins DE, Marshall JH. Evaluation of skin cleansing procedures using the wipe-rinse technique. *Health Lab Sci* 1974;11:182–97.
1243. Schalkowsky S, Hall LB, Kline RC. Potential effects of recent findings on spacecraft sterilization requirements. *Space Life Sci* 1969;1:520–30.
1244. Hall LB, Lyle RG. Foundations of planetary quarantine. *Environ Biol Med* 1971;1:5–8.
1245. Rutala WA, Weber DJ. Uses of inorganic hypochlorite (bleach) in health-care facilities. *Clin Microbiol Rev* 1997;10:597–610.
1246. Mallison GF. Central services and linens and laundry. In: Bennett JV, Brachman PS, eds. Hospital infections. Boston, MA: Little, Brown, & Co, 1986;251–6.
1247. (365) Blaser MJ, Smith PE, Cody HJ, Wang W-LL, LaForce FM. Killing of fabric-associated bacteria in hospital laundry by low-temperature washing. *J Infect Dis* 1984;149:48–57.
1248. Centers for Disease Control. Outbreak of viral hepatitis in the staff of a pediatric ward — California. *MMWR* 1977;28:77–9.

1249. Shah PC, Krajden S, Kane J, Summerbell RC. Tinea corporis caused by *Microsporum canis*: report of a nosocomial outbreak. *Eur J Epidemiol* 1988;4:33–7.
1250. (353) Barrie D, Hoffman PN, Wilson JA, Kramer JM. Contamination of hospital linen by *Bacillus cereus*. *Epidemiol Infect* 1994;113:297–306.
1251. Standaert SM, Hutcheson RH, Schaffner W. Nosocomial transmission of *Salmonella gastroenteritis* to laundry workers in a nursing home. *Infect Control Hosp Epidemiol* 1994;15:22–6.
1252. Pasternak J, Richtmann R, Ganme APP, et al. Scabies epidemic: price and prejudice. *Infect Control Hosp Epidemiol* 1994;15:540–2.
1253. (357) Association for the Advancement of Medical Instrumentation. Processing of reusable surgical textiles for use in health care facilities: ANSI/AAMI recommended practice ST65. Arlington, VA: Association for the Advancement of Medical Instrumentation, 2000;16.
1254. Association of Operating Room Nurses. Recommended practices for surgical attire: AORN standards and recommended practices. *AORN J* 1995;62:141–2.
1255. Loh W, Ng VV, Holton J. Bacterial flora on the white coats of medical students. *J Hosp Infect* 2000;45:65–8.
1256. Belkin NL. Use of scrubs and related apparel in healthcare facilities. *Am J Infect Control* 1997;25:401–4.
1257. Belkin NL. Home laundering of soiled surgical scrubs: surgical site infections and the home environment. *Am J Infect Control* 2000;29:58–64.
1258. (355) Joint Committee on Healthcare Laundry Guidelines. Guidelines for healthcare linen service. Hallendale, FL: Textile Rental Service Association of America, 1999.
1259. (356) Greene VW. Microbiological contamination control in hospitals: part 6 — roles of central service and the laundry. *Hospitals JAHA* 1970;44:98–103.
1260. (350) Wagner RA. Partitioned laundry improves bacteria control. *Hospitals JAHA* 1966;40:148–51.
1261. (351) Hambraeus A, Malmborg AS. Is a bed centre in a hospital a hygienic hazard? *J Hyg (Camb)* 1982;88:143–7.
1262. (352) McDonald LL, Pugliese G. Textile processing service. In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;1031–4.
1263. (354) Legnani PP, Leoni E. Factors affecting the bacteriological contamination of commercial washing machines. *Zentralbl Hyg Umweltmed* 1997;200:319–33.
1264. Maki DG, Alvarado C, Hassemer C. Double-bagging of items from isolation rooms is unnecessary as an infection control measure: a comparative study of surface contamination with single- and double-bagging. *Infect Control* 1986;7:535–7.
1265. Garner JS, Simmons BP. CDC guideline for isolation precautions in hospitals. *Infect Control* 1983;4:245–325 and *Am J Infect Control* 1984;12:103–63.
1266. Weinstein SA, Gantz NM, Pelletier C, Hibert D. Bacterial surface contamination of patients' linen: isolation precautions versus standard care. *Am J Infect Control* 1989;17:264–7.
1267. (358) Hughes HG. Chutes in hospitals. *Can Hosp* 1964;41:56–7, 87.
1268. (359) Michaelsen GS. Designing linen chutes to reduce spread of infectious organisms. *Hospitals JAHA* 1965;39 (3):116–9.
1269. (360) Hoch KW. Laundry chute cleaning recommendations [letter]. *Infect Control* 1982;3:360.
1270. (361) Whyte W, Baird G, Annand R. Bacterial contamination on the surface of hospital linen chutes. *J Hyg (Camb)* 1969;67:427–35.
1271. (363) Walter WG, Schillinger JE. Bacterial survival in laundered fabrics. *Appl Microbiol* 1975;29:368–73.
1272. (362) Taylor LJ. Segregation, collection, and disposal of hospital laundry and waste. *J Hosp Infect* 1988;11 (Suppl. A):57–63.
1273. Barrie D. How hospital linen and laundry services are provided. *J Hosp Infect* 1994;27:219–35.
1274. Riggs CH, Sherrill JC. Textile laundering technology. Hallendale FL: Textile Rental Service Association; 1999;92–7.
1275. Mouton RP, Bekkers JH. Bacteriological results of routine procedures in a hospital laundry. *Folia Med Neerl* 1967;10:71–6.
1276. Nicholes PS. Bacteria in laundered fabrics. *Am J Public Health* 1970;60:2175–80.
1277. Arnold L. A sanitary study of commercial laundry practices. *Am J Public Health* 1938;28:839–44.

1278. (364) Belkin NL. Aseptics and aesthetics of chlorine bleach: can its use in laundering be safely abandoned? *Am J Infect Control* 1998;26:149–51.
1279. Jordan WE, Jones DV. Antiviral effectiveness of chlorine bleach in household laundry use. *Am J Dis Child* 1969;117:313–6.
1280. Hittman Associates, Inc. Energy efficient water use in hospitals [Final summary report (H-W8000-78-756FR)]. Prepared for the University of California, Lawrence Berkeley Laboratory, 1979. Contract No. P.O. 4627702.
1281. (366) Jaska JM, Fredell DL. Impact of detergent systems on bacterial survival on laundered fabrics. *Appl Environ Microbiol* 1980;39:743–8.
1282. (367) Battles DR, Vesley D. Wash water temperature and sanitation in the hospital laundry. *J Environ Health* 1981;43:244–50.
1283. (368) Christian RR, Manchester JT, Mellor MT. Bacteriological quality of fabrics washed at lower-than-standard temperatures in a hospital laundry facility. *Appl Environ Microbiol* 1983;45:591–7.
1284. (369) Smith JA, Neil KR, Davidson CG, Davidson RW. Effect of water temperature on bacterial killing in laundry. *Infect Control* 1987;8:204–9.
1285. (370) Tompkins DS, Johnson P, Fittall BR. Low-temperature washing of patients' clothing: effects of detergent with disinfectant and a tunnel drier on bacterial survival. *J Hosp Infect* 1988;12:51–8.
1286. (371) Ayliffe GAJ, Collins BJ, Taylor LJ. Laundering. In: Wright PSG, ed. *Hospital-acquired Infection: principles and Prevention*. Bristol, UK: 1982;101–6.
1287. Koller W, Wewalka G. A new method for microbiological evaluation of disinfecting laundering processes. *Zbl Bakt Hyg I Abt Orig B* 1982;176:463–71.
1288. (372) Meyer CL, Eitzen HE, Schreiner RL, Gfell MA, Moye L, Kleiman MB. Should linen in newborn intensive care units be autoclaved? *Pediatrics* 1981;67:362–4.
1289. (373) Wagg RE. Disinfection of textiles in laundering and dry cleaning. *Chem Ind* 1965;44:1830–4.
1290. (374) Bates CJ, Wilcox MH, Smith TL, Spencer RC. The efficacy of a hospital dry cleaning cycle in disinfecting material contaminated with bacteria and viruses. *J Hosp Infect* 1993;23:255–62.
1291. (375) Oehnel E. Drycleaning in the hospital laundry. *Can Hosp* 1971;September:66–7.
1292. DiGacomo JC, Odom JW, Ritoto PC, Swan KC. Cost containment in the operating room: use of reusables versus disposable clothing. *Am Surg* 1992;58:654–6.
1293. American Society for Testing Materials. Standard test method for resistance of materials used in protective clothing to penetration by synthetic blood. ASTM, 1998;F1670–98.
1294. American Society for Testing Materials. Standard test method for resistance of materials used in protective clothing to penetration by bloodborne pathogens using phi-X174 bacteriophage penetration as a test system. ASTM 1997;F1671–976.
1295. Belkin NL. Are “impervious” surgical gowns really liquid-proof? *Bull Am Col Surgeons* 1999;84:19–36.
1296. Belkin NL. OR gowns — even a “pass” can fail. *AORN J* 1999;70:302–4.
1297. Laufman H, Belkin NL, Meyer KK. A critical review of a century's progress in surgical apparel: how far have we come? *J Am Col Surgeons* 2000;191:554–68.
1298. Leonas KK, Jinkins RS. The relationship of selected fabric characteristics and the barrier effectiveness of surgical gown fabrics. *Am J Infect Control* 1997;25:16–23.
1299. Meyer KK, Beck WC. Gown-glove interface: a possible solution to the danger zone. *Infect Control Hosp Epidemiol* 1995;16:488–90.
1300. McCullough EA. Methods for determining the barrier efficacy of surgical gowns. *Am J Infect Control* 1993;21:368–74.
1301. Pissiotis CA, Komborozos V, Papoutsi C, Skrekas G. Factors that influence the effectiveness of surgical gowns in the operating theater. *Eur J Surg* 1997;163:597–604.
1302. Association of Operating Room Nurses (AORN). Recommended practices for use and selection of barrier materials for surgical gowns and drapes. Association of Operating Room Nurses. *AORN J* 1996;63:650, 653–4.
1303. Belkin NL, Koch FT. OR barrier materials — Necessity or extravagance? *AORN J* 1998;67:443–5.
1304. Rutala WA, Weber DJ. A review of single-use and reusable gowns and drapes in health care. *Infect Control Hosp Epidemiol* 2001;22:248–57.
1305. Murphy L. Cost/benefit study of reusable and disposable OR draping materials. *J Healthc Mater Manage* 1993;11:44–8.

1306. (376) U.S. Environmental Protection Agency. Consumer products treated with pesticides. Office of Pesticide Programs. Available at: www.epa.gov/opp00001/citizens/treatart.htm
1307. Kalyon BD, Olgun U. Antibacterial efficacy of triclosan-incorporated polymers. *Am J Infect Control* 2001;29:124–5.
1308. U.S. Environmental Protection Agency. Clarification of treated articles exemption. Availability of draft PR notice. *Federal Register* 1998;63:19256–8.
1309. Mayer CE. FTC Challenges antibacterial product. Washington, DC: Washington Post, September 17, 1999;A09.
1310. (377) Fujita K, Lilly HA, Kidson A, Ayliffe GAJ. Gentamicin-resistant *Pseudomonas aeruginosa* infection from mattresses in a burns unit. *Br Med J* 1981;283:219–20.
1311. (378) Grubb DJ, Watson KC. *Pseudomonas* septicaemia from plastic mattresses [letter]. *Lancet* 1982;1:518.
1312. (379) Sherertz RJ, Sullivan ML. An outbreak of infections with *Acinetobacter calcoaceticus* in burn patients: contamination of patients' mattresses. *J Infect Dis* 1985;151:252–8.
1313. (380) Ndawula EM, Brown L. Mattresses as reservoirs of epidemic methicillin-resistant *Staphylococcus aureus* [letter]. *Lancet* 1991;337:488.
1314. (381) O'Donoghue MAT, Allen KD. Costs of an outbreak of wound infections in an orthopaedic ward. *J Hosp Infect* 1992;22:73–9.
1315. (382) Weernink A, Severin WPJ, Thernberg T, Dijkshoorn L. Pillows, an unexpected source of *Acinetobacter*. *J Hosp Infect* 1995;29:189–99.
1316. Newsome TW, Johns LA, Pruitt BA Jr. Use of an air-fluidized bed in the care of patients with extensive burns. *Am J Surg* 1972;124:52–6.
1317. (383) Scheidt A, Drusin LM. Bacteriologic contamination in an air-fluidized bed. *J Trauma* 1983;23:241–2.
1318. (384) Freeman R, Gould FK, Ryan DW, Chamberlain J, Sisson PR. Nosocomial infection due to enterococci attributed to a fluidized microsphere bed: the value of pyrolysis mass spectrometry. *J Hosp Infect* 1994;27:187–93.
1319. Sharbaugh RJ, Hargest TS. Bactericidal effect of the air-fluidized bed. *Am Surgeon* 1971;37:583–6.
1320. Sharbaugh RJ, Hargest TS, Wright FA. Further studies on the bactericidal effect of the air-fluidized bed. *Am Surgeon* 1973;39:253–6.
1321. Winters WD. A new perspective of microbial survival and dissemination in a prospectively contaminated air-fluidized bed model. *Am J Infect Control* 1990;18:307–15.
1322. (385) Clancy MJ. Nosocomial infection and microsphere beds [letter]. *Lancet* 1993;342:680–1.
1323. (386) Clancy MJ. Nosocomial infection due to enterococci attributed to a fluidized microsphere bed [letter]. *J Hosp Infect* 1994;28:324–5.
1324. Vesley D, Hankinson SE, Lauer JL. Microbial survival and dissemination associated with an air-fluidized therapy unit. *Am J Infect Control* 1986;14:35–40.
1325. Bolyard EA, Townsend TR, Horan T. Airborne contamination associated with in-use air-fluidized beds: a descriptive study. *Am J Infect Control* 1987;15:75–8.
1326. (387) Jacobsen E, Gurevich I, Cunha BA. Air-fluidized beds and negative-pressure isolation rooms [letter]. *Am J Infect Control* 1993;21:217–8.
1327. Cooper JE. Pets in hospitals. *Br Med J* 1976;1:698–700.
1328. Egerton JR. Pets and zoonoses. *Med J Aust* 1982;2:311.
1329. Yamauchi T. Pet programs in hospitals. *Pediatr Infect Dis J* 1993;12:707.
1330. Khan MA, Farrag N. Animal-assisted activity and infection control implications in a healthcare setting. *J Hosp Infect* 2000;46:4–11.
1331. Weber DJ, Rutala WA. Epidemiology and prevention of nosocomial infections associated with animals in the hospital. In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;1399–421.
1332. Acha PN, Szyfres B. Zoonoses and communicable diseases common to man and animals, 2nd ed. Washington, DC: Pan American Health Organization, 1987. Scientific publication No. 503.
1333. Elliot DL, Tolle SW, Goldberg L, Miller JB. Pet-associated illness. *N Engl J Med* 1985;313:985–95.
1334. Marx MB. Parasites, pets, and people. *Primary Care* 1991;18:153–65.
1335. Goldstein EJ. Household pets and human infections. *Infect Dis Clin North Am* 1991;5:117–30.
1336. Chomel BB. Zoonoses of house pets other than dogs, cats, and birds. *Pediatr Infect Dis J* 1992;11:479–87.

1337. Gnann JW Jr, Bressler GS, Bodet CA III, Avent CK. Human blastomycosis after a dog bite. *Ann Intern Med* 1983;98:48–9.
1338. Garcia VF. Animal bites and *Pasteurella* infections. *Pediatr Rev* 1997;18:127–30.
1339. Crowder HR, Dorn CR, Smith RE. Group A *Streptococcus* in pets and group A streptococcal disease in man. *Int J Zoonoses* 1978;5:45–54.
1340. (397) CDC. Reptile-associated salmonellosis — selected states, 1996–1998. *MMWR* 1999;48:1009–13.
1341. Devriese LA, Ieven M, Goossens H, et al. Presence of vancomycin-resistant enterococci in farm and pet animals. *Antimicrob Agent Chemother* 1996;40:2285–7.
1342. Scott GM, Thomson R, Malone-Lee J, Ridgway GL. Cross-infection between animals and man: Possible feline transmission of *Staphylococcus aureus* infection in humans? *J Hosp Infect* 1988;12:29–34.
1343. Weinberg A. Ecology and epidemiology of zoonotic pathogens. *Infect Dis Clin North Am* 1991;5:1–6.
1344. Yu V, Meissner C. Zoonoses. In: Schaechter M, Medoff G, Schlessinger D, eds. *Mechanisms of microbial diseases*. Baltimore, MD: Williams & Wilkins, 1989;749–64.
1345. Ryan KJ. Some bacteria causing zoonotic diseases. In: Sherris JC, ed. *Medical microbiology*, 2nd ed. New York, NY: Elsevier, 1990;489–98.
1346. Chang HJ, Miller HL, Watkins N, et al. An epidemic of *Malassezia pachydermatis* in an intensive care nursery associated with colonization of healthcare workers' pet dog. *N Engl J Med* 1998;338:706–11.
1347. Drusin LM, Ross BG, Rhodes KH, Krause AN, Scott RA. Nosocomial ringworm in a neonatal intensive care unit: a nurse and her cat. *Infect Control Hosp Epidemiol* 2000;21:605–7.
1348. Richet HM, Craven PC, Brown JM, et al. A cluster of *Rhodococcus (Gordona) bronchialis* sternal-wound infections after coronary-artery bypass surgery. *N Engl J Med* 1991;324:104–9.
1349. (394) Saylor K. Pet visitation program. *J Gerontol Nurs* 1998;24:36–8.
1350. Corson SA, O'Leary Corson E. Pets as mediators of therapy. *Curr Psychiatr Ther* 1978;18:195–205.
1351. Fick KM. The influence of an animal on social interactions of nursing home residents in a group setting. *Am J Occup Ther* 1993;47:529–34.
1352. Gunby P. Patient progressing well? He must have a pet. *JAMA* 1979;241:438.
1353. Culliton BJ. Take two pets and call me in the morning. *Science* 1987;237:1560–1.
1354. Wilkes CN, Shalko TK, Trahan M. Pet Rx: Implications for good health. *Health Educ* 1989;20:6–9.
1355. Doyle K, Kukowski T. Utilization of pets in a hospice program. *Health Educ* 1989;20:10–1.
1356. Teeter LM. Pet therapy program. *J Amer Vet Med Assoc* 1997;210:1435–8.
1357. Gammonley J, Yates J. Pet projects: animal assisted therapy in nursing homes. *J Gerontol Nurs* 1991;17:12–5.
1358. (395) Draper RJ, Gerber GJ, Layng EM. Defining the role of pet animals in psychotherapy. *Psychiat J Univ Ottawa* 1990;15:169–72.
1359. Allen DT. Effects of dogs on human health. *J Amer Vet Med Assoc* 1997;210:1136–9.
1360. (391) Delta Society. *Standards of practice for animal-assisted activities and animal-assisted therapy*. Renton, WA: Delta Society, 1996.
1361. (392) Fox JG. Transmissible drug resistance in *Shigella* and *Salmonella* isolated from pet monkeys and their owners. *J Med Primatol* 1975;4:165–71.
1362. (393) Ostrowski SR, Leslie MJ, Parrott T, Abelt S, Piercy PE. B-virus from pet macaque monkeys: an emerging threat in the United States? *Emerg Infect Dis* 1998;4:117–21.
1363. CDC. How to prevent transmission of intestinal roundworms from pets to people. Available at: www.cdc.gov/ncidod/diseases/roundworm/roundworm.htm
1364. (146) Boyce JM, Pittet D. Guideline for hand hygiene in health-care settings: recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force. *Infect Control Hosp Epidemiol* 2002;23 (Suppl):S1–S40.
1365. (388) American Academy of Allergy, Asthma, and Immunology. Tips to remember: indoor allergens. Available at: www.aaaai.org/patients/publicedmat/tips/indoorallergens.stm
1366. (389) Duncan SL, APIC Guideline Committee. APIC State-of-the-art report: the implications of service animals in healthcare settings. *Am J Infect Control* 2000;28:170–80.
1367. (390) Murray AB, Ferguson A, Morrison BJ. The frequency and severity of cat allergy vs. dog allergy in atopic children. *J Allergy Clin Immunol* 1983;72:145–9.
1368. Hodson T, Custovic A, Simpson A, Chapman M, Woodcock A, Green R. Washing the dog reduces dog allergen levels, but the dog needs to be washed twice a week. *J Allergy Clin Immunol* 1999;103:581–5.
1369. Brickel CN. The therapeutic roles of cat mascots with a hospital based population: a staff survey. *Gerontologist* 1979;19:368–72.

1370. Thomas W, Stermer M. Eden alternative principles hold promise for the future of long-term care. *Balance* 1999;3:14–7.
1371. Tavormina CE. Embracing the Eden alternative in long-term care environments. *Geriatr Nurs* 1999;20:158–61.
1372. Brook I, Fish CH, Schantz PM, Cotton DD. Toxocariasis in an institution for the mentally retarded. *Infect Control* 1981;2:317–9.
1373. Huminer D, Symon R, Groskopf I, et al. Seroepidemiological study of toxocariasis and strongyloidiasis in adult mentally retarded institutionalized subjects. *Am J Trop Med Hyg* 1992;46:278–81.
1374. Huminer D, Pitlik SD, Block C, Kaufman L, Amit S, Rosenfeld JB. Aquarium-borne *Mycobacterium marinum* skin infection: report of a case and review of the literature. *Arch Dermatol* 1986;122:698–703.
1375. Lewis FMT, Marsh BJ, von Reyn CF. Fish tank exposure and cutaneous infections due to *Mycobacterium marinum*: tuberculin skin testing, treatment, and prevention. *Clin Infect Dis* 2003;37:390–7.
1376. (398) U.S. Department of Justice. Americans with Disabilities Act. Public Law 101-336 (28 CFR 36.101 et seq.). Title III, Public Accommodations Operated by Private Entities, Sect. 302, Prohibition of Discrimination by Public Accommodations;42 USC 12101 et seq. July 26, 1990.
1377. U.S. Department of Justice, Civil Rights Division, Disability Rights Section. Commonly asked questions about service animals in places of business, 1996.
1378. Schachter J, Sung M, Meyer KF. Potential danger of Q fever in a university hospital environment. *J Infect Dis* 1971;123:301–4.
1379. Konkle DM, Nelson KN, Lunn DP. Nosocomial transmission of *Cryptosporidium* in a veterinary hospital. *J Vet Intern Med* 1997;11:340–3.
1380. House JK, Mainar-Jaime RC, Smith BP, House AM, Kamiya DY. Risk factors for nosocomial *Salmonella* infection among hospitalized horses. *J Am Vet Med Assoc* 1999;214:1511–6.
1381. Weese JS, Staempfli HR, Prescott JF. Isolation of environmental *Clostridium difficile* from a veterinary teaching hospital. *J Vet Diagn Invest* 2000;12:449–52.
1382. Boerlin P, Eugster S, Gaschen F, Straub R, Schawalder P. Transmission of opportunistic pathogens in a veterinary teaching hospital. *Vet Microbiol* 2001;82:347–9.
1383. Schott HC II, Ewart SL, Walker RD, et al. An outbreak of salmonellosis among horses at a veterinary teaching hospital. *J Am Vet Med Assoc* 2001;218:1152–9,1170.
1384. Kim LM, Morley PS, Traub-Dargatz JL, Salman MD, Gentry-Weeks C. Factors associated with *Salmonella* shedding among equine colic patients at a veterinary teaching hospital. *J Am Vet Med Assoc* 2001;218:740–8.
1385. Seguin JC, Walker RD, Caron JP, et al. Methicillin-resistant *Staphylococcus aureus* outbreak in a veterinary teaching hospital: potential human-to-animal transmission. *J Clin Microbiol* 1999;37:1459–63.
1386. Shen DT, Crawford TB, Gorham JR, et al. Inactivation of equine infectious anemia virus by chemical disinfectants. *Am J Vet Res* 1977;38:1217–9.
1387. Scott FW. Virucidal disinfectants and feline viruses. *Am J Vet Res* 1980;41:410–4.
1388. Brown TT. Laboratory evaluation of selected disinfectants as virucidal agents against porcine parvovirus, pseudorabies virus, and transmissible gastroenteritis virus. *Am J Vet Res* 1981;42:1033–6.
1389. Saknimit M, Inatsuki I, Sugiyama Y, et al. Virucidal efficacy of physico-chemical treatments against coronaviruses and parvoviruses of laboratory animals. *Exp Anim* 1988;37:341–5.
1390. Bruins G, Dyer JA. Environmental considerations of disinfectants used in agriculture. *Rev Sci Tech* 1995;14:81–94.
1391. Quinn PJ, Markey BK. Disinfection and disease prevention in veterinary medicine. In: Block SS, ed. *Disinfection, sterilization, and preservation*, 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2001;1069–103.
1392. Fox JG, Lipman NS. Infections transmitted by large and small laboratory animals. *Infect Dis Clin North Am* 1991;5:131–63.
1393. (401) U.S. Department of Labor, Occupational Safety and Health Administration. Personal Protective Equipment for General Industry, Final Rule (29 CFR 1910 §1910.132, 1910.138). *Federal Register* 1994;59:16334–64.
1394. U.S. Department of Agriculture. Public Law 89-544 (The Animal Welfare Act of 1966). 7 USC § 2131-2156.

1395. **(399)** U.S. Department of Agriculture. Public Law 99-198 Food Security Act of 1985, Subtitle F - Animal Welfare. 7 USC § 2131.
1396. Althaus H, Sauerwald M, Schrammeck E. Waste from hospitals and sanatoria. *Zbl Bakteriol Hyg I Abt Orig B* 1983;178:1–29.
1397. Kalnowski G, Wiegand H, Henning R. The microbial contamination of hospital waste. *Zbl Bakteriol Hyg I Abt Orig B* 1983;178:364–79.
1398. Mose JR, Reinthaler F. Microbial contamination of hospital waste and household refuse. *Zbl Bakteriol Hyg I Abt Orig B* 1985;181:98–110.
1399. Collins CH, Kennedy DA. The microbiological hazards of municipal and clinical wastes. *J Appl Bacteriol* 1992;73:1–6.
1400. Rutala WA, Odette RL, Samsa GP. Management of infectious waste by U.S. hospitals. *JAMA* 1989;262:1635–40.
1401. Agency for Toxic Substances and Disease Registry. The public health implications of medical waste: a report to Congress. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, 1990.
1402. Hedrick ER. Infectious waste management — will science prevail? *Infect Control Hosp Epidemiol* 1988;9:488–90.
1403. Keene J. Medical waste management: public pressure vs. sound science. *Hazard Mat Control* 1989;Sept/Oct:29–36.
1404. Keene J. Medical waste: a minimal hazard. *Infect Control Hosp Epidemiol* 1991;12:682–5.
1405. Rutala WA, Weber DJ. Mismatch between science and policy. *N Engl J Med* 1991;325:578–82.
1406. U.S. Environmental Protection Agency. EPA guide for infectious waste management. Washington, DC: U.S. Government Printing Office, 1986. EPA Publication No. 530SW86014.
1407. **(402)** U.S. Department of Transportation. Hazardous materials regulations. 49 CFR Parts 171–180, Division 6.2; and Hazardous Materials: Revision to Standards for Infectious Substances and Genetically-Modified Microorganisms: Proposed Rule. *Federal Register* 1998;63:46843–59.
1408. **(403)** U.S. Postal Service. C 023.8.0 Infectious substances (Hazard Class 6, Division 6.2) et seq. At: <http://pe.usps.gov/text/dmm/c023.htm>
1409. **(404)** Greene R, Miele DJ, Slavik NS. Technical assistance manual: state regulatory oversight of medical waste treatment technologies, 2nd ed: a report of the State and Territorial Association on Alternative Treatment Technologies, 1994.
1410. U.S. Environmental Protection Agency (EPA). 40 CFR Part 60. Standards of performance for new stationary sources and emission guidelines for existing sources: hospital/medical/infectious waste incinerators; Final Rule. *Federal Register* 1997;62:48347–91.
1411. CDC/National Institutes of Health. Biosafety in microbiological and biomedical laboratories. Washington, DC: U.S. Government Printing Office, 1985. DHHS Publication No. (CDC) 93–8395.
1412. **(409)** U.S. Department of Health and Human Services, Office of Inspector General, CDC. Possession, use, and transfer of select agents and toxins, interim final rule (42 CFR Part 73). *Federal Register*, December 13, 2002;67(240):76885–905.
1413. U.S. Department of Agriculture, Animal and Plant Health Inspection Service. Agricultural Bioterrorism Protection Act of 2002; Possession, use, and transfer of biological agents and toxins: interim final rule (9 CFR Part 121). *Federal Register*. December 13, 2002;67(240):76907–38.
1414. **(410)** CDC. Recommendations on infective waste. Atlanta, GA: Office of Biosafety and Hospital Infections Program, 1988;1–6.
1415. **(405)** CDC. National Institute for Occupational Safety and Health. NIOSH Alert: Preventing needlestick injuries in health care settings. Cincinnati, OH: DHHS, 1999. DHHS-NIOSH Publication No. 2000–108.
1416. Rutala WA, Stiegel MM, Sarubbi FA. Decontamination of laboratory microbiological waste by steam sterilization. *Appl Environ Microbiol* 1982;43:1311–6.
1417. Lauer JL, Battles DR, Vesley D. Decontaminating infectious laboratory waste by autoclaving. *Appl Environ Microbiol* 1982;44:690–4.
1418. Palenik CJ, Cumberlander ND. Effects of steam sterilization on the contents of sharps containers. *Am J Infect Control* 1993;21:28–33.
1419. **(406)** Weber AM, Boudreau Y, Mortimer VD. Stericycle, Inc., Morton, WA. HETA 98-0027-2709. NIOSH, CDC, Cincinnati, OH, 1998.

1420. (407) Johnson KR, Braden CR, Cairns KL, et al. Transmission of *Mycobacterium tuberculosis* from medical waste. *JAMA* 2000;284:1683–8.
1421. (408) Emery R, Sprau D, Lao YJ, Pryor W. Release of bacterial aerosols during infectious waste compaction: An initial hazard evaluation for health care workers. *Am Ind Hyg Assoc J* 1992;53:339–45.
1422. National Committee for Clinical Laboratory Standards (NCCLS). Protection of laboratory workers from instrument biohazards and infectious disease transmitted by blood, body fluids, and tissue. Approved guideline. 1997, NCCLS Document M29-A (ISBN1-56238-339-6).
1423. Snyder JW, Check W. Bioterrorism threats to our future: the role of the clinical microbiology laboratory in detection, identification, and confirmation of biological agents. Report of the October 27–29, 2000 Colloquium, American Academy of Microbiology, American College of Microbiology. Available at: www.asm.org
1424. CDC. List of select agents and biological toxins. At: www.cdc.gov/od/sap/docs/salist.pdf
1425. Bond WW. Survival of hepatitis B virus in the environment. *JAMA* 1984;252:397–8.
1426. Slade JE, Pike EB, Eglin RP, Colbourne JS, Kurtz JB. The survival of human immunodeficiency virus in water, sewage, and sea water. *Water Sci Technol* 1989;21:55–9.
1427. Geertsma RE, Van Asten JAAM. Sterilization of prions. *Zentr Steril* 1995;3:385–94.
1428. (52) Johnson MW, Mitch WE, Heller AH, Spector R. The impact of an educational program on gentamicin use in a teaching hospital. *Am J Med* 1982;73:9–14.
1429. (53) Soumerai SB, Salem-Schatz S, Avorn J, Casteris CS, Ross-Degnan D, Popovsky MA. A controlled trial of educational outreach to improve blood transfusion practice. *JAMA* 1993;270:961–6.
1430. (54) Eisenberg JM. An education program to modify laboratory use by house staff. *J Med Educ* 1977;52:578–81.
1431. (55) Rello J, Quintana E, Ausina V, Puzo V, Puzo C, Net A, Prats G. Risk factors for *Staphylococcus aureus* nosocomial pneumonia in critically ill patients. *Am Rev Respir Dis* 1990;142:1320–4.
1432. (56) McWhinney PHM, Kibbler CC, Hamon MD, et al. Progress in the diagnosis and management of aspergillosis in bone marrow transplantation: 13 years' experience. *Clin Infect Dis* 1993;17:397–404.
1433. (74) Aisner J, Murillo J, Schimpff SC, Steere AC. Invasive aspergillosis in acute leukemia: Correlation with nose cultures and antibiotic use. *Ann Intern Med* 1979;90:4–9.
1434. (102) Rhome FS. Endemic nosocomial filamentous fungal disease: a proposed structure for conceptualizing and studying the environmental hazard. *Infect Control* 1986;7S:124–5.
1435. (Table 1) Mutchler JE. Principles of ventilation. In: NIOSH. The industrial environment — its evaluation and control. Washington, DC: U.S. Department of Health, Education, and Welfare, Public Health Service, NIOSH, 1973. Publication #74-117. Available at: www.cdc.gov/niosh/74-117.html
1436. Breiman RF, Cozen W, Fields BS, et al. Role of air sampling in investigation of an outbreak of Legionnaires' disease associated with exposure to aerosols from an evaporative condenser. *J Infect Dis* 1990;161:1257–61.
1437. (Appendix; 9) CDC. Procedures for the recovery of *Legionella* from the environment. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, CDC, 1992;1–13.
1438. Costerton JW, Lewandowski Z, Caldwell DE, Korber DR, Lappin-Scott HM. Microbial biofilms. *Ann Rev Microbiol* 1995;49:711–45.
1439. LeChevallier MW, Babcock TM, Lee RG. Examination and characterization of distribution system biofilms. *Appl Environ Microbiol* 1987;53:2714–24.
1440. Nagy LA, Olson BH. Occurrence and significance of bacteria, fungi, and yeasts associated with distribution pipe surfaces. *Proceeds of the Water Quality Technology Conference*. Portland, OR: 1985;213–38.
1441. Maki DG, Martin WT. Nationwide epidemic of septicemia caused by contaminated infusion products. IV growth of microbial pathogens in fluids for intravenous infusion. *J Infect Dis* 1975;131:267–72.
1442. Costerton JW, Stewart PS, Greenberg EP. Bacterial biofilms: a common cause of persistent infections. *Science* 1999;284:1318–22.
1443. Costerton JW, Khoury AE, Ward KH, Anwar H. Practical measures to control device-related bacterial infections. *Int J Artif Organs* 1993;16:765–70.
1444. Nickel JC, Costerton JW, McLean RJC, Olson M. Bacterial biofilms: influence on the pathogenesis, diagnosis, and treatment of urinary tract infections. *J Antimicrobial Chemother* 1994;33 (Suppl. A):31–41.
1445. LeChevallier MW, Cawthon CD, Lee RG. Inactivation of biofilm bacteria. *Appl Environ Microbiol* 1988;54:2492–9.

1446. Anwar J, Strap JL, Costerton JW. Establishment of aging biofilms: possible mechanism of bacterial resistance to antimicrobial therapy. *Antimicrobiol Agents Chemotherapy* 1992;36:1347–51.
1447. Stewart PS. Biofilm accumulation model that predicts antibiotic resistance to *Pseudomonas aeruginosa* biofilms. *Antimicrobiol Agents Chemotherapy* 1994;38:1052–8.
1448. Chen X, Stewart PS. Chlorine penetration into artificial biofilm is limited by a reaction-diffusion interaction. *Environ Sci Technol* 1996;30:2078–83.
1449. Huang C-T, Yu FP, McFeters GA, Stewart PS. Non-uniform spatial patterns of respiratory activity with biofilms during disinfection. *Appl Environ Microbiol* 1995;61:2252–6.
1450. Donlan RM, Pipes WO. Selected drinking water characteristics and attached microbial population density. *JAWWA* 1988;80:70–6.
1451. Reasoner DJ, Geldreich EE. A new medium for the enumeration and subculture of bacteria from potable water. *Appl Environ Microbiol* 1985;49:1–7.
1452. Pass T, Wright R, Sharp B, Harding GB. Culture of dialysis fluids on nutrient-rich media for short periods at elevated temperatures underestimates microbial contamination. *Blood Purif* 1996;14:136–45.
1453. Arduino MJ, Bland LA, Agüero SM, et al. Effects of incubation time and temperature on microbiologic sampling procedures for hemodialysis fluids. *J Clin Microbiol* 1991;29:1462–5.
1454. (234) Klein E, Pass T, Harding GB, Wright R, Million C. Microbial and endotoxin contamination in water and dialysate in the central United States. *Artif Organs* 1990;14:85–94.
1455. (235) Man N-K, Degremont A, Darbord J-C, Collet M, Vaillant P. Evidence of bacterial biofilm in tubing from hydraulic pathway of hemodialysis system. *Artif Organs* 1998;22:596–600.
1456. Pearson FC, Weary ME, Sargent HE, et al. Comparison of several control standard endotoxins to the National Reference Standard Endotoxin — an HIMA collaborative study. *Appl Environ Microbiol* 1985;50:91–3.
1457. (Appendix; 1) Arnow PM, Weil D, Para MF. Prevalence of significance of *Legionella pneumophila* contamination of residential hot-tap water systems. *J Infect Dis* 1985;152:145–51.
1458. (Appendix; 2) Shelton BG, Morris GK, Gorman GW. Reducing risks associated with *Legionella* bacteria in building water systems. In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;279–81.
1459. (Appendix; 3) Joly JR. Monitoring for the presence of *Legionella*: where, when, and how? In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;211–6.
1460. (Appendix; 7) Brenner DJ, Feeley JC, Weaver RE. Family VII. *Legionellaceae*. In: Krieg NR, Holt JG, eds. *Bergey's manual of systemic bacteriology*, volume 1. Baltimore, MD: Williams & Wilkins, 1984;279–88.
1461. (Appendix; 8) Katz SM, Hammel JM. The effect of drying, heat, and pH on the survival of *Legionella pneumophila*. *Ann Clin Lab Sci* 1987;17:150–6.
1462. (Box 2) Alary MA, Joly JR. Comparison of culture methods and an immunofluorescence assay for the detection of *Legionella pneumophila* in domestic hot water devices. *Curr Microbiol* 1992;25:19–25.
1463. (Box 2) Vickers RM, Stout JE, Yu VL. Failure of a diagnostic monoclonal immunofluorescent reagent to detect *Legionella pneumophila* in environmental samples. *Appl Environ Microbiol* 1990;56:2912–4.
1464. (Box 2) Flournoy DJ, Belobraydic KA, Silberg SL, Lawrence CH, Guthrie PJ. False positive *Legionella pneumophila* direct immunofluorescence monoclonal antibody test caused by *Bacillus cereus* spores. *Diag Microbiol Infect Dis* 1988;9:123–5.
1465. (Box 2) Bej AK, Majbubani MH, Atlas RM. Detection of viable *Legionella pneumophila* in water by polymerase chain reaction and gene probe methods. *Appl Environ Microbiol* 1991;57:597–600.
1466. Schulze-Röbbecke R, Jung KD, Pullman H, Hundgeburth J. Control of *Legionella pneumophila* in a hospital hot water system. *Zbl Hyg* 1990;190:84–100.
1467. Colbourne JS, Pratt DJ, Smith MG, Fisher-Hoch SP, Harper D. Water fittings as sources of *Legionella pneumophila* in a hospital plumbing system. *Lancet* 1984;1:210–3.
1468. U.S. Environmental Protection Agency. National interim primary drinking water regulations: control of trihalomethanes in drinking water: final rules. *Federal Register* 1979;44:68624–705.
1469. U.S. Environmental Protection Agency. National interim primary drinking water regulations: Trihalomethanes. *Federal Register* 1983;48:8406–14.

Part IV. Appendices

Appendix A. Glossary of Terms

Acceptable indoor air quality: air in which there are no known contaminants at harmful concentrations as determined by knowledgeable authorities and with which a substantial majority ($\geq 80\%$) of the people exposed do not express dissatisfaction.

ACGIH: American Conference of Governmental Industrial Hygienists.

Action level: the concentration of a contaminant at which steps should be taken to interrupt the trend toward higher, unacceptable levels.

Aerosol: particles of respirable size generated by both humans and environmental sources and that have the capability of remaining viable and airborne for extended periods in the indoor environment.

AIA: American Institute of Architects, a professional group responsible for publishing the *Guidelines for Design and Construction of Hospitals and Healthcare Facilities*, a consensus document for design and construction of health-care facilities endorsed by the U.S. Department of Health and Human Services, health-care professionals, and professional organizations.

Air changes per hour (ACH): the ratio of the volume of air flowing through a space in a certain period of time (the airflow rate) to the volume of that space (the room volume). This ratio is expressed as the number of air changes per hour (ACH).

Air mixing: the degree to which air supplied to a room mixes with the air already in the room, usually expressed as a mixing factor. This factor varies from 1 (for perfect mixing) to 10 (for poor mixing). It is used as a multiplier to determine the actual airflow required (i.e., the recommended ACH multiplied by the mixing factor equals the actual ACH required).

Airborne transmission: a means of spreading infection when airborne droplet nuclei (small particle residue of evaporated droplets $\leq 5 \mu\text{m}$ in size containing microorganisms that remain suspended in air for long periods of time) are inhaled by the susceptible host.

Air-cleaning system: a device or combination of devices applied to reduce the concentration of airborne contaminants (e.g., microorganisms, dusts, fumes, aerosols, other particulate matter, and gases).

Air conditioning: the process of treating air to meet the requirements of a conditioned space by controlling its temperature, humidity, cleanliness, and distribution.

Allogeneic: non-twin, non-self. The term refers to transplanted tissue from a donor closely matched to a recipient but not related to that person.

Ambient air: the air surrounding an object.

Anemometer: a flow meter which measures the wind force and velocity of air. An anemometer is often used as a means of determining the volume of air being drawn into an air sampler.

Anteroom: a small room leading from a corridor into an isolation room. This room can act as an airlock, preventing the escape of contaminants from the isolation room into the corridor.

ASHE: American Society for Healthcare Engineering, an association affiliated with the American Hospital Association.

ASHRAE: American Society of Heating, Refrigerating, and Air-Conditioning Engineers Inc.

Autologous: self. The term refers to transplanted tissue whose source is the same as the recipient, or an identical twin.

Automated cyler: a machine used during peritoneal dialysis which pumps fluid into and out of the patient while he/she sleeps.

Biochemical oxygen demand (BOD): a measure of the amount of oxygen removed from aquatic environments by aerobic microorganisms for their metabolic requirements. Measurement of BOD is used to determine the level of organic pollution of a stream or lake. The greater the BOD, the greater

the degree of water pollution. The term is also referred to as Biological Oxygen Demand (BOD).

Biological oxygen demand (BOD): an indirect measure of the concentration of biologically degradable material present in organic wastes (pertaining to water quality). It usually reflects the amount of oxygen consumed in five days by biological processes breaking down organic waste (BOD5).

Biosafety level: a combination of microbiological practices, laboratory facilities, and safety equipment determined to be sufficient to reduce or prevent occupational exposures of laboratory personnel to the microbiological agents they work with. There are four biosafety levels based on the hazards associated with the various microbiological agents.

BOD5: the amount of dissolved oxygen consumed in five days by biological processes breaking down organic matter.

Bonneting: a floor cleaning method for either carpeted or hard surface floors that uses a circular motion of a large fibrous disc to lift and remove soil and dust from the surface.

Capped spur: a pipe leading from the water recirculating system to an outlet that has been closed off ("capped"). A capped spur cannot be flushed, and it might not be noticed unless the surrounding wall is removed.

CFU/m³: colony forming units per cubic meter (of air).

Chlamydo spores: thick-walled, typically spherical or ovoid resting spores asexually produced by certain types of fungi from cells of the somatic hyphae.

Chloramines: compounds containing nitrogen, hydrogen, and chlorine. These are formed by the reaction between hypochlorous acid (HOCl) and ammonia (NH₃) and/or organic amines in water. The formation of chloramines in drinking water treatment extends the disinfecting power of chlorine. The term is also referred to as Combined Available Chlorine.

Cleaning: the removal of visible soil and organic contamination from a device or surface, using either the physical action of scrubbing with a surfactant or detergent and water, or an energy-based process (e.g., ultrasonic cleaners) with appropriate chemical agents.

Coagulation-flocculation: coagulation is the clumping of particles that results in the settling of impurities. It may be induced by coagulants (e.g., lime, alum, and iron salts). Flocculation in water and wastewater treatment is the agglomeration or clustering of colloidal and finely-divided suspended matter after coagulation by gentle stirring by either mechanical or hydraulic means, such that they can be separated from water or sewage.

Commissioning (a room): testing a system or device to ensure that it meets the pre-use specifications as indicated by the manufacturer or predetermined standard, or air sampling in a room to establish a pre-occupancy baseline standard of microbial or particulate contamination. The term is also referred to as benchmarking at 77°F (25°C).

Completely packaged: functionally packaged, as for laundry.

Conidia: asexual spores of fungi borne externally.

Conidiophores: specialized hyphae that bear conidia in fungi.

Conditioned space: that part of a building that is heated or cooled, or both, for the comfort of the occupants.

Contaminant: an unwanted airborne constituent that may reduce the acceptability of air.

Convection: the transfer of heat or other atmospheric properties within the atmosphere or in the airspace of an enclosure by the circulation of currents from one region to another, especially by such motion directed upward.

Cooling tower: a structure engineered to receive accumulated heat from ventilation systems and equipment and transfer this heat to water, which then releases the stored heat to the atmosphere through evaporative cooling.

Critical item (medical instrument): a medical instrument or device that contacts normally sterile areas of the body or enters the vascular system. There is a high risk of infection from such devices if they are microbiologically contaminated prior to use. These devices must be sterilized before use.

Dead legs: areas in the water system where water stagnates. A dead leg is a pipe or spur, leading from the water recirculating system to an outlet that is used infrequently, resulting in inadequate flow of

water from the recirculating system to the outlet. This inadequate flow reduces the perfusion of heat or chlorine into this part of the water distribution system, thereby adversely affecting the disinfection of the water system in that area.

Deionization: removal of ions from water by exchange with other ions associated with fixed charges on a resin bed. Cations are usually removed and H^+ ions are exchanged; OH^- ions are exchanged for anions.

Detritus: particulate matter produced by or remaining after the wearing away or disintegration of a substance or tissue.

Dew point: the temperature at which a gas or vapor condenses to form a liquid; the point at which moisture begins to condense out of the air. At dew point, air is cooled to the point where it is at 100% relative humidity or saturation.

Dialysate: the aqueous electrolyte solution, usually containing dextrose, used to make a concentration gradient between the solution and blood in the hemodialyzer (dialyzer).

Dialyzer: a device that consists of two compartments (blood and dialysate) separated by a semipermeable membrane. A dialyzer is usually referred to as an artificial kidney.

Diffuser: the grille plate that disperses the air stream coming into the conditioned air space.

Direct transmission: involves direct body surface-to-body surface contact and physical transfer of microorganisms between a susceptible host and an infected/colonized person, or exposure to cloud of infectious particles within 3 feet of the source; the aerosolized particles are $>5 \mu m$ in size.

Disability: as defined by the Americans with Disabilities Act, a disability is any physical or mental impairment that substantially limits one or more major life activities, including but not limited to walking, talking, seeing, breathing, hearing, or caring for oneself.

Disinfection: a generally less lethal process of microbial inactivation (compared to sterilization) that eliminates virtually all recognized pathogenic microorganisms but not necessarily all microbial forms (e.g., bacterial spores).

Drain pans: pans that collect water within the HVAC system and remove it from the system. Condensation results when air and steam come together.

Drift: circulating water lost from the cooling tower in the form as liquid droplets entrained in the exhaust air stream (i.e., exhaust aerosols from a cooling tower).

Drift eliminators: an assembly of baffles or labyrinth passages through which the air passes prior to its exit from the cooling tower. The purpose of a drift eliminator is to remove entrained water droplets from the exhaust air.

Droplets: particles of moisture, such as are generated when a person coughs or sneezes, or when water is converted to a fine mist by a device such as an aerator or shower head. These particles may contain infectious microorganisms. Intermediate in size between drops and droplet nuclei, these particles tend to quickly settle out from the air so that any risk of disease transmission is generally limited to persons in close proximity to the droplet source.

Droplet nuclei: sufficiently small particles ($1-5 \mu m$ in diameter) that can remain airborne indefinitely and cause infection when a susceptible person is exposed at or beyond 3 feet of the source of these particles.

Dual duct system: an HVAC system that consists of parallel ducts that produce a cold air stream in one and a hot air stream in the other.

Dust: an air suspension of particles (aerosol) of any solid material, usually with particle sizes $\leq 100 \mu m$ in diameter.

Dust-spot test: a procedure that uses atmospheric air or a defined dust to measure a filter's ability to remove particles. A photometer is used to measure air samples on either side of the filter, and the difference is expressed as a percentage of particles removed.

Effective leakage area: the area through which air can enter or leave the room. This does not include supply, return, or exhaust ducts. The smaller the effective leakage area, the better isolated the room.

Endotoxin: the lipopolysaccharides of gram-negative bacteria, the toxic character of which resides in the lipid portion. Endotoxins generally produce pyrogenic reactions in persons exposed to these

bacterial components.

Enveloped virus: a virus whose outer surface is derived from a membrane of the host cell (either nuclear or the cell's outer membrane) during the budding phase of the maturation process. This membrane-derived material contains lipid, a component that makes these viruses sensitive to the action of chemical germicides.

Evaporative condenser: a wet-type, heat-rejection unit that produces large volumes of aerosols during the process of removing heat from conditioned space air.

Exhaust air: air removed from a space and not reused therein.

Exposure: the condition of being subjected to something (e.g., infectious agents) that could have a harmful effect.

Fastidious: having complex nutritional requirements for growth, as in microorganisms.

Fill: that portion of a cooling tower which makes up its primary heat transfer surface. Fill is alternatively known as "packing."

Finished water: treated, or potable water.

Fixed room-air HEPA recirculation systems: nonmobile devices or systems that remove airborne contaminants by recirculating air through a HEPA filter. These may be built into the room and permanently ducted or may be mounted to the wall or ceiling within the room. In either situation, they are fixed in place and are not easily movable.

Fomite: an inanimate object that may be contaminated with microorganisms and serves in their transmission.

Free and available chlorine: the term applied to the three forms of chlorine that may be found in solution (i.e., chlorine [Cl₂], hypochlorite [OCl⁻], and hypochlorous acid [HOCl]).

Germicide: a chemical that destroys microorganisms. Germicides may be used to inactivate microorganisms in or on living tissue (antiseptics) or on environmental surfaces (disinfectants).

Health-care-associated: an outcome, usually an infection, that occurs in any health-care facility as a result of medical care. The term "health-care-associated" replaces "nosocomial," the latter term being limited to adverse infectious outcomes occurring only in hospitals.

Hemodiafiltration: a form of renal replacement therapy in which waste solutes in the patient's blood are removed by both diffusion and convection through a high-flux membrane.

Hemodialysis: a treatment for renal replacement therapy in which waste solutes in the patient's blood are removed by diffusion and/or convection through the semipermeable membrane of an artificial kidney or dialyzer.

Hemofiltration: cleansing of waste products or other toxins from the blood by convection across a semipermeable, high-flux membrane where fluid balance is maintained by infusion of sterile, pyrogen-free substitution fluid pre- or post-hemodialyzer.

HEPA filter: High Efficiency Particulate Air filters capable of removing 99.97% of particles 0.3 μm in diameter and may assist in controlling the transmission of airborne disease agents. These filters may be used in ventilation systems to remove particles from the air or in personal respirators to filter air before it is inhaled by the person wearing the respirator. The use of HEPA filters in ventilation systems requires expertise in installation and maintenance. To test this type of filter, 0.3 μm particles of dioctylphthalate (DOP) are drawn through the filter. Efficiency is calculated by comparing the downstream and upstream particle counts. The optimal HEPA filter allows only three particles to pass through for every 10,000 particles that are fed to the filter.

Heterotrophic (heterotroph): that which requires some nutrient components from exogenous sources. Heterotrophic bacteria cannot synthesize all of their metabolites and therefore require certain nutrients from other sources.

High-efficiency filter: a filter with a particle-removal efficiency of 90%–95%.

High flux: a type of dialyzer or hemodialysis treatment in which large molecules (>8,000 daltons [e.g., β₂ microglobulin]) are removed from blood.

High-level disinfection: a disinfection process that inactivates vegetative bacteria, mycobacteria, fungi, and viruses, but not necessarily high numbers of bacterial spores.

Housekeeping surfaces: environmental surfaces (e.g., floors, walls, ceilings, and tabletops) that are not involved in direct delivery of patient care in health-care facilities.

Hoyer lift: an apparatus that facilitates the repositioning of the non-ambulatory patient from bed to wheelchair or gurney and subsequently to therapy equipment (immersion tanks).

Hubbard tank: a tank used in hydrotherapy that may accommodate whole-body immersion (e.g., as may be indicated for burn therapy). Use of a Hubbard tank has been replaced largely by bedside post-lavage therapy for wound care management.

HVAC: Heating, Ventilation, Air Conditioning.

Iatrogenic: induced in a patient by a physician's activity, manner, or therapy. The term is used especially in reference to an infectious complication or other adverse outcome of medical treatment.

Impactor: an air-sampling device in which particles and microorganisms are directed onto a solid surface and retained there for assay.

Impingement: an air-sampling method during which particles and microorganisms are directed into a liquid and retained there for assay.

Indirect transmission: involves contact of a susceptible host with a contaminated intermediate object, usually inanimate (a fomite).

Induction unit: the terminal unit of an in-room ventilation system. Induction units take centrally conditioned air and further moderate its temperature. Induction units are not appropriate for areas with high exhaust requirements (e.g., research laboratories).

Intermediate-level disinfection: a disinfection process that inactivates vegetative bacteria, most fungi, mycobacteria, and most viruses (particularly the enveloped viruses), but does not inactivate bacterial spores.

Isoform: a possible configuration (tertiary structure) of a protein molecule. With respect to prion proteins, the molecules with large amounts of α -conformation are the normal isoform of that particular protein, whereas those prions with large amounts of β -sheet conformation are the proteins associated with the development of spongiform encephalopathy (e.g., Creutzfeldt-Jakob disease [CJD]).

Laminar flow: HEPA-filtered air that is blown into a room at a rate of 90 ± 10 feet/min in a unidirectional pattern with 100 ACH–400 ACH.

Large enveloped virus: viruses whose particle diameter is >50 nm and whose outer surface is covered by a lipid-containing structure derived from the membranes of the host cells. Examples of large enveloped viruses include influenza viruses, herpes simplex viruses, and poxviruses.

Laser plume: the transfer of electromagnetic energy into tissues which results in a release of particles, gases, and tissue debris.

Lipid-containing viruses: viruses whose particle contains lipid components. The term is generally synonymous with enveloped viruses whose outer surface is derived from host cell membranes. Lipid-containing viruses are sensitive to the inactivating effects of liquid chemical germicides.

Lithotriptors: instruments used for crushing calculi (i.e., calcified stones, and sand) in the bladder or kidneys.

Low efficiency filter: the prefilter with a particle-removal efficiency of approximately 30% through which incoming air first passes. See also Prefilter.

Low-level disinfection: a disinfection process that will inactivate most vegetative bacteria, some fungi, and some viruses, but cannot be relied upon to inactivate resistant microorganisms (e.g., mycobacteria or bacterial spores).

Makeup air: outdoor air supplied to the ventilation system to replace exhaust air.

Makeup water: a cold water supply source for a cooling tower.

Manometer: a device that measures the pressure of liquids and gases. A manometer is used to verify air filter performance by measuring pressure differentials on either side of the filter.

Membrane filtration: an assay method suitable for recovery and enumeration of microorganisms from liquid samples. This method is used when sample volume is large and anticipated microbial contamination levels are low.

Mesophilic: that which favors a moderate temperature. For mesophilic bacteria, a temperature range of

68°F–131°F (20°C–55°C) is favorable for their growth and proliferation.

Mixing box: the site where the cold and hot air streams mix in the HVAC system, usually situated close to the air outlet for the room.

Mixing faucet: a faucet that mixes hot and cold water to produce water at a desired temperature.

MMAD: Mass Median Aerodynamic Diameter. This is the unit used by ACGIH to describe the size of particles when particulate air sampling is conducted.

Moniliaceous: hyaline or brightly colored. This is a laboratory term for the distinctive characteristics of certain opportunistic fungi in culture (e.g., *Aspergillus* spp. and *Fusarium* spp.).

Monochloramine: the result of the reaction between chlorine and ammonia that contains only one chlorine atom. Monochloramine is used by municipal water systems as a water treatment.

Natural ventilation: the movement of outdoor air into a space through intentionally provided openings (i.e., windows, doors, or nonpowered ventilators).

Negative pressure: air pressure differential between two adjacent airspaces such that air flow is directed into the room relative to the corridor ventilation (i.e., room air is prevented from flowing out of the room and into adjacent areas).

Neutropenia: a medical condition in which the patient's concentration of neutrophils is substantially less than that in the normal range. Severe neutropenia occurs when the concentration is <1,000 polymorphonuclear cells/ μ L for 2 weeks or <100 polymorphonuclear cells /mL for 1 week, particularly for hematopoietic stem cell transplant (HSCT) recipients.

Noncritical devices: medical devices or surfaces that come into contact with only intact skin. The risk of infection from use of these devices is low.

Non-enveloped virus: a virus whose particle is not covered by a structure derived from a membrane of the host cell. Non-enveloped viruses have little or no lipid compounds in their biochemical composition, a characteristic that is significant to their inherent resistance to the action of chemical germicides.

Nosocomial: an occurrence, usually an infection, that is acquired in a hospital as a result of medical care.

NTM: nontuberculous mycobacteria. These organisms are also known as atypical mycobacteria, or as "Mycobacteria other than tuberculosis" (MOTT). This descriptive term refers to any of the fast- or slow-growing *Mycobacterium* spp. found in primarily in natural or man-made waters, but it excludes *Mycobacterium tuberculosis* and its variants.

Nuisance dust: generally innocuous dust, not recognized as the direct cause of serious pathological conditions.

Oocysts: a cyst in which sporozoites are formed; a reproductive aspect of the life cycle of a number of parasitic agents (e.g., *Cryptosporidium* spp., and *Cyclospora* spp.).

Outdoor air: air taken from the external atmosphere and, therefore, not previously circulated through the ventilation system.

Parallel streamlines: a unidirectional airflow pattern achieved in a laminar flow setting, characterized by little or no mixing of air.

Particulate matter (particles): a state of matter in which solid or liquid substances exist in the form of aggregated molecules or particles. Airborne particulate matter is typically in the size range of 0.01–100 μ m diameter.

Pasteurization: a disinfecting method for liquids during which the liquids are heated to 140°F (60°C) for a short time (\geq 30 mins.) to significantly reduce the numbers of pathogenic or spoilage microorganisms.

Plinth: a treatment table or a piece of equipment used to reposition the patient for treatment.

Portable room-air HEPA recirculation units: free-standing portable devices that remove airborne contaminants by recirculating air through a HEPA filter.

Positive pressure: air pressure differential between two adjacent air spaces such that air flow is directed from the room relative to the corridor ventilation (i.e., air from corridors and adjacent areas is prevented from entering the room).

Potable (drinking) water: water that is fit to drink. The microbiological quality of this water as defined by EPA microbiological standards from the Surface Water Treatment Rule: a) *Giardia lamblia*: 99.9% killed/inactivated; b) viruses: 99.9% inactivated; c) *Legionella* spp.: no limit, but if *Giardia* and viruses are inactivated, *Legionella* will also be controlled; d) heterotrophic plate count [HPC]: ≤ 500 CFU/mL; and e) $>5\%$ of water samples total coliform-positive in a month.

PPE: Personal Protective Equipment.

ppm: parts per million. The term is a measure of concentration in solution. Chlorine bleaches (undiluted) that are available in the U.S. (5.25%–6.15% sodium hypochlorite) contain approximately 50,000–61,500 parts per million of free and available chlorine.

Prefilter: the first filter for incoming fresh air in a HVAC system. This filter is approximately 30% efficient in removing particles from the air. See also Low-Efficiency Filter.

Prion: a class of agent associated with the transmission of diseases known as transmissible spongiform encephalopathies (TSEs). Prions are considered to consist of protein only, and the abnormal isoform of this protein is thought to be the agent that causes diseases such as Creutzfeldt-Jakob disease (CJD), kuru, scrapie, bovine spongiform encephalopathy (BSE), and the human version of BSE which is variant CJD (vCJD).

Product water: water produced by a water treatment system or individual component of that system.

Protective environment: a special care area, usually in a hospital, designed to prevent transmission of opportunistic airborne pathogens to severely immunosuppressed patients.

Pseudoepidemic (pseudo-outbreak): a cluster of positive microbiologic cultures in the absence of clinical disease. A pseudoepidemic usually results from contamination of the laboratory apparatus and process used to recover microorganisms.

Pyrogenic: an endotoxin burden such that a patient would receive ≥ 5 endotoxin units (EU) per kilogram of body weight per hour, thereby causing a febrile response. In dialysis this usually refers to water or dialysate having endotoxin concentrations of ≥ 5 EU/mL.

Rank order: a strategy for assessing overall indoor air quality and filter performance by comparing airborne particle counts from lowest to highest (i.e., from the best filtered air spaces to those with the least filtration).

RAPD: a method of genotyping microorganisms by randomly amplified polymorphic DNA. This is one version of the polymerase chain reaction method.

Recirculated air: air removed from the conditioned space and intended for reuse as supply air.

Relative humidity: the ratio of the amount of water vapor in the atmosphere to the amount necessary for saturation at the same temperature. Relative humidity is expressed in terms of percent and measures the percentage of saturation. At 100% relative humidity, the air is saturated. The relative humidity decreases when the temperature is increased without changing the amount of moisture in the air.

Reprocessing (of medical instruments): the procedures or steps taken to make a medical instrument safe for use on the next patient. Reprocessing encompasses both cleaning and the final or terminal step (i.e., sterilization or disinfection) which is determined by the intended use of the instrument according to the Spaulding classification.

Residuals: the presence and concentration of a chemical in media (e.g., water) or on a surface after the chemical has been added.

Reservoir: a nonclinical source of infection.

Respirable particles: those particles that penetrate into and are deposited in the nonciliated portion of the lung. Particles $>10 \mu\text{m}$ in diameter are not respirable.

Return air: air removed from a space to be then recirculated.

Reverse osmosis (RO): an advanced method of water or wastewater treatment that relies on a semi-permeable membrane to separate waters from pollutants. An external force is used to reverse the normal osmotic process resulting in the solvent moving from a solution of higher concentration to one of lower concentration.

Riser: water piping that connects the circulating water supply line, from the level of the base of the tower or supply header, to the tower's distribution system.

RODAC: Replicate Organism Direct Agar Contact. This term refers to a nutrient agar plate whose convex agar surface is directly pressed onto an environmental surface for the purpose of microbiologic sampling of that surface.

Room-air HEPA recirculation systems and units: devices (either fixed or portable) that remove airborne contaminants by recirculating air through a HEPA filter.

Routine sampling: environmental sampling conducted without a specific, intended purpose and with no action plan dependent on the results obtained.

Sanitizer: an agent that reduces microbial contamination to safe levels as judged by public health standards or requirements.

Saprophytic: a naturally-occurring microbial contaminant.

Sedimentation: the act or process of depositing sediment from suspension in water. The term also refers to the process whereby solids settle out of wastewater by gravity during treatment.

Semicritical devices: medical devices that come into contact with mucous membranes or non-intact skin.

Service animal: any animal individually trained to do work or perform tasks for the benefit of a person with a disability.

Shedding: the generation and dispersion of particles and spores by sources within the patient area, through activities such as patient movement and airflow over surfaces.

Single-pass ventilation: ventilation in which 100% of the air supplied to an area is exhausted to the outside.

Small, non-enveloped viruses: viruses whose particle diameter is <50 nm and whose outer surface is the protein of the particle itself and not that of host cell membrane components. Examples of small, non-enveloped viruses are polioviruses and hepatitis A virus.

Spaulding Classification: the categorization of inanimate medical device surfaces in the medical environment as proposed in 1972 by Dr. Earle Spaulding. Surfaces are divided into three general categories, based on the theoretical risk of infection if the surfaces are contaminated at time of use. The categories are “critical,” “semicritical,” and “noncritical.”

Specific humidity: the mass of water vapor per unit mass of moist air. It is expressed as grains of water per pound of dry air, or pounds of water per pound of dry air. The specific humidity changes as moisture is added or removed. However, temperature changes do not change the specific humidity unless the air is cooled below the dew point.

Splatter: visible drops of liquid or body fluid that are expelled forcibly into the air and settle out quickly, as distinguished from particles of an aerosol which remain airborne indefinitely.

Steady state: the usual state of an area.

Sterilization: the use of a physical or chemical procedure to destroy all microbial life, including large numbers of highly-resistant bacterial endospores.

Stop valve: a valve that regulates the flow of fluid through a pipe. The term may also refer to a faucet.

Substitution fluid: fluid that is used for fluid management of patients receiving hemodiafiltration. This fluid can be prepared on-line at the machine through a series of ultrafilters or with the use of sterile peritoneal dialysis fluid.

Supply air: air that is delivered to the conditioned space and used for ventilation, heating, cooling, humidification, or dehumidification.

Tensile strength: the resistance of a material to a force tending to tear it apart, measured as the maximum tension the material can withstand without tearing.

Therapy animal: an animal (usually a personal pet) that, with their owners or handlers, provide supervised, goal-directed intervention to clients in hospitals, nursing homes, special-population schools, and other treatment sites.

Thermophilic: capable of growing in environments warmer than body temperature.

Thermotolerant: capable of withstanding high temperature conditions.

TLV®: an exposure level under which most people can work consistently for 8 hours a day, day after day, without adverse effects. The term is used by the ACGIH to designate degree of exposure to

contaminants. TLV® can be expressed as approximate milligrams of particulate per cubic meter of air (mg/m^3). TLVs® are listed as either an 8-hour TWA (time weighted average) or a 15-minute STEL (short term exposure limit).

TLV-TWA: Threshold Limit Value-Time Weighted Average. The term refers to the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed repeatedly, day after day, without adverse effects. The TLV-TWA for “particulates (insoluble) not otherwise classified” (PNOC) - (sometimes referred to as nuisance dust) - are those particulates containing no asbestos and <1% crystalline silica. A TLV-TWA of $10 \text{ mg}/\text{m}^3$ for inhalable particulates and a TLV-TWA of $3 \text{ mg}/\text{m}^3$ for respirable particulates (particulates $\leq 5 \mu\text{m}$ in aerodynamic diameter) have been established.

Total suspended particulate matter: the mass of particles suspended in a unit of volume of air when collected by a high-volume air sampler.

Transient: a change in the condition of the steady state that takes a very short time compared with the steady state. Opening a door, and shaking bed linens are examples of transient activities.

TWA: average exposure for an individual over a given working period, as determined by sampling at given times during the period. TWA is usually presented as the average concentration over an 8-hour workday for a 40-hour workweek.

Ultraclean air: air in laminar flow ventilation that has also passed through a bank of HEPA filters.

Ultrafilter: a membrane filter with a pore size in the range of $0.001\text{--}0.05 \mu\text{m}$, the performance of which is usually rated in terms of a nominal molecular weight cut-off (defined as the smallest molecular weight species for which the filter membrane has more than 90% rejection).

Ultrafiltered dialysate: the process by which dialysate is passed through a filter having a molecular weight cut-off of approximately 1 kilodalton for the purpose of removing bacteria and endotoxin from the bath.

Ultraviolet germicidal irradiation (UVGI): the use of ultraviolet radiation to kill or inactivate microorganisms.

Ultraviolet germicidal irradiation lamps: lamps that kill or inactivate microorganisms by emitting ultraviolet germicidal radiation, predominantly at a wavelength of 254 nm. UVGI lamps can be used in ceiling or wall fixtures or within air ducts of ventilation systems.

Vapor pressure: the pressure exerted by free molecules at the surface of a solid or liquid. Vapor pressure is a function of temperature, increasing as the temperature rises.

Vegetative bacteria: bacteria that are actively growing and metabolizing, as opposed to a bacterial state of quiescence that is achieved when certain bacteria (gram-positive bacilli) convert to spores when the environment can no longer support active growth.

Vehicle: any object, person, surface, fomite, or media that may carry and transfer infectious microorganisms from one site to another.

Ventilation: the process of supplying and removing air by natural or mechanical means to and from any space. Such air may or may not be conditioned.

Ventilation air: that portion of the supply air consisting of outdoor air plus any recirculated air that has been treated for the purpose of maintaining acceptable indoor air quality.

Ventilation, dilution: an engineering control technique to dilute and remove airborne contaminants by the flow of air into and out of an area. Air that contains droplet nuclei is removed and replaced by contaminant-free air. If the flow is sufficient, droplet nuclei become dispersed, and their concentration in the air is diminished.

Ventilation, local exhaust: ventilation used to capture and removed airborne contaminants by enclosing the contaminant source (the patient) or by placing an exhaust hood close to the contaminant source.

v/v: volume to volume. This term is an expression of concentration of a percentage solution when the principle component is added as a liquid to the diluent.

w/v: weight to volume. This term is an expression of concentration of a percentage solution when the principle component is added as a solid to the diluent.

Weight-arrestance: a measure of filter efficiency, used primarily when describing the performance of low- and medium-efficiency filters. The measurement of weight-arrestance is performed by feeding a standardized synthetic dust to the filter and weighing the fraction of the dust removed.

Appendix B. Air

1. Airborne Contaminant Removal

Table B.1. Air changes/hour (ACH) and time required for airborne-contaminant removal efficiencies of 99% and 99.9%*

ACH+ § ¶	Time (mins.) required for removal:	
	99% efficiency	99.9% efficiency
2	138	207
4	69	104
6	46	69
8	35	52
10	28	41
12	23	35
15	18	28
20	14	21
50	6	8

* This table is revised from Table S3-1 in reference 4 and has been adapted from the formula for the rate of purging airborne contaminants presented in reference 1435.

+ Shaded entries denote frequently cited ACH for patient-care areas.

§ Values were derived from the formula:

$$t_2 - t_1 = -[\ln(C_2 / C_1) / (Q / V)] \times 60, \text{ with } t_1 = 0 \text{ and where}$$

t_1 = initial timepoint in minutes

t_2 = final timepoint in minutes

C_1 = initial concentration of contaminant

C_2 = final concentration of contaminant

$C_2 / C_1 = 1 - (\text{removal efficiency} / 100)$

Q = air flow rate in cubic feet/hour

V = room volume in cubic feet

$Q / V = \text{ACH}$

¶ Values apply to an empty room with no aerosol-generating source. With a person present and generating aerosol, this table would not apply. Other equations are available that include a constant generating source. However, certain diseases (e.g., infectious tuberculosis) are not likely to be aerosolized at a constant rate. The times given assume perfect mixing of the air within the space (i.e., mixing factor = 1). However, perfect mixing usually does not occur. Removal times will be longer in rooms or areas with imperfect mixing or air stagnation.²¹³ Caution should be exercised in using this table in such situations. For booths or other local ventilation enclosures, manufacturers' instructions should be consulted.

2. Air Sampling for Aerosols Containing Legionellae

Air sampling is an insensitive means of detecting *Legionella pneumophila*, and is of limited practical value in environmental sampling for this pathogen. In certain instances, however, it can be used to a) demonstrate the presence of legionellae in aerosol droplets associated with suspected bacterial

reservoirs; b) define the role of certain devices [e.g., showers, faucets, decorative fountains, or evaporate condensers] in disease transmission; and c) quantitate and determine the size of the droplets containing legionellae.¹⁴³⁶ Stringent controls and calibration are necessary when sampling is used to determine particle size and numbers of viable bacteria.¹⁴³⁷ Samplers should be placed in locations where human exposure to aerosols is anticipated, and investigators should wear a NIOSH-approved respirator (e.g., N95 respirator) if sampling involves exposure to potentially infectious aerosols.

Methods used to sample air for legionellae include impingement in liquid, impaction on solid medium, and sedimentation using settle plates.¹⁴³⁶ The Chemical Corps.-type all-glass impingers (AGI) with the stem 30 mm from the bottom of the flask have been used successfully to sample for legionellae.¹⁴³⁶ Because of the velocity at which air samples are collected, clumps tend to become fragmented, leading to a more accurate count of bacteria present in the air. The disadvantages of this method are a) the velocity of collection tends to destroy some vegetative cells; b) the method does not differentiate particle sizes; and c) AGIs are easily broken in the field. Yeast extract broth (0.25%) is the recommended liquid medium for AGI sampling of legionellae;¹⁴³⁷ standard methods for water samples can be used to culture these samples.

Andersen samplers are viable particle samplers in which particles pass through jet orifices of decreasing size in cascade fashion until they impact on an agar surface.¹²¹⁸ The agar plates are then removed and incubated. The stage distribution of the legionellae should indicate the extent to which the bacteria would have penetrated the respiratory system. The advantages of this sampling method are a) the equipment is more durable during use; b) the sampler can determine the number and size of droplets containing legionellae; c) the agar plates can be placed directly in an incubator with no further manipulations; and d) both selective and nonselective BCYE agar can be used. If the samples must be shipped to a laboratory, they should be packed and shipped without refrigeration as soon as possible.

3. Calculation of Air Sampling Results

Assuming that each colony on the agar plate is the growth from a single bacteria-carrying particle, the contamination of the air being sampled is determined from the number of colonies counted. The airborne microorganisms may be reported in terms of the number per cubic foot of air sampled. The following formulas can be applied to convert colony counts to organisms per cubic foot of air sampled.¹²¹⁸

For solid agar impactor samplers:

$$C / (R \times P) = N \quad \text{where}$$

N = number of organisms collected per cubic foot of air sampled
 C = total plate count
 R = airflow rate in cubic feet per minute
 P = duration of sampling period in minutes

For liquid impingers:

$$(C \times V) / (Q \times P \times R) = N \quad \text{where}$$

C = total number of colonies from all aliquots plated
 V = final volume in mL of collecting media
 Q = total number of mL plated
 P, R, and N are defined as above

Area designation	Air movement relationship to adjacent area ²	Minimum air changes of outdoor air per hour ³	Minimum total air changes per hour ^{4, 5}	All air exhausted directly to outdoors ⁶	Recirculated by means of room units ⁷	Relative humidity ⁸ (%)	Design temperature ⁹ (degrees F [C])
<u>Ancillary</u>							
Radiology¹⁹							
X-ray (surgical/critical care and catheterization)	Out	3	15	–	No	30-60	70–75 (21–24)
X-ray (diagnostic & treatment)	–	–	6	–	–	–	75 (24)
Darkroom	In	–	10	Yes	No	–	–
Laboratory							
General ¹⁹	–	–	6	–	–	–	75 (24)
Biochemistry ¹⁹	Out	–	6	–	No	–	75 (24)
Cytology	In	–	6	Yes	No	–	75 (24)
Glass washing	In	–	10	Yes	–	–	–
Histology	In	–	6	Yes	No	–	75 (24)
Microbiology ¹⁹	In	–	6	Yes	No	–	75 (24)
Nuclear medicine	In	–	6	Yes	No	–	75 (24)
Pathology	In	–	6	Yes	No	–	75 (24)
Serology	Out	–	6	–	No	–	75 (24)
Sterilizing	In	–	10	Yes	–	–	–
Autopsy room ¹¹	–	–	12	Yes	No	–	–
Nonrefrigerated body-holding room	In	–	10	Yes	–	–	70 (21)
Pharmacy	Out	–	4	–	–	–	–
<u>Diagnostic and treatment</u>							
Examination room	–	–	6	–	–	–	75 (24)
Medication room	Out	–	4	–	–	–	–
Treatment room	–	–	6	–	–	–	75 (24)
Physical therapy and hydrotherapy	In	–	6	–	–	–	75 (24)
Soiled workroom or soiled holding	In	–	10	Yes	No	–	–
Clean workroom or clean holding	Out	–	4	–	–	–	–
<u>Sterilizing and supply</u>							
ETO-sterilizer room	In	–	10	Yes	No	30-60	75 (24)
Sterilizer equipment room	In	–	10	Yes	–	–	–
Central medical and surgical supply							
Soiled or decontamination room	In	–	6	Yes	No	–	68–73 (20–23)
Clean workroom	Out	–	4	–	No	30-60	75 (24)
Sterile storage	Out	–	4	–	–	(Max.) 70	–

Area designation	Air movement relationship to adjacent area ²	Minimum air changes of outdoor air per hour ³	Minimum total air changes per hour ^{4, 5}	All air exhausted directly to outdoors ⁶	Recirculated by means of room units ⁷	Relative humidity ⁸ (%)	Design temperature ⁹ (degrees F [C])
Service							
Food preparation center ²⁰	–	–	10	–	No	–	–
Ware washing	In	–	10	Yes	No	–	–
Dietary day storage	In	–	2	–	–	–	–
Laundry, general	–	–	10	Yes	–	–	–
Soiled linen (sorting and storage)	In	–	10	Yes	No	–	–
Clean linen storage	Out	–	2	–	–	–	–
Soiled linen and trash chute room	In	–	10	Yes	No	–	–
Bedpan room	In	–	10	Yes	–	–	–
Bathroom	In	–	10	–	–	–	75 (24)
Janitor's closet	In	–	10	Yes	No	–	–

Notes:

1. The ventilation rates in this table cover ventilation for comfort, as well as for asepsis and odor control in areas of acute care hospitals that directly affect patient care and are determined based on health-care facilities being predominantly “No Smoking” facilities. Where smoking may be allowed, ventilation rates will need adjustment. Areas where specific ventilation rates are not given in the table shall be ventilated in accordance with ASHRAE Standard 62, *Ventilation for Acceptable Indoor Air Quality*, and ASHRAE *Handbook - HVAC Applications*. Specialized patient care areas, including organ transplant units, burn units, specialty procedure rooms, etc., shall have additional ventilation provisions for air quality control as may be appropriate. OSHA standards and/or NIOSH criteria require special ventilation requirements for employee health and safety within health-care facilities.
2. Design of the ventilation system shall provide air movement which is generally from clean to less clean areas. If any form of variable air volume or load shedding system is used for energy conservation, it must not compromise the corridor-to-room pressure balancing relationships or the minimum air changes required by the table.
3. To satisfy exhaust needs, replacement air from the outside is necessary. Table B2 does not attempt to describe specific amounts of outside air to be supplied to individual spaces except for certain areas such as those listed. Distribution of the outside air, added to the system to balance required exhaust, shall be as required by good engineering practice. Minimum outside air quantities shall remain constant while the system is in operation.
4. Number of air changes may be reduced when the room is unoccupied if provisions are made to ensure that the number of air changes indicated is reestablished any time the space is being utilized. Adjustments shall include provisions so that the direction of air movement shall remain the same when the number of air changes is reduced. Areas not indicated as having continuous directional control may have ventilation systems shut down when space is unoccupied and ventilation is not otherwise needed, if the maximum infiltration or exfiltration permitted in Note 2 is not exceeded and if adjacent pressure balancing relationships are not compromised. Air quantity calculations must account for filter loading such that the indicated air change rates are provided up until the time of filter change-out.
5. Air change requirements indicated are minimum values. Higher values should be used when required to maintain indicated room conditions (temperature and humidity), based on the cooling load of the space (lights, equipment, people, exterior walls and windows, etc.).

6. Air from areas with contamination and/or odor problems shall be exhausted to the outside and not recirculated to other areas. Note that individual circumstances may require special consideration for air exhaust to the outside, (e.g., in intensive care units in which patients with pulmonary infection are treated) and rooms for burn patients.
7. Recirculating room HVAC units refer to those local units that are used primarily for heating and cooling of air, and not disinfection of air. Because of cleaning difficulty and potential for buildup of contamination, recirculating room units shall not be used in areas marked “No.” However, for airborne infection control, air may be recirculated within individual isolation rooms if HEPA filters are used. Isolation and intensive care unit rooms may be ventilated by reheat induction units in which only the primary air supplied from a central system passes through the reheat unit. Gravity-type heating or cooling units such as radiators or convectors shall not be used in operating rooms and other special care areas. See this table’s Appendix I for a description of recirculation units to be used in isolation rooms (A7).
8. The ranges listed are the minimum and maximum limits where control is specifically needed. The maximum and minimum limits are not intended to be independent of a space’s associated temperature. The humidity is expected to be at the higher end of the range when the temperature is also at the higher end, and vice versa.
9. Where temperature ranges are indicated, the systems shall be capable of maintaining the rooms at any point within the range during normal operation. A single figure indicates a heating or cooling capacity of at least the indicated temperature. This is usually applicable when patients may be undressed and require a warmer environment. Nothing in these guidelines shall be construed as precluding the use of temperatures lower than those noted when the patients' comfort and medical conditions make lower temperatures desirable. Unoccupied areas such as storage rooms shall have temperatures appropriate for the function intended.
10. National Institute for Occupational Safety and Health (NIOSH) criteria documents regarding “Occupational Exposure to Waste Anesthetic Gases and Vapors,” and “Control of Occupational Exposure to Nitrous Oxide” indicate a need for both local exhaust (scavenging) systems and general ventilation of the areas in which the respective gases are utilized.
11. Differential pressure shall be a minimum of 0.01" water gauge (2.5 Pa). If alarms are installed, allowances shall be made to prevent nuisance alarms of monitoring devices.
12. Some surgeons may require room temperatures which are outside of the indicated range. All operating room design conditions shall be developed in consultation with surgeons, anesthesiologists, and nursing staff.
13. The term “trauma room” as used here is the operating room space in the emergency department or other trauma reception area that is used for emergency surgery. The “first aid room” and/or “emergency room” used for initial treatment of accident victims may be ventilated as noted for the “treatment room.” Treatment rooms used for bronchoscopy shall be treated as Bronchoscopy rooms. Treatment rooms used for cryosurgery procedures with nitrous oxide shall contain provisions for exhausting waste gases.
14. In a ventilation system that recirculates air, HEPA filters can be used in lieu of exhausting the air from these spaces to the outside. In this application, the return air shall be passed through the HEPA filters before it is introduced into any other spaces.
15. If it is not practical to exhaust the air from the airborne infection isolation room to the outside, the air may be returned through HEPA filters to the air-handling system exclusively serving the isolation room.
16. Total air changes per room for patient rooms, labor/delivery/recovery rooms, and labor/delivery/recovery/postpartum rooms may be reduced to 4 when supplemental heating and/or cooling systems (radiant heating and cooling, baseboard heating, etc.) are used.
17. The protective environment airflow design specifications protect the patient from common environmental airborne infectious microbes (i.e., *Aspergillus* spores). These special ventilation areas shall be designed to provide directed airflow from the cleanest patient care area to less clean areas. These rooms shall be protected with HEPA filters at 99.97 percent efficiency for a 0.3 µm sized particle in the supply airstream. These interrupting filters protect patient rooms from maintenance-derived release of environmental microbes from the ventilation system components. Recirculation HEPA filters can be used to increase the equivalent room air exchanges. Constant volume airflow is required for consistent ventilation for the protected environment. If the facility determines that airborne infection isolation is necessary for protective environment patients, an anteroom should be

provided. Rooms with reversible airflow provisions for the purpose of switching between protective environment and airborne infection isolation functions are not acceptable.

18. The infectious disease isolation room described in these guidelines is to be used for isolating the airborne spread of infectious diseases, such as measles, varicella, or tuberculosis. The design of airborne infection isolation (AII) rooms should include the provision for normal patient care during periods not requiring isolation precautions. Supplemental recirculating devices may be used in the patient room to increase the equivalent room air exchanges; however, such recirculating devices do not provide the outside air requirements. Air may be recirculated within individual isolation rooms if HEPA filters are used. Rooms with reversible airflow provisions for the purpose of switching between protective environment and AII functions are not acceptable.

19. When required, appropriate hoods and exhaust devices for the removal of noxious gases or chemical vapors shall be provided (see Section 7.31.D14 and 7.31.D15 in the AIA guideline [reference 120] and NFPA 99).

20. Food preparation centers shall have ventilation systems whose air supply mechanisms are interfaced appropriately with exhaust hood controls or relief vents so that exfiltration or infiltration to or from exit corridors does not compromise the exit corridor restrictions of NFPA 90A, the pressure requirements of NFPA 96, or the maximum defined in the table. The number of air changes may be reduced or varied to any extent required for odor control when the space is not in use. See Section 7.31.D1.p in the AIA guideline (reference 120).

Appendix I:

A7. Recirculating devices with HEPA filters may have potential uses in existing facilities as interim, supplemental environmental controls to meet requirements for the control of airborne infectious agents. Limitations in design must be recognized. The design of either portable or fixed systems should prevent stagnation and short circuiting of airflow. The supply and exhaust locations should direct clean air to areas where health-care workers are likely to work, across the infectious source, and then to the exhaust, so that the health-care worker is not in position between the infectious source and the exhaust location. The design of such systems should also allow for easy access for scheduled preventative maintenance and cleaning.

A11. The verification of airflow direction can include a simple visual method such as smoke trail, ball-in-tube, or flutterstrip. These devices will require a minimum differential air pressure to indicate airflow direction.

Table B.3. Pressure relationships and ventilation of certain areas of nursing facilities¹

Notes: This table is Table 8.1 in the AIA guidelines, 2001 edition. Superscripts used in this table refer to notes following the table.

Area designation	Air movement relationship to adjacent area ²	Minimum air changes of outdoor air per hour ³	Minimum total air changes per hour ⁴	All air exhausted directly to outdoors ⁵	Recirculated by means of room units ⁶	Relative humidity ⁷ (%)	Design temperature ⁸ (degrees F [C])
Resident room	–	2	2	–	–	⁹	70–75 (21–24)
Resident unit corridor	–	–	4	–	–	⁹	
Resident gathering areas	–	4	4	–	–	–	–
Toilet room	In	–	10	Yes	No	–	–
Dining rooms	–	2	4	–	–	–	75 (24)
Activity rooms, if provided	–	4	4	–	–	–	–
Physical therapy	In	2	6	–	–	–	75 (24)
Occupational therapy	In	2	6	–	–	–	75.(24)
Soiled workroom or soiled holding	In	2	10	Yes	No	–	–
Clean workroom or clean holding	Out	2	4	–	–	(Max. 70)	75 (24)
Sterilizer exhaust room	In	–	10	Yes	No	–	–
Linen and trash chute room, if provided	In	–	10	Yes	No	–	–
Laundry, general, if provided	–	2	10	Yes	No	–	–
Soiled linen sorting and storage	In	–	10	Yes	No	–	–
Clean linen storage	Out	–	2	Yes	No	–	–
Food preparation facilities ¹⁰	–	2	10	Yes	No	–	–
Dietary warewashing	In	–	10	Yes	No	–	–
Dietary storage areas	–	–	2	Yes	No	–	–
Housekeeping rooms	In	–	10	Yes	No	–	–
Bathing rooms	In	–	10	Yes	No	–	75 (24)

Notes:

1. The ventilation rates in this table cover ventilation for comfort, as well as for asepsis and odor control in areas of nursing facilities that directly affect resident care and are determined based on nursing facilities being predominantly “No Smoking” facilities. Where smoking may be allowed, ventilation rates will need adjustment. Areas where specific ventilation rates are not given in the table shall be ventilated in accordance with ASHRAE Standard 62, *Ventilation for Acceptable Indoor Air Quality*, and ASHRAE *Handbook - HVAC Applications*. OSHA standards and/or NIOSH criteria require special ventilation requirements for employee health and safety within nursing facilities.

2. Design of the ventilation system shall, insofar as possible, provide that air movement is from clean to less clean areas. However, continuous compliance may be impractical with full utilization of some forms of variable air volume and load shedding systems that may be used for energy conservation. Areas that do require positive and continuous control are noted with “Out” or “In” to indicate the required direction of air movement in relation to the space named. Rate of air movement may, of course, be varied as needed

within the limits required for positive control. Where indication of air movement direction is enclosed in parentheses, continuous directional control is required only when the specialized equipment or device is in use or where room use may otherwise compromise the intent of movement from clean to less clean. Air movement for rooms with dashes and nonpatient areas may vary as necessary to satisfy the requirements of those spaces. Additional adjustments may be needed when space is unused or unoccupied and air systems are deenergized or reduced.

3. To satisfy exhaust needs, replacement air from outside is necessary. Table B.3 does not attempt to describe specific amounts of outside air to be supplied to individual spaces except for certain areas such as those listed. Distribution of the outside air, added to the system to balance required exhaust, shall be as required by good engineering practice.
4. Number of air changes may be reduced when the room is unoccupied if provisions are made to ensure that the number of air changes indicated is reestablished any time the space is being utilized. Adjustments shall include provisions so that the direction of air movement shall remain the same when the number of air changes is reduced. Areas not indicated as having continuous directional control may have ventilation systems shut down when space is unoccupied and ventilation is not otherwise needed.
5. Air from areas with contamination and/or odor problems shall be exhausted to the outside and not recirculated to other areas. Note that individual circumstances may require special consideration for air exhaust to outside.
6. Because of cleaning difficulty and potential for buildup of contamination, recirculating room units shall not be used in areas marked "No." Isolation rooms may be ventilated by reheat induction units in which only the primary air supplied from a central system passes through the reheat unit. Gravity-type heating or cooling units such as radiators or convectors shall not be used in special care areas.
7. The ranges listed are the minimum and maximum limits where control is specifically needed. See A8.31.D in the AIA guideline (reference 120) for additional information.
8. Where temperature ranges are indicated, the systems shall be capable of maintaining the rooms at any point within the range. A single figure indicates a heating or cooling capacity of at least the indicated temperature. This is usually applicable where residents may be undressed and require a warmer environment. Nothing in these guidelines shall be construed as precluding the use of temperatures lower than those noted when the residents' comfort and medical conditions make lower temperatures desirable. Unoccupied areas such as storage rooms shall have temperatures appropriate for the function intended.
9. See A8.31.D1 in the AIA guideline (reference 120).
10. Food preparation facilities shall have ventilation systems whose air supply mechanisms are interfaced appropriately with exhaust hood controls or relief vents so that exfiltration or infiltration to or from exit corridors does not compromise the exit corridor restrictions of NFPA 90A, the pressure requirements of NFPA 96, or the maximum defined in the table. The number of air changes may be reduced or varied to any extent required for odor control when the space is not in use.

Table B.4. Filter efficiencies for central ventilation and air conditioning systems in general hospitals*

Note: This table is Table 7.3 in the AIA guidelines, 2001 edition.

Area designation	Number of filter beds	Filter bed No.1 (%)	Filter bed No. 2 (%)
All areas for inpatient care, treatment, and diagnosis, and those areas providing direct service or clean supplies, such as sterile and clean processing, etc.	2	30	90
Protective environment room	2	30	99.97
Laboratories	1	80	–
Administrative, bulk storage, soiled holding areas, food preparation areas, and laundries	1	30	–

* Additional roughing or prefilters should be considered to reduce maintenance required for filters with efficiency higher than 75 percent. The filtration efficiency ratings are based on average dust sopt efficiency per ASHRAE 52.1–1992.

Table B.5. Filter efficiencies for central ventilation and air conditioning systems in outpatient facilities*

Note: This table is Table 9.1 in the AIA guidelines, 2001 edition.

Area designation	Number of filter beds	Filter bed No. 1 (%)	Filter bed No. 2+ (%)
All areas for patient care, treatment, and/or diagnosis, and those areas providing direct service or clean supplies such as sterile and clean processing, etc.	2	30	90
Laboratories	1	80	–
Administrative, bulk storage, soiled holding areas, food preparation areas, and laundries	1	30	–

* Additional roughing or prefilters should be considered to reduce maintenance required for main filters. The filtration efficiency ratings are based on dust spot efficiency per ASHRAE 52.1–1992.

+ These requirements do not apply to small primary (e.g., neighborhood) outpatient facilities or outpatient facilities that do not perform invasive applications or procedures.

Table B.6. Filter efficiencies for central ventilation and air conditioning systems in nursing facilities

Note: This table is Table 8.2 in the AIA guidelines, 2001 edition.

Area designation	Minimum number of filter beds	Filter bed No. 1 (%)*	Filter bed No. 2 (%)*
All areas for inpatient care, treatment, and/or diagnosis, and those areas providing direct service or clean supplies	2	30	80
Administrative, bulk storage, soiled holding, laundries, and food preparation areas	1	30	–

* The filtration efficiency ratings are based on average dust spot efficiency as per ASHRAE 52.1–1992.

Table B.7. Filter efficiencies for central ventilation and air conditioning systems in psychiatric hospitals

Note: This table is Table 11.1 in the AIA guidelines, 2001 edition.

Area designation	Minimum number of filter beds	Filter bed No. 1 (%)*	Filter bed No. 2 (%)*
All areas for inpatient care, treatment, and diagnosis, and those areas providing direct services	2	30	90
Administrative, bulk storage, soiled holding, laundries, and food preparation areas	1	30	–

* The filtration efficiency ratings are based on average dust spot efficiency as per ASHRAE 52.1–1992.

Appendix C. Water

1. Biofilms

Microorganisms have a tendency to associate with and stick to surfaces. These adherent organisms can initiate and develop biofilms, which are comprised of cells embedded in a matrix of extracellularly produced polymers and associated abiotic particles.¹⁴³⁸ It is inevitable that biofilms will form in most water systems. In the health-care facility environment, biofilms may be found in the potable water supply piping, hot water tanks, air conditioning cooling towers, or in sinks, sink traps, aerators, or shower heads. Biofilms, especially in water systems, are not present as a continuous slime or film, but

are more often scanty and heterogeneous in nature.¹⁴³⁹ Biofilms may form under stagnant as well as flowing conditions, so storage tanks, in addition to water system piping, may be vulnerable to the development of biofilm, especially if water temperatures are low enough to allow the growth of thermophilic bacteria (e.g., *Legionella* spp.). Favorable conditions for biofilm formation are present if these structures and equipment are not cleaned for extended periods of time.¹⁴⁴⁰

Algae, protozoa, and fungi may be present in biofilms, but the predominant microorganisms of water system biofilms are gram-negative bacteria. Although most of these organisms will not normally pose a problem for healthy individuals, certain biofilm bacteria (e.g., *Pseudomonas aeruginosa*, *Klebsiella* spp., *Pantoea agglomerans*, and *Enterobacter cloacae*) all may be agents for opportunistic infections for immunocompromised individuals.^{1441, 1442} These biofilm organisms may easily contaminate indwelling medical devices or intravenous (IV) fluids, and they could be transferred on the hands of health-care workers.^{1441–1444} Biofilms may potentially provide an environment for the survival of pathogenic organisms, such as *Legionella pneumophila* and *E. coli* O157:H7. Although the association of biofilms and medical devices provides a plausible explanation for a variety of health-care-associated infections, it is not clear how the presence of biofilms in the water system may influence the rates of health-care-associated waterborne infection.

Organisms within biofilms behave quite differently than their planktonic (i.e., free floating) counterparts. Research has shown that biofilm-associated organisms are more resistant to antibiotics and disinfectants than are planktonic organisms, either because the cells are protected by the polymer matrix, or because they are physiologically different.^{1445–1450} Nevertheless, municipal water utilities attempt to maintain a chlorine residual in the distribution system to discourage microbiological growth. Though chlorine in its various forms is a proven disinfectant, it has been shown to be less effective against biofilm bacteria.¹⁴⁴⁸ Higher levels of chlorine for longer contact times are necessary to eliminate biofilms.

Routine sampling of health-care facility water systems for biofilms is not warranted. If an epidemiologic investigation points to the water supply system as a possible source of infection, then water sampling for biofilm organisms should be considered so that prevention and control strategies can be developed. An established biofilm is difficult to remove totally in existing piping. Strategies to remediate biofilms in a water system would include flushing the system piping, hot water tank, dead legs, and those areas of the facility's water system subject to low or intermittent flow. The benefits of this treatment would include a) elimination of corrosion deposits and sludge from the bottom of hot water tanks, b) removal of biofilms from shower heads and sink aerators, and c) circulation of fresh water containing elevated chlorine residuals into the health-care facility water system.

The general strategy for evaluating water system biofilm depends on a comparison of the bacteriological quality of the incoming municipal water and that of water sampled from within facility's distribution system. Heterotrophic plate counts and coliform counts, both of which are routinely run by the municipal water utility, will at least provide an indication of the potential for biofilm formation. Heterotrophic plate count levels in potable water should be <500 CFU/mL. These levels may increase on occasion, but counts consistently >500 CFU/mL would indicate a general decrease in water quality. A direct correlation between heterotrophic plate count and biofilm levels has been demonstrated.¹⁴⁵⁰ Therefore, an increase in heterotrophic plate count would suggest a greater rate and extent of biofilm formation in a health-care facility water system. The water supplied to the facility should also contain <1 coliform bacteria/100 mL. Coliform bacteria are organisms whose presence in the distribution system could indicate fecal contamination. It has been shown that coliform bacteria can colonize biofilms within drinking water systems. Intermittant contamination of a water system with these organisms could lead to colonization of the system.

Water samples can be collected from throughout the health-care facility system, including both hot and cold water sources; samples should be cultured by standard methods.⁹⁴⁵ If heterotrophic plate counts in samples from the facility water system are higher than those from samples collected at the point of water entry to the building, it can be concluded that the facility water quality has diminished. If biofilms are detected in the facility water system and determined by an epidemiologic and environmental investigation to be a reservoir for health-care-associated pathogens, the municipal water supplier could be contacted with a request to provide higher chlorine residuals in the distribution system, or the health-care facility could consider installing a supplemental chlorination system.

Sample collection sites for biofilm in health-care facilities include a) hot water tanks; b) shower heads; and c) faucet aerators, especially in immunocompromised patient-care areas. Swabs should be placed into tubes containing phosphate buffered water, pH 7.2 or phosphate buffered saline, shipped to the laboratory under refrigeration and processed within 24 hrs. of collection. Samples are suspended by vortexing with sterile glass beads and plated onto a nonselective medium (e.g., Plate Count Agar or R2A medium) and selective media (e.g., media for *Legionella* spp. isolation) after serial dilution. If the plate counts are elevated above levels in the water (i.e. comparing the plate count per square centimeter of swabbed surface to the plate count per milliliter of water), then biofilm formation can be suspected. In the case of an outbreak, it would be advisable to isolate organisms from these plates to determine whether the suspect organisms are present in the biofilm or water samples and compare them to the organisms isolated from patient specimens.

2. Water and Dialysate Sampling Strategies in Dialysis

In order to detect the low, total viable heterotrophic plate counts outlined by the current AAMI standards for water and dialysate in dialysis settings, it is necessary to use standard quantitative culture techniques with appropriate sensitivity levels.^{792, 832, 833} The membrane filter technique is particularly suited for this application because it permits large volumes of water to be assayed.^{792, 834} Since the membrane filter technique may not be readily available in clinical laboratories, the spread plate assay can be used as an alternative.⁸³⁴ If the spread plate assay is used, however, the standard prohibits the use of a calibrated loop when applying sample to the plate.⁷⁹² The prohibition is based on the low sensitivity of the calibrated loop. A standard calibrated loop transfers 0.001 mL of sample to the culture medium, so that the minimum sensitivity of the assay is 1,000 CFU/mL. This level of sensitivity is unacceptable when the maximum allowable limit for microorganisms is 200 CFU/mL. Therefore, when the spread plate method is used, a pipette must be used to place 0.1–0.5 mL of water on the culture medium.

The current AAMI standard specifically prohibits the use of nutrient-rich media (e.g., blood agar, and chocolate agar) in dialysis water and dialysate assays because these culture media are too rich for growth of the naturally occurring organisms found in water.⁷⁹² Debate continues within AAMI, however, as to the most appropriate culture medium and incubation conditions to be used. The original clinical observations on which the microbiological requirements of this standard were based used Standard Methods Agar (SMA), a medium containing relatively few nutrients.⁶⁶⁶ The use of tryptic soy agar (TSA), a general purpose medium for isolating and cultivating microorganisms was recommended in later versions of the standard because it was thought to be more appropriate for culturing bicarbonate-containing dialysate.^{788, 789, 835} Moreover, culturing systems based on TSA are readily available from commercial sources. Several studies, however, have shown that the use of nutrient-poor media, such as R2A, results in an increased recovery of bacteria from water.^{1451, 1452} The original standard also specified incubation for 48 hours at 95°F–98.6°F (35°C–37°C) before enumeration of bacterial colonies. Extending the culturing time up to 168 hours, or 7 days and using incubation temperatures of 73.4°F–82.4°F (23°C–28°C) have also been shown to increase the recovery of bacteria.^{1451, 1452} Other

investigators, however, have not found such clear cut differences between culturing techniques.^{835, 1453} After considerable discussion, the AAMI Committee has not reached a consensus regarding changes in the assay technique, and the use of TSA or its equivalent for 48 hours at 95°F–98.6°F (35°C–37°C) remains the recommended method. It should be recognized, however, that these culturing conditions may underestimate the bacterial burden in the water and fail to identify the presence of some organisms. Specifically, the recommended method may not detect the presence of various NTM that have been associated with several outbreaks of infection in dialysis units.^{31, 32} In these instances, however, the high numbers of mycobacteria in the water were related to the total heterotrophic plate counts, each of which was significantly greater than that allowable by the AAMI standard. Additionally, the recommended method will not detect fungi and yeast, which have been shown to contaminate water used for hemodialysis applications.¹⁴⁵⁴ Biofilm on the surface of the pipes may hide viable bacterial colonies, even though no viable colonies are detected in the water using sensitive culturing techniques.¹⁴⁵⁵ Many disinfection processes remove biofilm poorly, and a rapid increase in the level of bacteria in the water following disinfection may indicate significant biofilm formation. Therefore, although the results of microbiological surveillance obtained using the test methods outlined above may be useful in guiding disinfection schedules and in demonstrating compliance with AAMI standards, they should not be taken as an indication of the absolute microbiological purity of the water.⁷⁹²

Endotoxin can be tested by one of two types of assays a) a kinetic test method [e.g., colorimetric or turbidimetric] or b) a gel-clot assay. Endotoxin units are assayed by the *Limulus* Amebocyte Lysate (LAL) method. Because endotoxins differ in their activity on a mass basis, their activity is referred to a standard *Escherichia coli* endotoxin. The current standard (EC-6) is prepared from *E. coli* O113:H10. The relationship between mass of endotoxin and its activity varies with both the lot of LAL and the lot of control standard endotoxin used. Since standards for endotoxin were harmonized in 1983 with the introduction of EC-5, the relationship between mass and activity of endotoxin has been approximately 5–10 EU/ng. Studies to harmonize standards have led to the measurement of endotoxin units (EU) where 5 EU is equivalent to 1 ng *E. coli* O55:B5 endotoxin.¹⁴⁵⁶

In summary, water used to prepare dialysate and to reprocess hemodialyzers should not contain a total microbial count >200 CFU/mL as determined by assay on TSA agar for 48 hrs. at 96.8°F (36°C), and ≤2 endotoxin units (EU) per mL. The dialysate at the end of a dialysis treatment should not contain >2,000 CFU/mL.^{31, 32, 668, 789, 792}

3. Water Sampling Strategies and Culture Techniques for Detecting Legionellae

Legionella spp. are ubiquitous and can be isolated from 20%–40% of freshwater environments, including man-made water systems.^{1457, 1458} In health-care facilities, where legionellae in potable water rarely result in disease among immunocompromised patients, courses of remedial action are unclear.

Scheduled microbiologic monitoring for legionellae remains controversial because the presence of legionellae is not necessarily evidence of a potential for causing disease.¹⁴⁵⁹ CDC recommends aggressive disinfection measures for cleaning and maintaining devices known to transmit legionellae, but does not recommend regularly scheduled microbiologic assays for the bacteria.³⁹⁶ However, scheduled monitoring of potable water within a hospital might be considered in certain settings where persons are highly susceptible to illness and mortality from *Legionella* infection (e.g., hematopoietic stem cell transplantation units and solid organ transplant units).⁹ Also, after an outbreak of

legionellosis, health officials agree monitoring is necessary to identify the source and to evaluate the efficacy of biocides or other prevention measures.

Examination of water samples is the most efficient microbiologic method for identifying sources of legionellae and is an integral part of an epidemiologic investigation into health-care–associated Legionnaires disease. Because of the diversity of plumbing and HVAC systems in health-care facilities, the number and types of sites to be tested must be determined before collection of water samples. One environmental sampling protocol that addresses sampling site selection in hospitals might serve as a prototype for sampling in other institutions.¹²⁰⁹ Any water source that might be aerosolized should be considered a potential source for transmission of legionellae. The bacteria are rarely found in municipal water supplies and tend to colonize plumbing systems and point-of-use devices. To colonize, legionellae usually require a temperature range of 77°F–108°F (25°C–42.2°C) and are most commonly located in hot water systems.¹⁴⁶⁰ Legionellae do not survive drying. Therefore, air-conditioning equipment condensate, which frequently evaporates, is not a likely source.¹⁴⁶¹

Water samples and swabs from point-of-use devices or system surfaces should be collected when sampling for legionellae (Box C.1).¹⁴³⁷ Swabs of system surfaces allow sampling of biofilms, which frequently contain legionellae. When culturing faucet aerators and shower heads, swabs of surface areas should be collected first; water samples are collected after aerators or shower heads are removed from their pipes. Collection and culture techniques are outlined (Box C.2). Swabs can be streaked directly onto buffered charcoal yeast extract agar (BCYE) plates if the plates are available at the collection site. If the swabs and water samples must be transported back to a laboratory for processing, immersing individual swabs in sample water minimizes drying during transit. Place swabs and water samples in insulated coolers to protect specimens from temperature extremes.

Box C.1. Potential sampling sites for *Legionella* spp. in health-care facilities*

-
- **Potable water systems**
incoming water main, water softener unit, holding tanks, cisterns, water heater tanks
(at the inflows and outflows)
 - **Potable water outlets, especially those in or near patient rooms**
faucets or taps, showers
 - **Cooling towers and evaporative condensers**
makeup water (e.g., added to replace water lost because of evaporation, drift, or leakage),
basin (i.e., area under the tower for collection of cooled water), sump (i.e., section of basin
from which cooled water returns to heat source), heat sources (e.g., chillers)
 - **Humidifiers (e.g., nebulizers)**
bubblers for oxygen, water used for respiratory therapy equipment
 - **Other sources**
decorative fountains, irrigation equipment, fire sprinkler system (if recently used), whirlpools,
spas
-

* Material in this box is adapted from reference 1209.

Box C.2. Procedures for collecting and processing environmental specimens for *Legionella* spp.*

1. Collect water (1-liter samples, if possible) in sterile, screw-top bottles.
2. Collect culture swabs of internal surfaces of faucets, aerators, and shower heads in a sterile, screw-top container (e.g., 50 mL plastic centrifuge tube). Submerge each swab in 5–10 mL of sample water taken from the same device from which the sample was obtained.
3. Transport samples and process in a laboratory proficient at culturing water specimens for *Legionella* spp. as soon as possible after collection.+
4. Test samples for the presence of *Legionella* spp. by using semiselective culture media using procedures specific to the cultivation and detection of *Legionella* spp.§¶

* Material in this table is compiled from references 1209, 1437, 1462–1465.

+ Samples may be transported at room temperature but must be protected from temperature extremes. Samples not processed within 24 hours of collection should be refrigerated.

§ Detection of *Legionella* spp. antigen by the direct fluorescent antibody technique is not suitable for environmental samples.

¶ Use of polymerase chain reaction for identification of *Legionella* spp. is not recommended until more data regarding the sensitivity and specificity of this procedure are available.

4. Procedure for Cleaning Cooling Towers and Related Equipment

- I. Perform these steps prior to chemical disinfection and mechanical cleaning.
 - A. Provide protective equipment to workers who perform the disinfection, to prevent their exposure to chemicals used for disinfection and aerosolized water containing *Legionella* spp. Protective equipment may include full-length protective clothing, boots, gloves, goggles, and a full- or half-face mask that combines a HEPA filter and chemical cartridges to protect against airborne chlorine levels of up to 10 mg/L.
 - B. Shut off cooling tower.
 1. Shut off the heat source, if possible.
 2. Shut off fans, if present, on the cooling tower/evaporative condenser (CT/EC).
 3. Shut off the system blowdown (i.e., purge) valve.
 4. Shut off the automated blowdown controller, if present, and set the system controller to manual.
 5. Keep make-up water valves open.
 6. Close building air-intake vents within at least 30 meters of the CT/EC until after the cleaning procedure is complete.
 7. Continue operating pumps for water circulation through the CT/EC.
- II. Perform these chemical disinfection procedures.
 - A. Add fast-release, chlorine-containing disinfectant in pellet, granular, or liquid form, and follow safety instructions on the product label. Use EPA-registered products, if available. Examples of disinfectants include sodium hypochlorite (NaOCl) or calcium hypochlorite (Ca[OCl]₂), calculated to achieve initial free residual chlorine (FRC) of 50 mg/L: either a) 3.0 lbs [1.4 kg] industrial grade NaOCl [12%–15% available Cl] per 1,000 gallons of CT/EC water; b) 10.5 lbs [4.8 kg] domestic grade NaOCl [3%–5% available Cl] per 1,000 gallons of CT/EC water; or c)

0.6 lb [0.3 kg] $\text{Ca}[\text{OCl}]_2$ per 1,000 gallons of CT/EC water. If significant biodeposits are present, additional chlorine may be required. If the volume of water in the CT/EC is unknown, it can be estimated (in gallons) by multiplying either the recirculation rate in gallons per minute by 10 or the refrigeration capacity in tons by 30. Other appropriate compounds may be suggested by a water-treatment specialist.

- B. Record the type and quality of all chemicals used for disinfection, the exact time the chemicals were added to the system, and the time and results of FRC and pH measurements.
- C. Add dispersant simultaneously with or within 15 minutes of adding disinfectant. The dispersant is best added by first dissolving it in water and adding the solution to a turbulent zone in the water system. Automatic-dishwasher compounds are examples of low- or nonfoaming, silicate-based dispersants. Dispersants are added at 10–25 lbs (4.5–11.25 kg) per 1,000 gallons of CT/EC water.
- D. After adding disinfectant and dispersant, continue circulating the water through the system. Monitor the FRC by using an FRC-measuring device with the DPD method (e.g., a swimming-pool test kit), and measure the pH with a pH meter every 15 minutes for 2 hours. Add chlorine as needed to maintain the FRC at ≥ 10 mg/L. Because the biocidal effect of chlorine is reduced at a higher pH, adjust the pH to 7.5–8.0. The pH may be lowered by using any acid (e.g., muriatic acid or sulfuric acid used for maintenance of swimming pools) that is compatible with the treatment chemicals.
- E. Two hours after adding disinfectant and dispersant or after the FRC level is stable at ≥ 10 mg/L, monitor at 2-hour intervals and maintain the FRC at ≥ 10 mg/L for 24 hours.
- F. After the FRC level has been maintained at ≥ 10 mg/L for 24 hours, drain the system. CT/EC water may be drained safely into the sanitary sewer. Municipal water and sewerage authorities should be contacted regarding local regulations. If a sanitary sewer is not available, consult local or state authorities (e.g., a department of natural resources or environmental protection) regarding disposal of water. If necessary, the drain-off may be dechlorinated by dissipation or chemical neutralization with sodium bisulfite.
- G. Refill the system with water and repeat the procedure outline in steps 2–7 in I-B above.

III. Perform mechanical cleaning.

- A. After water from the second chemical disinfection has been drained, shut down the CT/EC.
- B. Inspect all water-contact areas for sediment, sludge, and scale. Using brushes and/or a low-pressure water hose, thoroughly clean all CT/EC water-contact areas, including the basin, sump, fill, spray nozzles, and fittings. Replace components as needed.
- C. If possible, clean CT/EC water-contact areas within the chillers.

IV. Perform these procedures after mechanical cleaning.

- A. Fill the system with water and add chlorine to achieve an FRC level of 10 mg/L.
- B. Circulate the water for 1 hour, then open the blowdown valve and flush the entire system until the water is free of turbidity.
- C. Drain the system.
- D. Open any air-intake vents that were closed before cleaning.
- E. Fill the system with water. The CT/EC may be put back into service using an effective water-treatment program.

5. Maintenance Procedures Used to Decrease Survival and Multiplications of *Legionella* spp. in Potable-Water Distribution Systems

Wherever allowable by state code, provide water at $\geq 124^{\circ}\text{F}$ ($\geq 51^{\circ}\text{C}$) at all points in the heated water system, including the taps. This requires that water in calorifiers (e.g., water heaters) be maintained at $\geq 140^{\circ}\text{F}$ ($\geq 60^{\circ}\text{C}$). In the United Kingdom, where maintenance of water temperatures at $\geq 122^{\circ}\text{F}$ ($\geq 50^{\circ}\text{C}$) in hospitals has been mandated, installation of blending or mixing valves at or near taps to reduce the water temperature to $\leq 109.4^{\circ}\text{F}$ ($\leq 63^{\circ}\text{C}$) has been recommended in certain settings to reduce the risk for scald injury to patients, visitors, and health care workers.⁷²⁶ However, *Legionella* spp. can multiply even in short segments of pipe containing water at this temperature. Increasing the flow rate from the hot-water-circulation system may help lessen the likelihood of water stagnation and cooling.^{711, 1465} Insulation of plumbing to ensure delivery of cold ($< 68^{\circ}\text{F}$ [$< 20^{\circ}\text{C}$]) water to water heaters (and to cold-water outlets) may diminish the opportunity for bacterial multiplication.⁴⁵⁶ Both dead legs and capped spurs within the plumbing system provide areas of stagnation and cooling to $< 122^{\circ}\text{F}$ ($< 50^{\circ}\text{C}$) regardless of the circulating water temperature; these segments may need to be removed to prevent colonization.⁷⁰⁴ Rubber fittings within plumbing systems have been associated with persistent colonization, and replacement of these fittings may be required for *Legionella* spp. eradication.¹⁴⁶⁷

Continuous chlorination to maintain concentrations of free residual chlorine at 1–2 mg/L (1–2 ppm) at the tap is an alternative option for treatment. This requires the placement of flow-adjusted, continuous injectors of chlorine throughout the water distribution system. Adverse effects of continuous chlorination can include accelerated corrosion of plumbing (resulting in system leaks) and production of potentially carcinogenic trihalomethanes. However, when levels of free residual chlorine are below 3 mg/L (3 ppm), trihalomethane levels are kept below the maximum safety level recommended by the EPA.^{727, 1468, 1469}

Appendix D. Insects and Microorganisms

Table D.1. Microorganisms isolated from arthropods in health-care settings

Insect	Microorganism category	Microorganisms	References
Cockroaches	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Citrobacter freundii</i> ; <i>Enterobacter</i> spp., <i>E. cloacae</i> ; <i>Escherichia coli</i> ; <i>Flavobacterium</i> spp.; <i>Klebsiella</i> spp.; <i>Proteus</i> spp.; <i>Pseudomonas</i> spp., <i>P. aeruginosa</i> , <i>P. fluorescens</i> , <i>P. putida</i> ; <i>Salmonella</i> spp.; <i>Serratia</i> spp., <i>S. marcescens</i> ; <i>Shigella boydii</i>	1048, 1051, 1056, 1058, 1059, 1062
	Gram-positive bacteria	<i>Bacillus</i> spp.; <i>Enterococcus faecalis</i> ; <i>Micrococcus</i> spp.; <i>Staphylococcus aureus</i> , <i>S. epidermidis</i> ; <i>Streptococcus</i> spp., <i>S. viridans</i>	1056, 1058, 1059
	Acid-fast bacteria	<i>Mycobacterium tuberculosis</i>	1065
	Fungi	<i>Aspergillus niger</i> ; <i>Mucor</i> spp.; <i>Rhizopus</i> spp.	1052, 1059
	Parasites	<i>Endolimax nana</i> ; <i>Entamoeba coli</i>	1059
Houseflies	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Campulobacter fetus</i> subsp. <i>Jejuni</i> ; <i>Chlamydia</i> spp.; <i>Citrobacter freundii</i> ; <i>Enterobacter</i> spp.; <i>Escherichia coli</i> ; <i>Helicobacter pylori</i> ; <i>Klebsiella</i> spp.; <i>Proteus</i> spp.; <i>Pseudomonas aeruginosa</i> ; <i>Serratia marcescens</i> ; <i>Shigella</i> spp.	1047, 1048, 1050, 1053–1055, 1060
	Gram-positive bacteria	<i>Bacillus</i> spp.; <i>Enterococcus faecalis</i> ; <i>Micrococcus</i> spp.; <i>Staphylococcus</i> spp. (coagulase-negative), <i>S. aureus</i> ; <i>Streptococcus</i> spp., <i>S. viridans</i>	1048, 1060
	Fungi / yeasts	<i>Candida</i> spp.; <i>Geotrichum</i> spp.	1060
	Parasites	<i>Endolimax nana</i> ; <i>Entamoeba coli</i>	1060
	Viruses	Rotaviruses	1049
Ants	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Escherichia coli</i> ; <i>Klebsiella</i> spp.; <i>Neisseria sicca</i> ; <i>Proteus</i> spp.; <i>Providencia</i> spp.; <i>Pseudomonas aeruginosa</i> , <i>P. fluorescens</i>	1057
	Gram-positive bacteria	<i>Bacillus</i> spp., <i>B. cereus</i> , <i>B. pumilis</i> ; <i>Clostridium cochlearium</i> , <i>C. welchii</i> ; <i>Enterococcus faecalis</i> ; <i>Staphylococcus</i> spp. (coagulase-negative), <i>S. aureus</i> ; <i>Streptococcus pyrogenes</i>	1057
Spiders	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Citrobacter freundii</i> ; <i>Enterobacter aerogenes</i> ; <i>Morganella morganii</i>	1048
	Gram-positive bacteria	<i>Staphylococcus</i> spp. (coagulase-negative)	1048
Mites, midges	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Burkholderia cepacia</i> ; <i>Enterobacter agglomerans</i> , <i>E. aerogenes</i> ; <i>Hafnia alvei</i> ; <i>Pseudomonas aeruginosa</i>	1048
	Gram-positive bacteria	<i>Staphylococcus</i> spp. (coagulase-negative)	1048
Mosquitoes	Gram-negative bacteria	<i>Acinetobacter calcoaceticus</i> ; <i>Enterobacter cloacae</i>	1048
	Gram-positive bacteria	<i>Enterococcus</i> spp.; <i>Staphylococcus</i> spp. (coagulase-negative)	1048

Appendix E. Information Resources

The following sources of information may be helpful to the reader. Some of these are available at no charge, while others are available for purchase from the publisher.

Air and Water

- Jensen PA, Schafer MP. Sampling and characterization of bioaerosols. NIOSH Manual of Analytical Methods; revised 6/99. www.cdc.gov/niosh/nmam/pdfs/chapter-j.pdf
- American Institutes of Architects. *Guidelines for Design and Construction of Hospital and Health Care Facilities*. Washington DC; American Institute of Architects Press; 2001. AIA, 1735 New York Avenue, NW, Washington DC 20006. 1-800-AIA-3837 or (202) 626-7541
- ASHRAE. Standard 62, and Standard 12-2000. These documents may be purchased from: American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc. 1791 Tullie Circle, NE, Atlanta GA 30329 1-800-527-4723 or (404) 636-8400.
- University of Minnesota websites: www.dehs.umn.edu Indoor air quality site: www.dehs.umn.edu/resources.htm#indoor Water infiltration and use of the wet test (moisture) meter: www.dehs.umn.edu/remangi.html
- The CDC website for bioterrorism information contains the interim intervention plan for smallpox. The plan discusses infection control issues both for home-based care and hospital-based patient management. www.bt.cdc.gov/agent/smallpox/response-plan/index.asp

Environmental Sampling

- ISO. Sterilization of medical devices – microbiological methods, Part 1. ISO standard 11737-1. Paramus NJ; International Organization for Standardization; 1995.

Animals in Health-Care Facilities

- Service animal information with respect to the Americans with Disabilities Act. Contact the U.S. Department of Justice ADA Information Line at (800) 514-0301 (voice) or (800) 514-0383 (TDD), or visit the ADA website at: www.usdoj.gov/crt/ada/adahom1.htm

Regulated Medical Waste

- U.S. Environmental Protection Agency. This is the Internet address on their Internet web site that will link to any state for information about medical waste rules and regulations at the state level: www.epa.gov/epaoswer/other/medical/stregs.htm

General Resources

- APIC Text of Infection Control and Epidemiology. Association for Professionals in Infection Control and Epidemiology, Inc. Washington DC; 2000. (Two binder volumes, or CD-ROM)
- Abrutyn E, Goldmann DA, Scheckler WE. Saunders Infection Control Reference Service, 2nd Edition. Philadelphia PA; WB Saunders; 2000.
- ECRI publications are available on a variety of healthcare topics. Contact ECRI at (610) 825-6000. CRI, 5200 Butler Pike, Plymouth Meeting, PA 19462-1298.

Appendix F. Areas of Future Research

Air

- Standardize the methodology and interpretation of microbiologic air sampling (e.g., determine action levels or minimum infectious dose for aspergillosis, and evaluate the significance of airborne bacteria and fungi in the surgical field and the impact on postoperative SSI).
- Develop new molecular typing methods to better define the epidemiology of health-care–associated outbreaks of aspergillosis and to associate isolates recovered from both clinical and environmental sources.
- Develop new methods for the diagnosis of aspergillosis that can lead reliably to early recognition of infection.
- Assess the value of laminar flow technology for surgeries other than for joint replacement surgery.
- Determine if particulate sampling can be routinely performed in lieu of microbiologic sampling for purposes such as determining air quality of clean environments (e.g., operating rooms, HSCT units).

Water

- Evaluate new methods of water treatment, both in the facility and at the water utility (e.g., ozone, chlorine dioxide, copper/silver/monochloramine) and perform cost-benefit analyses of treatment in preventing health-care–associated legionellosis.
- Evaluate the role of biofilms in overall water quality and determine the impact of water treatments for the control of biofilm in distribution systems.
- Determine if the use of ultrapure fluids in dialysis is feasible and warranted, and determine the action level for the final bath.
- Develop quality assurance protocols and validated methods for sampling filtered rinse water used with AERs and determine acceptable microbiologic quality of AER rinse water.

Environmental Services

- Evaluate the innate resistance of microorganisms to the action of chemical germicides, and determine what, if any, linkage there may be between antibiotic resistance and resistance to disinfectants.

Laundry and Bedding

- Evaluate the microbial inactivation capabilities of new laundry detergents, bleach substitutes, other laundry additives, and new laundry technologies.

Animals in Health-Care Facilities

- Conduct surveillance to monitor incidence of infections among patients in facilities that use animal programs, and conduct investigations to determine new infection control strategies to prevent these infections.
- Evaluate the epidemiologic impact of performing procedures on animals (e.g., surgery or imaging) in human health-care facilities.

Regulated Medical Waste

- Determine the efficiency of current medical waste treatment technologies to inactivate emerging pathogens that may be present in medical waste (e.g., SARS-coV).
- Explore options to enable health-care facilities to reinstate the capacity to inactivate microbiological cultures and stocks on-site.

Index—Parts I and IV

A

AAMI standards 59, 60, 62, 222, 223
Acinetobacter spp. 11, 20, 43, 44, 99, 104
aerators 47, 48, 94, 220–222, 224, 225
aerosols... 12, 27, 41, 47, 56, 59, 67, 75, 76, 78, 80, 85, 89,
90, 98, 106, 111, 113, 114
AIA guidelines..... 17, 18, 19, 25, 37, 39, 99
AII rooms 35–37
air changes per hour (ACH)..... 6, 12, 16, 18, 31, 111, 210
air conditioners 8, 22
air conditioning systems 13, 20, 57, 59
air filtration..... 111
air intakes 31, 226
air sampling 26, 29, 89, 90, 91, 93, 210
airborne infection isolation (AII)..... 6, 19
airborne transmission..... 12
air-fluidized beds 104
alcohol-based hand rubs 53
alkaline glutaraldehyde..... 70
allergens..... 17, 80, 107
American Institute of Architects (AIA)..... 13
Americans with Disabilities Act..... 108, 110
amplified stocks and cultures..... 114, 115
Animal Assisted Activities 106, 107
Animal Assisted Therapy 106, 107
animal bites..... 107
animal handler 107
animal patient 110
Animal Welfare Act..... 112
anterooms 12, 25, 33, 36–38
ants 81
ASHRAE 13, 47, 49
aspergillosis 7, 8, 16, 19, 21, 35, 79, 80
Aspergillus fumigatus 7, 8, 29
Aspergillus spp. 5, 7, 20, 21, 28, 32, 34, 81
automated cyclers 65
automated endoscope reprocessor..... 50, 69, 70
autopsy suites/rooms..... 12, 87

B

bacterial spores 73, 84, 89
bank of filters..... 14
barrier 34
barrier precautions/protection..... 74, 109, 116
barriers..... 27, 31, 33
bassinets..... 76
biofilms..... 46, 54, 64, 71, 220–222, 224
biosafety level..... 114
bioterrorism 89, 114
bird droppings..... 9, 20, 22
birthing tanks..... 67, 69

blood.. 12, 64, 69, 75, 77–79, 86, 87, 98, 99, 102, 113, 116
bloodborne pathogens..... 73, 116
boil water advisory 51, 52

C

calibrated loop 222
carpet cleaning 79
carpet tiles..... 79
carpeting 22, 25, 52, 78, 79
cats..... 105, 106, 108, 109
chain of infection 4, 87
chemical germicides 73, 74, 77, 80, 84
chloramine/chloramine-T 68
chlorine..... 46, 50, 53, 69, 84, 221, 226
chlorine bleach..... 78, 101
chlorine residual 50, 68, 69, 94, 101, 221
cleaning .. 68, 70–72, 74, 78, 80, 83, 85, 86, 107, 109, 112,
225
cleaning cloths 75
cleaning solutions 75, 76
Clostridium difficile 5, 84
cloth chairs..... 79
cockroaches 81
coliform bacteria..... 221
colonization 42–44, 68, 70, 83, 99, 106, 227
colony counts..... 211
commissioning..... 29, 89
construction 7, 13, 14, 21, 23, 26, 27, 29, 31, 37, 76, 89
construction workers..... 24, 26, 31
contact precautions 85
contact time 74, 84, 88, 221
contaminants..... 14, 18, 19, 59, 210
contaminated fabrics..... 98, 101, 102
contingency plans 21, 50
continuous chlorination 227
cooling tower..... 41, 53, 55, 57–59, 220, 225
copper/silver ions..... 54
copper-8-quinolinolate..... 35
Creutzfeldt-Jakob disease 86, 116
Cryptosporidium parvum..... 46

D

dead legs 59, 64, 221
decorative fountains..... 47, 49, 224
demolition..... 23, 25, 26, 29
dental unit water lines..... 71
detergent/disinfectant..... 74–76
dialysate 59–62
dialysis machines 64
dialysis water 222
dialyzer 62
dialyzer membranes..... 61

dialyzer reprocessing 59
 dioctylphthalate (DOP) particle test 15
 direct contact 6, 41, 67, 85, 86, 98, 108, 111
 direct threat 109
 disinfectant fogging 75
 disinfectant residuals 96
 disinfectants 21, 76, 225
 disinfecting 71, 74, 80, 83, 85, 86, 112, 226
 disinfection 63, 64, 68, 70
 dispersant 226
 disposal (of medical waste) 113
 distribution system 94, 221
 dogs 105, 106, 108, 109
 drift eliminators 58
 drinking water 71
 droplet nuclei 6, 7, 10, 12, 89
 droplets 6, 55, 85, 86, 89
 dry cleaning 102
 drying 11
 dual-duct system 20
 duct cleaning 21
 ductwork 20, 22
 dust 8, 20, 24, 27, 30, 32, 74, 79, 93
 dust-spot test 15

E

education 24
 electrical generators 53
 emergency 53
 endotoxin 60–62, 64, 223
 engineering controls 36
 enteric viruses 85
 environmental cultures 83, 88
 Environmental Protection Agency (EPA) 21, 73–75, 77, 103, 227
 environmental sampling 88, 95
 environmental surfaces 11, 44, 71, 72, 74, 82–86, 88, 98, 107
 environmental surveillance 54, 55
 EPA registration 73, 76, 83
 EPA-registered germicides 75, 78, 85, 86
 evaporative condensers 41, 57–59
 exclusion (of a service animal) 109
 exotic animals 110

F

fan-coil units 18
 faucets 47, 54, 94, 222, 224, 225
 fecal contamination 84
 FIFRA 75, 103
 filter efficiency 27, 29
 filtration 15
 fire codes 31
 fish 105, 108
 fish tanks 108
 flies 81
 flooding 51

floors 25, 75, 82, 83
 flowers 80
 flush times 51
 flutter strips 20, 34, 36
 fomites 3, 4, 7, 85
 Food and Drug Administration (FDA) 69, 73, 103
 free residual chlorine 51, 54, 225–227
 fungal spores 8, 15, 16, 19–21, 26–28, 31, 34, 38, 79, 89, 93
 fungi 8
 furniture 52, 79, 82

G

gram-negative bacteria 11, 41, 42, 48, 50, 60, 63, 64, 221
 gram-positive bacteria 11, 84

H

hand hygiene 25, 71, 107, 109
 hand transferral 3, 44, 65, 82–84, 106, 221
 handwashing 25, 80, 84, 99, 107
 hantaviruses 12
 hematopoietic stem cell transplant 6
 hemodiafiltration 62
 hemodialysis 59, 60, 62, 64, 223
 hemodialysis patients 7
 hemofiltration 62
 HEPA filtration/filters 6, 12, 14, 15, 17, 31, 32, 36, 76
 hepatitis B virus 40, 73, 98
 heterotrophic plate counts 51, 62, 66, 95, 221–223
 high-flux membranes 61
 high-level disinfectants 73
 high-level disinfection 60, 69, 72
 high-temperature flushing 50
 high-touch surfaces 75, 83–85
 holding tank 47
 hospital disinfectant 73
 hot water system 51, 54
 hot water tanks 53, 220–222
 hot water temperature 49
 housekeeping surfaces 3, 64, 72, 74–77, 83
 HSCT patients 6, 37
 HSCT units 11, 26, 79, 80, 107
 Hubbard tanks 68
 human health-care facilities 110, 111
 human immunodeficiency virus 73
 humidifiers 17, 23, 41
 humidity 13, 14, 17, 20, 38, 90
 HVAC systems 13, 14, 16, 17, 19–21, 27, 30, 51
 hydrotherapy equipment 67–69
 hydrotherapy pools 68
 hydrotherapy tanks 67, 68, 82
 hygienically-clean laundry 98–100, 102
 hyperchlorination 50, 53, 54, 59

I, J

iatrogenic cases.....	87
ice machines and ice.....	25, 48, 65, 66
ice-storage chests.....	66
immunocompromised patients.....	6, 7, 9, 26, 29–31, 34, 42, 47, 56, 66, 80, 107, 108, 223
impaction.....	90, 211
impactors.....	28, 93
impingement.....	90, 93, 211
impingers.....	211
inactivation studies.....	87
incineration.....	113, 114
incubators (nursery).....	76
indirect transmission.....	6
indirect contact.....	41
indoor air.....	21, 24, 26, 27, 90
industrial-grade HEPA filter.....	16, 31, 38, 39
infection-control risk assessment (ICRA)....	26, 29, 31, 35, 108, 111
influenza viruses.....	6, 12, 73, 85
innate resistance.....	72, 73, 84
insects.....	67, 81
insulation material.....	20
intermediate-level disinfectants.....	73, 78, 83, 85, 86
intermediate-level disinfection.....	72
isolation/isolation areas.....	11, 36, 100
JCAHO.....	13, 14, 51, 59

L

laboratories.....	12, 13, 32, 47, 78, 79, 83, 105, 111, 112, 114, 222
laboratory confirmation.....	55
laminar airflow.....	18, 34, 38
laser plumes.....	40
laundry.....	49
laundry bags.....	100
laundry chutes.....	100
laundry cycles.....	101
laundry disinfection.....	101
laundry facility.....	99
laundry packaging.....	100, 101
laundry process.....	98, 99, 102
laundry services.....	99
laundry transport.....	100, 101
<i>Legionella pneumophila</i>	210, 221
<i>Legionella</i> spp.	41, 42, 50, 54–57, 59, 71, 222, 223, 225, 227
legionellae.....	41, 54, 211, 223
legionellosis.....	53–56, 224
Legionnaires disease.....	41, 47, 57, 58, 224
liquid chemical sterilant.....	70
low-level disinfectants.....	72, 73, 83, 86
low-level disinfection.....	60, 64

M

manufacturer's instructions.....	67, 69, 74, 86, 102, 116
material safety data sheets (MSDS).....	75, 87
mattress cover.....	77, 104
mattresses.....	77, 104
medical equipment.....	74, 83
medical equipment surfaces.....	72
medical gas piping.....	30
medical records.....	51
medical waste.....	112, 113, 117
medical waste management.....	112
membrane filtration.....	70, 95, 96, 222
methicillin-resistant <i>Staphylococcus aureus</i> (MRSA) ...	82, 83, 104, 105
microbial inactivation.....	72
microbial resistance.....	70
microbiologic air sampling.....	27
microbiologic cultures and stocks.....	112
microbiologic sampling.....	64
microbiologic sampling of laundry.....	102
microbiological wastes.....	112, 114
moisture.....	20, 24, 32, 51, 70, 96
moisture meters.....	51
molecular typing.....	28
monochloramine.....	54
mop heads.....	75
multidisciplinary team.....	23, 91
municipal water.....	47, 50, 224
municipal water systems/utilities.....	45, 221
<i>Mycobacterium tuberculosis</i>	5, 7, 10, 73, 114
myiasis.....	81

N

negative air pressure.....	6, 12, 18, 19, 21, 36, 99, 100, 104, 111
neutralizer chemicals.....	96
NIOSH.....	40
nontuberculous mycobacteria (NTM).....	5, 41, 44–46, 60, 63, 70, 71, 223

O

operating rooms.....	13, 15, 17, 34, 38, 76, 82, 87, 109, 111
opportunistic infections.....	4, 5
organic matter.....	78
OSHA.....	13, 73, 77, 79, 98, 100, 113
outdoor air.....	14, 15, 18, 25, 91
oxygen-based laundry detergents.....	101

P

particle sampling.....	27, 33, 89
performance measures.....	2
periodic culturing.....	57
peritoneal dialysis.....	64, 65

personal protective equipment	77, 98, 99, 112, 114, 225
persons with disabilities	108, 109
person-to-person transmission	12, 85
pest control	82
phenolics	76
pillows	104
pipes	64, 69, 221, 223, 224
planktonic organisms	221
plastic enclosures	31
plastic wrapping	74
<i>Pneumocystis carinii</i>	9
pneumonia	42, 55
point-of-use fixtures	47, 51, 224
polyvinylchloride (PVC)	46, 64
pools	67
positive air pressure	18, 38
potable water	220
potted plants	8, 80
pressure differentials	18, 19, 25, 30, 38
primates	105, 106, 111
prions	86, 116
privacy curtains	75
product water	64, 70
protective environment (PE)	6, 18, 19, 34, 56, 108
<i>Pseudomonas aeruginosa</i>	5, 11, 20, 42, 68, 70, 71, 73, 79, 80, 96, 104, 221
pseudo-outbreaks	44, 70
pyrogenic reactions	60, 61

Q

quality assurance	89, 94, 95
-------------------	------------

R

R2A media	222
rank order	27
recirculation	16, 18
recirculation loops	46
recreational equipment	69
reduced nutrient media	94
reducing agent	94
relative humidity	17
renovation	13, 14, 23, 37
repairs	31
reprocess hemodialyzers	61, 223
research animals	111
reservoirs	3, 6, 41, 42, 71, 79, 83, 95, 105, 211
resident animals	107
respirable particles	27, 28, 90
respirators	26, 40
respiratory protection	36, 78
respiratory syncytial virus (RSV)	6, 12, 85
respiratory therapy equipment	224
return air	14
return temperature	54
reverse osmosis (RO)	52, 54, 59, 60, 63
rewiring	25
rinse water monitoring	70

RODAC plates	102
rodents	67
rooftops	30

S

Sabouraud dextrose agar	96
sample/rinse methods	97
sanitary sewer	116, 117
SARS	86
SARS-CoV	86
scalding	49, 51
screens	82
scrub suits	98, 99
sealed windows	19, 26, 29, 89
sedimentation	90, 93, 211
select agents	114, 115
self-closing doors	19
semicritical device	70
service animal	105, 108–110
settle plates	28, 90, 93, 211
sewage spills	51
sharps containers	113
shock decontamination	51
shower heads	47, 49, 54, 220, 221, 222, 224, 225
showers	47, 48
skin antiseptics	73
smallpox	36
smallpox virus	7, 12
smoke tubes	20, 34, 36
sodium hydroxide	87
sodium hypochlorite	67, 69, 73, 77, 83, 84, 87, 88, 94, 225
solid-organ transplant program	56
sorting (laundry)	98, 100
Spaulding classification	71, 98
spills	75, 77, 79
standard precautions	100, 111
standards	2, 14, 71, 88, 90, 112, 223
<i>Staphylococcus aureus</i>	10, 11, 38, 64, 73, 99, 104
state codes/regulations	55, 69
steam jet	101
steam sterilization (of medical waste)	113, 114
sterile water	55
storage tanks	63, 64
streptococci	10, 38
supplemental treatment methods	53
surgical gowns and drapes	103
surgical site infections (SSI)	11, 38, 65
surgical smoke	40
surveillance	26, 51, 57, 99, 223
swabs	224

T

tacky mats	76
tap water	42, 44, 57, 65, 66, 70
TB patients	38
temperature (air)	13, 14, 17, 89
temperature (water)	40, 45, 49, 68, 101, 221, 227

thermostatic mixing valves	49
transport and storage (of medical waste)	113
treated items/products	79, 103
tryptic soy agar	94, 96, 222
tub liners	68
tuberculocidal claim	73
tuberculosis (TB)	35

U

ultrapure dialysate	61
ultraviolet germicidal irradiation (UVGI)	14, 16, 17, 36, 38
uniforms	98, 99

V

vacuum breakers	47, 50
vacuum cleaners	76, 79
vacuuming	79
vancomycin-resistant enterococci (VRE)	3, 5, 82, 83, 105
vancomycin-resistant <i>Staphylococcus aureus</i> (VRSA)	83
variable air ventilation	20, 38
varicella-zoster virus (VZV)	5, 7, 40
vase water	81
vegetative bacteria	73
ventilation rates	18
ventilation systems	8, 9, 111
viable particles	9, 91

viral hemorrhagic fever	12
viral particles	11
viruses	11, 85
visual monitoring device	34
volumetric air samplers	29
volumetric sampling methods	28

W

wallboard	8, 22, 52
walls	25
washing machines and dryers	102
water conditioning	68
water distribution systems	64, 221, 227
water droplets	58
water pipes	46
water pressure	50
water quality	71, 94
water sampling	54, 94, 221, 224
water stagnation	227
water treatment system	63
waterborne transmission	46
weight-arrestance test	15
wet cleaning	79
whirlpool spas	59, 67, 69
whirlpools	68, 69
window chutes	33
windows	22, 59
wood	8, 9, 15, 35

GUIDE TO INFECTION PREVENTION FOR OUTPATIENT SETTINGS:

Minimum Expectations for Safe Care



National Center for Emerging and Zoonotic Infectious Diseases
Division of Healthcare Quality Promotion



NOTE TO READERS

The following document is a summary guide of infection prevention recommendations for outpatient (ambulatory care) settings. The recommendations included in this document are not new but rather reflect existing evidence-based guidelines produced by the Centers for Disease Control and Prevention and the Healthcare Infection Control Practices Advisory Committee. This summary guide is based primarily upon elements of Standard Precautions and represents the minimum infection prevention expectations for safe care in ambulatory care settings. Readers are urged to use the *Infection Prevention Checklist for Outpatient Settings* (Appendix A), a companion to the summary guide, and to consult the full guidelines for additional background, rationale, and evidence behind each recommendation.

All guidelines are available at:

http://www.cdc.gov/HAI/prevent/prevent_pubs.html

The transition of healthcare delivery from acute care hospitals to outpatient (ambulatory care) settings, along with ongoing outbreaks and patient notification events, have demonstrated the need for greater understanding and implementation of basic infection prevention guidance. *Guide to Infection Prevention for Outpatient Settings: Minimum Expectations for Safe Care* distills existing infection prevention guidance from the Centers for Disease Control and Prevention (CDC) and the Healthcare Infection Control Practices Advisory Committee (HICPAC).

Over the past several decades, we have witnessed a significant shift in healthcare delivery from the acute, inpatient hospital setting to a variety of ambulatory and community-based settings. Ambulatory care is provided in hospital-based outpatient clinics, nonhospital-based clinics and physician offices, ambulatory surgical centers, and many other specialized settings. Americans have frequent encounters with ambulatory care. For example, more than three-quarters of all operations in the United States are performed in settings outside the hospital¹. In addition, between 1995 and 2007, the average person made three visits each year to physician offices². By 2007, the total number of physician offices visits approached one billion³. Vulnerable patient populations rely on frequent and intensive use of ambulatory care to maintain or improve their health. For example, each year more than one million cancer patients receive outpatient chemotherapy, radiation therapy, or both⁴. It is critical that all of this care be provided under conditions that minimize or eliminate risks of healthcare-associated infections (HAI).

Compared to inpatient acute care settings, ambulatory care settings have traditionally lacked infrastructure and resources to support infection

prevention and surveillance activities^{5,6,7}. While data describing risks for HAI are lacking for most ambulatory settings, numerous outbreak reports have described transmission of gram-negative and gram-positive bacteria, mycobacteria, viruses, and parasites^{8,9}. In many instances, outbreaks and other adverse events were associated with breakdowns in basic infection prevention procedures (e.g., reuse of syringes leading to transmission of bloodborne viruses).

All healthcare settings, regardless of the level of care provided, must make infection prevention a priority and must be equipped to observe Standard Precautions. The 2007 CDC and HICPAC Guideline for Isolation Precautions was a first attempt to provide recommendations that can be applied in all healthcare settings. The Guide presented here is based primarily upon elements of Standard Precautions from that guideline and represents the minimum infection prevention expectations for safe care in ambulatory care settings. It is intended for use by anyone needing information about general infection prevention measures in ambulatory care settings. To assist with conducting periodic assessments of infection prevention policies and practices, the reader is referred to the *Infection Prevention Checklist for Outpatient Settings*, which appears at the end of this document as Appendix A.

For the purposes of this document, ambulatory care is defined as care provided in facilities where patients do not remain overnight (e.g., hospital-based outpatient clinics, non-hospital based clinics and physician offices, urgent care centers, ambulatory surgical centers, public health clinics, imaging centers, oncology clinics, ambulatory behavioral health and substance abuse clinics, physical therapy and rehabilitation centers). Healthcare personnel (HCP) are defined as all

persons, paid and unpaid, working in ambulatory care settings who have the potential for exposure to patients and/or to infectious materials, including body substances, contaminated medical supplies and equipment, contaminated environmental surfaces, or contaminated air. This includes persons not directly involved in patient care (e.g., clerical, house-keeping, and volunteers) but potentially exposed to infectious agents that can be transmitted to and from HCP and patients.

This document does not replace existing, more-detailed guidance for hemodialysis centers or dental practices. Further, the reader is referred to other CDC and HICPAC guidelines and websites for more detailed information and for recommendations concerning specialized infection prevention issues (e.g., sterilization and disinfection of equipment, multi-drug resistant organisms).

OBJECTIVES

By highlighting existing CDC and HICPAC recommendations, this summary guide: 1) provides basic infection prevention recommendations for outpatient (ambulatory care) settings; 2) reaffirms Standard Precautions as the foundation for preventing transmission of infectious agents during patient care in all healthcare settings; 3) provides links to full guidelines and source documents, which readers can reference for more detailed background and recommendations.

FUNDAMENTAL ELEMENTS NEEDED TO PREVENT TRANSMISSION OF INFECTIOUS AGENTS IN AMBULATORY CARE SETTINGS

Dedicate Resources to Infection Prevention (Administrative Measures)

Infection prevention must be made a priority in any setting where healthcare is delivered. Those with primary administrative oversight of the ambulatory care facility/setting must ensure that sufficient fiscal and human resources are available to develop and maintain infection prevention and occupational health programs. This includes the availability of sufficient and appropriate equipment and supplies necessary for the consistent observation of Standard Precautions, including hand hygiene products, injection equipment, and personal protective equipment (e.g., gloves, gowns, face and eye protection).

Infection prevention programs must extend beyond Occupational Safety and Health Administration (OSHA) bloodborne pathogen training to address patient protection. Facilities should assure that at least one individual with training in infection prevention is employed by or regularly available to the facility. This individual should be involved in the development of written infection prevention policies and have regular communication with HCP to address specific issues or concerns related to infection prevention. The development and ongoing refinement of infection prevention policies and procedures should be based on evidence-based guidelines, regulations, or standards. These policies and procedures should be tailored to the facility and re-assessed on a regular basis (e.g., annually), taking into consideration the types of services provided by the facility and the patient population that is served. This process (referred to as risk assessment by the Infection Prevention profession) will allow facilities to better prioritize

resources and focus extra attention on those areas that are determined to pose greater risk to their patients. For example, an ambulatory surgical center, which performs on-site sterilization of surgical equipment, would be expected to have more detailed policies regarding equipment reprocessing than a substance abuse clinic, where on-site sterilization is unlikely to be performed. However, both facilities should have policies and procedures addressing handling of reusable medical equipment. Similarly, a clinic primarily serving patients infected with tuberculosis will have infection prevention needs beyond those of a general pediatric office.

Facility administrators should also assure that facility policies and procedures address occupational health needs including vaccination of HCP, management of exposures or infections in personnel requiring post-exposure prophylaxis and/or work restrictions, and compliance with OSHA bloodborne pathogen standards. Recommendations for prevention of infections in HCP can be found in the following documents: Guideline for infection control in healthcare personnel (available at: <http://www.cdc.gov/hicpac/pdf/InfectControl98.pdf>), Immunization of Health-Care Workers: Recommendations of the Advisory Committee on Immunization (available at: <http://www.cdc.gov/mmwr/preview/mmwrhtml/00050577.htm>), and OSHA Bloodborne Pathogens and Needlestick Prevention (available at: <http://www.osha.gov/SLTC/bloodbornepathogens/index.html>).

Key administrative recommendations for ambulatory care settings:

1. Develop and maintain infection prevention and occupational health programs
2. Assure sufficient and appropriate supplies necessary for adherence to Standard Precautions (e.g., hand hygiene products, personal protective equipment, injection equipment)
3. Assure at least one individual with training in infection prevention is employed by or regularly available to the facility
4. Develop written infection prevention policies and procedures appropriate for the services provided by the facility and based upon evidence-based guidelines, regulations, or standards

Key recommendations for education and training of healthcare personnel in ambulatory care settings:

1. Provide job- or task-specific infection prevention education and training to all HCP
 - a. This includes those employed by outside agencies and available by contract or on a volunteer basis to the facility
2. Training should focus on principles of both HCP safety and patient safety
3. Training should be provided upon orientation and repeated regularly (e.g., annually)
4. Competencies should be documented initially and repeatedly, as appropriate for the specific HCP positions

Educate and Train Healthcare Personnel

Ongoing education and training of HCP are critical for ensuring that infection prevention policies and procedures are understood and followed. Education on the basic principles and practices for preventing the spread of infections should be provided to all HCP. Training should include both HCP safety (e.g., OSHA bloodborne pathogen training) and patient safety, emphasizing job- or task-specific needs. Education and training should be provided upon orientation to the facility and should be repeated regularly (e.g., annually) to maintain competency, including anytime policies or procedures are updated/revised. Competencies should be documented initially and as appropriate for the specific HCP positions. Refer to the *Infection Prevention Checklist for Outpatient Settings* (Appendix A) for an example checklist.

Monitor and Report Healthcare-associated Infections

Surveillance is defined as the ongoing, systematic collection, analysis, interpretation, and dissemination of data regarding a health-related event for use in public health action to reduce morbidity and mortality and to improve health. Surveillance typically refers to tracking of outcome measures (e.g., HAIs) but can also refer to tracking of adherence to specific process measures (e.g., hand hygiene, environmental cleaning) as a means to reduce infection transmission. Surveillance for outcome measures in ambulatory care settings is challenging because patient encounters may be brief or sporadic and evaluation and treatment of consequent infections may involve different healthcare settings (e.g., hospitals).

At a minimum, ambulatory care facilities need to adhere to local, state, and federal requirements regarding reportable disease and outbreak reporting. Certain types of facilities (e.g.,

ambulatory surgical centers) may also be subject to additional HAI surveillance or process measure reporting requirements, for example as part of accreditation, Medicare certification, or state/local statutes. Facilities should check the requirements for their state/region to assure that they are compliant with all regulations and should have contact information for their local and/or state health department available to ensure required reporting is done in a timely manner. (A list of state reportable disease websites is available at: <http://www.cste.org/?StateReportable>)

Regular focused practice surveys or audits (e.g., audits of infection prevention practices including hand hygiene, medication handling and preparation, reprocessing of patient equipment, environmental cleaning) offer a means to assess competencies of HCP as recommended under Education and Training. One example of an audit tool being used by federal surveyors to assess adherence to elements of Standard Precautions in ambulatory surgical centers is available at: http://www.cms.gov/manuals/downloads/som107_exhibit_351.pdf. Another example of a tool is the *Infection Prevention Checklist for Outpatient Settings* (Appendix A), which is a companion to this summary guide.

Key recommendations for HAI surveillance and reporting in ambulatory care settings:

- 1.** Adhere to local, state, and federal requirements regarding HAI surveillance, reportable diseases, and outbreak reporting
- 2.** Perform regular audits and competency evaluations of HCP adherence to infection prevention practices

Adhere to Standard Precautions

Standard Precautions are the minimum infection prevention practices that apply to all patient care, regardless of suspected or confirmed infection status of the patient, in any setting where healthcare is delivered. These practices are designed to both protect HCP and prevent HCP from spreading infections among patients. Standard Precautions include: 1) hand hygiene, 2) use of personal protective equipment (e.g., gloves, gowns, masks), 3) safe injection practices, 4) safe handling of potentially contaminated equipment or surfaces in the patient environment, and 5) respiratory hygiene/cough etiquette. Each of these elements of Standard Precautions are described in the sections that follow.

Education and training on the principles and rationale for recommended practices are critical elements of Standard Precautions because they facilitate appropriate decision-making and promote adherence. Further, at the facility level, an understanding of the specific procedures performed and typical patient interactions, as described above in Administrative Measures as part of policy and procedure development, will assure that necessary equipment is available.

The application of Standard Precautions and guidance on appropriate selection and an example of donning and removal of personal protective equipment is described in detail in the 2007 Guideline for Isolation Precautions (available at: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>).

Hand Hygiene

Good hand hygiene, including use of alcohol-based hand rubs and handwashing with soap and water, is critical to reduce the risk of spreading infections in ambulatory care settings. Use of alcohol-based hand rub as the primary mode of hand hygiene in healthcare settings is

recommended by the CDC and the World Health Organization (WHO) because of its activity against a broad spectrum of epidemiologically important pathogens, and because compared with soap and water, use of ABHR in healthcare settings can increase compliance with recommended hand hygiene practices by requiring less time, irritating hands less, and facilitating hand hygiene at the patient bedside. For these reasons, alcohol-based hand rub is the preferred method for hand hygiene except when hands are visibly soiled (e.g., dirt, blood, body fluids), or after caring for patients with known or suspected infectious diarrhea (e.g., *Clostridium difficile*, norovirus), in which case soap and water should be used.

Complete guidance on how and when hand hygiene should be performed, including recommendations regarding surgical hand antisepsis and artificial nails can be found in the Guideline for Hand Hygiene in Health-Care Settings (available at: <http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf>).

Personal Protective Equipment

Personal Protective Equipment (PPE) refers to wearable equipment that is intended to protect

HCP from exposure to or contact with infectious agents. Examples include gloves, gowns, face masks, respirators, goggles and face shields. The selection of PPE is based on the nature of the patient interaction and potential for exposure to blood, body fluids or infectious agents. Examples of appropriate use of PPE for adherence to Standard Precautions include: use of gloves in situations involving possible contact with blood or body fluids, mucous membranes, non-intact skin or potentially infectious material; use of a gown to protect skin and clothing during procedures or activities where contact with blood or body fluids is anticipated; use of mouth, nose and eye protection during procedures that are likely to generate splashes or sprays of blood or other body fluids. Hand hygiene is always the final step after removing and disposing of PPE.

In addition to protection of HCP, face masks are also effective in limiting the dispersal of oropharyngeal droplets and are recommended when placing a catheter or injecting materials into epidural or subdural spaces, as during myelography or spinal or epidural anesthesia. Failure to wear face masks during these procedures has resulted in development of

Key recommendations for hand hygiene in ambulatory care settings:

1. Key situations where hand hygiene should be performed include:
 - a. Before touching a patient, even if gloves will be worn
 - b. Before exiting the patient's care area after touching the patient or the patient's immediate environment
 - c. After contact with blood, body fluids or excretions, or wound dressings
 - d. Prior to performing an aseptic task (e.g., placing an IV, preparing an injection)
 - e. If hands will be moving from a contaminated-body site to a clean-body site during patient care
 - f. After glove removal
2. Use soap and water when hands are visibly soiled (e.g., blood, body fluids), or after caring for patients with known or suspected infectious diarrhea (e.g., *Clostridium difficile*, norovirus). Otherwise, the preferred method of hand decontamination is with an alcohol-based hand rub.

bacterial meningitis in patients undergoing these procedures¹⁰. Each ambulatory care facility/setting should evaluate the services they provide to determine specific needs and to assure that sufficient and appropriate PPE is available for adherence to Standard Precautions. All HCP at the facility should be educated regarding proper selection and use of PPE.

Complete guidance on the appropriate selection of PPE, including one approach for donning and removing PPE is provided in the 2007 Guideline for Isolation Precautions (available at: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>).

Injection Safety

Injection safety includes practices intended to prevent transmission of infectious diseases between one patient and another, or between a patient and healthcare provider during preparation and administration of parenteral medications.

Implementation of the OSHA Bloodborne Pathogens Standard has helped increase the protection of HCP from blood exposure and sharps injuries, but there is room for improvement in ambulatory care settings. For example, efforts to increase uptake of hepatitis B vaccination and implementation of safety devices that are designed to decrease risks of sharps injury are needed.

Further attention to patient protection is also needed as evidenced by continued outbreaks in ambulatory settings resulting from unsafe injection practices. Unsafe practices that have led to patient harm include 1) use of a single syringe, with or without the same needle, to administer medication to multiple patients, 2) reinsertion of a used syringe, with or without the same needle, into a medication vial or solution container (e.g., saline bag) to obtain additional medication for a single patient and then using that vial or solution container for subsequent patients, 3) preparation of medications in close proximity to contaminated supplies or equipment.

Key recommendations for use of PPE in ambulatory care settings:

1. Facilities should assure that sufficient and appropriate PPE is available and readily accessible to HCP
2. Educate all HCP on proper selection and use of PPE
3. Remove and discard PPE before leaving the patient's room or area
4. Wear gloves for potential contact with blood, body fluids, mucous membranes, non-intact skin or contaminated equipment
 - a. Do not wear the same pair of gloves for the care of more than one patient
 - b. Do not wash gloves for the purpose of reuse
 - c. Perform hand hygiene immediately after removing gloves
5. Wear a gown to protect skin and clothing during procedures or activities where contact with blood or body fluids is anticipated
 - a. Do not wear the same gown for the care of more than one patient
6. Wear mouth, nose and eye protection during procedures that are likely to generate splashes or sprays of blood or other body fluids
7. Wear a surgical mask when placing a catheter or injecting material into epidural or subdural space

Complete guidance on safe injection practices can be found in the 2007 Guideline for Isolation Precautions (available at: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>). Additional materials, including a list of frequently asked questions from providers and a patient notification toolkit, are also available (<http://www.cdc.gov/injectionsafety/>). The *One & Only Campaign* is a public health effort to eliminate unsafe medical injections. The Campaign is led by the Centers for Disease Control and Prevention (CDC) and the Safe Injection Practices Coalition (SIPC). To learn more about safe injection practices, and access training videos and resources, please visit OneandOnlyCampaign.org

Environmental Cleaning

Ambulatory care facilities should establish policies and procedures for routine cleaning and disinfection of environmental surfaces as part of

their infection prevention plan. Cleaning refers to the removal of visible soil and organic contamination from a device or environmental surface using the physical action of scrubbing with a surfactant or detergent and water, or an energy-based process (e.g., ultrasonic cleaners) with appropriate chemical agents. This process removes large numbers of microorganisms from surfaces and must always precede disinfection. Disinfection is generally a less lethal process of microbial inactivation (compared to sterilization) that eliminates virtually all recognized pathogenic microorganisms but not necessarily all microbial forms (e.g., bacterial spores).

Emphasis for cleaning and disinfection should be placed on surfaces that are most

Key recommendations for safe injection practices in ambulatory care settings:

1. Use aseptic technique when preparing and administering medications
2. Cleanse the access diaphragms of medication vials with 70% alcohol before inserting a device into the vial
3. Never administer medications from the same syringe to multiple patients, even if the needle is changed or the injection is administered through an intervening length of intravenous tubing
4. Do not reuse a syringe to enter a medication vial or solution
5. Do not administer medications from single-dose or single-use vials, ampoules, or bags or bottles of intravenous solution to more than one patient
6. Do not use fluid infusion or administration sets (e.g., intravenous tubing) for more than one patient
7. Dedicate multidose vials to a single patient whenever possible. If multidose vials will be used for more than one patient, they should be restricted to a centralized medication area and should not enter the immediate patient treatment area (e.g., operating room, patient room/cubicle)
8. Dispose of used syringes and needles at the point of use in a sharps container that is closable, puncture-resistant, and leak-proof.
9. Adhere to federal and state requirements for protection of HCP from exposure to bloodborne pathogens.

likely to become contaminated with pathogens, including those in close proximity to the patient (e.g., bedrails) and frequently-touched surfaces in the patient-care environment (e.g., doorknobs). Facility policies and procedures should also address prompt and appropriate cleaning and decontamination of spills of blood or other potentially infectious materials.

Responsibility for routine cleaning and disinfection of environmental surfaces should be assigned to appropriately trained HCP. Cleaning procedures can be periodically monitored or assessed to ensure that they are consistently and correctly performed. EPA-registered disinfectants or detergents/disinfectants with label claims for use in healthcare should be selected for disinfection. Disinfectant products should not be used as cleaners unless the label indicates the product is suitable for such use. Healthcare professionals should follow manufacturer's recommendations for use of products selected for cleaning and disinfection (e.g., amount, dilution, contact time, safe use, and disposal).

Complete guidance for the cleaning and disinfection of environmental surfaces, including for cleaning blood or body substance spills, is available in the Guidelines for Environmental Infection Control in Health-Care Facilities (available at: http://www.cdc.gov/hicpac/pdf/guidelines/eic_in_HCF_03.pdf) and the Guideline for Disinfection and Sterilization in Healthcare Facilities (available at: http://www.cdc.gov/hicpac/pdf/guidelines/Disinfection_Nov_2008.pdf).

Key recommendations for cleaning and disinfection of environmental surfaces in ambulatory care settings:

1. Establish policies and procedures for routine cleaning and disinfection of environmental surfaces in ambulatory care settings
 - a. Focus on those surfaces in proximity to the patient and those that are frequently touched
2. Select EPA-registered disinfectants or detergents/disinfectants with label claims for use in healthcare
3. Follow manufacturer's recommendations for use of cleaners and EPA-registered disinfectants (e.g., amount, dilution, contact time, safe use, and disposal)

Medical Equipment

Medical equipment is labeled by the manufacturer as either reusable or single-use. Reusable medical equipment (e.g., endoscopes) should be accompanied by instructions for cleaning and disinfection or sterilization as appropriate. Single-use devices (SUDs) are labeled by the manufacturer for only a single use and do not have reprocessing instructions. They may not be reprocessed except by entities which have complied with FDA regulatory requirements and have received FDA clearance to reprocess specific SUDs as outlined in FDA Guidance for Industry and FDA Staff (available at: <http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm071434>). Legally marketed SUDs are available from FDA-registered Third Party Reprocessors.

All reusable medical equipment must be cleaned and maintained according to the manufacturer's instructions to prevent patient-to-patient transmission of infectious agents. The Spaulding Classification is a traditional approach that has been used to determine the level of disinfection or sterilization required for reusable medical devices, based upon the degree of risk for transmitting infections if the device is contaminated at the time of use.

- ❑ Critical items (e.g., surgical instruments) are objects that enter sterile tissue or the vascular system and must be sterile prior to use.
- ❑ Semi-critical items (e.g., endoscopes used for upper endoscopy and colonoscopy) contact mucous membranes or non-intact skin and require, at a minimum, high-level disinfection prior to reuse.
- ❑ Noncritical items (e.g., blood pressure cuffs) are those that may come in contact with intact skin but not mucous membranes and should undergo low- or intermediate-level disinfection depending on the nature and degree of contamination.
- ❑ Environmental surfaces (e.g., floors, walls) are those that generally do not contact the patient during delivery of care. Cleaning may be all that is needed for the management of these surfaces but if disinfection is indicated, low-level disinfection is appropriate.

Cleaning to remove organic material must always precede disinfection or sterilization because residual debris reduces the effectiveness of the disinfection and sterilization processes.

Facilities should establish policies and procedures for containing, transporting, and handling equipment that may be contaminated with blood or body fluids. Manufacturer's instructions for reprocessing any reusable medical equipment in the facility (including point-of-care devices such as blood glucose meters) should be readily available and used to establish clear and appropriate policies and procedures. Instructions should be posted at the site where equipment reprocessing is performed. Responsibility for cleaning, disinfection and/or sterilization of medical equipment should be assigned to HCP with training in the required reprocessing steps and in the appropriate use of PPE necessary for handling of contaminated equipment. Competencies of HCP responsible for reprocessing of equipment should be documented initially upon assignment of those duties, whenever new equipment is introduced, and periodically (e.g., semi-annually).

Recommendations for the cleaning, disinfection, and sterilization of medical equipment, including general guidance on endoscope reprocessing are available in the Guideline for Disinfection and Sterilization in Healthcare Facilities (available at: http://www.cdc.gov/hicpac/pdf/guidelines/Disinfection_Nov_2008.pdf). Materials specific for the handling of blood glucose monitoring equipment are also available. (<http://www.cdc.gov/injectionsafety/blood-glucose-monitoring.html>)

FDA regulations on reprocessing of single-use devices are available at: <http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm071434> and <http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/ReprocessingofSingle-UseDevices/default.htm>.

Key recommendations for cleaning, disinfection, and/or sterilization of medical equipment in ambulatory care settings:

- 1.** Facilities should ensure that reusable medical equipment (e.g., blood glucose meters and other point-of-care devices, surgical instruments, endoscopes) is cleaned and reprocessed appropriately prior to use on another patient
- 2.** Reusable medical equipment must be cleaned and reprocessed (disinfection or sterilization) and maintained according to the manufacturer's instructions. If the manufacturer does not provide such instructions, the device may not be suitable for multi-patient use
- 3.** Assign responsibilities for reprocessing of medical equipment to HCP with appropriate training
 - a.** Maintain copies of the manufacturer's instructions for reprocessing of equipment in use at the facility; post instructions at locations where reprocessing is performed
 - b.** Observe procedures to document competencies of HCP responsible for equipment reprocessing upon assignment of those duties, whenever new equipment is introduced, and on an ongoing periodic basis (e.g., quarterly)
- 4.** Assure HCP have access to and wear appropriate PPE when handling and reprocessing contaminated patient equipment

Respiratory Hygiene/Cough Etiquette

Respiratory Hygiene/Cough Etiquette is an element of Standard Precautions that highlights the need for prompt implementation of infection prevention measures at the first point of encounter with the facility/ambulatory settings (e.g., reception and triage areas). This strategy is targeted primarily at patients and accompanying family members or friends with undiagnosed transmissible respiratory infections, and applies to any person with signs of illness including cough, congestion, rhinorrhea, or increased production of respiratory secretions when entering the facility.

Additional information related to respiratory hygiene/cough etiquette can be found in the 2007 Guideline for Isolation Precautions (available at: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>). Recommendations for preventing the spread of influenza are available at: <http://www.cdc.gov/flu/professionals/infectioncontrol/>.

Additional Considerations

The majority of ambulatory care settings are not designed to implement all of the isolation practices and other Transmission-Based Precautions (e.g., Airborne Precautions for patients with suspected tuberculosis, measles or chicken pox) that are recommended for hospital settings. Nonetheless, specific syndromes involving diagnostic uncertainty (e.g., diarrhea, febrile respiratory illness, febrile rash) are routinely encountered in ambulatory settings and deserve appropriate triage. Facilities should develop and implement systems for early detection and management of potentially infectious patients at initial points of entry to the facility. To the extent possible, this includes prompt placement

Key recommendations for Respiratory Hygiene/Cough Etiquette in ambulatory care settings:

- 1.** Implement measures to contain respiratory secretions in patients and accompanying individuals who have signs and symptoms of a respiratory infection, beginning at point of entry to the facility and continuing throughout the duration of the visit.
 - a.** Post signs at entrances with instructions to patients with symptoms of respiratory infection to:
 - i.** Cover their mouths/noses when coughing or sneezing
 - ii.** Use and dispose of tissues
 - iii.** Perform hand hygiene after hands have been in contact with respiratory secretions
 - b.** Provide tissues and no-touch receptacles for disposal of tissues
 - c.** Provide resources for performing hand hygiene in or near waiting areas
 - d.** Offer masks to coughing patients and other symptomatic persons upon entry to the facility
 - e.** Provide space and encourage persons with symptoms of respiratory infections to sit as far away from others as possible. If available, facilities may wish to place these patients in a separate area while waiting for care
- 2.** Educate HCP on the importance of infection prevention measures to contain respiratory secretions to prevent the spread of respiratory pathogens when examining and caring for patients with signs and symptoms of a respiratory infection.

of such patients into a single-patient room and a systematic approach to transfer when appropriate. When arranging for patient transfer, facilities should inform the transporting agency and the accepting facility of the suspected infection type.

Additional information related to Transmission-Based Precautions (contact precautions, droplet precautions and airborne precautions) can be found in the 2007 Guideline for Isolation Precautions (available at: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>). Recommendations regarding management of multidrug-resistant organisms can be found in the Guideline for the Management of Multidrug-Resistant Organisms in Healthcare Settings, 2006 available at: <http://www.cdc.gov/hicpac/pdf/guidelines/MDROGuideline2006.pdf>

Conclusions

The recommendations described in the preceding document represent the absolute minimum infection prevention expectations for safe care in outpatient (ambulatory care) settings. This guidance is not all-encompassing. Facilities and HCP are encouraged to refer to the original source documents, which provide more detailed guidance and references for the information included in this document.

SOURCE DOCUMENTS

Source Documents

All evidence-based recommendations for prevention of healthcare-associated infections from CDC/HICPAC can be found at the following site:
<http://www.cdc.gov/hicpac/pubs.html>

Guidelines available at this webpage include:

General

2008 Guideline for Disinfection, and Sterilization in Healthcare Facilities
http://www.cdc.gov/hicpac/Disinfection_Sterilization/1_sumIntroMethTerms.html

Guidelines for Environmental Infection Control in Healthcare Facilities
<http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5210a1.htm>

Guideline for Hand Hygiene in Healthcare Settings
<http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf>

2007 Guideline for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings
http://www.cdc.gov/hicpac/2007IP/2007ip_ExecSummary.html

Guideline for the Prevention of Surgical Site Infection, 1999
<http://www.cdc.gov/ncidod/dhqp/pdf/guidelines/SSI.pdf>

Guidelines for the Prevention of Intravascular Catheter-Related Infections, 2011
<http://www.cdc.gov/hicpac/pdf/guidelines/bsi-guidelines-2011.pdf>

Drug-resistant Organisms

Management of Multi-drug Resistant Organisms in Healthcare Settings, 2006
http://www.cdc.gov/hicpac/mdro/mdro_toc.html

Healthcare Personnel

Influenza Vaccination of Health-Care Personnel, 2006
<http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5502a1.htm>

Guideline for Infection Control in Healthcare Personnel 1998
<http://www.cdc.gov/hicpac/pdf/InfectControl98.pdf>

Specialized Settings

Recommendations for Preventing Transmission of Infections Among Chronic Hemodialysis Patients available at: <http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5005a1.htm>

Guidelines for Infection Control in Dental Health-Care Settings – 2003 available at: <http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5217a1.htm>

Key Links for Additional Information

CDC Website on Healthcare-associated infections:
www.cdc.gov/hai

CDC Website on Hand Hygiene in Healthcare facilities: www.cdc.gov/handhygiene

CDC Website on Injection Safety:
www.cdc.gov/injectionsafety

CDC's *One & Only Campaign*:
www.oneandonlycampaign.org

CDC Website on Influenza: www.cdc.gov/flu

REFERENCES

1. Barie PS. Infection Control Practices in Ambulatory Surgical Centers. *JAMA*. 2010;303:2295-2297.
2. National Center for Health Statistics. *Health, United States, 2009: With Special Features on Medical Technology*. Hyattsville, MD. 2010.
3. Hsiao CJ, Cherry DK, Beatty PC, Rechsteiner EA. National Ambulatory Medical Care Survey: 2007 summary. *National health statistics reports; no 27*. Hyattsville, MD: National Center for Health Statistics. 2010.
4. Halpern MT, Yabroff KR. Prevalence of Outpatient Cancer Treatment in the United States: Estimates from the Medical Panel Expenditures Survey (MEPS). *Cancer Investigation*. 2008;26:647-651.
5. Maki DG, Crnich CJ. History Forgotten is History Relived: Nosocomial Infection Control is also Essential in the Outpatient Setting. *Arch Int Med*. 2005;165:2565-2567.
6. Jarvis WR. Infection control and changing health-care delivery systems. *Emerg Infect Dis* 2001;7:17-173.
7. Williams IT, Perz JF, Bell BP. Viral hepatitis transmission in ambulatory health care settings. *Clin Infect Dis* 2004;38:1592-1598.
8. Goodman RA, Solomon SL. Transmission of Infectious Diseases in Outpatient Health Care Settings. *JAMA*. 1991;265:2377-2381.
9. Thompson ND, Perz JF, Moorman AC, Holmberg SD. Nonhospital Health Care-Associated Hepatitis B and C Virus Transmission: United States, 1998-2008. *Annals of Internal Medicine*. 2009;150: 33-39.
10. CDC. Bacterial Meningitis After Intrapartum Spinal Anesthesia – New York and Ohio, 2008-2009. *MMWR Morb Mortal Wkly Rep* 2010; 59(3):65-69.

APPENDIX A: INFECTION PREVENTION CHECKLIST FOR OUTPATIENT SETTINGS:

Minimum Expectations for Safe Care

The following checklist is a companion to the *Guide to Infection Prevention for Outpatient Settings: Minimum Expectations for Safe Care*. The checklist should be used:

1. To ensure that the facility has appropriate infection prevention policies and procedures in place and supplies to allow healthcare personnel to provide safe care.
2. To systematically assess personnel adherence to correct infection prevention practices. (Assessment of adherence should be conducted by direct observation of healthcare personnel during the performance of their duties.)

Facilities using this checklist should identify all procedures performed in their ambulatory setting and refer to appropriate sections to conduct their evaluation. Certain sections may not apply (e.g., some settings may not perform sterilization or high-level disinfection). If the answer to any of the listed questions is No, efforts should be made to correct the practice, appropriately educate healthcare personnel (if applicable), and determine why the correct practice was not being performed. Consideration should also be made for determining the risk posed to patients by the deficient practice. Certain infection control lapses (e.g., re-use of syringes on more than one patient or to access a medication container that is used for subsequent patients; re-use of lancets) can result in bloodborne pathogen transmission and should be halted immediately. Identification of such lapses warrants immediate consultation with the state or local health department and appropriate notification and testing of potentially affected patients.

Section I: Administrative Policies and Facility Practices

Facility Policies	Practice Performed	If answer is No, document plan for remediation
<p>A. Written infection prevention policies and procedures are available, current, and based on evidence-based guidelines (e.g., CDC/HICPAC), regulations, or standards</p> <p><i>Note: Policies and procedures should be appropriate for the services provided by the facility and should extend beyond OSHA bloodborne pathogen training</i></p>	Yes No	
<p>B. Infection prevention policies and procedures are re-assessed at least annually or according to state or federal requirements</p>	Yes No	
<p>C. At least one individual trained in infection prevention is employed by or regularly available to the facility</p>	Yes No	
<p>D. Supplies necessary for adherence to Standard Precautions are readily available</p> <p><i>Note: This includes hand hygiene products, personal protective equipment, and injection equipment.</i></p>	Yes No	

General Infection Prevention Education and Training

Facility Policies	Practice Performed	If answer is No, document plan for remediation
<p>A. Healthcare Personnel (HCP) receive job-specific training on infection prevention policies and procedures upon hire and at least annually or according to state or federal requirements</p> <p><i>Note: This includes those employed by outside agencies and available by contract or on a volunteer basis to the facility.</i></p>	Yes No	
<p>B. Competency and compliance with job-specific infection prevention policies and procedures are documented both upon hire and through annual evaluations/assessments</p>	Yes No	

Occupational Health

For additional guidance on occupational health recommendations consult the following resource(s):

Guideline for Infection Control in Healthcare Personnel available at:

<http://www.cdc.gov/hicpac/pdf/InfectControl98.pdf>

Immunization of HealthCare Personnel, guidance available at:

<http://www.cdc.gov/vaccines/spec-grps/hcw.htm>

Occupational Safety & Health Administration (OSHA) Bloodborne Pathogens and Needlestick Prevention Standards available at:

<http://www.osha.gov/SLTC/bloodbornepathogens/index.html>

Facility Policies	Practice Performed	If answer is No, document plan for remediation
<p>A. HCP are trained on the OSHA bloodborne pathogen standard upon hire and at least annually</p>	Yes No	
<p>B. The facility maintains a log of needlesticks, sharps injuries, and other employee exposure events</p>	Yes No	
<p>C. Following an exposure event, post-exposure evaluation and follow-up, including prophylaxis as appropriate, are available at no cost to employee and are supervised by a licensed healthcare professional</p>	Yes No	
<p>D. Hepatitis B vaccination is available at no cost to all employees who are at risk of occupational exposure</p>	Yes No	
<p>E. Post-vaccination screening for protective levels of hepatitis B surface antibody is conducted after third vaccine dose is administered</p>	Yes No	

Facility Policies	Practice Performed	If answer is No, document plan for remediation
F. All HCP are offered annual influenza vaccination at no cost	Yes No	
G. All HCP who have potential for exposure to tuberculosis (TB) are screened for TB upon hire and annually (if negative)	Yes No	
H. The facility has a respiratory protection program that details required worksite-specific procedures and elements for required respirator use	Yes No	
I. Respiratory fit testing is provided at least annually to appropriate HCP	Yes No	
J. Facility has written protocols for managing/preventing job-related and community-acquired infections or important exposures in HCP, including notification of appropriate Infection Prevention and Occupational Health personnel when applicable	Yes No	

Surveillance and Disease Reporting

Facility Policies	Practice Performed	If answer is No, document plan for remediation
A. An updated list of diseases reportable to the public health authority is readily available to all personnel	Yes No	
B. The facility can demonstrate compliance with mandatory reporting requirements for notifiable diseases, healthcare associated infections, and for potential outbreaks.	Yes No	

Hand Hygiene

For additional guidance on hand hygiene and resources for training and measurement of adherence, consult the following resource(s).

Guideline for Hand Hygiene in Healthcare Settings available at:

<http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf>

Hand Hygiene in Healthcare Settings available at: <http://www.cdc.gov/handhygiene/>

List of tools that can be used to measure adherence to hand hygiene available at:

http://www.jointcommission.org/assets/1/18/hh_monograph.pdf

Facility Policies	Practice Performed	If answer is No, document plan for remediation
A. The facility provides supplies necessary for adherence to hand hygiene (e.g., soap, water, paper towels, alcohol-based hand rub) and ensures they are readily accessible to HCP in patient care areas	Yes No	
B. HCP are educated regarding appropriate indications for hand washing with soap and water versus hand rubbing with alcohol-based hand rub <i>Note: Soap and water should be used when bare hands are visibly soiled (e.g., blood, body fluids) or after caring for a patient with known or suspected infectious diarrhea (e.g., Clostridium difficile or norovirus). In all other situations, alcohol-based hand rub may be used.</i>	Yes No	
C. The facility periodically monitors and records adherence to hand hygiene and provides feedback to personnel regarding their performance Examples of tools used to record adherence to hand hygiene: http://www.jointcommission.org/assets/1/18/hh_monograph.pdf	Yes No	

Personal Protective Equipment (PPE)

For additional guidance on personal protective equipment consult the following resource(s):

2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings available at: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>

Facility Policies	Practice Performed	If answer is No, document plan for remediation
A. The facility has sufficient and appropriate PPE available and readily accessible to HCP	Yes No	
B. HCP receive training on proper selection and use of PPE	Yes No	

Injection Safety

For additional guidance on injection safety consult the following resource(s): **2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings** available at: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>

CDC Injection Safety Web Materials available at: <http://www.cdc.gov/injectionsafety/>

Frequently Asked Questions (FAQs) regarding Safe Practices for Medical Injections available at: http://www.cdc.gov/injectionsafety/providers/provider_faqs.html

CDC's One & Only Campaign training videos and materials available at: <http://www.oneandonlycampaign.org>

Facility Policies	Practice Performed	If answer is No, document plan for remediation
A. Medication purchasing decisions at the facility reflect selection of vial sizes that most appropriately fit the procedure needs of the facility and limit need for sharing of multi-dose vials	Yes No	
B. Injections are required to be prepared using aseptic technique in a clean area free from contamination or contact with blood, body fluids or contaminated equipment	Yes No	
C. Facility has policies and procedures to track HCP access to controlled substances to prevent narcotics theft/diversion	Yes No	

Respiratory Hygiene/Cough Etiquette

For additional guidance on respiratory hygiene/cough etiquette consult the following resource(s):

2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings available at: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>

Recommendations for preventing the spread of influenza available at: <http://www.cdc.gov/flu/professionals/infectioncontrol/>

Facility Policies	Practice Performed	If answer is No, document plan for remediation
<p>A. The facility has policies and procedures to contain respiratory secretions in persons who have signs and symptoms of a respiratory infection, beginning at point of entry to the facility and continuing through the duration of the visit. Measures include:</p> <ul style="list-style-type: none"> i. Posting signs at entrances (with instructions to patients with symptoms of respiratory infection to cover their mouths/noses when coughing or sneezing, use and dispose of tissues, and perform hand hygiene after hands have been in contact with respiratory secretions.) ii. Providing tissues and no-touch receptacles for disposal of tissues iii. Providing resources for performing hand hygiene in or near waiting areas iv. Offering facemasks to coughing patients and other symptomatic persons upon entry to the facility v. Providing space and encouraging persons with symptoms of respiratory infections to sit as far away from others as possible. If available, facilities may wish to place these patients in a separate area while waiting for care 	<p>Yes No</p> <p>Yes No</p> <p>Yes No</p> <p>Yes No</p> <p>Yes No</p>	
<p>B. The facility educates HCP on the importance of infection prevention measures to contain respiratory secretions to prevent the spread of respiratory pathogens when examining and caring for patients with signs and symptoms of a respiratory infection.</p>	<p>Yes No</p>	

Environmental Cleaning

For additional guidance on environmental cleaning consult the following resource(s):

Guidelines for Environmental Infection Control in Healthcare Facilities available at:

http://www.cdc.gov/hicpac/pdf/guidelines/eic_in_HCF_03.pdf

Facility Policies	Practice Performed	If answer is No, document plan for remediation
A. Facility has written policies and procedures for routine cleaning and disinfection of environmental services, including identification of responsible personnel	Yes No	
B. Environmental services staff receive job-specific training and competency validation at hire and when procedures/policies change	Yes No	
C. Training and equipment are available to ensure that HCP wear appropriate PPE to preclude exposure to infectious agents or chemicals (PPE can include gloves, gowns, masks, and eye protection)	Yes No	
D. Cleaning procedures are periodically monitored and assessed to ensure that they are consistently and correctly performed	Yes No	
E. The facility has a policy/procedure for decontamination of spills of blood or other body fluids	Yes No	

Reprocessing of Reusable Medical Devices

The following basic information allows for a general assessment of policies and procedures related to reprocessing of reusable medical devices. Ambulatory facilities that are providing on-site sterilization or high-level disinfection of reusable medical equipment should refer to the more detailed checklists related to sterilization and high-level disinfection in separate sections of this document devoted to those issues.

Critical items (e.g., surgical instruments) are objects that enter sterile tissue or the vascular system and must be sterile prior to use (see Sterilization Section).

Semi-critical items (e.g., endoscopes for upper endoscopy and colonoscopy, vaginal probes) are objects that contact mucous membranes or non-intact skin and require, at a minimum, high-level disinfection prior to reuse (see High-level Disinfection Section).

Non-critical items (e.g., blood pressure cuffs) are objects that may come in contact with intact skin but not mucous membranes and should undergo cleaning and low- or intermediate-level disinfection depending on the nature and degree of contamination.

Single-use devices (SUDs) are labeled by the manufacturer for a single use and do not have reprocessing instructions. They may not be reprocessed for reuse except by entities which have complied with FDA regulatory requirements and have received FDA clearance to reprocess specific SUDs.

Note: Pre-cleaning must always be performed prior to sterilization and/or disinfection

For additional guidance on reprocessing of medical devices consult the manufacturer instructions for the device and the following resource(s):

Guideline for Disinfection and Sterilization in Healthcare Facilities available at:

http://www.cdc.gov/hicpac/pdf/guidelines/Disinfection_Nov_2008.pdf

FDA regulations on reprocessing of single-use medical devices available at:

<http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm071434>

Facility Policies	Practice Performed	If answer is No, document plan for remediation
<p>A. Facility has policies and procedures to ensure that reusable medical devices are cleaned and reprocessed appropriately prior to use on another patient</p> <p><i>Note: This includes clear delineation of responsibility among HCP.</i></p>	Yes No	
<p>B. Policies, procedures, and manufacturer reprocessing instructions for reusable medical devices used in the facility are available in the reprocessing area(s)</p>	Yes No	
<p>C. HCP responsible for reprocessing reusable medical devices are appropriately trained and competencies are regularly documented (at least annually and when new equipment is introduced)</p>	Yes No	
<p>D. Training and equipment are available to ensure that HCP wear appropriate PPE to prevent exposure to infectious agents or chemicals (PPE can include gloves, gowns, masks, and eye protection).</p> <p><i>Note: The exact type of PPE depends on infectious or chemical agent and anticipated type of exposure.</i></p>	Yes No	

Sterilization of Reusable Instruments and Devices

For additional guidance on sterilization of medical devices consult the manufacturer instructions for the device and the following resource(s):

Guideline for Disinfection and Sterilization in Healthcare Facilities available at:

http://www.cdc.gov/hicpac/pdf/guidelines/Disinfection_Nov_2008.pdf

Facility Policies	Practice Performed	If answer is No, document plan for remediation
A. All reusable critical instruments and devices are sterilized prior to reuse	Yes No	
B. Routine maintenance for sterilization equipment is performed according to manufacturer instructions (confirm maintenance records are available)	Yes No	
C. Policies and procedures are in place outlining facility response (i.e., recall of device and risk assessment) in the event of a reprocessing error/failure.	Yes No	

High-Level Disinfection of Reusable Instruments and Devices

For additional guidance on reprocessing of high-level disinfection devices consult the manufacturer instructions for the device and the following resource(s):

Guideline for Disinfection and Sterilization in Healthcare Facilities available at:

http://www.cdc.gov/hicpac/pdf/guidelines/Disinfection_Nov_2008.pdf

Facility Policies	Practice Performed	If answer is No, document plan for remediation
A. All reusable semi-critical items receive at least high-level disinfection prior to reuse	Yes No	
B. The facility has a system in place to identify which instrument (e.g., endoscope) was used on a patient via a log for each procedure	Yes No	
C. Routine maintenance for high-level disinfection equipment is performed according to manufacturer instructions; confirm maintenance records are available	Yes No	

Additional Resources and Evidence-based Guidelines available at:

http://www.cdc.gov/HAI/prevent/prevent_pubs.html

Section II: Personnel and Patient-care Observations

Hand hygiene performed correctly	Practice Performed	If answer is No, document plan for remediation
A. Before contact with the patient or their immediate care environment (even if gloves are worn)	Yes No	
B. Before exiting the patient's care area after touching the patient or the patient's immediate environment (even if gloves are worn)	Yes No	
C. Before performing an aseptic task (e.g., insertion of IV or preparing an injection) (even if gloves are worn)	Yes No	
D. After contact with blood, body fluids or contaminated surfaces (even if gloves are worn)	Yes No	
E. When hands move from a contaminated-body site to a clean-body site during patient care (even if gloves are worn)	Yes No	

Person Protective Equipment (PPE) is correctly used	Practice Performed	If answer is No, document plan for remediation
A. PPE is removed and discarded prior to leaving the patient's room or care area	Yes No	
B. Hand hygiene is performed immediately after removal of PPE	Yes No	
C. Gloves <ul style="list-style-type: none"> i. HCP wear gloves for potential contact with blood, body fluids, mucous membranes, non-intact skin, or contaminated equipment ii. HCP <u>do not</u> wear the same pair of gloves for the care of more than one patient iii. HCP <u>do not</u> wash gloves for the purpose of reuse 	Yes No Yes No Yes No	
D. Gowns: <ul style="list-style-type: none"> i. HCP wear gowns to protect skin and clothing during procedures or activities where contact with blood or body fluids is anticipated ii. HCP <u>do not</u> wear the same gown for the care of more than one patient 	Yes No Yes No	
E. Facial protection: <ul style="list-style-type: none"> i. HCP wear mouth, nose, and eye protection during procedures that are likely to generate splashes or sprays of blood or other body fluids ii. HCP wear a facemask (e.g., surgical mask) when placing a catheter or injecting material into the epidural or subdural space (e.g., during myelogram, epidural or spinal anesthesia) 	Yes No Yes No	

Injection Safety	Practice Performed	If answer is No, document plan for remediation
A. Needles and syringes are used for only one patient (this includes manufactured prefilled syringes and cartridge devices such as insulin pens)	Yes No	
B. The rubber septum on a medication vial is disinfected with alcohol prior to piercing	Yes No	
C. Medication vials are entered with a new needle and a new syringe, even when obtaining additional doses for the same patient	Yes No	
D. Single dose (single-use) medication vials, ampules, and bags or bottles of intravenous solution are used for only one patient	Yes No	
E. Medication administration tubing and connectors are used for only one patient	Yes No	
<p>F. Multi-dose vials are dated by HCP when they are first opened and discarded within 28 days unless the manufacturer specifies a different (shorter or longer) date for that opened vial</p> <p><i>Note: This is different from the expiration date printed on the vial.</i></p>	Yes No	
G. Multi-dose vials are dedicated to individual patients whenever possible.	Yes No	
<p>H. Multi-dose vials to be used for more than one patient are kept in a centralized medication area and <u>do not</u> enter the immediate patient treatment area (e.g., operating room, patient room/cubicle)</p> <p><i>Note: If multi-dose vials enter the immediate patient treatment area they should be dedicated for single-patient use and discarded immediately after use.</i></p>	Yes No	
I. All sharps are disposed of in a puncture-resistant sharps container	Yes No	
J. Filled sharps containers are disposed of in accordance with state regulated medical waste rules	Yes No	
K. All controlled substances (e.g., Schedule II, III, IV, V drugs) are kept locked within a secure area	Yes No	

Point-of-Care Testing (e.g., blood glucose meters, INR monitor)

For additional guidance on infection prevention during point-of-care testing consult the following resource(s):

Infection Prevention during Blood Glucose Monitoring and Insulin Administration available at:

<http://www.cdc.gov/injectionsafety/blood-glucose-monitoring.html>

Frequently Asked Questions (FAQs) regarding Assisted Blood Glucose Monitoring and Insulin Administration available at:

http://www.cdc.gov/injectionsafety/providers/blood-glucose-monitoring_faqs.html

Point-of-Care Testing	Practice Performed	If answer is No, document plan for remediation
A. New single-use, auto-disabling lancing device is used for each patient <i>Note: Lancet holder devices are not suitable for multi-patient use.</i>	Yes No	
B. If used for more than one patient, the point-of-care testing meter is cleaned and disinfected after every use according to manufacturer instructions <i>Note: If the manufacturer does not provide instructions for cleaning and disinfection, then the testing meter should not be used for >1 patient.</i>	Yes No	

Environmental Cleaning	Practice Performed	If answer is No, document plan for remediation
A. Environmental surfaces, with an emphasis on surfaces in proximity to the patient and those that are frequently touched, are cleaned and then disinfected with an EPA-registered disinfectant	Yes No	
B. Cleaners and disinfectants are used in accordance with manufacturer instructions (e.g., dilution, storage, shelf-life, contact time)	Yes No	

Reprocessing of Reusable Instruments and Devices	Practice Performed	If answer is No, document plan for remediation
<p>A. Reusable medical devices are cleaned, reprocessed (disinfection or sterilization) and maintained according to the manufacturer instructions.</p> <p><i>Note: If the manufacturer does not provide such instructions, the device may not be suitable for multi-patient use.</i></p>	Yes No	
<p>B. Single-use devices are discarded after use and not used for more than one patient.</p> <p><i>Note: If the facility elects to reuse single-use devices, these devices must be reprocessed prior to reuse by a third-party reprocessor that it is registered with the FDA as a third-party reprocessor and cleared by the FDA to reprocess the specific device in question. The facility should have documentation from the third party reprocessor confirming this is the case.</i></p>	Yes No	
<p>C. Reprocessing area has a workflow pattern such that devices clearly flow from high contamination areas to clean/sterile areas (i.e., there is clear separation between soiled and clean workspaces)</p>	Yes No	
<p>D. Medical devices are stored in a manner to protect from damage and contamination</p>	Yes No	

Sterilization of Reusable Instruments and Devices	Practice Performed	If answer is No, document plan for remediation
<p>A. Items are thoroughly pre-cleaned according to manufacturer instructions and visually inspected for residual soil prior to sterilization</p> <p><i>Note: For lumened instruments, device channels and lumens must be cleaned using appropriately sized cleaning brushes.</i></p>	Yes No	
<p>B. Enzymatic cleaner or detergent is used for pre-cleaning and discarded according to manufacturer instructions (typically after each use)</p>	Yes No	
<p>C. Cleaning brushes are disposable or cleaned and high-level disinfected or sterilized (per manufacturer instructions) after each use</p>	Yes No	
<p>D. After pre-cleaning, instruments are appropriately wrapped/packaged for sterilization (e.g., package system selected is compatible with the sterilization process being performed, hinged instruments are open, instruments are disassembled if indicated by the manufacturer)</p>	Yes No	
<p>E. A chemical indicator (process indicator) is placed correctly in the instrument packs in every load</p>	Yes No	
<p>F. A biological indicator is used at least weekly for each sterilizer and with every load containing implantable items</p>	Yes No	
<p>G. For dynamic air removal-type sterilizers, a Bowie-Dick test is performed each day the sterilizer is used to verify efficacy of air removal</p>	Yes No	
<p>H. Sterile packs are labeled with the sterilizer used, the cycle or load number, and the date of sterilization</p>	Yes No	
<p>I. Logs for each sterilizer cycle are current and include results from each load</p>	Yes No	
<p>J. After sterilization, medical devices and instruments are stored so that sterility is not compromised</p>	Yes No	
<p>K. Sterile packages are inspected for integrity and compromised packages are reprocessed prior to use</p>	Yes No	
<p>L. Immediate-use steam sterilization (flash sterilization), if performed, is only done in circumstances in which routine sterilization procedures cannot be performed</p>	Yes No	
<p>M. Instruments that are flash-sterilized are used immediately and not stored</p>	Yes No	

<p align="center">High-Level Disinfection of Resuable Instruments and Devices</p>	<p align="center">Practice Performed</p>	<p align="center">If answer is No, document plan for remediation</p>
<p>A. Flexible endoscopes are inspected for damage and leak tested as part of each reprocessing cycle</p>	<p align="center">Yes No</p>	
<p>B. Items are thoroughly pre-cleaned according to manufacturer instructions and visually inspected for residual soil prior to high-level disinfection</p> <p><i>Note: For lumened instruments, device channels and lumens must be cleaned using appropriately sized cleaning brushes.</i></p>	<p align="center">Yes No</p>	
<p>C. Enzymatic cleaner or detergent is used and discarded according to manufacturer instructions (typically after each use)</p>	<p align="center">Yes No</p>	
<p>D. Cleaning brushes are disposable or cleaned and high-level disinfected or sterilized (per manufacturer instructions) after each use.</p>	<p align="center">Yes No</p>	
<p>E. For chemicals used in high-level disinfection, manufacturer instructions are followed for:</p> <ul style="list-style-type: none"> i. preparation ii. testing for appropriate concentration iii. replacement (i.e., prior to expiration or loss of efficacy) 	<p align="center">Yes No Yes No Yes No</p>	
<p>F. If automated reprocessing equipment is used, proper connectors are used to assure that channels and lumens are appropriately disinfected</p>	<p align="center">Yes No</p>	
<p>G. Devices are disinfected for the appropriate length of time as specified by manufacturer instructions</p>	<p align="center">Yes No</p>	
<p>H. Devices are disinfected at the appropriate temperature as specified by manufacturer instructions</p>	<p align="center">Yes No</p>	
<p>I. After high-level disinfection, devices are rinsed with sterile water, filtered water, or tap water followed by a rinse with 70% - 90% ethyl or isopropyl alcohol</p>	<p align="center">Yes No</p>	
<p>J. Devices are dried thoroughly prior to reuse</p> <p><i>Note: Lumened instruments (e.g., endoscopes) require flushing channels with alcohol and forcing air through channels.</i></p>	<p align="center">Yes No</p>	
<p>K. After high-level disinfection, devices are stored in a manner to protect from damage or contamination</p> <p><i>Note: Endoscopes should be hung in a vertical position</i></p>	<p align="center">Yes No</p>	

Additional Resources and Evidence-based Guidelines available at:
http://www.cdc.gov/HAI/prevent/prevent_pubs.html

The recommendations in this guideline for Ebola Virus Disease have been superseded by CDC's [Infection Prevention and Control Recommendations for Hospitalized Patients with Known or Suspected Ebola Virus Disease in U.S. Hospitals](#).

This information is in [Appendix A](#).

Click here for current information on [how Ebola virus is transmitted](#).

The recommendations in this guideline for Measles have been superseded by [CDC's Immunization of Healthcare Personnel: Recommendations of the Advisory Committee on Immunization Practices \(ACIP\)](#).

2007 Guideline for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings

**Jane D. Siegel, MD; Emily Rhinehart, RN MPH CIC; Marguerite Jackson, PhD;
Linda Chiarello, RN MS; the Healthcare Infection Control Practices Advisory
Committee**

Acknowledgement: The authors and HICPAC gratefully acknowledge Dr. Larry Strausbaugh for his many contributions and valued guidance in the preparation of this guideline.

Suggested citation: Siegel JD, Rhinehart E, Jackson M, Chiarello L, and the Healthcare Infection Control Practices Advisory Committee, 2007 Guideline for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings
<http://www.cdc.gov/ncidod/dhqp/pdf/isolation2007.pdf>

**Healthcare Infection Control Practices
Advisory Committee (HICPAC):**

Chair

Patrick J. Brennan, MD
Professor of Medicine
Division of Infectious Diseases
University of Pennsylvania Medical School

Executive Secretary

Michael Bell, MD
Division of Healthcare Quality Promotion
National Center for Infectious Diseases
Centers for Disease Control and Prevention

Members

BRINSKO, Vicki L., RN, BA Infection
Control Coordinator Vanderbilt
University Medical Center

DELLINGER, E. Patchen., MD
Professor of Surgery
University of Washington School of
Medicine

ENGEL, Jeffrey, MD
Head General Communicable Disease
Control
Branch
North Carolina State Epidemiologist

GORDON, Steven M., MD
Chairman, Department of Infections
Diseases
Hospital Epidemiologist
Cleveland Clinic Foundation
Department of Infectious Disease

HARRELL, Lizzie J., PhD, D(ABMM)
Research Professor of Molecular Genetics,
Microbiology and Pathology
Associate Director, Clinical Microbiology
Duke University Medical Center

O'BOYLE, Carol, PhD, RN
Assistant Professor, School of Nursing
University of Minnesota

PEGUES, David Alexander, MD
Division of Infectious Diseases
David Geffen School of Medicine at UCLA

PERROTTA, Dennis M. PhD., CIC
Adjunct Associate Professor of
Epidemiology
University of Texas School of Public Health
Texas A&M University School of Rural
Public
Health

PITT, Harriett M., MS, CIC, RN
Director, Epidemiology
Long Beach Memorial Medical Center

RAMSEY, Keith M., MD
Professor of Medicine
Medical Director of Infection Control
The Brody School of Medicine at East
Carolina
University

SINGH, Nalini, MD, MPH
Professor of Pediatrics
Epidemiology and International Health
The George Washington University
Children's National
Medical Center

STEVENSON, Kurt Brown, MD, MPH
Division of Infectious Diseases
Department of Internal Medicine
The Ohio State University Medical Center

SMITH, Philip W., MD
Chief, Section of Infectious Diseases
Department of Internal Medicine
University of Nebraska Medical Center

HICPAC membership (past)

Robert A. Weinstein, MD (Chair)
Cook County Hospital
Chicago, IL

Jane D. Siegel, MD (Co-Chair)
University of Texas Southwestern Medical
Center
Dallas, TX

Michele L. Pearson, MD
(Executive Secretary)
Centers for Disease Control and Prevention
Atlanta, GA

Raymond Y.W. Chinn, MD
Sharp Memorial Hospital
San Diego, CA

Alfred DeMaria, Jr, MD
Massachusetts Department of Public Health
Jamaica Plain, MA

James T. Lee, MD, PhD
University of Minnesota
Minneapolis, MN

William A. Rutala, PhD, MPH
University of North Carolina Health Care
System
Chapel Hill, NC

William E. Scheckler, MD
University of Wisconsin
Madison, WI

Beth H. Stover, RN
Kosair Children's Hospital
Louisville, KY

Marjorie A. Underwood, RN, BSN CIC
Mt. Diablo Medical Center
Concord, CA

HICPAC Liaisons

William B. Baine, MD
Liaison to Agency for Healthcare Quality
Research

Joan Blanchard, RN, MSN, CNOR
Liaison to Association of periOperative
Registered Nurses

Patrick J. Brennan, MD
Liaison to Board of Scientific Counselors

Nancy Bjerke, RN, MPH, CIC
Liaison to Association of Professionals in
Infection Prevention and Control

Jeffrey P. Engel, MD
Liaison to Advisory Committee on
Elimination of
Tuberculosis

David Henderson, MD
Liaison to National Institutes of Health

Lorine J. Jay MPH, RN, CPHQ
Liaison to Healthcare Resources Services
Administration

Stephen F. Jencks, MD, MPH
Liaison to Center for Medicare and Medicaid
Services

Sheila A. Murphey, MD
Liaison to Food and Drug Administration

Mark Russi, MD, MPH
Liaison to American College of Occupational
and
Environmental Medicine

Rachel L. Stricof, MPH
Liaison to Advisory Committee on
Elimination of
Tuberculosis

Michael L. Tapper, MD
Liaison to Society for Healthcare
Epidemiology of
America

Robert A. Wise, MD
Liaison to Joint Commission on the
Accreditation of
Healthcare Organizations

Authors' Associations

Jane D. Siegel, MD
Professor of Pediatrics
Department of Pediatrics
University of Texas Southwestern Medical
Center

Emily Rhinehart RN MPH CIC CPHQ
Vice President
AIG Consultants, Inc.

Marguerite Jackson, RN PhD CIC
Director, Administrative Unit, National
Tuberculosis
Curriculum Consortium, Department of
Medicine
University of California San Diego

Linda Chiarello, RN MS
Division of Healthcare Quality Promotion
National Center for Infectious Diseases,
CDC

TABLE OF CONTENTS

Executive Summary	7
Abbreviations.....	11
Part I: Review of the Scientific Data Regarding Transmission of Infectious Agents in Healthcare Settings.....	12
I.A. Evolution of the 2007 document	12
I.B. Rationale for Standard and Transmission-Based Precautions in healthcare settings.....	14
I.B.1. Source of infectious agents.....	14
I.B.2. Susceptible hosts.....	14
I.B.3. Modes of transmission	15
I.B.3.a. Contact transmission.....	15
I.B.3.a.i. Direct contact transmission	16
I.B.3.a.ii. Indirect contact transmission	16
I.B.3.b. Droplet transmission	17
I.B.3.c. Airborne transmission.....	18
I.B.3.d. Emerging issues and controversies concerning bioaerosols and airborne transmission of infectious agents.....	19
I.B.3.d.i. Transmission from patients.....	19
I.B.3.d.ii. Transmission from the environment.....	20
I.B.3.e. Other sources of infection	20
I.C. Infectious agents of special infection control interest for healthcare settings.....	20
I.C.1. Epidemiologically important organisms.....	21
I.C.1.a. <i>Clostridium difficile</i>	21
I.C.1.b. Multidrug-resistant organisms(MDROs).....	22
I.C.2. Agents of bioterrorism.....	23
I.C.3. Prions	24
I.C.4. Severe acute respiratory syndrome (SARS)	26
I.C.5. Monkeypox	28
I.C.6. Noroviruses.....	28
I.C.7. Hemorrhagic fever viruses.....	29
I.D. Transmission risks associated with specific types of healthcare settings	31
I.D.1. Hospitals.....	31
I.D.1.a. Intensive care units	31
I.D.1.b. Burn units.....	32
I.D.1.c. Pediatrics	33
I.D.2. Non-acute care settings.....	34
I.D.2.a. Long term care	34
I.D.2.b. Ambulatory care settings	35
I.D.2.c. Home care	36
I.D.2.d. Other sites of healthcare delivery	37
I.E. Transmission risks associated with special patient populations.....	38
I.E.1. Immunocompromised patients	38
I.E.2. Cystic fibrosis patients	39

I.F. New therapies with potential transmissible infectious agents.....	39
I.F.1. Gene therapy	39
I.F.2. Infections Transmitted through Blood, Organs and Tissues.....	40
I.F.3. Xenotransplantation and tissue allografts	40

Part II. Fundamental Elements to Prevent Transmission of Infectious Agents in Healthcare Settings..... 41

II.A. Healthcare system components that influence the effectiveness of precautions to prevent transmission	41
II.A.1. Administrative measures.....	41
II.A.1.a. Scope of Work and Staffing Needs for Infection Control Professionals (ICP).....	42
II.A.1.a.i. Infection Control Liaison Nurse.....	43
II.A.1.b. Bedside nurse staffing.....	43
II.A.1.c. Clinical microbiology laboratory support.....	43
II.A.2. Institutional safety culture and organizational characteristics.....	45
II.A.3. Adherence of healthcare personnel to recommended guidelines....	45
II.B. Surveillance for healthcare-associated infections (HAIs).....	46
II.C. Education of healthcare workers, patients, and families	47
II.D. Hand hygiene.....	49
II.E. Personal protective equipment for healthcare personnel	49
II.E.1. Gloves.....	50
II.E.2. Isolation gowns	51
II.E.3. Face protection: masks, goggles, face shields.....	52
II.E.3.a. Masks.....	52
II.E.3.b. Goggles, face shields.....	52
II.E.4. Respiratory protection	53
II.F. Safe work practices to prevent HCW exposure to bloodborne pathogens	55
II.F.1. Prevention of needlesticks and other sharps-related injuries	55
II.F.2. Prevention of mucous membrane contact.....	56
II.F.2.a. Precautions during aerosol-generating procedures.....	56
II.G. Patient placement.....	56
II.G.1. Hospitals and long-term care settings	56
II.G.2. Ambulatory care settings	58
II.G.3. Home care	59
II.H. Transport of patients.....	59
II.I. Environmental measures.....	60
II.J. Patient care equipment, instruments/devices	61
II.K. Textiles and laundry	61
II.L. Solid waste.....	62
II.M. Dishware and eating utensils.....	62
II.N. Adjunctive measures	63
II.N.1. Chemoprophylaxis	63
II.N.2. Immunoprophylaxis.....	63

II.N.3. Management of visitors.....	64
II.N.3.a. Visitors as sources of infection.....	64
II.N.3.b. Use of barrier precautions by visitors.....	65
Part III. HICPAC Precautions to Prevent Transmission of Infectious Agents	66
III.A. Standard Precautions	66
III.A.1. New Standard Precautions for patients.....	67
III.A.1.a. Respiratory Hygiene/Cough Etiquette.....	67
III.A.1.b. Safe Injection Practices.....	68
III.A.1.c. Infection Control Practices for Special Lumbar Puncture Procedures.....	69
III.B. Transmission-Based Precautions	69
III.B.1. Contact Precautions.....	70
III.B.2. Droplet Precautions	70
III.B.3. Airborne Infection Isolation Precautions.....	71
III.C. Syndromic or empiric application of Transmission-Based Precautions.....	71
III.D. Discontinuation of precautions.....	72
III.E. Application of Transmission-Based Precautions in ambulatory and home care settings.....	72
III.F. Protective environment (PE)	73
Part IV: Recommendations.....	74
Appendix A. Type and duration of precautions needed for selected infections and conditions	93
Tables	
Table 1. Recent history of guidelines for prevention of healthcare-associated infections.....	
Table 2. Clinical syndromes or conditions warranting additional empiric transmission-based precautions pending confirmation of diagnosis.....	
Table 3. Infection control considerations for high-priority (CDC Category A) diseases that may result from bioterrorist attacks or are considered to be bioterrorist threats ..	
Table 4. Recommendations for application of Standard Precautions for the care of all patients in all healthcare settings.....	
Table 5. Components of a Protective Environment	
Figure Sequence for donning and removing PPE	
Glossary.....	
References.....	

EXECUTIVE SUMMARY

The *Guideline for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings 2007* updates and expands the *1996 Guideline for Isolation Precautions in Hospitals*. The following developments led to revision of the 1996 guideline:

1. The transition of healthcare delivery from primarily acute care hospitals to other healthcare settings (e.g., home care, ambulatory care, free-standing specialty care sites, long-term care) created a need for recommendations that can be applied in all healthcare settings using common principles of infection control practice, yet can be modified to reflect setting-specific needs. Accordingly, the revised guideline addresses the spectrum of healthcare delivery settings. Furthermore, the term “nosocomial infections” is replaced by “healthcare-associated infections” (HAIs) to reflect the changing patterns in healthcare delivery and difficulty in determining the geographic site of exposure to an infectious agent and/or acquisition of infection.
2. The emergence of new pathogens (e.g., SARS-CoV associated with the severe acute respiratory syndrome [SARS], Avian influenza in humans), renewed concern for evolving known pathogens (e.g., *C. difficile*, noroviruses, community-associated MRSA [CA-MRSA]), development of new therapies (e.g., gene therapy), and increasing concern for the threat of bioweapons attacks, established a need to address a broader scope of issues than in previous isolation guidelines.
3. The successful experience with Standard Precautions, first recommended in the 1996 guideline, has led to a reaffirmation of this approach as the foundation for preventing transmission of infectious agents in all healthcare settings. New additions to the recommendations for Standard Precautions are Respiratory Hygiene/Cough Etiquette and safe injection practices, including the use of a mask when performing certain high-risk, prolonged procedures involving spinal canal punctures (e.g., myelography, epidural anesthesia). The need for a recommendation for Respiratory Hygiene/Cough Etiquette grew out of observations during the SARS outbreaks where failure to implement simple source control measures with patients, visitors, and healthcare personnel with respiratory symptoms may have contributed to SARS coronavirus (SARS-CoV) transmission. The recommended practices have a strong evidence base. The continued occurrence of outbreaks of hepatitis B and hepatitis C viruses in ambulatory settings indicated a need to re-iterate safe injection practice recommendations as part of Standard Precautions. The addition of a mask for certain spinal injections grew from recent evidence of an associated risk for developing meningitis caused by respiratory flora.
4. The accumulated evidence that environmental controls decrease the risk of life-threatening fungal infections in the most severely immunocompromised patients (allogeneic hematopoietic stem-cell transplant patients) led to the update on the components of the Protective Environment (PE).
5. Evidence that organizational characteristics (e.g., nurse staffing levels and composition, establishment of a safety culture) influence healthcare personnel adherence to recommended infection control practices, and therefore are important factors in preventing transmission of infectious agents, led to a new

emphasis and recommendations for administrative involvement in the development and support of infection control programs.

6. Continued increase in the incidence of HAIs caused by multidrug-resistant organisms (MDROs) in all healthcare settings and the expanded body of knowledge concerning prevention of transmission of MDROs created a need for more specific recommendations for surveillance and control of these pathogens that would be practical and effective in various types of healthcare settings.

This document is intended for use by infection control staff, healthcare epidemiologists, healthcare administrators, nurses, other healthcare providers, and persons responsible for developing, implementing, and evaluating infection control programs for healthcare settings across the continuum of care. The reader is referred to other guidelines and websites for more detailed information and for recommendations concerning specialized infection control problems.

Parts I - III: Review of the Scientific Data Regarding Transmission of Infectious Agents in Healthcare Settings

Part I reviews the relevant scientific literature that supports the recommended prevention and control practices. As with the 1996 guideline, the modes and factors that influence transmission risks are described in detail. New to the section on transmission are discussions of bioaerosols and of how droplet and airborne transmission may contribute to infection transmission. This became a concern during the SARS outbreaks of 2003, when transmission associated with aerosol-generating procedures was observed. Also new is a definition of “epidemiologically important organisms” that was developed to assist in the identification of clusters of infections that require investigation (i.e. multidrug-resistant organisms, *C. difficile*). Several other pathogens that hold special infection control interest (i.e., norovirus, SARS, Category A bioterrorist agents, prions, monkeypox, and the hemorrhagic fever viruses) also are discussed to present new information and infection control lessons learned from experience with these agents. This section of the guideline also presents information on infection risks associated with specific healthcare settings and patient populations.

Part II updates information on the basic principles of hand hygiene, barrier precautions, safe work practices and isolation practices that were included in previous guidelines. However, new to this guideline, is important information on healthcare system components that influence transmission risks, including those under the influence of healthcare administrators. An important administrative priority that is described is the need for appropriate infection control staffing to meet the ever-expanding role of infection control professionals in the modern, complex healthcare system. Evidence presented also demonstrates another administrative concern, the importance of nurse staffing levels, including numbers of appropriately trained nurses in ICUs for preventing HAIs. The role of the clinical microbiology laboratory in supporting infection control is described to emphasize the need for this service in healthcare facilities. Other factors that influence transmission risks are discussed i.e., healthcare worker adherence to recommended infection control practices, organizational safety culture or climate, education and training. Discussed for the first time in an isolation guideline is surveillance of healthcare-associated infections. The information presented will be useful to new infection control professionals as

well as persons involved in designing or responding to state programs for public reporting of HAI rates.

Part III describes each of the categories of precautions developed by the Healthcare Infection Control Practices Advisory Committee (HICPAC) and the Centers for Disease Control and Prevention (CDC) and provides guidance for their application in various healthcare settings. The categories of Transmission-Based Precautions are unchanged from those in the 1996 guideline: Contact, Droplet, and Airborne. One important change is the recommendation to don the indicated personal protective equipment (gowns, gloves, mask) *upon entry into the patient's room* for patients who are on Contact and/or Droplet Precautions since the nature of the interaction with the patient cannot be predicted with certainty and contaminated environmental surfaces are important sources for transmission of pathogens.

In addition, the Protective Environment (PE) for allogeneic hematopoietic stem cell transplant patients, described in previous guidelines, has been updated.

Tables, Appendices, and other Information

There are several tables that summarize important information: 1) a summary of the evolution of this document; 2) guidance on using empiric isolation precautions according to a clinical syndrome; 3) a summary of infection control recommendations for category A agents of bioterrorism; 4) components of Standard Precautions and recommendations for their application; 5) components of the Protective Environment; and 6) a glossary of definitions used in this guideline. New in this guideline is a figure that shows a recommended sequence for donning and removing personal protective equipment used for isolation precautions to optimize safety and prevent self-contamination during removal.

Appendix A: Type and Duration of Precautions Recommended for Selected Infections and Conditions

Appendix A consists of an updated alphabetical list of most infectious agents and clinical conditions for which isolation precautions are recommended. A preamble to the Appendix provides a rationale for recommending the use of one or more Transmission-Based Precautions, in addition to Standard Precautions, based on a review of the literature and evidence demonstrating a real or potential risk for person-to-person transmission in healthcare settings. The type and duration of recommended precautions are presented with additional comments concerning the use of adjunctive measures or other relevant considerations to prevent transmission of the specific agent. Relevant citations are included.

Pre- Publication of the Guideline on Preventing Transmission of MDROs

New to this guideline is a comprehensive review and detailed recommendations for prevention of transmission of MDROs. This portion of the guideline was published electronically in October 2006 and updated in November, 2006 (Siegel JD, Rhinehart E, Jackson M, Chiarello L and HICPAC. Management of Multidrug-Resistant Organisms in Healthcare Settings 2006 www.cdc.gov/ncidod/dhqp/pdf/ar/mdroGuideline2006.pdf), and is considered a part of the Guideline for Isolation Precautions. This section provides a detailed review of the complex topic of MDRO control in healthcare settings and is intended to provide a context for evaluation of MDRO at individual healthcare settings. A rationale and

institutional requirements for developing an effective MDRO control program are summarized. Although the focus of this guideline is on measures to prevent *transmission* of MDROs in healthcare settings, information concerning the judicious use of antimicrobial agents is presented since such practices are intricately related to the size of the reservoir of MDROs which in turn influences transmission (e.g. colonization pressure). There are two tables that summarize recommended prevention and control practices using the following seven categories of interventions to control MDROs: administrative measures, education of healthcare personnel, judicious antimicrobial use, surveillance, infection control precautions, environmental measures, and decolonization. Recommendations for each category apply to and are adapted for the various healthcare settings. With the increasing incidence and prevalence of MDROs, all healthcare facilities must prioritize effective control of MDRO transmission. Facilities should identify prevalent MDROs at the facility, implement control measures, assess the effectiveness of control programs, and demonstrate decreasing MDRO rates. A set of intensified MDRO prevention interventions is presented to be added 1) if the incidence of transmission of a target MDRO is NOT decreasing despite implementation of basic MDRO infection control measures, and 2) when the *first* case(s) of an epidemiologically important MDRO is identified within a healthcare facility.

Summary

This updated guideline responds to changes in healthcare delivery and addresses new concerns about transmission of infectious agents to patients and healthcare workers in the United States and infection control. The primary objective of the guideline is to improve the safety of the nation's healthcare delivery system by reducing the rates of HAIs.

Abbreviations Used in the Guideline

AIIR	Airborne infection isolation room
CDC	Centers for Disease Control and Prevention
CF	Cystic fibrosis
CJD	Creutzfeld-Jakob Disease
CLSI	Clinical Laboratory Standards Institute
ESBL	Extended spectrum beta-lactamases
FDA	Food and Drug Administration
HAI	Healthcare-associated infections
HBV	Hepatitis B virus
HCV	Hepatitis C virus
HEPA	High efficiency particulate air [filtration]
HICPAC	Healthcare Infection Control Practices Advisory Committee
HIV	Human immunodeficiency virus
HCW	Healthcare worker
HSCT	Hematopoietic stem-cell transplant
ICU	Intensive care unit LTCF Long-term care facility
MDRO	Multidrug-resistant organism
MDR-GNB	Multidrug-resistant gram-negative bacilli
MRSA	Methicillin-resistant <i>Staphylococcus aureus</i>
NCCLS	National Committee for Clinical Laboratory Standards
NICU	Neonatal intensive care unit
NIOSH	National Institute for Occupational Safety and Health, CDC
NNIS	National Nosocomial Infection Surveillance
NSSP	Nonsusceptible <i>Streptococcus pneumoniae</i>
OSHA	Occupational Safety and Health Administration
PICU	Pediatric intensive care unit
PPE	Personal protective equipment
RSV	Respiratory syncytial virus
SARS	Severe acquired respiratory syndrome
vCJD	variant Creutzfeld-Jakob Disease
VRE	Vancomycin-resistant enterococci
WHO	World Health Organization

Part I: Review of Scientific Data Regarding Transmission of Infectious Agents in Healthcare Settings

I.A. Evolution of the 2007 Document

The *Guideline for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings 2007* builds upon a series of isolation and infection prevention documents promulgated since 1970. These previous documents are summarized and referenced in Table 1 and in Part I of the *1996 Guideline for Isolation Precautions in Hospitals*¹.

Objectives and methods The objectives of this guideline are to 1) provide infection control recommendations for all components of the healthcare delivery system, including hospitals, long-term care facilities, ambulatory care, home care and hospice; 2) reaffirm Standard Precautions as the foundation for preventing transmission during patient care in all healthcare settings; 3) reaffirm the importance of implementing Transmission-Based Precautions based on the clinical presentation or syndrome and likely pathogens until the infectious etiology has been determined (Table 2); and 4) provide epidemiologically sound and, whenever possible, evidence-based recommendations.

This guideline is designed for use by individuals who are charged with administering infection control programs in hospitals and other healthcare settings. The information also will be useful for other healthcare personnel, healthcare administrators, and anyone needing information about infection control measures to prevent transmission of infectious agents. Commonly used abbreviations are provided on page 12 and terms used in the guideline are defined in the Glossary (page 137).

Med-line and Pub Med were used to search for relevant studies published in English, focusing on those published since 1996. Much of the evidence cited for preventing transmission of infectious agents in healthcare settings is derived from studies that used “quasi-experimental designs”, also referred to as nonrandomized, pre- post-intervention study designs². Although these types of studies can provide valuable information regarding the effectiveness of various interventions, several factors decrease the certainty of attributing improved outcome to a specific intervention. These include: difficulties in controlling for important confounding variables; the use of multiple interventions during an outbreak; and results that are explained by the statistical principle of regression to the mean, (e.g., improvement over time without any intervention)³.

Observational studies remain relevant and have been used to evaluate infection control interventions^{4,5}. The quality of studies, consistency of results and correlation with results from randomized, controlled trials when available were considered during the literature review and assignment of evidence-based categories (See Part IV: Recommendations) to the recommendations in this guideline. Several authors have summarized properties to consider when evaluating studies for the purpose of determining if the results should change practice or in designing new studies^{2,6,7}.

Changes or clarifications in terminology This guideline contains four changes in terminology from the 1996 guideline:

- f The term *nosocomial infection* is retained to refer only to infections acquired in hospitals. The term *healthcare-associated infection (HAI)* is used to refer to infections associated with healthcare delivery in any setting (e.g., hospitals, long-term care facilities, ambulatory settings, home care). This term reflects the inability to determine with certainty where the pathogen is acquired since patients may be colonized with or exposed to potential pathogens outside of the healthcare setting, before receiving health care, or may develop infections caused by those pathogens when exposed to the conditions associated with delivery of healthcare. Additionally, patients frequently move among the various settings within a healthcare system⁸.
- f A new addition to the practice recommendations for Standard Precautions is *Respiratory Hygiene/Cough Etiquette*. While Standard Precautions generally apply to the recommended practices of healthcare personnel during patient care, Respiratory Hygiene/Cough Etiquette applies broadly to all persons who enter a healthcare setting, including healthcare personnel, patients and visitors. These recommendations evolved from observations during the SARS epidemic that failure to implement basic source control measures with patients, visitors, and healthcare personnel with signs and symptoms of respiratory tract infection may have contributed to SARS coronavirus (SARS-CoV) transmission. This concept has been incorporated into CDC planning documents for SARS and pandemic influenza^{9, 10}.
- f The term “*Airborne Precautions*” has been supplemented with the term “*Airborne Infection Isolation Room (AIIR)*” for consistency with the *Guidelines for Environmental Infection Control in Healthcare Facilities*¹¹, the *Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health-Care Settings 2005*¹² and the American Institute of Architects (AIA) guidelines for design and construction of hospitals, 2006¹³.
- f A set of prevention measures termed *Protective Environment* has been added to the precautions used to prevent HAIs. These measures, which have been defined in other guidelines, consist of engineering and design interventions that decrease the risk of exposure to environmental fungi for severely immunocompromised allogeneic hematopoietic stem cell transplant (HSCT) patients during their highest risk phase, usually the first 100 days post transplant, or longer in the presence of graft-versus-host disease^{11, 13-15}. Recommendations for a Protective Environment apply only to acute care hospitals that provide care to HSCT patients.

Scope This guideline, like its predecessors, focuses primarily on interactions between patients and healthcare providers. The Guidelines for the Prevention of MDRO Infection were published separately in November 2006, and are available online at www.cdc.gov/ncidod/dhqp/index.html. Several other HICPAC

guidelines to prevent transmission of infectious agents associated with healthcare delivery are cited; e.g., *Guideline for Hand Hygiene*, *Guideline for Environmental Infection Control*, *Guideline for Prevention of Healthcare-Associated Pneumonia*, and *Guideline for Infection Control in Healthcare Personnel*^{11, 14, 16, 17}. In combination, these provide comprehensive guidance on the primary infection control measures for ensuring a safe environment for patients and healthcare personnel.

This guideline does not discuss in detail specialized infection control issues in defined populations that are addressed elsewhere, (e.g., *Recommendations for Preventing Transmission of Infections among Chronic Hemodialysis Patients*, *Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health-Care Facilities 2005*, *Guidelines for Infection Control in Dental Health-Care Settings* and *Infection Control Recommendations for Patients with Cystic Fibrosis*^{12, 18-20}. An exception has been made by including abbreviated guidance for a Protective Environment used for allogeneic HSCT recipients because components of the Protective Environment have been more completely defined since publication of the *Guidelines for Preventing Opportunistic Infections Among HSCT Recipients in 2000* and the *Guideline for Environmental Infection Control in Healthcare Facilities*^{11, 15}.

I.B. Rationale for Standard and Transmission-Based Precautions in healthcare settings

Transmission of infectious agents within a healthcare setting requires three elements: a source (or reservoir) of infectious agents, a susceptible host with a portal of entry receptive to the agent, and a mode of transmission for the agent. This section describes the interrelationship of these elements in the epidemiology of HAIs.

I.B.1. Sources of infectious agents Infectious agents transmitted during healthcare derive primarily from human sources but inanimate environmental sources also are implicated in transmission. Human reservoirs include patients²⁰⁻²⁸, healthcare personnel^{29-35 17, 36-39}, and household members and other visitors⁴⁰⁻⁴⁵. Such source individuals may have active infections, may be in the asymptomatic and/or incubation period of an infectious disease, or may be transiently or chronically colonized with pathogenic microorganisms, particularly in the respiratory and gastrointestinal tracts. The endogenous flora of patients (e.g., bacteria residing in the respiratory or gastrointestinal tract) also are the source of HAIs⁴⁶⁻⁵⁴.

I.B.2. Susceptible hosts Infection is the result of a complex interrelationship between a potential host and an infectious agent. Most of the factors that influence infection and the occurrence and severity of disease are related to the host. However, characteristics of the host-agent interaction as it relates to

pathogenicity, virulence and antigenicity are also important, as are the infectious dose, mechanisms of disease production and route of exposure⁵⁵. There is a spectrum of possible outcomes following exposure to an infectious agent. Some persons exposed to pathogenic microorganisms never develop symptomatic disease while others become severely ill and even die. Some individuals are prone to becoming transiently or permanently colonized but remain asymptomatic. Still others progress from colonization to symptomatic disease either immediately following exposure, or after a period of asymptomatic colonization. The immune state at the time of exposure to an infectious agent, interaction between pathogens, and virulence factors intrinsic to the agent are important predictors of an individuals' outcome. Host factors such as extremes of age and underlying disease (e.g. diabetes^{56,57}), human immunodeficiency virus/acquired immune deficiency syndrome [HIV/AIDS]^{58,59}, malignancy, and transplants^{18,60,61} can increase susceptibility to infection as do a variety of medications that alter the normal flora (e.g., antimicrobial agents, gastric acid suppressants, corticosteroids, antirejection drugs, antineoplastic agents, and immunosuppressive drugs). Surgical procedures and radiation therapy impair defenses of the skin and other involved organ systems. Indwelling devices such as urinary catheters, endotracheal tubes, central venous and arterial catheters^{62,64} and synthetic implants facilitate development of HAIs by allowing potential pathogens to bypass local defenses that would ordinarily impede their invasion and by providing surfaces for development of biofilms that may facilitate adherence of microorganisms and protect from antimicrobial activity⁶⁵. Some infections associated with invasive procedures result from transmission within the healthcare facility; others arise from the patient's endogenous flora⁴⁶⁻⁵⁰. High-risk patient populations with noteworthy risk factors for infection are discussed further in Sections I.D, I.E., and I.F.

I.B.3. Modes of transmission Several classes of pathogens can cause infection, including bacteria, viruses, fungi, parasites, and prions. The modes of transmission vary by type of organism and some infectious agents may be transmitted by more than one route: some are transmitted primarily by direct or indirect contact, (e.g., *Herpes simplex* virus [HSV], respiratory syncytial virus, *Staphylococcus aureus*), others by the droplet, (e.g., influenza virus, *B. pertussis*) or airborne routes (e.g., *M. tuberculosis*). Other infectious agents, such as bloodborne viruses (e.g., hepatitis B and C viruses [HBV, HCV] and HIV are transmitted rarely in healthcare settings, via percutaneous or mucous membrane exposure. Importantly, not all infectious agents are transmitted from person to person. These are distinguished in Appendix A. The three principal routes of transmission are summarized below.

I.B.3.a. Contact transmission The most common mode of transmission, contact transmission is divided into two subgroups: direct contact and indirect contact.

I.B.3.a.i. Direct contact transmission Direct transmission occurs when microorganisms are transferred from one infected person to another person without a contaminated intermediate object or person. Opportunities for direct contact transmission between patients and healthcare personnel have been summarized in the Guideline for Infection Control in Healthcare Personnel, 1998¹⁷ and include:

- blood or other blood-containing body fluids from a patient directly enters a caregiver's body through contact with a mucous membrane⁶⁶ or breaks (i.e., cuts, abrasions) in the skin⁶⁷.
- mites from a scabies-infested patient are transferred to the skin of a caregiver while he/she is having direct ungloved contact with the patient's skin^{68, 69}.
- a healthcare provider develops herpetic whitlow on a finger after contact with HSV when providing oral care to a patient without using gloves or HSV is transmitted to a patient from a herpetic whitlow on an ungloved hand of a healthcare worker (HCW)^{70, 71}.

I.B.3.a.ii. Indirect contact transmission Indirect transmission involves the transfer of an infectious agent through a contaminated intermediate object or person. In the absence of a point-source outbreak, it is difficult to determine how indirect transmission occurs. However, extensive evidence cited in the Guideline for Hand Hygiene in Health-Care Settings suggests that the contaminated hands of healthcare personnel are important contributors to indirect contact transmission¹⁶. Examples of opportunities for indirect contact transmission include:

- Hands of healthcare personnel may transmit pathogens after touching an infected or colonized body site on one patient or a contaminated inanimate object, if hand hygiene is not performed before touching another patient.^{72, 73}
- Patient-care devices (e.g., electronic thermometers, glucose monitoring devices) may transmit pathogens if devices contaminated with blood or body fluids are shared between patients without cleaning and disinfecting between patients^{74 75-77}.
- Shared toys may become a vehicle for transmitting respiratory viruses (e.g., respiratory syncytial virus^{24, 78, 79} or pathogenic bacteria (e.g., *Pseudomonas aeruginosa*⁸⁰) among pediatric patients.
- Instruments that are inadequately cleaned between patients before disinfection or sterilization (e.g., endoscopes or surgical instruments)⁸¹⁻⁸⁵ or that have manufacturing defects that interfere with the effectiveness of reprocessing^{86, 87} may transmit bacterial and viral pathogens.

Clothing, uniforms, laboratory coats, or isolation gowns used as personal protective equipment (PPE), may become contaminated with potential pathogens after care of a patient colonized or infected with an infectious agent, (e.g., MRSA⁸⁸, VRE⁸⁹, and *C. difficile*⁹⁰). Although contaminated clothing has not been

implicated directly in transmission, the potential exists for soiled garments to transfer infectious agents to successive patients.

I.B.3.b. Droplet transmission Droplet transmission is, technically, a form of contact transmission, and some infectious agents transmitted by the droplet route also may be transmitted by the direct and indirect contact routes. However, in contrast to contact transmission, respiratory droplets carrying infectious pathogens transmit infection when they travel directly from the respiratory tract of the infectious individual to susceptible mucosal surfaces of the recipient, generally over short distances, necessitating facial protection. Respiratory droplets are generated when an infected person coughs, sneezes, or talks^{91, 92} or during procedures such as suctioning, endotracheal intubation,⁹³⁻⁹⁶ cough induction by chest physiotherapy⁹⁷ and cardiopulmonary resuscitation^{98, 99}. Evidence for droplet transmission comes from epidemiological studies of disease outbreaks¹⁰⁰⁻¹⁰³, experimental studies¹⁰⁴ and from information on aerosol dynamics^{91, 105}. Studies have shown that the nasal mucosa, conjunctivae and less frequently the mouth, are susceptible portals of entry for respiratory viruses¹⁰⁶. The maximum distance for droplet transmission is currently unresolved, although pathogens transmitted by the droplet route have not been transmitted through the air over long distances, in contrast to the airborne pathogens discussed below. Historically, the area of defined risk has been a distance of ≤ 3 feet around the patient and is based on epidemiologic and simulated studies of selected infections^{103, 104}. Using this distance for donning masks has been effective in preventing transmission of infectious agents via the droplet route. However, experimental studies with smallpox^{107, 108} and investigations during the global SARS outbreaks of 2003¹⁰¹ suggest that droplets from patients with these two infections could reach persons located 6 feet or more from their source. It is likely that the distance droplets travel depends on the velocity and mechanism by which respiratory droplets are propelled from the source, the density of respiratory secretions, environmental factors such as temperature and humidity, and the ability of the pathogen to maintain infectivity over that distance¹⁰⁵. Thus, a distance of ≤ 3 feet around the patient is best viewed as an *example* of what is meant by “a short distance from a patient” and should not be used as the sole *criterion* for deciding when a mask should be donned to protect from droplet exposure. Based on these considerations, it may be prudent to don a mask when within 6 to 10 feet of the patient or upon entry into the patient’s room, especially when exposure to emerging or highly virulent pathogens is likely. More studies are needed to improve understanding of droplet transmission under various circumstances.

Droplet size is another variable under discussion. Droplets traditionally have been defined as being $>5 \mu\text{m}$ in size. Droplet nuclei, particles arising from desiccation of suspended droplets, have been associated with airborne transmission and defined as $\leq 5 \mu\text{m}$ in size¹⁰⁵, a reflection of the pathogenesis of pulmonary tuberculosis which is not generalizable to other organisms. Observations of particle dynamics have demonstrated that a range of droplet sizes, including those with diameters of $30\mu\text{m}$ or greater, can remain suspended

in the air¹⁰⁹. The behavior of droplets and droplet nuclei affect recommendations for preventing transmission. Whereas fine airborne particles containing pathogens that are able to remain infective may transmit infections over long distances, requiring AIIR to prevent its dissemination within a facility; organisms transmitted by the droplet route do not remain infective over long distances, and therefore do not require special air handling and ventilation. Examples of infectious agents that are transmitted via the droplet route include *Bordetella pertussis*¹¹⁰, influenza virus²³, adenovirus¹¹¹, rhinovirus¹⁰⁴, *Mycoplasma pneumoniae*¹¹², SARS-associated coronavirus (SARS-CoV)^{21, 96, 113}, group A streptococcus¹¹⁴, and *Neisseria meningitidis*^{95, 103, 115}. Although respiratory syncytial virus may be transmitted by the droplet route, direct contact with infected respiratory secretions is the most important determinant of transmission and consistent adherence to Standard plus Contact Precautions prevents transmission in healthcare settings^{24, 116, 117}.

Rarely, pathogens that are not transmitted routinely by the droplet route are dispersed into the air over short distances. For example, although *S. aureus* is transmitted most frequently by the contact route, viral upper respiratory tract infection has been associated with increased dispersal of *S. aureus* from the nose into the air for a distance of 4 feet under both outbreak and experimental conditions and is known as the “cloud baby” and “cloud adult” phenomenon¹¹⁸⁻¹²⁰.

I.B.3.c. Airborne transmission Airborne transmission occurs by dissemination of either airborne droplet nuclei or small particles in the respirable size range containing infectious agents that remain infective over time and distance (e.g., spores of *Aspergillus* spp, and *Mycobacterium tuberculosis*). Microorganisms carried in this manner may be dispersed over long distances by air currents and may be inhaled by susceptible individuals who have not had face-to-face contact with (or been in the same room with) the infectious individual¹²¹⁻¹²⁴. Preventing the spread of pathogens that are transmitted by the airborne route requires the use of special air handling and ventilation systems (e.g., AIIRs) to contain and then safely remove the infectious agent^{11, 12}. Infectious agents to which this applies include *Mycobacterium tuberculosis*¹²⁴⁻¹²⁷, rubeola virus (measles)¹²², and varicella-zoster virus (chickenpox)¹²³. In addition, published data suggest the possibility that variola virus (smallpox) may be transmitted over long distances through the air under unusual circumstances and AIIRs are recommended for this agent as well; however, droplet and contact routes are the more frequent routes of transmission for smallpox^{108, 128, 129}. In addition to AIIRs, respiratory protection with NIOSH certified N95 or higher level respirator is recommended for healthcare personnel entering the AIIR to prevent acquisition of airborne infectious agents such as *M. tuberculosis*¹².

For certain other respiratory infectious agents, such as influenza^{130, 131} and rhinovirus¹⁰⁴, and even some gastrointestinal viruses (e.g., norovirus¹³² and rotavirus¹³³) there is some evidence that the pathogen may be transmitted via small-particle aerosols, under natural and experimental conditions. Such transmission has occurred over distances longer than 3 feet but within a defined

airspace (e.g., patient room), suggesting that it is unlikely that these agents remain viable on air currents that travel long distances. AIIRs are not required routinely to prevent transmission of these agents. Additional issues concerning examples of small particle aerosol transmission of agents that are most frequently transmitted by the droplet route are discussed below.

I.B.3.d. Emerging issues concerning airborne transmission of infectious agents.

I.B.3.d.i. *Transmission from patients* The emergence of SARS in 2002, the importation of monkeypox into the United States in 2003, and the emergence of avian influenza present challenges to the assignment of isolation categories because of conflicting information and uncertainty about possible routes of transmission. Although SARS-CoV is transmitted primarily by contact and/or droplet routes, airborne transmission over a limited distance (e.g. within a room), has been suggested, though not proven¹³⁴⁻¹⁴¹. This is true of other infectious agents such as influenza virus¹³⁰ and noroviruses^{132, 142, 143}. Influenza viruses are transmitted primarily by close contact with respiratory droplets^{23, 102} and acquisition by healthcare personnel has been prevented by Droplet Precautions, even when positive pressure rooms were used in one center¹⁴⁴. However, inhalational transmission could not be excluded in an outbreak of influenza in the passengers and crew of a single aircraft¹³⁰. Observations of a protective effect of UV lights in preventing influenza among patients with tuberculosis during the influenza pandemic of 1957-'58 have been used to suggest airborne transmission^{145, 146}.

In contrast to the strict interpretation of an airborne route for transmission (i.e., long distances beyond the patient room environment), short distance transmission by small particle aerosols generated under specific circumstances (e.g., during endotracheal intubation) to persons in the immediate area near the patient has been demonstrated. Also, aerosolized particles <100 µm can remain suspended in air when room air current velocities exceed the terminal settling velocities of the particles¹⁰⁹. SARS-CoV transmission has been associated with endotracheal intubation, noninvasive positive pressure ventilation, and cardio•pulmonary resuscitation^{93, 94, 96, 98, 141}. Although the most frequent routes of transmission of noroviruses are contact and food and waterborne routes, several reports suggest that noroviruses may be transmitted through aerosolization of infectious particles from vomitus or fecal material^{142, 143, 147, 148}. It is hypothesized that the aerosolized particles are inhaled and subsequently swallowed.

Roy and Milton proposed a new classification for aerosol transmission when evaluating routes of SARS transmission: 1) *obligate*: under natural conditions, disease occurs following transmission of the agent only through inhalation of small particle aerosols (e.g., tuberculosis); 2) *preferential*: natural infection results from transmission through multiple routes, but small particle aerosols are the predominant route (e.g. measles, varicella); and 3) *opportunistic*: agents that naturally cause disease through other routes, but under special circumstances

may be transmitted via fine particle aerosols¹⁴⁹. This conceptual framework can explain rare occurrences of airborne transmission of agents that are transmitted most frequently by other routes (e.g., smallpox, SARS, influenza, noroviruses). Concerns about unknown or possible routes of transmission of agents associated with severe disease and no known treatment often result in more extreme prevention strategies than may be necessary; therefore, recommended precautions could change as the epidemiology of an emerging infection is defined and controversial issues are resolved.

I.B.3.d.ii. *Transmission from the environment* Some airborne infectious agents are derived from the environment and do not usually involve person-to-person transmission. For example, anthrax spores present in a finely milled powdered preparation can be aerosolized from contaminated environmental surfaces and inhaled into the respiratory tract^{150, 151}. Spores of environmental fungi (e.g., *Aspergillus spp.*) are ubiquitous in the environment and may cause disease in immunocompromised patients who inhale aerosolized (e.g., via construction dust) spores^{152, 153}. As a rule, neither of these organisms is subsequently transmitted from infected patients. However, there is one well-documented report of person-to-person transmission of *Aspergillus sp.* in the ICU setting that was most likely due to the aerosolization of spores during wound debridement¹⁵⁴. A Protective Environment refers to isolation practices designed to decrease the risk of exposure to environmental fungal agents in allogeneic HSCT patients^{11, 14, 15, 155-158}.

Environmental sources of respiratory pathogens (eg. Legionella) transmitted to humans through a common aerosol source is distinct from direct patient-to-patient transmission.

I.B.3.e. Other sources of infection Transmission of infection from sources other than infectious individuals include those associated with *common environmental sources or vehicles* (e.g. contaminated food, water, or medications (e.g. intravenous fluids). Although *Aspergillus spp.* have been recovered from hospital water systems¹⁵⁹, the role of water as a reservoir for immunosuppressed patients remains uncertain. *Vectorborne transmission* of infectious agents from mosquitoes, flies, rats, and other vermin also can occur in healthcare settings. Prevention of vector borne transmission is not addressed in this document.

I.C. Infectious agents of special infection control interest for healthcare settings

Several infectious agents with important infection control implications that either were not discussed extensively in previous isolation guidelines or have emerged recently are discussed below. These are epidemiologically important organisms (e.g., *C. difficile*), agents of bioterrorism, prions, SARS-CoV, monkeypox, noroviruses, and the hemorrhagic fever viruses. Experience with these agents has broadened the understanding of modes of transmission and effective

preventive measures. These agents are included for purposes of information and, for some (i.e., SARS-CoV, monkeypox), because of the lessons that have been learned about preparedness planning and responding effectively to new infectious agents.

I.C.1. Epidemiologically important organisms Any infectious agents transmitted in healthcare settings may, under defined conditions, become targeted for control because they are epidemiologically important. *C. difficile* is specifically discussed below because of wide recognition of its current importance in U.S. healthcare facilities. In determining what constitutes an “epidemiologically important organism”, the following characteristics apply:

- A propensity for transmission within healthcare facilities based on published reports and the occurrence of temporal or geographic clusters of > 2 patients, (e.g., *C. difficile*, norovirus, respiratory syncytial virus (RSV), influenza, rotavirus, *Enterobacter* spp; *Serratia* spp., group A streptococcus). A single case of healthcare-associated invasive disease caused by certain pathogens (e.g., group A streptococcus post-operatively¹⁶⁰, in burn units¹⁶¹, or in a LTCF¹⁶²; *Legionella* sp.^{14, 163}, *Aspergillus* sp.¹⁶⁴) is generally considered a trigger for investigation and enhanced control measures because of the risk of additional cases and severity of illness associated with these infections. Antimicrobial resistance
- Resistance to first-line therapies (e.g., MRSA, VISA, VRSA, VRE, ESBL-producing organisms).
- Common and uncommon microorganisms with unusual patterns of resistance within a facility (e.g., the first isolate of *Burkholderia cepacia* complex or *Ralstonia* spp. in non-CF patients or a quinolone-resistant strain of *Pseudomonas aeruginosa* in a facility).
- Difficult to treat because of innate or acquired resistance to multiple classes of antimicrobial agents (e.g., *Stenotrophomonas maltophilia*, *Acinetobacter* spp.).
- Association with serious clinical disease, increased morbidity and mortality (e.g., MRSA and MSSA, group A streptococcus)
- A newly discovered or reemerging pathogen

I.C.1.a. *C. difficile* *C. difficile* is a spore-forming gram positive anaerobic bacillus that was first isolated from stools of neonates in 1935¹⁶⁵ and identified as the most commonly identified causative agent of antibiotic-associated diarrhea and pseudomembranous colitis in 1977¹⁶⁶. This pathogen is a major cause of healthcare-associated diarrhea and has been responsible for many large outbreaks in healthcare settings that were extremely difficult to control. Important factors that contribute to healthcare-associated outbreaks include environmental contamination, persistence of spores for prolonged periods of time, resistance of spores to routinely used disinfectants and antiseptics, hand carriage by healthcare personnel to other patients, and exposure of patients to frequent courses of antimicrobial agents¹⁶⁷. Antimicrobials most frequently associated

with increased risk of *C. difficile* include third generation cephalosporins, clindamycin, vancomycin, and fluoroquinolones.

Since 2001, outbreaks and sporadic cases of *C. difficile* with increased morbidity and mortality have been observed in several U.S. states, Canada, England and the Netherlands¹⁶⁸⁻¹⁷². The same strain of *C. difficile* has been implicated in these outbreaks¹⁷³. This strain, toxinotype III, North American PFGE type 1, and PCR-ribotype 027 (NAP1/027). has been found to hyperproduce toxin A (16 fold increase) and toxin B (23 fold increase) compared with isolates from 12 different pulsed-field gel electrophoresis PFGE types. A recent survey of U.S. infectious disease physicians found that 40% perceived recent increases in the incidence and severity of *C. difficile* disease¹⁷⁴. Standardization of testing methodology and surveillance definitions is needed for accurate comparisons of trends in rates among hospitals¹⁷⁵. It is hypothesized that the incidence of disease and apparent heightened transmissibility of this new strain may be due, at least in part, to the greater production of toxins A and B, increasing the severity of diarrhea and resulting in more environmental contamination. Considering the greater morbidity, mortality, length of stay, and costs associated with *C. difficile* disease in both acute care and long term care facilities, control of this pathogen is now even more important than previously. Prevention of transmission focuses on syndromic application of Contact Precautions for patients with diarrhea, accurate identification of patients, environmental measures (e.g., rigorous cleaning of patient rooms) and consistent hand hygiene. Use of soap and water, rather than alcohol based handrubs, for mechanical removal of spores from hands, and a bleach-containing disinfectant (5000 ppm) for environmental disinfection, may be valuable when there is transmission in a healthcare facility. See Appendix A for specific recommendations.

I.C.1. b. Multidrug-Resistant Organisms (MDROs) In general, MDROs are defined as microorganisms – predominantly bacteria – that are resistant to one or more classes of antimicrobial agents¹⁷⁶. Although the names of certain MDROs suggest resistance to only one agent (e.g., methicillin-resistant *Staphylococcus aureus* [MRSA], vancomycin resistant enterococcus [VRE]), these pathogens are usually resistant to all but a few commercially available antimicrobial agents. This latter feature defines MDROs that are considered to be epidemiologically important and deserve special attention in healthcare facilities¹⁷⁷. Other MDROs of current concern include multidrug-resistant *Streptococcus pneumoniae* (MDRSP) which is resistant to penicillin and other broad-spectrum agents such as macrolides and fluoroquinolones, multidrug-resistant gram-negative bacilli (MDR- GNB), especially those producing extended spectrum beta-lactamases (ESBLs); and strains of *S. aureus* that are intermediate or resistant to vancomycin (i.e., VISA and VRSA)^{178-197 198}.

MDROs are transmitted by the same routes as antimicrobial susceptible infectious agents. Patient-to-patient transmission in healthcare settings, usually via hands of HCWs, has been a major factor accounting for the increase in MDRO incidence and prevalence, especially for MRSA and VRE in acute care

facilities¹⁹⁹⁻²⁰¹. Preventing the emergence and transmission of these pathogens requires a comprehensive approach that includes administrative involvement and measures (e.g., nurse staffing, communication systems, performance improvement processes to ensure adherence to recommended infection control measures), education and training of medical and other healthcare personnel, judicious antibiotic use, comprehensive surveillance for targeted MDROs, application of infection control precautions during patient care, environmental measures (e.g., cleaning and disinfection of the patient care environment and equipment, dedicated single-patient-use of non-critical equipment), and decolonization therapy when appropriate.

The prevention and control of MDROs is a national priority - one that requires that all healthcare facilities and agencies assume responsibility and participate in community-wide control programs^{176, 177}. A detailed discussion of this topic and recommendations for prevention was published in 2006 may be found at <http://www.cdc.gov/ncidod/dhqp/pdf/ar/mdroGuideline2006.pdf>

I.C.2. Agents of bioterrorism CDC has designated the agents that cause anthrax, smallpox, plague, tularemia, viral hemorrhagic fevers, and botulism as Category A (high priority) because these agents can be easily disseminated environmentally and/or transmitted from person to person; can cause high mortality and have the potential for major public health impact; might cause public panic and social disruption; and require special action for public health preparedness²⁰². General information relevant to infection control in healthcare settings for Category A agents of bioterrorism is summarized in Table 3. Consult www.bt.cdc.gov for additional, updated Category A agent information as well as information concerning Category B and C agents of bioterrorism and updates. Category B and C agents are important but are not as readily disseminated and cause less morbidity and mortality than Category A agents.

Healthcare facilities confront a different set of issues when dealing with a suspected bioterrorism event as compared with other communicable diseases. An understanding of the epidemiology, modes of transmission, and clinical course of each disease, as well as carefully drafted plans that provide an approach and relevant websites and other resources for disease-specific guidance to healthcare, administrative, and support personnel, are essential for responding to and managing a bioterrorism event. Infection control issues to be addressed include: 1) identifying persons who may be exposed or infected; 2) preventing transmission among patients, healthcare personnel, and visitors; 3) providing treatment, chemoprophylaxis or vaccine to potentially large numbers of people; 4) protecting the environment including the logistical aspects of securing sufficient numbers of AIIRs or designating areas for patient cohorts when there are an insufficient number of AIIRs available; 5) providing adequate quantities of appropriate personal protective equipment; and 6) identifying appropriate staff to care for potentially infectious patients (e.g., vaccinated healthcare personnel for

care of patients with smallpox). The response is likely to differ for exposures resulting from an intentional release compared with naturally occurring disease because of the large number of persons that can be exposed at the same time and possible differences in pathogenicity.

A variety of sources offer guidance for the management of persons exposed to the most likely agents of bioterrorism. Federal agency websites (e.g., www.usamriid.army.mil/publications/index.html, www.bt.cdc.gov) and state and county health department web sites should be consulted for the most up-to-date information. Sources of information on specific agents include: anthrax²⁰³; smallpox²⁰⁴⁻²⁰⁶; plague^{207, 208}; botulinum toxin²⁰⁹; tularemia²¹⁰; and hemorrhagic fever viruses:^{211, 212}

I.C.2.a. Pre-event administration of smallpox (vaccinia) vaccine to healthcare personnel Vaccination of personnel in preparation for a possible smallpox exposure has important infection control implications²¹³⁻²¹⁵. These include the need for meticulous screening for vaccine contraindications in persons who are at increased risk for adverse vaccinia events; containment and monitoring of the vaccination site to prevent transmission in the healthcare setting and at home; and the management of patients with vaccinia-related adverse events^{216, 217}. The pre-event U.S. smallpox vaccination program of 2003 is an example of the effectiveness of carefully developed recommendations for both screening potential vaccinees for contraindications and vaccination site care and monitoring. Approximately 760,000 individuals were vaccinated in the Department of Defense and 40,000 in the civilian or public health populations from December 2002 to February 2005, including approximately 70,000 who worked in healthcare settings. There were no cases of eczema vaccinatum, progressive vaccinia, fetal vaccinia, or contact transfer of vaccinia in healthcare settings or in military workplaces^{218, 219}. Outside the healthcare setting, there were 53 cases of contact transfer from military vaccinees to close personal contacts (e.g., bed partners or contacts during participation in sports such as wrestling²²⁰). All contact transfers were from individuals who were not following recommendations to cover their vaccination sites. Vaccinia virus was confirmed by culture or PCR in 30 cases, and two of the confirmed cases resulted from tertiary transfer. All recipients, including one breast-fed infant, recovered without complication. Subsequent studies using viral culture and PCR techniques have confirmed the effectiveness of semipermeable dressings to contain vaccinia²²¹⁻²²⁴. This experience emphasizes the importance of ensuring that newly vaccinated healthcare personnel adhere to recommended vaccination-site care, especially if they are to care for high-risk patients. Recommendations for pre-event smallpox vaccination of healthcare personnel and vaccinia-related infection control recommendations are published in the MMWR^{216, 225} with updates posted on the CDC bioterrorism web site²⁰⁵.

I.C.3. Prions Creutzfeldt-Jakob disease (CJD) is a rapidly progressive, degenerative, neurologic disorder of humans with an incidence in the United States of approximately 1 person/million population/year^{226, 227}

(www.cdc.gov/ncidod/diseases/cjd/cjd.htm). CJD is believed to be caused by a transmissible proteinaceous infectious agent termed a prion. Infectious prions are isoforms of a host-encoded glycoprotein known as the prion protein. The incubation period (i.e., time between exposure and onset of symptoms) varies from two years to many decades. However, death typically occurs within 1 year of the onset of symptoms. Approximately 85% of CJD cases occur sporadically with no known environmental source of infection and 10% are familial. Iatrogenic transmission has occurred with most resulting from treatment with human cadaveric pituitary-derived growth hormone or gonadotropin^{228, 229}, from implantation of contaminated human dura mater grafts²³⁰ or from corneal transplants²³¹). Transmission has been linked to the use of contaminated neurosurgical instruments or stereotactic electroencephalogram electrodes^{232, 233, 234, 235}.

Prion diseases in animals include scrapie in sheep and goats, bovine spongiform encephalopathy (BSE, or “mad cow disease”) in cattle, and chronic wasting disease in deer and elk²³⁶. BSE, first recognized in the United Kingdom (UK) in 1986, was associated with a major epidemic among cattle that had consumed contaminated meat and bone meal.

The possible transmission of BSE to humans causing variant CJD (vCJD) was first described in 1996 and subsequently found to be associated with consumption of BSE-contaminated cattle products primarily in the United Kingdom. There is strong epidemiologic and laboratory evidence for a causal association between the causative agent of BSE and vCJD²³⁷. Although most cases of vCJD have been reported from the UK, a few cases also have been reported from Europe, Japan, Canada, and the United States. Most vCJD cases worldwide lived in or visited the UK during the years of a large outbreak of BSE (1980-96) and may have consumed contaminated cattle products during that time (www.cdc.gov/ncidod/diseases/cjd/cjd.htm). Although there has been no indigenously acquired vCJD in the United States, the sporadic occurrence of BSE in cattle in North America has heightened awareness of the possibility that such infections could occur and have led to increased surveillance activities. Updated information may be found on the following website: www.cdc.gov/ncidod/diseases/cjd/cjd.htm. The public health impact of prion diseases has been reviewed²³⁸.

vCJD in humans has different clinical and pathologic characteristics from sporadic or classic CJD²³⁹, including the following: 1) younger median age at death: 28 (range 16-48) vs. 68 years; 2) longer duration of illness: median 14 months vs. 4-6 months; 3) increased frequency of sensory symptoms and early psychiatric symptoms with delayed onset of frank neurologic signs; and 4) detection of prions in tonsillar and other lymphoid tissues from vCJD patients but not from sporadic CJD patients²⁴⁰. Similar to sporadic CJD, there have been no reported cases of direct human-to-human transmission of vCJD by casual or environmental contact, droplet, or airborne routes. Ongoing blood safety surveillance in the U.S. has not detected sporadic CJD transmission through

blood transfusion²⁴¹⁻²⁴³. However, bloodborne transmission of vCJD is believed to have occurred in two UK patients^{244, 245}. The following FDA websites provide information on steps that are being taken in the US to protect the blood supply from CJD and vCJD: <http://www.fda.gov/cber/gdlns/cjdvcjd.htm>;

<http://www.fda.gov/cber/gdlns/cjdvcjdq&a.htm>.

Standard Precautions are used when caring for patients with suspected or confirmed CJD or vCJD. However, special precautions are recommended for tissue handling in the histology laboratory and for conducting an autopsy, embalming, and for contact with a body that has undergone autopsy²⁴⁶. Recommendations for reprocessing surgical instruments to prevent transmission of CJD in healthcare settings have been published by the World Health Organization (WHO) and are currently under review at CDC.

Questions concerning notification of patients potentially exposed to CJD or vCJD through contaminated instruments and blood products from patients with CJD or vCJD or at risk of having vCJD may arise. The risk of transmission associated with such exposures is believed to be extremely low but may vary based on the specific circumstance. Therefore consultation on appropriate options is advised. The United Kingdom has developed several documents that clinicians and patients in the US may find useful (http://www.hpa.org.uk/infections/topics_az/cjd/information_documents.htm).

I.C.4. Severe Acute Respiratory Syndrome (SARS) SARS is a newly discovered respiratory disease that emerged in China late in 2002 and spread to several countries^{135, 140}; Mainland China, Hong Kong, Hanoi, Singapore, and Toronto were affected significantly. SARS is caused by SARS CoV, a previously unrecognized member of the coronavirus family^{247, 248}. The incubation period from exposure to the onset of symptoms is 2 to 7 days but can be as long as 10 days and uncommonly even longer²⁴⁹. The illness is initially difficult to distinguish from other common respiratory infections. Signs and symptoms usually include fever >38.0°C and chills and rigors, sometimes accompanied by headache, myalgia, and mild to severe respiratory symptoms. Radiographic finding of atypical pneumonia is an important clinical indicator of possible SARS. Compared with adults, children have been affected less frequently, have milder disease, and are less likely to transmit SARS-CoV^{135, 249-251}. The overall case fatality rate is approximately 6.0%; underlying disease and advanced age increase the risk of mortality (www.who.int/csr/sarsarchive/2003_05_07a/en/).

Outbreaks in healthcare settings, with transmission to large numbers of healthcare personnel and patients have been a striking feature of SARS; undiagnosed, infectious patients and visitors were important initiators of these outbreaks^{21, 252-254}. The relative contribution of potential modes of transmission is not precisely known. There is ample evidence for droplet and contact transmission^{96, 101, 113}; however, opportunistic airborne transmission cannot be excluded^{101, 135-139, 149, 255}. For example, exposure to aerosol-generating

procedures (e.g., endotracheal intubation, suctioning) was associated with transmission of infection to large numbers of healthcare personnel outside of the United States^{93, 94, 96, 98, 253}. Therefore, aerosolization of small infectious particles generated during these and other similar procedures could be a risk factor for transmission to others within a multi-bed room or shared airspace. A review of the infection control literature generated from the SARS outbreaks of 2003 concluded that the greatest risk of transmission is to those who have close contact, are not properly trained in use of protective infection control procedures, do not consistently use PPE; and that N95 or higher respirators may offer additional protection to those exposed to aerosol- generating procedures and high risk activities^{256, 257}. Organizational and individual factors that affected adherence to infection control practices for SARS also were identified²⁵⁷.

Control of SARS requires a coordinated, dynamic response by multiple disciplines in a healthcare setting⁹. Early detection of cases is accomplished by screening persons with symptoms of a respiratory infection for history of travel to areas experiencing community transmission or contact with SARS patients, followed by implementation of Respiratory Hygiene/Cough Etiquette (i.e., placing a mask over the patient's nose and mouth) and physical separation from other patients in common waiting areas. The precise combination of precautions to protect healthcare personnel has not been determined. At the time of this publication, CDC recommends Standard Precautions, with emphasis on the use of hand hygiene, Contact Precautions with emphasis on environmental cleaning due to the detection of SARS CoV RNA by PCR on surfaces in rooms occupied by SARS patients^{138, 254, 258}, Airborne Precautions, including use of fit-tested NIOSH-approved N95 or higher level respirators, and eye protection²⁵⁹. In Hong Kong, the use of Droplet and Contact Precautions, which included use of a mask but not a respirator, was effective in protecting healthcare personnel¹¹³. However, in Toronto, consistent use of an N95 respirator was slightly more protective than a mask⁹³. It is noteworthy that there was no transmission of SARS-CoV to public hospital workers in Vietnam despite inconsistent use of infection control measures, including use of PPE, which suggests other factors (e.g., severity of disease, frequency of high risk procedures or events, environmental features) may influence opportunities for transmission²⁶⁰.

SARS-CoV also has been transmitted in the laboratory setting through breaches in recommended laboratory practices. Research laboratories where SARS-CoV was under investigation were the source of most cases reported after the first series of outbreaks in the winter and spring of 2003^{261, 262}. Studies of the SARS outbreaks of 2003 and transmissions that occurred in the laboratory re-affirm the effectiveness of recommended infection control precautions and highlight the importance of consistent adherence to these measures.

Lessons from the SARS outbreaks are useful for planning to respond to future public health crises, such as pandemic influenza and bioterrorism events. Surveillance for cases among patients and healthcare personnel, ensuring availability of adequate supplies and staffing, and limiting access to healthcare

facilities were important factors in the response to SARS that have been summarized⁹. Guidance for infection control precautions in various settings is available at www.cdc.gov/ncidod/sars.

I.C.5. Monkeypox Monkeypox is a rare viral disease found mostly in the rain forest countries of Central and West Africa. The disease is caused by an orthopoxvirus that is similar in appearance to smallpox but causes a milder disease. The only recognized outbreak of human monkeypox in the United States was detected in June 2003 after several people became ill following contact with sick pet prairie dogs. Infection in the prairie dogs was subsequently traced to their contact with a shipment of animals from Africa, including giant Gambian rats²⁶³. This outbreak demonstrates the importance of recognition and prompt reporting of unusual disease presentations by clinicians to enable prompt identification of the etiology; and the potential of epizootic diseases to spread from animal reservoirs to humans through personal and occupational exposure²⁶⁴.

Limited data on transmission of monkeypox are available. Transmission from infected animals and humans is believed to occur primarily through direct contact with lesions and respiratory secretions; airborne transmission from animals to humans is unlikely but cannot be excluded, and may have occurred in veterinary practices (e.g., during administration of nebulized medications to ill prairie dogs²⁶⁵). Among humans, four instances of monkeypox transmission within hospitals have been reported in Africa among children, usually related to sharing the same ward or bed^{266, 267}. Additional recent literature documents transmission of Congo Basin monkeypox in a hospital compound for an extended number of generations²⁶⁸.

There has been no evidence of airborne or any other person-to-person transmission of monkeypox in the United States, and no new cases of monkeypox have been identified since the outbreak in June 2003²⁶⁹. The outbreak strain is a clade of monkeypox distinct from the Congo Basin clade and may have different epidemiologic properties (including human-to-human transmission potential) from monkeypox strains of the Congo Basin²⁷⁰; this awaits further study. Smallpox vaccine is 85% protective against Congo Basin monkeypox²⁷¹. Since there is an associated case fatality rate of $\leq 10\%$, administration of smallpox vaccine within 4 days to individuals who have had direct exposure to patients or animals with monkeypox is a reasonable consideration²⁷². For the most current information on monkeypox, see www.cdc.gov/ncidod/monkeypox/clinicians.htm.

I.C.6. Noroviruses Noroviruses, formerly referred to as Norwalk-like viruses, are members of the *Caliciviridae* family. These agents are transmitted via contaminated food or water and from person-to-person, causing explosive outbreaks of gastrointestinal disease²⁷³. Environmental contamination also has been documented as a contributing factor in ongoing transmission during outbreaks^{274, 275}. Although noroviruses cannot be propagated in cell culture,

DNA detection by molecular diagnostic techniques has facilitated a greater appreciation of their role in outbreaks of gastrointestinal disease²⁷⁶. Reported outbreaks in hospitals^{132, 142, 277}, nursing homes^{275, 278-283}, cruise ships^{284, 285}, hotels^{143, 147}, schools¹⁴⁸, and large crowded shelters established for hurricane evacuees²⁸⁶, demonstrate their highly contagious nature, the disruptive impact they have in healthcare facilities and the community, and the difficulty of controlling outbreaks in settings where people share common facilities and space. Of note, there is nearly a 5 fold increase in the risk to patients in outbreaks where a patient is the index case compared with exposure of patients during outbreaks where a staff member is the index case²⁸⁷.

The average incubation period for gastroenteritis caused by noroviruses is 12-48 hours and the clinical course lasts 12-60 hours²⁷³. Illness is characterized by acute onset of nausea, vomiting, abdominal cramps, and/or diarrhea. The disease is largely self-limited; rarely, death caused by severe dehydration can occur, particularly among the elderly with debilitating health conditions.

The epidemiology of norovirus outbreaks shows that even though primary cases may result from exposure to a fecally-contaminated food or water, secondary and tertiary cases often result from person-to-person transmission that is facilitated by contamination of fomites^{273, 288} and dissemination of infectious particles, especially during the process of vomiting^{132, 142, 143, 147, 148, 273, 279, 280}. Widespread, persistent and inapparent contamination of the environment and fomites can make outbreaks extremely difficult to control^{147, 275, 284}. These clinical observations and the detection of norovirus DNA on horizontal surfaces 5 feet above the level that might be touched normally suggest that, under certain circumstances, aerosolized particles may travel distances beyond 3 feet¹⁴⁷. It is hypothesized that infectious particles may be aerosolized from vomitus, inhaled, and swallowed. In addition, individuals who are responsible for cleaning the environment may be at increased risk of infection. Development of disease and transmission may be facilitated by the low infectious dose (i.e., <100 viral particles)²⁸⁹ and the resistance of these viruses to the usual cleaning and disinfection agents (i.e., may survive ≤ 10 ppm chlorine)²⁹⁰⁻²⁹². An alternate phenolic agent that was shown to be effective against feline calicivirus was used for environmental cleaning in one outbreak^{275, 293}. There are insufficient data to determine the efficacy of alcohol-based hand rubs against noroviruses when the hands are not visibly soiled²⁹⁴. Absence of disease in certain individuals during an outbreak may be explained by protection from infection conferred by the B histo-blood group antigen²⁹⁵. Consultation on outbreaks of gastroenteritis is available through CDC's Division of Viral and Rickettsial Diseases²⁹⁶.

I.C.7. Hemorrhagic fever viruses (HFV) The hemorrhagic fever viruses are a mixed group of viruses that cause serious disease with high fever, skin rash, bleeding diathesis, and in some cases, high mortality; the disease caused is referred to as viral hemorrhagic fever (VHF). Among the more commonly known HFVs are Ebola and Marburg viruses (Filoviridae), Lassa virus (Arenaviridae), Crimean-Congo hemorrhagic fever and Rift Valley Fever virus (Bunyaviridae),

and Dengue and Yellow fever viruses (Flaviviridae)^{212, 297}. These viruses are transmitted to humans via contact with infected animals or via arthropod vectors. While none of these viruses is endemic in the United States, outbreaks in affected countries provide potential opportunities for importation by infected humans and animals. Furthermore, there are concerns that some of these agents could be used as bioweapons²¹². Person-to-person transmission is documented for Ebola, Marburg, Lassa and Crimean-Congo hemorrhagic fever viruses. In resource-limited healthcare settings, transmission of these agents to healthcare personnel, patients and visitors has been described and in some outbreaks has accounted for a large proportion of cases²⁹⁸⁻³⁰⁰. Transmissions within households also have occurred among individuals who had direct contact with ill persons or their body fluids, but not to those who did not have such contact³⁰¹.

Evidence concerning the transmission of HFVs has been summarized^{212, 302}. Person-to-person transmission is associated primarily with direct blood and body fluid contact. Percutaneous exposure to contaminated blood carries a particularly high risk for transmission and increased mortality^{303, 304}. The finding of large numbers of Ebola viral particles in the skin and the lumina of sweat glands has raised concern that transmission could occur from direct contact with intact skin though epidemiologic evidence to support this is lacking³⁰⁵. Postmortem handling of infected bodies is an important risk for transmission^{301, 306, 307}. In rare situations, cases in which the mode of transmission was unexplained among individuals with no known direct contact, have led to speculation that airborne transmission could have occurred²⁹⁸. However, airborne transmission of naturally occurring HFVs in humans has not been seen. In one study of airplane passengers exposed to an in-flight index case of Lassa fever, there was no transmission to any passengers³⁰⁸.

In the laboratory setting, animals have been infected experimentally with Marburg or Ebola viruses via direct inoculation of the nose, mouth and/or conjunctiva^{309, 310} and by using mechanically generated virus-containing aerosols^{311, 312}. Transmission of Ebola virus among laboratory primates in an animal facility has been described³¹³. Secondarily infected animals were in individual cages and separated by approximately 3 meters. Although the possibility of airborne transmission was suggested, the authors were not able to exclude droplet or indirect contact transmission in this incidental observation.

Guidance on infection control precautions for HFVs that are transmitted person-to-person have been published by CDC^{1, 211} and by the Johns Hopkins Center for Civilian Biodefense Strategies²¹². The most recent recommendations at the time of publication of this document were posted on the CDC website on 5/19/05³¹⁴. Inconsistencies among the various recommendations have raised questions about the appropriate precautions to use in U.S. hospitals. In less developed countries, outbreaks of HFVs have been controlled with basic hygiene, barrier precautions, safe injection practices, and safe burial practices^{299, 306}. The preponderance of evidence on HFV transmission indicates that Standard, Contact and Droplet Precautions with eye protection are effective in protecting

healthcare personnel and visitors who may attend an infected patient. Single gloves are adequate for routine patient care; double-gloving is advised during invasive procedures (e.g., surgery) that pose an increased risk for blood exposure. Routine eye protection (i.e. goggles or face shield) is particularly important. Fluid-resistant gowns should be worn for all patient contact. Airborne Precautions are not required for routine patient care; however, use of AIIRs is prudent when procedures that could generate infectious aerosols are performed (e.g., endotracheal intubation, bronchoscopy, suctioning, autopsy procedures involving oscillating saws). N95 or higher level respirators may provide added protection for individuals in a room during aerosol-generating procedures (Table 3, Appendix A). When a patient with a syndrome consistent with hemorrhagic fever also has a history of travel to an endemic area, precautions are initiated upon presentation and then modified as more information is obtained (Table 2). Patients with hemorrhagic fever syndrome in the setting of a suspected bioweapon attack should be managed using Airborne Precautions, including AIIRs, since the epidemiology of a potentially weaponized hemorrhagic fever virus is unpredictable.

I.D. Transmission risks associated with specific types of healthcare settings

Numerous factors influence differences in transmission risks among the various healthcare settings. These include the population characteristics (e.g., increased susceptibility to infections, type and prevalence of indwelling devices), intensity of care, exposure to environmental sources, length of stay, and frequency of interaction between patients/residents with each other and with HCWs. These factors, as well as organizational priorities, goals, and resources, influence how different healthcare settings adapt transmission prevention guidelines to meet their specific needs^{315, 316}. Infection control management decisions are informed by data regarding institutional experience/epidemiology, trends in community and institutional HAIs, local, regional, and national epidemiology, and emerging infectious disease threats.

I.D.1. Hospitals Infection transmission risks are present in all hospital settings. However, certain hospital settings and patient populations have unique conditions that predispose patients to infection and merit special mention. These are often sentinel sites for the emergence of new transmission risks that may be unique to that setting or present opportunities for transmission to other settings in the hospital.

I.D.1.a. Intensive Care Units Intensive care units (ICUs) serve patients who are immunocompromised by disease state and/or by treatment modalities, as well as patients with major trauma, respiratory failure and other life-threatening conditions (e.g., myocardial infarction, congestive heart failure, overdoses, strokes, gastrointestinal bleeding, renal failure, hepatic failure, multi-organ system failure, and the extremes of age). Although ICUs account for a relatively

small proportion of hospitalized patients, infections acquired in these units accounted for >20% of all HAIs³¹⁷. In the National Nosocomial Infection Surveillance (NNIS) system, 26.6% of HAIs were reported from ICU and high risk nursery (NICU) patients in 2002 (NNIS, unpublished data). This patient population has increased susceptibility to colonization and infection, especially with MDROs and *Candida* sp.^{318, 319}, because of underlying diseases and conditions, the invasive medical devices and technology used in their care (e.g. central venous catheters and other intravascular devices, mechanical ventilators, extracorporeal membrane oxygenation (ECMO), hemodialysis/-filtration, pacemakers, implantable left ventricular assist devices), the frequency of contact with healthcare personnel, prolonged length of stay, and prolonged exposure to antimicrobial agents³²⁰⁻³³¹. Furthermore, adverse patient outcomes in this setting are more severe and are associated with a higher mortality³³². Outbreaks associated with a variety of bacterial, fungal and viral pathogens due to common-source and person-to-person transmissions are frequent in adult and pediatric ICUs^{31, 333-336, 337, 338}.

I.D.1.b. Burn Units Burn wounds can provide optimal conditions for colonization, infection, and transmission of pathogens; infection acquired by burn patients is a frequent cause of morbidity and mortality^{320, 339, 340}. In patients with a burn injury involving $\geq 30\%$ of the total body surface area (TBSA), the risk of invasive burn wound infection is particularly high^{341, 342}. Infections that occur in patients with burn injury involving <30% TBSA are usually associated with the use of invasive devices. Methicillin-susceptible *Staphylococcus aureus*, MRSA, enterococci, including VRE, gram-negative bacteria, and candida are prevalent pathogens in burn infections^{53, 340, 343-350} and outbreaks of these organisms have been reported³⁵¹⁻³⁵⁴. Shifts over time in the predominance of pathogens causing infections among burn patients often lead to changes in burn care practices^{343, 355-358}. Burn wound infections caused by *Aspergillus* sp. or other environmental molds may result from exposure to supplies contaminated during construction³⁵⁹ or to dust generated during construction or other environmental disruption³⁶⁰.

Hydrotherapy equipment is an important environmental reservoir of gram-negative organisms. Its use for burn care is discouraged based on demonstrated associations between use of contaminated hydrotherapy equipment and infections. Burn wound infections and colonization, as well as bloodstream infections, caused by multidrug-resistant *P. aeruginosa*³⁶¹, *A. baumannii*³⁶², and MRSA³⁵² have been associated with hydrotherapy; excision of burn wounds in operating rooms is preferred.

Advances in burn care, specifically early excision and grafting of the burn wound, use of topical antimicrobial agents, and institution of early enteral feeding, have led to decreased infectious complications. Other advances have included prophylactic antimicrobial usage, selective digestive decontamination (SDD), and use of antimicrobial-coated catheters (ACC), but few epidemiologic studies and no efficacy studies have been performed to show the relative benefit of these measures³⁵⁷.

There is no consensus on the most effective infection control practices to prevent transmission of infections to and from patients with serious burns (e.g., single-bed rooms³⁵⁸, laminar flow³⁶³ and high efficiency particulate air filtration [HEPA]³⁶⁰ or maintaining burn patients in a separate unit without exposure to patients or equipment from other units³⁶⁴). There also is controversy regarding the need for and type of barrier precautions for routine care of burn patients. One retrospective study demonstrated efficacy and cost effectiveness of a simplified barrier isolation protocol for wound colonization, emphasizing handwashing and use of gloves, caps, masks and plastic impermeable aprons (rather than isolation gowns) for direct patient contact³⁶⁵. However, there have been no studies that define the most effective combination of infection control precautions for use in burn settings. Prospective studies in this area are needed.

I.D.1.c. Pediatrics Studies of the epidemiology of HAIs in children have identified unique infection control issues in this population^{63, 64, 366-370}. Pediatric intensive care unit (PICU) patients and the lowest birthweight babies in the high-risk nursery (HRN) monitored in the NNIS system have had high rates of central venous catheter-associated bloodstream infections^{64, 320, 369-372}. Additionally, there is a high prevalence of community-acquired infections among hospitalized infants and young children who have not yet become immune either by vaccination or by natural infection. The result is more patients and their sibling visitors with transmissible infections present in pediatric healthcare settings, especially during seasonal epidemics (e.g., pertussis^{36, 40, 41}, respiratory viral infections including those caused by RSV²⁴, influenza viruses³⁷³, parainfluenza virus³⁷⁴, human metapneumovirus³⁷⁵, and adenoviruses³⁷⁶, rubeola [measles]³⁴, varicella [chickenpox]³⁷⁷, and rotavirus^{38, 378}).

Close physical contact between healthcare personnel and infants and young children (eg. cuddling, feeding, playing, changing soiled diapers, and cleaning copious uncontrolled respiratory secretions) provides abundant opportunities for transmission of infectious material. Practices and behaviors such as congregation of children in play areas where toys and bodily secretions are easily shared and family members rooming-in with pediatric patients can further increase the risk of transmission. Pathogenic bacteria have been recovered from toys used by hospitalized patients³⁷⁹; contaminated bath toys were implicated in an outbreak of multidrug-resistant *P. aeruginosa* on a pediatric oncology unit⁸⁰. In addition, several patient factors increase the likelihood that infection will result from exposure to pathogens in healthcare settings (e.g., immaturity of the neonatal immune system, lack of previous natural infection and resulting immunity, prevalence of patients with congenital or acquired immune deficiencies, congenital anatomic anomalies, and use of life-saving invasive devices in neonatal and pediatric intensive care units)⁶³. There are theoretical concerns that infection risk will increase in association with innovative practices used in the NICU for the purpose of improving developmental outcomes. Such factors include co-bedding³⁸⁰ and kangaroo care³⁸¹ that may increase opportunity for skin-to-skin exposure of multiple gestation infants to each other and to their mothers, respectively; although infection risk may actually be

reduced among infants receiving kangaroo care³⁸². Children who attend child care centers^{383, 384} and pediatric rehabilitation units³⁸⁵ may increase the overall burden of antimicrobial resistance (eg. by contributing to the reservoir of community-associated MRSA [CA-MRSA])³⁸⁶⁻³⁹¹. Patients in chronic care facilities may have increased rates of colonization with resistant GNBs and may be sources of introduction of resistant organisms to acute care settings⁵⁰.

I.D.2. Nonacute healthcare settings Healthcare is provided in various settings outside of hospitals including facilities, such as long-term care facilities (LTCF) (e.g. nursing homes), homes for the developmentally disabled, settings where behavioral health services are provided, rehabilitation centers and hospices³⁹². In addition, healthcare may be provided in nonhealthcare settings such as workplaces with occupational health clinics, adult day care centers, assisted living facilities, homeless shelters, jails and prisons, school clinics and infirmaries. Each of these settings has unique circumstances and population risks to consider when designing and implementing an infection control program. Several of the most common settings and their particular challenges are discussed below. While this Guideline does not address each setting, the principles and strategies provided may be adapted and applied as appropriate.

I.D.2.a. Long-term care The designation LTCF applies to a diverse group of residential settings, ranging from institutions for the developmentally disabled to nursing homes for the elderly and pediatric chronic-care facilities³⁹³⁻³⁹⁵. Nursing homes for the elderly predominate numerically and frequently represent long-term care as a group of facilities. Approximately 1.8 million Americans reside in the nation's 16,500 nursing homes³⁹⁶. Estimates of HAI rates of 1.8 to 13.5 per 1000 resident-care days have been reported with a range of 3 to 7 per 1000 resident-care days in the more rigorous studies³⁹⁷⁻⁴⁰¹. The infrastructure described in the Department of Veterans Affairs nursing home care units is a promising example for the development of a nationwide HAI surveillance system for LTCFs⁴⁰².

LTCFs are different from other healthcare settings in that elderly patients at increased risk for infection are brought together in one setting and remain in the facility for extended periods of time; for most residents, it is their home. An atmosphere of community is fostered and residents share common eating and living areas, and participate in various facility-sponsored activities^{403, 404}. Since able residents interact freely with each other, controlling transmission of infection in this setting is challenging⁴⁰⁵. Residents who are colonized or infected with certain microorganisms are, in some cases, restricted to their room. However, because of the psychosocial risks associated with such restriction, it has been recommended that psychosocial needs be balanced with infection control needs in the LTCF setting⁴⁰⁶⁻⁴⁰⁹. Documented LTCF outbreaks have been caused by various viruses (e.g., influenza virus^{35, 410-412}, rhinovirus⁴¹³, adenovirus (conjunctivitis)⁴¹⁴, norovirus^{278, 279 275, 281}) and bacteria, including group A streptococcus¹⁶², *B. pertussis*⁴¹⁵, non-susceptible *S. pneumoniae*^{197, 198}, other MDROs, and *Clostridium difficile*⁴¹⁶) These pathogens can lead to substantial

morbidity and mortality, and increased medical costs; prompt detection and implementation of effective control measures are required.

Risk factors for infection are prevalent among LTCF residents^{395, 417, 418}. Age-related declines in immunity may affect responses to immunizations for influenza and other infectious agents, and increase susceptibility to tuberculosis. Immobility, incontinence, dysphagia, underlying chronic diseases, poor functional status, and age-related skin changes increase susceptibility to urinary, respiratory and cutaneous and soft tissue infections, while malnutrition can impair wound healing⁴¹⁹⁻⁴²³. Medications (e.g., drugs that affect level of consciousness, immune function, gastric acid secretions, and normal flora, including antimicrobial therapy) and invasive devices (e.g., urinary catheters and feeding tubes) heighten susceptibility to infection and colonization in LTCF residents⁴²⁴⁻⁴²⁶. Finally, limited functional status and total dependence on healthcare personnel for activities of daily living have been identified as independent risk factors for infection^{401, 417, 427} and for colonization with MRSA^{428, 429} and ESBL-producing *K. pneumoniae*⁴³⁰. Several position papers and review articles have been published that provide guidance on various aspects of infection control and antimicrobial resistance in LTCFs^{406-408, 431-436}. The Centers for Medicare and Medicaid Services (CMS) have established regulations for the prevention of infection in LTCFs⁴³⁷.

Because residents of LTCFs are hospitalized frequently, they can transfer pathogens between LTCFs and healthcare facilities in which they receive care^{8, 438-441}. This is also true for pediatric long-term care populations. Pediatric chronic care facilities have been associated with importing extended-spectrum cephalosporin-resistant, gram-negative bacilli into one PICU⁵⁰. Children from pediatric rehabilitation units may contribute to the reservoir of community-associated MRSA^{385, 389-391}.

I.D.2.b. Ambulatory Care In the past decade, healthcare delivery in the United States has shifted from the acute, inpatient hospital to a variety of ambulatory and community-based settings, including the home. Ambulatory care is provided in hospital-based outpatient clinics, nonhospital-based clinics and physician offices, public health clinics, free-standing dialysis centers, ambulatory surgical centers, urgent care centers, and many others. In 2000, there were 83 million visits to hospital outpatient clinics and more than 823 million visits to physician offices⁴⁴²; ambulatory care now accounts for most patient encounters with the health care system⁴⁴³. In these settings, adapting transmission prevention guidelines is challenging because patients remain in common areas for prolonged periods waiting to be seen by a healthcare provider or awaiting admission to the hospital, examination or treatment rooms are turned around quickly with limited cleaning, and infectious patients may not be recognized immediately. Furthermore, immunocompromised patients often receive chemotherapy in infusion rooms where they stay for extended periods of time along with other types of patients.

There are few data on the risk of HAIs in ambulatory care settings, with the exception of hemodialysis centers^{18, 444, 445}. Transmission of infections in outpatient settings has been reviewed in three publications⁴⁴⁶⁻⁴⁴⁸. Goodman and Solomon summarized 53 clusters of infections associated with the outpatient setting from 1961-1990⁴⁴⁶. Overall, 29 clusters were associated with common source transmission from contaminated solutions or equipment, 14 with person-to-person transmission from or involving healthcare personnel and ten associated with airborne or droplet transmission among patients and healthcare workers. Transmission of bloodborne pathogens (i.e., hepatitis B and C viruses and, rarely, HIV) in outbreaks, sometimes involving hundreds of patients, continues to occur in ambulatory settings. These outbreaks often are related to common source exposures, usually a contaminated medical device, multi-dose vial, or intravenous solution^{82, 449-453}. In all cases, transmission has been attributed to failure to adhere to fundamental infection control principles, including safe injection practices and aseptic technique. This subject has been reviewed and recommended infection control and safe injection practices summarized⁴⁵⁴.

Airborne transmission of *M. tuberculosis* and measles in ambulatory settings, most frequently emergency departments, has been reported^{34, 127, 446, 448, 455-457}. Measles virus was transmitted in physician offices and other outpatient settings during an era when immunization rates were low and measles outbreaks in the community were occurring regularly^{34, 122, 458}. Rubella has been transmitted in the outpatient obstetric setting³³; there are no published reports of varicella transmission in the outpatient setting. In the ophthalmology setting, adenovirus type 8 epidemic keratoconjunctivitis has been transmitted via incompletely disinfected ophthalmology equipment and/or from healthcare workers to patients, presumably by contaminated hands^{17, 446, 448, 459-462}.

If transmission in outpatient settings is to be prevented, screening for potentially infectious symptomatic and asymptomatic individuals, especially those who may be at risk for transmitting airborne infectious agents (e.g., *M. tuberculosis*, varicella-zoster virus, rubeola [measles]), is necessary at the start of the initial patient encounter. Upon identification of a potentially infectious patient, implementation of prevention measures, including prompt separation of potentially infectious patients and implementation of appropriate control measures (e.g., Respiratory Hygiene/Cough Etiquette and Transmission-Based Precautions) can decrease transmission risks^{9, 12}. Transmission of MRSA and VRE in outpatient settings has not been reported, but the association of CA-MRSA in healthcare personnel working in an outpatient HIV clinic with environmental CA-MRSA contamination in that clinic, suggests the possibility of transmission in that setting⁴⁶³. Patient-to-patient transmission of *Burkholderia species* and *Pseudomonas aeruginosa* in outpatient clinics for adults and children with cystic fibrosis has been confirmed^{464, 465}.

I.D.2.c. Home Care Home care in the United States is delivered by over 20,000 provider agencies that include home health agencies, hospices, durable medical equipment providers, home infusion therapy services, and personal care and

support services providers. Home care is provided to patients of all ages with both acute and chronic conditions. The scope of services ranges from assistance with activities of daily living and physical and occupational therapy to the care of wounds, infusion therapy, and chronic ambulatory peritoneal dialysis (CAPD).

The incidence of infection in home care patients, other than those associated with infusion therapy is not well studied⁴⁶⁶⁻⁴⁷¹. However, data collection and calculation of infection rates have been accomplished for central venous catheter-associated bloodstream infections in patients receiving home infusion therapy⁴⁷⁰⁻⁴⁷⁴ and for the risk of blood contact through percutaneous or mucosal exposures, demonstrating that surveillance can be performed in this setting⁴⁷⁵. Draft definitions for home care associated infections have been developed⁴⁷⁶.

Transmission risks during home care are presumed to be minimal. The main transmission risks to home care patients are from an infectious healthcare provider or contaminated equipment; providers also can be exposed to an infectious patient during home visits. Since home care involves patient care by a limited number of personnel in settings without multiple patients or shared equipment, the potential reservoir of pathogens is reduced. Infections of home care providers, that could pose a risk to home care patients include infections transmitted by the airborne or droplet routes (e.g., chickenpox, tuberculosis, influenza), and skin infestations (e.g., scabies⁶⁹ and lice) and infections (e.g., impetigo) transmitted by direct or indirect contact. There are no published data on indirect transmission of MDROs from one home care patient to another, although this is theoretically possible if contaminated equipment is transported from an infected or colonized patient and used on another patient. Of note, investigation of the first case of VISA in homecare¹⁸⁶ and the first 2 reported cases of VRSA^{178, 180, 181, 183} found no evidence of transmission of VISA or VRSA to other home care recipients. Home health care also may contribute to antimicrobial resistance; a review of outpatient vancomycin use found 39% of recipients did not receive the antibiotic according to recommended guidelines⁴⁷⁷.

Although most home care agencies implement policies and procedures to prevent transmission of organisms, the current approach is based on the adaptation of the *1996 Guideline for Isolation Precautions in Hospitals*¹ as well as other professional guidance^{478, 479}. This issue has been very challenging in the home care industry and practice has been inconsistent and frequently not evidence-based. For example, many home health agencies continue to observe “nursing bag technique,” a practice that prescribes the use of barriers between the nursing bag and environmental surfaces in the home⁴⁸⁰. While the home environment may not always appear clean, the use of barriers between two non-critical surfaces has been questioned^{481, 482}. Opportunities exist to conduct research in home care related to infection transmission risks⁴⁸³.

I.D.2.d. Other sites of healthcare delivery Facilities that are not primarily healthcare settings but in which healthcare is delivered include clinics in correctional facilities and shelters. Both settings can have suboptimal features,

such as crowded conditions and poor ventilation. Economically disadvantaged individuals who may have chronic illnesses and healthcare problems related to alcoholism, injection drug use, poor nutrition, and/or inadequate shelter often receive their primary healthcare at sites such as these ⁴⁸⁴. Infectious diseases of special concern for transmission include tuberculosis, scabies, respiratory infections (e.g., *N. meningitides*, *S. pneumoniae*), sexually transmitted and bloodborne diseases (e.g., HIV, HBV, HCV, syphilis, gonorrhea), hepatitis A virus (HAV), diarrheal agents such as norovirus, and foodborne diseases ^{286, 485-488}. A high index of suspicion for tuberculosis and CA-MRSA in these populations is needed as outbreaks in these settings or among the populations they serve have been reported ⁴⁸⁹⁻⁴⁹⁷.

Patient encounters in these types of facilities provide an opportunity to deliver recommended immunizations and screen for *M. tuberculosis* infection in addition to diagnosing and treating acute illnesses ⁴⁹⁸. Recommended infection control measures in these non-traditional areas designated for healthcare delivery are the same as for other ambulatory care settings. Therefore, these settings must be equipped to observe Standard Precautions and, when indicated, Transmission-based Precautions.

I.E. Transmission risks associated with special patient populations

As new treatments emerge for complex diseases, unique infection control challenges associated with special patient populations need to be addressed.

I.E.1. Immunocompromised patients Patients who have congenital primary immune deficiencies or acquired disease (eg. treatment-induced immune deficiencies) are at increased risk for numerous types of infections while receiving healthcare and may be located throughout the healthcare facility. The specific defects of the immune system determine the types of infections that are most likely to be acquired (e.g., viral infections are associated with T-cell defects and fungal and bacterial infections occur in patients who are neutropenic). As a general group, immunocompromised patients can be cared for in the same environment as other patients; however, it is always advisable to minimize exposure to other patients with transmissible infections such as influenza and other respiratory viruses ^{499, 500}. The use of more intense chemotherapy regimens for treatment of childhood leukemia may be associated with prolonged periods of neutropenia and suppression of other components of the immune system, extending the period of infection risk and raising the concern that additional precautions may be indicated for select groups ^{501, 502}. With the application of newer and more intense immunosuppressive therapies for a variety of medical conditions (e.g., rheumatologic disease ^{503, 504}, inflammatory bowel disease ⁵⁰⁵), immunosuppressed patients are likely to be more widely distributed throughout a healthcare facility rather than localized to single patient units (e.g.

hematology-oncology). Guidelines for preventing infections in certain groups of immunocompromised patients have been published ^{15, 506, 507}.

Published data provide evidence to support placing allogeneic HSCT patients in a Protective Environment ^{15, 157, 158}. Also, three guidelines have been developed that address the special requirements of these immunocompromised patients, including use of antimicrobial prophylaxis and engineering controls to create a Protective Environment for the prevention of infections caused by *Aspergillus* spp. and other environmental fungi ^{11, 14, 15}. As more intense chemotherapy regimens associated with prolonged periods of neutropenia or graft-versus-host disease are implemented, the period of risk and duration of environmental protection may need to be prolonged beyond the traditional 100 days ⁵⁰⁸.

I.E.2. Cystic fibrosis patients Patients with cystic fibrosis (CF) require special consideration when developing infection control guidelines. Compared to other patients, CF patients require additional protection to prevent transmission from contaminated respiratory therapy equipment ⁵⁰⁹⁻⁵¹³. Infectious agents such as *Burkholderia cepacia* complex and *P. aeruginosa* ^{464, 465, 514, 515} have unique clinical and prognostic significance. In CF patients, *B. cepacia* infection has been associated with increased morbidity and mortality ⁵¹⁶⁻⁵¹⁸, while delayed acquisition of chronic *P. aeruginosa* infection may be associated with an improved long-term clinical outcome ^{519, 520}.

Person-to-person transmission of *B. cepacia* complex has been demonstrated among children ⁵¹⁷ and adults ⁵²¹ with CF in healthcare settings ^{464, 522}, during various social contacts ⁵²³, most notably attendance at camps for patients with CF ⁵²⁴, and among siblings with CF ⁵²⁵. Successful infection control measures used to prevent transmission of respiratory secretions include segregation of CF patients from each other in ambulatory and hospital settings (including use of private rooms with separate showers), environmental decontamination of surfaces and equipment contaminated with respiratory secretions, elimination of group chest physiotherapy sessions, and disbanding of CF camps ^{97, 526}. The Cystic Fibrosis Foundation published a consensus document with evidence-based recommendations for infection control practices for CF patients ²⁰.

I.F. New therapies associated with potentially transmissible infectious agents

I.F.1. Gene therapy Gene therapy has been attempted using a number of different viral vectors, including nonreplicating retroviruses, adenoviruses, adeno-associated viruses, and replication-competent strains of poxviruses. Unexpected adverse events have restricted the prevalence of gene therapy protocols.

The infectious hazards of gene therapy are theoretical at this time, but require meticulous surveillance due to the possible occurrence of in vivo recombination

and the subsequent emergence of a transmissible genetically altered pathogen. Greatest concern attends the use of replication-competent viruses, especially vaccinia. As of the time of publication, no reports have described transmission of a vector virus from a gene therapy recipient to another individual, but surveillance is ongoing. Recommendations for monitoring infection control issues throughout the course of gene therapy trials have been published⁵²⁷⁻⁵²⁹.

I.F.2. Infections transmitted through blood, organs and other tissues The potential hazard of transmitting infectious pathogens through biologic products is a small but ever present risk, despite donor screening. Reported infections transmitted by transfusion or transplantation include West Nile Virus infection⁵³⁰, cytomegalovirus infection⁵³¹, Creutzfeldt-Jacob disease²³⁰, hepatitis C⁵³², infections with *Clostridium* spp.⁵³³ and group A streptococcus⁵³⁴, malaria⁵³⁵, babesiosis⁵³⁶, Chagas disease⁵³⁷, lymphocytic choriomeningitis⁵³⁸, and rabies^{539, 540}. Therefore, it is important to consider receipt of biologic products when evaluating patients for potential sources of infection.

I.F.3. Xenotransplantation The transplantation of nonhuman cells, tissues, and organs into humans potentially exposes patients to zoonotic pathogens. Transmission of known zoonotic infections (e.g., trichinosis from porcine tissue), constitutes one concern, but also of concern is the possibility that transplantation of nonhuman cells, tissues, or organs may transmit previously unknown zoonotic infections (xenozoonoses) to immunosuppressed human recipients. Potential infections that might accompany transplantation of porcine organs have been described⁵⁴¹. Guidelines from the U.S. Public Health Service address many infectious diseases and infection control issues that surround the developing field of xenotransplantation⁵⁴²); work in this area is ongoing.

Part II:

Fundamental elements needed to prevent transmission of infectious agents in healthcare settings

II.A. Healthcare system components that influence the effectiveness of precautions to prevent transmission

II.A.1. Administrative measures Healthcare organizations can demonstrate a commitment to preventing transmission of infectious agents by incorporating infection control into the objectives of the organization's patient and occupational safety programs⁵⁴³⁻⁵⁴⁷. An infrastructure to guide, support, and monitor adherence to Standard and Transmission-Based Precautions^{434, 548, 549} will facilitate fulfillment of the organization's mission and achievement of the Joint Commission on Accreditation of Healthcare Organization's patient safety goal to decrease HAIs⁵⁵⁰. Policies and procedures that explain how Standard and Transmission-Based Precautions are applied, including systems used to identify and communicate information about patients with potentially transmissible infectious agents, are essential to ensure the success of these measures and may vary according to the characteristics of the organization.

A key administrative measure is provision of fiscal and human resources for maintaining infection control and occupational health programs that are responsive to emerging needs. Specific components include bedside nurse⁵⁵¹ and infection prevention and control professional (ICP) staffing levels⁵⁵², inclusion of ICPs in facility construction and design decisions¹¹, clinical microbiology laboratory support^{553, 554}, adequate supplies and equipment including facility ventilation systems¹¹, adherence monitoring⁵⁵⁵, assessment and correction of system failures that contribute to transmission^{556, 557}, and provision of feedback to healthcare personnel and senior administrators^{434, 548, 549, 558}. The positive influence of institutional leadership has been demonstrated repeatedly in studies of HCW adherence to recommended hand hygiene practices^{176, 177, 434, 548, 549, 559-564}. Healthcare administrator involvement in infection control processes can improve administrators' awareness of the rationale and resource requirements for following recommended infection control practices.

Several administrative factors may affect the transmission of infectious agents in healthcare settings: institutional culture, individual worker behavior, and the work environment. Each of these areas is suitable for performance improvement monitoring and incorporation into the organization's patient safety goals^{543, 544, 546, 565}.

II.A.1.a.Scope of work and staffing needs for infection control professionals

The effectiveness of infection surveillance and control programs in preventing nosocomial infections in United States hospitals was assessed by the CDC through the Study on the Efficacy of Nosocomial Infection Control (SENIC Project) conducted 1970-76⁵⁶⁶. In a representative sample of US general hospitals, those with a trained infection control physician or microbiologist involved in an infection control program, and at least one infection control nurse per 250 beds, were associated with a 32% lower rate of four infections studied (CVC-associated bloodstream infections, ventilator-associated pneumonias, catheter-related urinary tract infections, and surgical site infections).

Since that landmark study was published, responsibilities of ICPs have expanded commensurate with the growing complexity of the healthcare system, the patient populations served, and the increasing numbers of medical procedures and devices used in all types of healthcare settings. The scope of work of ICPs was first assessed in 1982⁵⁶⁷⁻⁵⁶⁹ by the Certification Board of Infection Control (CBIC), and has been re-assessed every five years since that time^{558, 570-572}. The findings of these task analyses have been used to develop and update the Infection Control Certification Examination, offered for the first time in 1983. With each survey, it is apparent that the role of the ICP is growing in complexity and scope, beyond traditional infection control activities in acute care hospitals. Activities currently assigned to ICPs in response to emerging challenges include: 1) surveillance and infection prevention at facilities other than acute care hospitals e.g., ambulatory clinics, day surgery centers, long term care facilities, rehabilitation centers, home care; 2) oversight of employee health services related to infection prevention, e.g. assessment of risk and administration of recommended treatment following exposure to infectious agents, tuberculosis screening, influenza vaccination, respiratory protection fit testing, and administration of other vaccines as indicated, such as smallpox vaccine in 2003; 3) preparedness planning for annual influenza outbreaks, pandemic influenza, SARS, bioweapons attacks; 4) adherence monitoring for selected infection control practices; 5) oversight of risk assessment and implementation of prevention measures associated with construction and renovation; 6) prevention of transmission of MDROs; 7) evaluation of new medical products that could be associated with increased infection risk. e.g., intravenous infusion materials; 9) communication with the public, facility staff, and state and local health departments concerning infection control-related issues; and 10) participation in local and multi-center research projects^{434, 549, 552, 558, 573, 574}.

None of the CBIC job analyses addressed specific staffing requirements for the identified tasks, although the surveys did include information about hours worked; the 2001 survey included the number of ICPs assigned to the responding facilities⁵⁵⁸. There is agreement in the literature that 1 ICP per 250 acute care beds is no longer adequate to meet current infection control needs; a Delphi project that assessed staffing needs of infection control programs in the 21st century concluded that a ratio of 0.8 to 1.0 ICP per 100 occupied acute care beds is an appropriate level of staffing⁵⁵². A survey of participants in the National

Nosocomial Infections Surveillance (NNIS) system found the average daily census per ICP was 115³¹⁶. Results of other studies have been similar: 3 per 500 beds for large acute care hospitals, 1 per 150-250 beds in long term care facilities, and 1.56 per 250 in small rural hospitals^{573, 575}. The foregoing demonstrates that infection control staffing can no longer be based on patient census alone, but rather must be determined by the scope of the program, characteristics of the patient population, complexity of the healthcare system, tools available to assist personnel to perform essential tasks (e.g., electronic tracking and laboratory support for surveillance), and unique or urgent needs of the institution and community⁵⁵². Furthermore, appropriate training is required to optimize the quality of work performed^{558, 572, 576}.

II.A.1.a.i. Infection Control Nurse Liaison Designating a bedside nurse on a patient care unit as an infection control liaison or “link nurse” is reported to be an effective adjunct to enhance infection control at the unit level⁵⁷⁷⁻⁵⁸². Such individuals receive training in basic infection control and have frequent communication with the ICPs, but maintain their primary role as bedside caregiver on their units. The infection control nurse liaison increases the awareness of infection control at the unit level. He or she is especially effective in implementation of new policies or control interventions because of the rapport with individuals on the unit, an understanding of unit-specific challenges, and ability to promote strategies that are most likely to be successful in that unit. This position is an adjunct to, not a replacement for, fully trained ICPs. Furthermore, the infection control liaison nurses should not be counted when considering ICP staffing.

II.A.1.b. Bedside nurse staffing There is increasing evidence that the level of bedside nurse-staffing influences the quality of patient care^{583, 584}. If there are adequate nursing staff, it is more likely that infection control practices, including hand hygiene and Standard and Transmission-Based Precautions, will be given appropriate attention and applied correctly and consistently⁵⁵². A national multicenter study reported strong and consistent inverse relationships between nurse staffing and five adverse outcomes in medical patients, two of which were HAIs: urinary tract infections and pneumonia⁵⁸³. The association of nursing staff shortages with increased rates of HAIs has been demonstrated in several outbreaks in hospitals and long term care settings, and with increased transmission of hepatitis C virus in dialysis units^{22, 418, 551, 585-597}. In most cases, when staffing improved as part of a comprehensive control intervention, the outbreak ended or the HAI rate declined. In two studies^{590, 596}, the composition of the nursing staff (“pool” or “float” vs. regular staff nurses) influenced the rate of primary bloodstream infections, with an increased infection rate occurring when the proportion of regular nurses decreased and pool nurses increased.

II.A.1.c. Clinical microbiology laboratory support The critical role of the clinical microbiology laboratory in infection control and healthcare epidemiology is described well^{553, 554, 598-600} and is supported by the Infectious Disease Society

of America policy statement on consolidation of clinical microbiology laboratories published in 2001⁵⁵³. The clinical microbiology laboratory contributes to preventing transmission of infectious diseases in healthcare settings by promptly detecting and reporting epidemiologically important organisms, identifying emerging patterns of antimicrobial resistance, and assisting in assessment of the effectiveness of recommended precautions to limit transmission during outbreaks⁵⁹⁸. Outbreaks of infections may be recognized first by laboratorians¹⁶². Healthcare organizations need to ensure the availability of the recommended scope and quality of laboratory services, a sufficient number of appropriately trained laboratory staff members, and systems to promptly communicate epidemiologically important results to those who will take action (e.g., providers of clinical care, infection control staff, healthcare epidemiologists, and infectious disease consultants)⁶⁰¹. As concerns about emerging pathogens and bioterrorism grow, the role of the clinical microbiology laboratory takes on even greater importance. For healthcare organizations that outsource microbiology laboratory services (e.g., ambulatory care, home care, LTCFs, smaller acute care hospitals), it is important to specify by contract the types of services (e.g., periodic institution-specific aggregate susceptibility reports) required to support infection control.

Several key functions of the clinical microbiology laboratory are relevant to this guideline:

- Antimicrobial susceptibility by testing and interpretation in accordance with current guidelines developed by the National Committee for Clinical Laboratory Standards (NCCLS), known as the Clinical and Laboratory Standards Institute (CLSI) since 2005⁶⁰², for the detection of emerging resistance patterns^{603, 604}, and for the preparation, analysis, and distribution of periodic cumulative antimicrobial susceptibility summary reports⁶⁰⁵⁻⁶⁰⁷. While not required, clinical laboratories ideally should have access to rapid genotypic identification of bacteria and their antibiotic resistance genes⁶⁰⁸.
- Performance of surveillance cultures when appropriate (including retention of isolates for analysis) to assess patterns of infection transmission and effectiveness of infection control interventions at the facility or organization. Microbiologists assist in decisions concerning the indications for initiating and discontinuing active surveillance programs and optimize the use of laboratory resources.
- Molecular typing, on-site or outsourced, in order to investigate and control healthcare-associated outbreaks⁶⁰⁹.
- Application of rapid diagnostic tests to support clinical decisions involving patient treatment, room selection, and implementation of control measures including barrier precautions and use of vaccine or chemoprophylaxis agents (e.g., influenza⁶¹⁰⁻⁶¹², B. pertussis⁶¹³, RSV^{614, 615}, and enteroviruses⁶¹⁶). The microbiologist provides guidance to limit rapid testing to clinical situations in which rapid results influence patient

- management decisions, as well as providing oversight of point-of-care testing performed by non-laboratory healthcare workers ⁶¹⁷.
- Detection and rapid reporting of epidemiologically important organisms, including those that are reportable to public health agencies.
 - Implementation of a quality control program that ensures testing services are appropriate for the population served, and stringently evaluated for sensitivity, specificity, applicability, and feasibility.
 - Participation in a multidisciplinary team to develop and maintain an effective institutional program for the judicious use of antimicrobial agents ^{618, 619}.

II.A.2. Institutional safety culture and organizational characteristics Safety culture (or safety climate) refers to a work environment where a shared commitment to safety on the part of management and the workforce is understood and followed ^{557, 620, 621}. The authors of the Institute of Medicine Report, *To Err is Human* ⁵⁴³, acknowledge that causes of medical error are multifaceted but emphasize repeatedly the pivotal role of system failures and the benefits of a safety culture. A safety culture is created through 1) the actions management takes to improve patient and worker safety; 2) worker participation in safety planning; 3) the availability of appropriate protective equipment; 4) influence of group norms regarding acceptable safety practices; and 5) the organization's socialization process for new personnel. Safety and patient outcomes can be enhanced by improving or creating organizational characteristics within patient care units as demonstrated by studies of surgical ICUs ^{622, 623}. Each of these factors has a direct bearing on adherence to transmission prevention recommendations ²⁵⁷. Measurement of an institutional culture of safety is useful for designing improvements in healthcare ^{624, 625}. Several hospital-based studies have linked measures of safety culture with both employee adherence to safe practices and reduced exposures to blood and body fluids ⁶²⁶⁻⁶³². One study of hand hygiene practices concluded that improved adherence requires integration of infection control into the organization's safety culture ⁵⁶¹. Several hospitals that are part of the Veterans Administration Healthcare System have taken specific steps toward improving the safety culture, including error reporting mechanisms, performing root cause analysis on problems identified, providing safety incentives, and employee education. ⁶³³⁻⁶³⁵.

II.A.3. Adherence of healthcare personnel to recommended guidelines Adherence to recommended infection control practices decreases transmission of infectious agents in healthcare settings ^{116, 562, 636-640}. However, several observational studies have shown limited adherence to recommended practices by healthcare personnel ^{559, 640-657}. Observed adherence to universal precautions ranged from 43% to 89% ^{641, 642, 649, 651, 652}. However, the degree of adherence depended frequently on the practice that was assessed and, for glove use, the circumstance in which they were used. Appropriate glove use has ranged from a low of 15% ⁶⁴⁵ to a high of 82% ⁶⁵⁰. However, 92% and 98% adherence with glove use have been reported during arterial blood gas collection and

resuscitation, respectively, procedures where there may be considerable blood contact^{643, 656}. Differences in observed adherence have been reported among occupational groups in the same healthcare facility⁶⁴¹ and between experienced and nonexperienced professionals⁶⁴⁵. In surveys of healthcare personnel, self-reported adherence was generally higher than that reported in observational studies. Furthermore, where an observational component was included with a self-reported survey, self-perceived adherence was often greater than observed adherence⁶⁵⁷. Among nurses and physicians, increasing years of experience is a negative predictor of adherence^{645, 651}. Education to improve adherence is the primary intervention that has been studied. While positive changes in knowledge and attitude have been demonstrated,^{640, 658} there often has been limited or no accompanying change in behavior^{642, 644}. Self-reported adherence is higher in groups that have received an educational intervention^{630, 659}. Educational interventions that incorporated videotaping and performance feedback were successful in improving adherence during the period of study; the long-term effect of these interventions is not known⁶⁵⁴. The use of videotape also served to identify system problems (e.g., communication and access to personal protective equipment) that otherwise may not have been recognized.

Use of engineering controls and facility design concepts for improving adherence is gaining interest. While introduction of automated sinks had a negative impact on consistent adherence to hand washing⁶⁶⁰, use of electronic monitoring and voice prompts to remind healthcare workers to perform hand hygiene, and improving accessibility to hand hygiene products, increased adherence and contributed to a decrease in HAIs in one study⁶⁶¹. More information is needed regarding how technology might improve adherence.

Improving adherence to infection control practices requires a multifaceted approach that incorporates continuous assessment of both the individual and the work environment^{559, 561}. Using several behavioral theories, Kretzer and Larson concluded that a single intervention (e.g., a handwashing campaign or putting up new posters about transmission precautions) would likely be ineffective in improving healthcare personnel adherence⁶⁶². Improvement requires that the organizational leadership make prevention an institutional priority and integrate infection control practices into the organization's safety culture⁵⁶¹. A recent review of the literature concluded that variations in organizational factors (e.g., safety climate, policies and procedures, education and training) and individual factors (e.g., knowledge, perceptions of risk, past experience) were determinants of adherence to infection control guidelines for protection against SARS and other respiratory pathogens²⁵⁷.

II.B. Surveillance for healthcare-associated infections (HAIs)

Surveillance is an essential tool for case-finding of single patients or clusters of patients who are infected or colonized with epidemiologically important organisms (e.g., susceptible bacteria such as *S. aureus*, *S. pyogenes* [Group A streptococcus] or *Enterobacter-Klebsiella* spp; MRSA, VRE, and other MDROs; *C. difficile*; RSV; influenza virus) for which transmission-based precautions may

be required. Surveillance is defined as the ongoing, systematic collection, analysis, interpretation, and dissemination of data regarding a health-related event for use in public health action to reduce morbidity and mortality and to improve health⁶⁶³. The work of Ignaz Semmelweis that described the role of person-to-person transmission in puerperal sepsis is the earliest example of the use of surveillance data to reduce transmission of infectious agents⁶⁶⁴. Surveillance of both process measures and the infection rates to which they are linked are important for evaluating the effectiveness of infection prevention efforts and identifying indications for change^{555, 665-668}.

The Study on the Efficacy of Nosocomial Infection Control (SENIC) found that different combinations of infection control practices resulted in reduced rates of nosocomial surgical site infections, pneumonia, urinary tract infections, and bacteremia in acute care hospitals⁵⁶⁶; however, surveillance was the only component essential for reducing all four types of HAIs. Although a similar study has not been conducted in other healthcare settings, a role for surveillance and the need for novel strategies have been described in LTCFs^{398, 434, 669, 670} and in home care⁴⁷⁰⁻⁴⁷³. The essential elements of a surveillance system are: 1) standardized definitions; 2) identification of patient populations at risk for infection; 3) statistical analysis (e.g. risk-adjustment, calculation of rates using appropriate denominators, trend analysis using methods such as statistical process control charts); and 4) feedback of results to the primary caregivers⁶⁷¹⁻⁶⁷⁶. Data gathered through surveillance of high-risk populations, device use, procedures, and/or facility locations (e.g., ICUs) are useful for detecting transmission trends⁶⁷¹⁻⁶⁷³. Identification of clusters of infections should be followed by a systematic epidemiologic investigation to determine commonalities in persons, places, and time; and guide implementation of interventions and evaluation of the effectiveness of those interventions.

Targeted surveillance based on the highest risk areas or patients has been preferred over facility-wide surveillance for the most effective use of resources^{673, 676}. However, surveillance for certain epidemiologically important organisms may need to be facility-wide. Surveillance methods will continue to evolve as healthcare delivery systems change^{392, 677} and user-friendly electronic tools become more widely available for electronic tracking and trend analysis^{674, 678, 679}. Individuals with experience in healthcare epidemiology and infection control should be involved in selecting software packages for data aggregation and analysis to assure that the need for efficient and accurate HAI surveillance will be met. Effective surveillance is increasingly important as legislation requiring public reporting of HAI rates is passed and states work to develop effective systems to support such legislation⁶⁸⁰.

II.C. Education of HCWs, patients, and families

Education and training of healthcare personnel are a prerequisite for ensuring that policies and procedures for Standard and Transmission-Based Precautions are understood and practiced. Understanding the scientific rationale for the

precautions will allow HCWs to apply procedures correctly, as well as safely modify precautions based on changing requirements, resources, or healthcare settings^{14, 655, 681-688}. In one study, the likelihood of HCWs developing SARS was strongly associated with less than 2 hours of infection control training and lack of understanding of infection control procedures⁶⁸⁹. Education about the important role of vaccines (e.g., influenza, measles, varicella, pertussis, pneumococcal) in protecting healthcare personnel, their patients, and family members can help improve vaccination rates⁶⁹⁰⁻⁶⁹³.

Education on the principles and practices for preventing transmission of infectious agents should begin during training in the health professions and be provided to anyone who has an opportunity for contact with patients or medical equipment (e.g., nursing and medical staff; therapists and technicians, including respiratory, physical, occupational, radiology, and cardiology personnel; phlebotomists; housekeeping and maintenance staff; and students). In healthcare facilities, education and training on Standard and Transmission-Based Precautions are typically provided at the time of orientation and should be repeated as necessary to maintain competency; updated education and training are necessary when policies and procedures are revised or when there is a special circumstance, such as an outbreak that requires modification of current practice or adoption of new recommendations. Education and training materials and methods appropriate to the HCW's level of responsibility, individual learning habits, and language needs, can improve the learning experience^{658, 694-702}.

Education programs for healthcare personnel have been associated with sustained improvement in adherence to best practices and a related decrease in device-associated HAIs in teaching and non-teaching settings^{639, 703} and in medical and surgical ICUs {Coopersmith, 2002 #2149; Babcock, 2004 #2126; Berenholtz, 2004 #2289; www.ihl.org/IHI/Programs/Campaign, #2563}. Several studies have shown that, in addition to targeted education to improve specific practices, periodic assessment and feedback of the HCWs knowledge, and adherence to recommended practices are necessary to achieve the desired changes and to identify continuing education needs^{562, 704-708}. Effectiveness of this approach for isolation practices has been demonstrated for control of RSV^{116, 684}.

Patients, family members, and visitors can be partners in preventing transmission of infections in healthcare settings^{9, 42, 709-711}. Information about Standard Precautions, especially hand hygiene, Respiratory Hygiene/Cough Etiquette, vaccination (especially against influenza) and other routine infection prevention strategies may be incorporated into patient information materials that are provided upon admission to the healthcare facility. Additional information about Transmission-Based Precautions is best provided at the time they are initiated. Fact sheets, pamphlets, and other printed material may include information on the rationale for the additional precautions, risks to household members, room assignment for Transmission-Based Precautions purposes, explanation about the use of personal protective equipment by HCWs, and directions for use of

such equipment by family members and visitors. Such information may be particularly helpful in the home environment where household members often have primary responsibility for adherence to recommended infection control practices. Healthcare personnel must be available and prepared to explain this material and answer questions as needed.

II.D. Hand hygiene

Hand hygiene has been cited frequently as the single most important practice to reduce the transmission of infectious agents in healthcare settings^{559, 712, 713} and is an essential element of Standard Precautions. The term “hand hygiene” includes both handwashing with either plain or antiseptic-containing soap and water, and use of alcohol-based products (gels, rinses, foams) that do not require the use of water. In the absence of visible soiling of hands, approved alcohol-based products for hand disinfection are preferred over antimicrobial or plain soap and water because of their superior microbicidal activity, reduced drying of the skin, and convenience⁵⁵⁹. Improved hand hygiene practices have been associated with a sustained decrease in the incidence of MRSA and VRE infections primarily in the ICU^{561, 562, 714-717}. The scientific rationale, indications, methods, and products for hand hygiene are summarized in other publications^{559, 717}.

The effectiveness of hand hygiene can be reduced by the type and length of fingernails^{559, 718, 719}. Individuals wearing artificial nails have been shown to harbor more pathogenic organisms, especially gram negative bacilli and yeasts, on the nails and in the subungual area than those with native nails^{720, 721}. In 2002, CDC/HICPAC recommended (Category IA) that artificial fingernails and extenders not be worn by healthcare personnel who have contact with high-risk patients (e.g., those in ICUs, ORs) due to the association with outbreaks of gram-negative bacillus and candidal infections as confirmed by molecular typing of isolates^{30, 31, 559, 722-725}. The need to restrict the wearing of artificial fingernails by all healthcare personnel who provide direct patient care or by healthcare personnel who have contact with other high risk groups (e.g., oncology, cystic fibrosis patients), has not been studied, but has been recommended by some experts²⁰. At this time such decisions are at the discretion of an individual facility’s infection control program. There is less evidence that jewelry affects the quality of hand hygiene. Although hand contamination with potential pathogens is increased with ring-wearing^{559, 726}, no studies have related this practice to HCW-to-patient transmission of pathogens.

II.E. Personal protective equipment (PPE) for healthcare personnel

PPE refers to a variety of barriers and respirators used alone or in combination to protect mucous membranes, airways, skin, and clothing from contact with infectious agents. The selection of PPE is based on the nature of the patient

interaction and/or the likely mode(s) of transmission. Guidance on the use of PPE is discussed in Part III. A suggested procedure for donning and removing PPE that will prevent skin or clothing contamination is presented in the Figure. Designated containers for used disposable or reusable PPE should be placed in a location that is convenient to the site of removal to facilitate disposal and containment of contaminated materials. Hand hygiene is always the final step after removing and disposing of PPE. The following sections highlight the primary uses and methods for selecting this equipment.

II.E.1. Gloves Gloves are used to prevent contamination of healthcare personnel hands when 1) anticipating direct contact with blood or body fluids, mucous membranes, nonintact skin and other potentially infectious material; 2) having direct contact with patients who are colonized or infected with pathogens transmitted by the contact route e.g., VRE, MRSA, RSV^{559, 727, 728}; or 3) handling or touching visibly or potentially contaminated patient care equipment and environmental surfaces^{72, 73, 559}. Gloves can protect both patients and healthcare personnel from exposure to infectious material that may be carried on hands⁷³. The extent to which gloves will protect healthcare personnel from transmission of bloodborne pathogens (e.g., HIV, HBV, HCV) following a needlestick or other puncture that penetrates the glove barrier has not been determined. Although gloves may reduce the volume of blood on the external surface of a sharp by 46-86%⁷²⁹, the residual blood in the lumen of a hollowbore needle would not be affected; therefore, the effect on transmission risk is unknown. Gloves manufactured for healthcare purposes are subject to FDA evaluation and clearance⁷³⁰. Nonsterile disposable medical gloves made of a variety of materials (e.g., latex, vinyl, nitrile) are available for routine patient care⁷³¹. The selection of glove type for non-surgical use is based on a number of factors, including the task that is to be performed, anticipated contact with chemicals and chemotherapeutic agents, latex sensitivity, sizing, and facility policies for creating a latex-free environment^{17, 732-734}. For contact with blood and body fluids during non-surgical patient care, a single pair of gloves generally provides adequate barrier protection⁷³⁴. However, there is considerable variability among gloves; both the quality of the manufacturing process and type of material influence their barrier effectiveness⁷³⁵. While there is little difference in the barrier properties of unused intact gloves⁷³⁶, studies have shown repeatedly that vinyl gloves have higher failure rates than latex or nitrile gloves when tested under simulated and actual clinical conditions^{731, 735-738}. For this reason either latex or nitrile gloves are preferable for clinical procedures that require manual dexterity and/or will involve more than brief patient contact. It may be necessary to stock gloves in several sizes. Heavier, reusable utility gloves are indicated for non-patient care activities, such as handling or cleaning contaminated equipment or surfaces^{11, 14, 739}.

During patient care, transmission of infectious organisms can be reduced by adhering to the principles of working from “clean” to “dirty”, and confining or limiting contamination to surfaces that are directly needed for patient care. It may be necessary to change gloves during the care of a single patient to prevent

cross-contamination of body sites^{559, 740}. It also may be necessary to change gloves if the patient interaction also involves touching portable computer keyboards or other mobile equipment that is transported from room to room. Discarding gloves between patients is necessary to prevent transmission of infectious material. Gloves must not be washed for subsequent reuse because microorganisms cannot be removed reliably from glove surfaces and continued glove integrity cannot be ensured. Furthermore, glove reuse has been associated with transmission of MRSA and gram-negative bacilli⁷⁴¹⁻⁷⁴³.

When gloves are worn in combination with other PPE, they are put on last. Gloves that fit snugly around the wrist are preferred for use with an isolation gown because they will cover the gown cuff and provide a more reliable continuous barrier for the arms, wrists, and hands. Gloves that are removed properly will prevent hand contamination (Figure). Hand hygiene following glove removal further ensures that the hands will not carry potentially infectious material that might have penetrated through unrecognized tears or that could contaminate the hands during glove removal^{559, 728, 741}.

II.E.2. Isolation gowns Isolation gowns are used as specified by Standard and Transmission-Based Precautions, to protect the HCW's arms and exposed body areas and prevent contamination of clothing with blood, body fluids, and other potentially infectious material^{24, 88, 262, 744-746}. The need for and type of isolation gown selected is based on the nature of the patient interaction, including the anticipated degree of contact with infectious material and potential for blood and body fluid penetration of the barrier. The wearing of isolation gowns and other protective apparel is mandated by the OSHA Bloodborne Pathogens Standard⁷³⁹. Clinical and laboratory coats or jackets worn over personal clothing for comfort and/or purposes of identity are not considered PPE.

When applying Standard Precautions, an isolation gown is worn only if contact with blood or body fluid is anticipated. However, when Contact Precautions are used (i.e., to prevent transmission of an infectious agent that is not interrupted by Standard Precautions alone and that is associated with environmental contamination), donning of both gown and gloves upon room entry is indicated to address unintentional contact with contaminated environmental surfaces^{54, 72, 73, 88}. The routine donning of isolation gowns upon entry into an intensive care unit or other high-risk area does not prevent or influence potential colonization or infection of patients in those areas^{365, 747-750}.

Isolation gowns are always worn in combination with gloves, and with other PPE when indicated. Gowns are usually the first piece of PPE to be donned. Full coverage of the arms and body front, from neck to the mid-thigh or below will ensure that clothing and exposed upper body areas are protected. Several gown sizes should be available in a healthcare facility to ensure appropriate coverage for staff members. Isolation gowns should be removed before leaving the patient care area to prevent possible contamination of the environment outside the patient's room. Isolation gowns should be removed in a manner that prevents contamination of clothing or skin (Figure). The outer, "contaminated", side of the

gown is turned inward and rolled into a bundle, and then discarded into a designated container for waste or linen to contain contamination.

II.E.3. Face protection: masks, goggles, face shields

II.E.3.a. Masks Masks are used for three primary purposes in healthcare settings: 1) placed on healthcare personnel to protect them from contact with infectious material from patients e.g., respiratory secretions and sprays of blood or body fluids, consistent with Standard Precautions and Droplet Precautions; 2) placed on healthcare personnel when engaged in procedures requiring sterile technique to protect patients from exposure to infectious agents carried in a healthcare worker's mouth or nose, and 3) placed on coughing patients to limit potential dissemination of infectious respiratory secretions from the patient to others (i.e., Respiratory Hygiene/Cough Etiquette). Masks may be used in combination with goggles to protect the mouth, nose and eyes, or a face shield may be used instead of a mask and goggles, to provide more complete protection for the face, as discussed below. **Masks should not be confused with particulate respirators that are used to prevent inhalation of small particles that may contain infectious agents transmitted via the airborne route as described below.**

The mucous membranes of the mouth, nose, and eyes are susceptible portals of entry for infectious agents, as can be other skin surfaces if skin integrity is compromised (e.g., by acne, dermatitis)^{66, 751-754}. Therefore, use of PPE to protect these body sites is an important component of Standard Precautions. The protective effect of masks for exposed healthcare personnel has been demonstrated^{93, 113, 755, 756}. Procedures that generate splashes or sprays of blood, body fluids, secretions, or excretions (e.g., endotracheal suctioning, bronchoscopy, invasive vascular procedures) require either a face shield (disposable or reusable) or mask and goggles^{93-95, 96, 113, 115, 262, 739, 757}. The wearing of masks, eye protection, and face shields in specified circumstances when blood or body fluid exposures are likely to occur is mandated by the OSHA Bloodborne Pathogens Standard⁷³⁹. Appropriate PPE should be selected based on the anticipated level of exposure.

Two mask types are available for use in healthcare settings: surgical masks that are cleared by the FDA and required to have fluid-resistant properties, and procedure or isolation masks^{758 #2688}. No studies have been published that compare mask types to determine whether one mask type provides better protection than another. Since procedure/isolation masks are not regulated by the FDA, there may be more variability in quality and performance than with surgical masks. Masks come in various shapes (e.g., molded and non-molded), sizes, filtration efficiency, and method of attachment (e.g., ties, elastic, ear loops). Healthcare facilities may find that different types of masks are needed to meet individual healthcare personnel needs.

II.E.3.b. Goggles, face shields Guidance on eye protection for infection control has been published⁷⁵⁹. The eye protection chosen for specific work situations (e.g., goggles or face shield) depends upon the circumstances of exposure, other

PPE used, and personal vision needs. Personal eyeglasses and contact lenses are NOT considered adequate eye protection (www.cdc.gov/niosh/topics/eye/eye-infectious.html). NIOSH states that, eye protection must be comfortable, allow for sufficient peripheral vision, and must be adjustable to ensure a secure fit. It may be necessary to provide several different types, styles, and sizes of protective equipment. Indirectly-vented goggles with a manufacturer's anti-fog coating may provide the most reliable practical eye protection from splashes, sprays, and respiratory droplets from multiple angles. Newer styles of goggles may provide better indirect airflow properties to reduce fogging, as well as better peripheral vision and more size options for fitting goggles to different workers. Many styles of goggles fit adequately over prescription glasses with minimal gaps. While effective as eye protection, goggles do not provide splash or spray protection to other parts of the face.

The role of goggles, in addition to a mask, in preventing exposure to infectious agents transmitted via respiratory droplets has been studied only for RSV. Reports published in the mid-1980s demonstrated that eye protection reduced occupational transmission of RSV^{760, 761}. Whether this was due to preventing hand-eye contact or respiratory droplet-eye contact has not been determined. However, subsequent studies demonstrated that RSV transmission is effectively prevented by adherence to Standard plus Contact Precautions and that for this virus routine use of goggles is not necessary^{24, 116, 117, 684, 762}. It is important to remind healthcare personnel that even if Droplet Precautions are not recommended for a specific respiratory tract pathogen, protection for the eyes, nose and mouth by using a mask and goggles, or face shield alone, is necessary when it is likely that there will be a splash or spray of any respiratory secretions or other body fluids as defined in Standard Precautions

Disposable or non-disposable face shields may be used as an alternative to goggles⁷⁵⁹. As compared with goggles, a face shield can provide protection to other facial areas in addition to the eyes. Face shields extending from chin to crown provide better face and eye protection from splashes and sprays; face shields that wrap around the sides may reduce splashes around the edge of the shield.

Removal of a face shield, goggles and mask can be performed safely after gloves have been removed, and hand hygiene performed. The ties, ear pieces and/or headband used to secure the equipment to the head are considered "clean" and therefore safe to touch with bare hands. The front of a mask, goggles and face shield are considered contaminated (Figure).

II.E.4. Respiratory protection The subject of respiratory protection as it applies to preventing transmission of airborne infectious agents, including the need for and frequency of fit-testing is under scientific review and was the subject of a CDC workshop in 2004⁷⁶³. Respiratory protection currently requires the use of a respirator with N95 or higher filtration to prevent inhalation of infectious particles. Information about respirators and respiratory protection programs is summarized

in the *Guideline for Preventing Transmission of Mycobacterium tuberculosis in Health-care Settings, 2005* (CDC.MMWR 2005; 54: RR-17¹²).

Respiratory protection is broadly regulated by OSHA under the general industry standard for respiratory protection (29CFR1910.134)⁷⁶⁴ which requires that U.S. employers in all employment settings implement a program to protect employees from inhalation of toxic materials. OSHA program components include medical clearance to wear a respirator; provision and use of appropriate respirators, including fit-tested NIOSH-certified N95 and higher particulate filtering respirators; education on respirator use and periodic re-evaluation of the respiratory protection program. When selecting particulate respirators, models with inherently good fit characteristics (i.e., those expected to provide protection factors of 10 or more to 95% of wearers) are preferred and could theoretically relieve the need for fit testing^{765, 766}. Issues pertaining to respiratory protection remain the subject of ongoing debate. Information on various types of respirators may be found at www.cdc.gov/niosh/npptl/respirators/respsars.html and in published studies^{765, 767, 768}. A user-seal check (formerly called a “fit check”) should be performed by the wearer of a respirator each time a respirator is donned to minimize air leakage around the facepiece⁷⁶⁹. The optimal frequency of fit-testing has not been determined; re-testing may be indicated if there is a change in facial features of the wearer, onset of a medical condition that would affect respiratory function in the wearer, or a change in the model or size of the initially assigned respirator¹².

Respiratory protection was first recommended for protection of preventing U.S. healthcare personnel from exposure to *M. tuberculosis* in 1989. That recommendation has been maintained in two successive revisions of the Guidelines for Prevention of Transmission of Tuberculosis in Hospitals and other Healthcare Settings^{12, 126}. The incremental benefit from respirator use, in addition to administrative and engineering controls (i.e., AIIRs, early recognition of patients likely to have tuberculosis and prompt placement in an AIIR, and maintenance of a patient with suspected tuberculosis in an AIIR until no longer infectious), for preventing transmission of airborne infectious agents (e.g., *M. tuberculosis*) is undetermined. Although some studies have demonstrated effective prevention of *M. tuberculosis* transmission in hospitals where surgical masks, instead of respirators, were used in conjunction with other administrative and engineering controls^{637, 770, 771}, CDC currently recommends N95 or higher level respirators for personnel exposed to patients with suspected or confirmed tuberculosis. Currently this is also true for other diseases that could be transmitted through the airborne route, including SARS²⁶² and smallpox^{108, 129, 772}, until inhalational transmission is better defined or healthcare-specific protective equipment more suitable for preventing infection are developed. Respirators are also currently recommended to be worn during the performance of aerosol-generating procedures (e.g., intubation, bronchoscopy, suctioning) on patients with SARS Co-V infection, avian influenza and pandemic influenza (See Appendix A).

Although Airborne Precautions are recommended for preventing airborne transmission of measles and varicella-zoster viruses, there are no data upon

which to base a recommendation for respiratory protection to protect susceptible personnel against these two infections; transmission of varicella-zoster virus has been prevented among pediatric patients using negative pressure isolation alone⁷⁷³. Whether respiratory protection (i.e., wearing a particulate respirator) would enhance protection from these viruses has not been studied. Since the majority of healthcare personnel have natural or acquired immunity to these viruses, only immune personnel generally care for patients with these infections⁷⁷⁴⁻⁷⁷⁷. Although there is no evidence to suggest that masks are not adequate to protect healthcare personnel in these settings, for purposes of consistency and simplicity, or because of difficulties in ascertaining immunity, some facilities may require the use of respirators for entry into all AIRs, regardless of the specific infectious agent.

Procedures for safe removal of respirators are provided (Figure). In some healthcare settings, particulate respirators used to provide care for patients *with M. tuberculosis* are reused by the same HCW. This is an acceptable practice providing the respirator is not damaged or soiled, the fit is not compromised by change in shape, and the respirator has not been contaminated with blood or body fluids. There are no data on which to base a recommendation for the length of time a respirator may be reused.

II.F. Safe work practices to prevent HCW exposure to bloodborne pathogens

II.F.1. Prevention of needlesticks and other sharps-related injuries Injuries due to needles and other sharps have been associated with transmission of HBV, HCV and HIV to healthcare personnel^{778, 779}. The prevention of sharps injuries has always been an essential element of Universal and now Standard Precautions^{1, 780}. These include measures to handle needles and other sharp devices in a manner that will prevent injury to the user and to others who may encounter the device during or after a procedure. These measures apply to routine patient care and do not address the prevention of sharps injuries and other blood exposures during surgical and other invasive procedures that are addressed elsewhere⁷⁸¹⁻⁷⁸⁵.

Since 1991, when OSHA first issued its Bloodborne Pathogens Standard to protect healthcare personnel from blood exposure, the focus of regulatory and legislative activity has been on implementing a hierarchy of control measures. This has included focusing attention on removing sharps hazards through the development and use of engineering controls. The federal Needlestick Safety and Prevention Act signed into law in November, 2000 authorized OSHA's revision of its Bloodborne Pathogens Standard to more explicitly require the use of safety-engineered sharp devices⁷⁸⁶. CDC has provided guidance on sharps injury prevention^{787, 788}, including for the design, implementation and evaluation of a comprehensive sharps injury prevention program⁷⁸⁹.

II.F.2. Prevention of mucous membrane contact Exposure of mucous membranes of the eyes, nose and mouth to blood and body fluids has been associated with the transmission of bloodborne viruses and other infectious agents to healthcare personnel^{66, 752, 754, 779}. The prevention of mucous membrane exposures has always been an element of Universal and now Standard Precautions for routine patient care^{1, 753} and is subject to OSHA bloodborne pathogen regulations. Safe work practices, in addition to wearing PPE, are used to protect mucous membranes and non-intact skin from contact with potentially infectious material. These include keeping gloved and ungloved hands that are contaminated from touching the mouth, nose, eyes, or face; and positioning patients to direct sprays and splatter away from the face of the caregiver. Careful placement of PPE before patient contact will help avoid the need to make PPE adjustments and possible face or mucous membrane contamination during use.

In areas where the need for resuscitation is unpredictable, mouthpieces, pocket resuscitation masks with one-way valves, and other ventilation devices provide an alternative to mouth-to-mouth resuscitation, preventing exposure of the caregiver's nose and mouth to oral and respiratory fluids during the procedure.

II.F.2.a. Precautions during aerosol-generating procedures The performance of procedures that can generate small particle aerosols (aerosol-generating procedures), such as bronchoscopy, endotracheal intubation, and open suctioning of the respiratory tract, have been associated with transmission of infectious agents to healthcare personnel, including *M. tuberculosis*⁷⁹⁰, SARS-CoV^{93, 94, 98} and *N. meningitidis*⁹⁵. Protection of the eyes, nose and mouth, in addition to gown and gloves, is recommended during performance of these procedures in accordance with Standard Precautions. Use of a particulate respirator is recommended during aerosol-generating procedures when the aerosol is likely to contain *M. tuberculosis*, SARS-CoV, or avian or pandemic influenza viruses.

II.G. Patient placement

II.G.1. Hospitals and long-term care settings Options for patient placement include single patient rooms, two patient rooms, and multi-bed wards. Of these, single patient rooms are preferred when there is a concern about transmission of an infectious agent. Although some studies have failed to demonstrate the efficacy of single patient rooms to prevent HAIs⁷⁹¹, other published studies, including one commissioned by the American Institute of Architects and the Facility Guidelines Institute, have documented a beneficial relationship between private rooms and reduction in infectious and noninfectious adverse patient outcomes^{792, 793}. The AIA notes that private rooms are the trend in hospital planning and design. However, most hospitals and long-term care facilities have multi-bed rooms and must consider many competing priorities when determining the appropriate room placement for patients (e.g., reason for admission; patient characteristics, such as age, gender, mental status; staffing needs; family

requests; psychosocial factors; reimbursement concerns). In the absence of obvious infectious diseases that require specified airborne infection isolation rooms (e.g., tuberculosis, SARS, chickenpox), the risk of transmission of infectious agents is not always considered when making placement decisions. When there are only a limited number of single-patient rooms, it is prudent to prioritize them for those patients who have conditions that facilitate transmission of infectious material to other patients (e.g., draining wounds, stool incontinence, uncontained secretions) and for those who are at increased risk of acquisition and adverse outcomes resulting from HAI (e.g., immunosuppression, open wounds, indwelling catheters, anticipated prolonged length of stay, total dependence on HCWs for activities of daily living)^{15, 24, 43, 430, 794, 795}. Single-patient rooms are always indicated for patients placed on Airborne Precautions and in a Protective Environment and are preferred for patients who require Contact or Droplet Precautions^{23, 24, 410, 435, 796, 797}. During a suspected or proven outbreak caused by a pathogen whose reservoir is the gastrointestinal tract, use of single patient rooms with private bathrooms limits opportunities for transmission, especially when the colonized or infected patient has poor personal hygiene habits, fecal incontinence, or cannot be expected to assist in maintaining procedures that prevent transmission of microorganisms (e.g., infants, children, and patients with altered mental status or developmental delay). In the absence of continued transmission, it is not necessary to provide a private bathroom for patients colonized or infected with enteric pathogens as long as personal hygiene practices and Standard Precautions, especially hand hygiene and appropriate environmental cleaning, are maintained. Assignment of a dedicated commode to a patient, and cleaning and disinfecting fixtures and equipment that may have fecal contamination (e.g., bathrooms, commodes⁷⁹⁸, scales used for weighing diapers) and the adjacent surfaces with appropriate agents may be especially important when a single-patient room can not be used since environmental contamination with intestinal tract pathogens is likely from both continent and incontinent patients^{54, 799}. Results of several studies to determine the benefit of a single-patient room to prevent transmission of *Clostridium difficile* are inconclusive^{167, 800-802}. Some studies have shown that being in the same room with a colonized or infected patient is not necessarily a risk factor for transmission^{791, 803-805}. However, for children, the risk of healthcare-associated diarrhea is increased with the increased number of patients per room⁸⁰⁶. Thus, patient factors are important determinants of infection transmission risks, and the need for a single-patient room and/or private bathroom for any patient is best determined on a case-by-case basis.

Cohorting is the practice of grouping together patients who are colonized or infected with the same organism to confine their care to one area and prevent contact with other patients. Cohorts are created based on clinical diagnosis, microbiologic confirmation when available, epidemiology, and mode of transmission of the infectious agent. It is generally preferred not to place severely immunosuppressed patients in rooms with other patients. Cohorting has been used extensively for managing outbreaks of MDROs including MRSA^{22, 807}, VRE^{638, 808, 809}, MDR-ESBLs⁸¹⁰, *Pseudomonas aeruginosa*²⁹; methicillin-susceptible

*Staphylococcus aureus*⁸¹¹; RSV^{812, 813}; adenovirus keratoconjunctivitis⁸¹⁴; rotavirus⁸¹⁵; and SARS⁸¹⁶. Modeling studies provide additional support for cohorting patients to control outbreaks Talon⁸¹⁷⁻⁸¹⁹. However, cohorting often is implemented only after routine infection control measures have failed to control an outbreak.

Assigning or cohorting healthcare personnel to care only for patients infected or colonized with a single target pathogen limits further transmission of the target pathogen to uninfected patients^{740, 819} but is difficult to achieve in the face of current staffing shortages in hospitals⁵⁸³ and residential healthcare sites⁸²⁰⁻⁸²². However, when continued transmission is occurring after implementing routine infection control measures and creating patient cohorts, cohorting of healthcare personnel may be beneficial.

During the seasons when RSV, human metapneumovirus⁸²³, parainfluenza, influenza, other respiratory viruses⁸²⁴, and rotavirus are circulating in the community, cohorting based on the presenting clinical syndrome is often a priority in facilities that care for infants and young children⁸²⁵. For example, during the respiratory virus season, infants may be cohorted based solely on the clinical diagnosis of bronchiolitis due to the logistical difficulties and costs associated with requiring microbiologic confirmation prior to room placement, and the predominance of RSV during most of the season. However, when available, single patient rooms are always preferred since a common clinical presentation (e.g., bronchiolitis), can be caused by more than one infectious agent^{823, 824, 826}. Furthermore, the inability of infants and children to contain body fluids, and the close physical contact that occurs during their care, increases infection transmission risks for patients and personnel in this setting^{24, 795}.

II.G.2. Ambulatory settings Patients actively infected with or incubating transmissible infectious diseases are seen frequently in ambulatory settings (e.g., outpatient clinics, physicians' offices, emergency departments) and potentially expose healthcare personnel and other patients, family members and visitors^{21, 34, 127, 135, 142, 827}. In response to the global outbreak of SARS in 2003 and in preparation for pandemic influenza, healthcare providers working in outpatient settings are urged to implement source containment measures (e.g., asking coughing patients to wear a surgical mask or cover their coughs with tissues) to prevent transmission of respiratory infections, beginning at the point of initial patient encounter^{9, 262, 828} as described below in section III.A.1.a. Signs can be posted at the entrance to facilities or at the reception or registration desk requesting that the patient or individuals accompanying the patient promptly inform the receptionist if there are symptoms of a respiratory infection (e.g., cough, flu-like illness, increased production of respiratory secretions). The presence of diarrhea, skin rash, or known or suspected exposure to a transmissible disease (e.g., measles, pertussis, chickenpox, tuberculosis) also could be added. Placement of potentially infectious patients without delay in an examination room limits the number of exposed individuals, e.g., in the common waiting area.

In waiting areas, maintaining a distance between symptomatic and non-symptomatic patients (e.g., >3 feet), in addition to source control measures, may limit exposures. However, infections transmitted via the airborne route (e.g., *M. tuberculosis*, measles, chickenpox) require additional precautions^{12, 125, 829}. Patients suspected of having such an infection can wear a surgical mask for source containment, if tolerated, and should be placed in an examination room, preferably an AIIR, as soon as possible. If this is not possible, having the patient wear a mask and segregate him/herself from other patients in the waiting area will reduce opportunities to expose others. Since the person(s) accompanying the patient also may be infectious, application of the same infection control precautions may need to be extended to these persons if they are symptomatic^{21, 252, 830}. For example, family members accompanying children admitted with suspected *M. tuberculosis* have been found to have unsuspected pulmonary tuberculosis with cavitory lesions, even when asymptomatic^{42, 831}. Patients with underlying conditions that increase their susceptibility to infection (e.g., those who are immunocompromised^{43, 44} or have cystic fibrosis²⁰) require special efforts to protect them from exposures to infected patients in common waiting areas. By informing the receptionist of their infection risk upon arrival, appropriate steps may be taken to further protect them from infection. In some cystic fibrosis clinics, in order to avoid exposure to other patients who could be colonized with *B. cepacia*, patients have been given beepers upon registration so that they may leave the area and receive notification to return when an examination room becomes available⁸³².

II.G.3. Home care In home care, the patient placement concerns focus on protecting others in the home from exposure to an infectious household member. For individuals who are especially vulnerable to adverse outcomes associated with certain infections, it may be beneficial to either remove them from the home or segregate them within the home. Persons who are not part of the household may need to be prohibited from visiting during the period of infectivity. For example, if a patient with pulmonary tuberculosis is contagious and being cared for at home, very young children (<4 years of age)⁸³³ and immunocompromised persons who have not yet been infected should be removed or excluded from the household. During the SARS outbreak of 2003, segregation of infected persons during the communicable phase of the illness was beneficial in preventing household transmission^{249, 834}.

II.H. Transport of patients

Several principles are used to guide transport of patients requiring Transmission-Based Precautions. In the inpatient and residential settings these include 1) limiting transport of such patients to essential purposes, such as diagnostic and therapeutic procedures that cannot be performed in the patient's room; 2) when transport is necessary, using appropriate barriers on the patient (e.g., mask, gown, wrapping in sheets or use of impervious dressings to cover the affected area(s) when infectious skin lesions or drainage are present, consistent with the route and risk of transmission; 3) notifying healthcare personnel in the receiving

area of the impending arrival of the patient and of the precautions necessary to prevent transmission; and 4) for patients being transported outside the facility, informing the receiving facility and the medi-van or emergency vehicle personnel in advance about the type of Transmission-Based Precautions being used. For tuberculosis, additional precautions may be needed in a small shared air space such as in an ambulance ¹².

II.I. Environmental measures

Cleaning and disinfecting non-critical surfaces in patient-care areas are part of Standard Precautions. In general, these procedures do not need to be changed for patients on Transmission-Based Precautions. The cleaning and disinfection of all patient-care areas is important for frequently touched surfaces, especially those closest to the patient, that are most likely to be contaminated (e.g., bedrails, bedside tables, commodes, doorknobs, sinks, surfaces and equipment in close proximity to the patient) ^{11, 72, 73, 835}. The frequency or intensity of cleaning may need to change based on the patient's level of hygiene and the degree of environmental contamination and for certain for infectious agents whose reservoir is the intestinal tract ⁵⁴. This may be especially true in LTCFs and pediatric facilities where patients with stool and urine incontinence are encountered more frequently. Also, increased frequency of cleaning may be needed in a Protective Environment to minimize dust accumulation ¹¹. Special recommendations for cleaning and disinfecting environmental surfaces in dialysis centers have been published ¹⁸. In all healthcare settings, administrative, staffing and scheduling activities should prioritize the proper cleaning and disinfection of surfaces that could be implicated in transmission. During a suspected or proven outbreak where an environmental reservoir is suspected, routine cleaning procedures should be reviewed, and the need for additional trained cleaning staff should be assessed. Adherence should be monitored and reinforced to promote consistent and correct cleaning is performed.

EPA-registered disinfectants or detergents/disinfectants that best meet the overall needs of the healthcare facility for routine cleaning and disinfection should be selected ^{11, 836}. In general, use of the existing facility detergent/disinfectant according to the manufacturer's recommendations for amount, dilution, and contact time is sufficient to remove pathogens from surfaces of rooms where colonized or infected individuals were housed. This includes those pathogens that are resistant to multiple classes of antimicrobial agents (e.g., *C. difficile*, VRE, MRSA, MDR-GNB ^{11, 24, 88, 435, 746, 796, 837}). Most often, environmental reservoirs of pathogens during outbreaks are related to a failure to follow recommended procedures for cleaning and disinfection rather than the specific cleaning and disinfectant agents used ⁸³⁸⁻⁸⁴¹.

Certain pathogens (e.g., rotavirus, noroviruses, *C. difficile*) may be resistant to some routinely used hospital disinfectants ^{275, 292, 842-847}. The role of specific disinfectants in limiting transmission of rotavirus has been demonstrated experimentally ⁸⁴². Also, since *C. difficile* may display increased levels of spore production when exposed to non-chlorine-based cleaning agents, and the spores are more resistant than vegetative cells to commonly used surface disinfectants,

some investigators have recommended the use of a 1:10 dilution of 5.25% sodium hypochlorite (household bleach) and water for routine environmental disinfection of rooms of patients with *C. difficile* when there is continued transmission^{844, 848}. In one study, the use of a hypochlorite solution was associated with a decrease in rates of *C. difficile* infections⁸⁴⁷. The need to change disinfectants based on the presence of these organisms can be determined in consultation with the infection control committee^{11, 847, 848}. Detailed recommendations for disinfection and sterilization of surfaces and medical equipment that have been in contact with prion-containing tissue or high risk body fluids, and for cleaning of blood and body substance spills, are available in the Guidelines for Environmental Infection Control in Health-Care Facilities¹¹ and in the Guideline for Disinfection and Sterilization⁸⁴⁸.

II.J. Patient care equipment and instruments/devices

Medical equipment and instruments/devices must be cleaned and maintained according to the manufacturers' instructions to prevent patient-to-patient transmission of infectious agents^{86, 87, 325, 849}. Cleaning to remove organic material must always precede high level disinfection and sterilization of critical and semi-critical instruments and devices because residual proteinaceous material reduces the effectiveness of the disinfection and sterilization processes^{836, 848}. Noncritical equipment, such as commodes, intravenous pumps, and ventilators, must be thoroughly cleaned and disinfected before use on another patient. All such equipment and devices should be handled in a manner that will prevent HCW and environmental contact with potentially infectious material. It is important to include computers and personal digital assistants (PDAs) used in patient care in policies for cleaning and disinfection of non-critical items. The literature on contamination of computers with pathogens has been summarized⁸⁵⁰ and two reports have linked computer contamination to colonization and infections in patients^{851, 852}. Although keyboard covers and washable keyboards that can be easily disinfected are in use, the infection control benefit of those items and optimal management have not been determined.

In all healthcare settings, providing patients who are on Transmission-Based Precautions with dedicated noncritical medical equipment (e.g., stethoscope, blood pressure cuff, electronic thermometer) has been beneficial for preventing transmission^{74, 89, 740, 853, 854}. When this is not possible, disinfection after use is recommended. Consult other guidelines for detailed guidance in developing specific protocols for cleaning and reprocessing medical equipment and patient care items in both routine and special circumstances^{11, 14, 18, 20, 740, 836, 848}.

In home care, it is preferable to remove visible blood or body fluids from durable medical equipment before it leaves the home. Equipment can be cleaned on-site using a detergent/disinfectant and, when possible, should be placed in a single plastic bag for transport to the reprocessing location^{20, 739}.

II.K. Textiles and laundry

Soiled textiles, including bedding, towels, and patient or resident clothing may be contaminated with pathogenic microorganisms. However, the risk of disease

transmission is negligible if they are handled, transported, and laundered in a safe manner ^{11, 855, 856}. Key principles for handling soiled laundry are 1) not shaking the items or handling them in any way that may aerosolize infectious agents; 2) avoiding contact of one's body and personal clothing with the soiled items being handled; and 3) containing soiled items in a laundry bag or designated bin. When laundry chutes are used, they must be maintained to minimize dispersion of aerosols from contaminated items ¹¹. The methods for handling, transporting, and laundering soiled textiles are determined by organizational policy and any applicable regulations ⁷³⁹; guidance is provided in the Guidelines for Environmental Infection Control ¹¹. Rather than rigid rules and regulations, hygienic and common sense storage and processing of clean textiles is recommended ^{11, 857}. When laundering occurs outside of a healthcare facility, the clean items must be packaged or completely covered and placed in an enclosed space during transport to prevent contamination with outside air or construction dust that could contain infectious fungal spores that are a risk for immunocompromised patients ¹¹.

Institutions are required to launder garments used as personal protective equipment and uniforms visibly soiled with blood or infective material ⁷³⁹. There are few data to determine the safety of home laundering of HCW uniforms, but no increase in infection rates was observed in the one published study ⁸⁵⁸ and no pathogens were recovered from home- or hospital-laundered scrubs in another study ⁸⁵⁹. In the home, textiles and laundry from patients with potentially transmissible infectious pathogens do not require special handling or separate laundering, and may be washed with warm water and detergent ^{11, 858, 859}.

II.L. Solid waste

The management of solid waste emanating from the healthcare environment is subject to federal and state regulations for medical and non-medical waste ^{860, 861}. No additional precautions are needed for non-medical solid waste that is being removed from rooms of patients on Transmission-Based Precautions. Solid waste may be contained in a single bag (as compared to using two bags) of sufficient strength. ⁸⁶².

II.M. Dishware and eating utensils

The combination of hot water and detergents used in dishwashers is sufficient to decontaminate dishware and eating utensils. Therefore, no special precautions are needed for dishware (e.g., dishes, glasses, cups) or eating utensils; reusable dishware and utensils may be used for patients requiring Transmission-Based Precautions. In the home and other communal settings, eating utensils and drinking vessels that are being used should not be shared, consistent with principles of good personal hygiene and for the purpose of preventing transmission of respiratory viruses, *Herpes simplex* virus, and infectious agents that infect the gastrointestinal tract and are transmitted by the fecal/oral route (e.g., hepatitis A virus, noroviruses). If adequate resources for cleaning utensils and dishes are not available, disposable products may be used.

II.N. Adjunctive measures

Important adjunctive measures that are not considered primary components of programs to prevent transmission of infectious agents, but improve the effectiveness of such programs, include 1) antimicrobial management programs; 2) postexposure chemoprophylaxis with antiviral or antibacterial agents; 3) vaccines used both for pre and postexposure prevention; and 4) screening and restricting visitors with signs of transmissible infections. Detailed discussion of judicious use of antimicrobial agents is beyond the scope of this document; however the topic is addressed in the MDRO section (Management of Multidrug-Resistant Organisms in Healthcare Settings 2006.

www.cdc.gov/ncidod/dhqp/pdf/ar/mdroGuideline2006.pdf).

II.N.1. Chemoprophylaxis Antimicrobial agents and topical antiseptics may be used to prevent infection and potential outbreaks of selected agents. Infections for which postexposure chemoprophylaxis is recommended under defined conditions include *B. pertussis*^{17, 863}, *N. meningitidis*⁸⁶⁴, *B. anthracis* after environmental exposure to aerosolizable material⁸⁶⁵, influenza virus⁶¹¹, HIV⁸⁶⁶, and group A streptococcus¹⁶⁰. Orally administered antimicrobials may also be used under defined circumstances for MRSA decolonization of patients or healthcare personnel⁸⁶⁷.

Another form of chemoprophylaxis is the use of topical antiseptic agents. For example, triple dye is used routinely on the umbilical cords of term newborns to reduce the risk of colonization, skin infections, and omphalitis caused by *S. aureus*, including MRSA, and group A streptococcus^{868, 869}. Extension of the use of triple dye to low birth weight infants in the NICU was one component of a program that controlled one longstanding MRSA outbreak²². Topical antiseptics are also used for decolonization of healthcare personnel or selected patients colonized with MRSA, using mupirocin as discussed in the MDRO guideline⁸⁷⁰
^{867, 871-873}.

II.N.2. Immunoprophylaxis Certain immunizations recommended for susceptible healthcare personnel have decreased the risk of infection and the potential for transmission in healthcare facilities^{17, 874}. The OSHA mandate that requires employers to offer hepatitis B vaccination to HCWs played a substantial role in the sharp decline in incidence of occupational HBV infection^{778, 875}. The use of varicella vaccine in healthcare personnel has decreased the need to place susceptible HCWs on administrative leave following exposure to patients with varicella⁷⁷⁵. Also, reports of healthcare-associated transmission of rubella in obstetrical clinics^{33, 876} and measles in acute care settings³⁴ demonstrate the importance of immunization of susceptible healthcare personnel against childhood diseases. Many states have requirements for HCW vaccination for measles and rubella in the absence of evidence of immunity. Annual influenza vaccine campaigns targeted to patients and healthcare personnel in LTCFs and acute-care settings have been instrumental in preventing or limiting institutional

outbreaks and increasing attention is being directed toward improving influenza vaccination rates in healthcare personnel ^{35, 611, 690, 877, 878, 879}.

Transmission of *B. pertussis* in healthcare facilities has been associated with large and costly outbreaks that include both healthcare personnel and patients ^{17, 36, 41, 100, 683, 827, 880, 881}.

HCWs who have close contact with infants with pertussis are at particularly high risk because of waning immunity and, until 2005, the absence of a vaccine that could be used in adults. However, two acellular pertussis vaccines were licensed in the United States in 2005, one for use in individuals aged 11-18 and one for use in ages 10-64 years ⁸⁸². Provisional ACIP recommendations at the time of publication of this document include adolescents and adults, especially those with contact with infants < 12 months of age and healthcare personnel with direct patient contact ^{883 884}.

Immunization of children and adults will help prevent the introduction of vaccine-preventable diseases into healthcare settings. The recommended immunization schedule for children is published annually in the January issues of the *Morbidity Mortality Weekly Report* with interim updates as needed ^{885, 886}. An adult immunization schedule also is available for healthy adults and those with special immunization needs due to high risk medical conditions ⁸⁸⁷.

Some vaccines are also used for postexposure prophylaxis of susceptible individuals, including varicella ⁸⁸⁸, influenza ⁶¹¹, hepatitis B ⁷⁷⁸, and smallpox ²²⁵ vaccines ^{17, 874}. In the future, administration of a newly developed *S. aureus* conjugate vaccine (still under investigation) to selected patients may provide a novel method of preventing healthcare-associated *S. aureus*, including MRSA, infections in high-risk groups (e.g., hemodialysis patients and candidates for selected surgical procedures) ^{889, 890}.

Immune globulin preparations also are used for postexposure prophylaxis of certain infectious agents under specified circumstances (e.g., varicella-zoster virus [VZIG], hepatitis B virus [HBIG], rabies [RIG], measles and hepatitis A virus [IG] ^{17, 833, 874}). The RSV monoclonal antibody preparation, Palivizumab, may have contributed to controlling a nosocomial outbreak of RSV in one NICU, but there is insufficient evidence to support a routine recommendation for its use in this setting ⁸⁹¹.

II.N. 3. Management of visitors

II.N.3.a. Visitors as sources of infection Visitors have been identified as the source of several types of HAIs (e.g., pertussis ^{40, 41}, *M. tuberculosis* ^{42, 892}, influenza, and other respiratory viruses ^{24, 43, 44, 373} and SARS ^{21, 252-254}). However, effective methods for visitor screening in healthcare settings have not been studied. Visitor screening is especially important during community outbreaks of infectious diseases and for high risk patient units. Sibling visits are often encouraged in birthing centers, post partum rooms and in pediatric inpatient units, ICUs, and in residential settings for children; in hospital settings, a child visitor should visit only his or her own sibling. Screening of visiting siblings and other children before they are allowed into clinical areas is necessary to prevent the introduction of childhood illnesses and common respiratory infections.

Screening may be passive through the use of signs to alert family members and visitors with signs and symptoms of communicable diseases not to enter clinical areas. More active screening may include the completion of a screening tool or questionnaire which elicits information related to recent exposures or current symptoms. That information is reviewed by the facility staff and the visitor is either permitted to visit or is excluded⁸³³.

Family and household members visiting pediatric patients with pertussis and tuberculosis may need to be screened for a history of exposure as well as signs and symptoms of current infection. Potentially infectious visitors are excluded until they receive appropriate medical screening, diagnosis, or treatment. If exclusion is not considered to be in the best interest of the patient or family (i.e., primary family members of critically or terminally ill patients), then the symptomatic visitor must wear a mask while in the healthcare facility and remain in the patient's room, avoiding exposure to others, especially in public waiting areas and the cafeteria.

Visitor screening is used consistently on HSCT units^{15, 43}. However, considering the experience during the 2003 SARS outbreaks and the potential for pandemic influenza, developing effective visitor screening systems will be beneficial⁹.

Education concerning Respiratory Hygiene/Cough Etiquette is a useful adjunct to visitor screening.

II.N.3.b. Use of barrier precautions by visitors The use of gowns, gloves, or masks by visitors in healthcare settings has not been addressed specifically in the scientific literature. Some studies included the use of gowns and gloves by visitors in the control of MDRO's, but did not perform a separate analysis to determine whether their use by visitors had a measurable impact⁸⁹³⁻⁸⁹⁵. Family members or visitors who are providing care or having very close patient contact (e.g., feeding, holding) may have contact with other patients and could contribute to transmission if barrier precautions are not used correctly. Specific recommendations may vary by facility or by unit and should be determined by the level of interaction.

Part III:

Precautions to Prevent Transmission of Infectious Agents There are two tiers of HICPAC/CDC precautions to prevent transmission of infectious agents, Standard Precautions and Transmission-Based Precautions. Standard Precautions are intended to be applied to the care of all patients in all healthcare settings, regardless of the suspected or confirmed presence of an infectious agent. **Implementation of *Standard Precautions* constitutes the primary strategy for the prevention of healthcare-associated transmission of infectious agents among patients and healthcare personnel.**

Transmission-Based Precautions are for patients who are known or suspected to be infected or colonized with infectious agents, including certain epidemiologically important pathogens, which require additional control measures to effectively prevent transmission. Since the infecting agent often is not known at the time of admission to a healthcare facility, Transmission-Based Precautions are used empirically, according to the clinical syndrome and the likely etiologic agents at the time, and then modified when the pathogen is identified or a transmissible infectious etiology is ruled out. Examples of this syndromic approach are presented in Table 2. The HICPAC/CDC Guidelines also include recommendations for creating a Protective Environment for allogeneic HSCT patients.

The specific elements of Standard and Transmission-Based Precautions are discussed in Part II of this guideline. In Part III, the circumstances in which Standard Precautions, Transmission-Based Precautions, and a Protective Environment are applied are discussed. See Tables 4 and 5 for summaries of the key elements of these sets of precautions

III.A. Standard Precautions Standard Precautions combine the major features of Universal Precautions (UP)^{780, 896} and Body Substance Isolation (BSI)⁶⁴⁰ and are based on the principle that all blood, body fluids, secretions, excretions except sweat, nonintact skin, and mucous membranes may contain transmissible infectious agents. Standard Precautions include a group of infection prevention practices that apply to all patients, regardless of suspected or confirmed infection status, in any setting in which healthcare is delivered (Table 4). These include: hand hygiene; use of gloves, gown, mask, eye protection, or face shield, depending on the anticipated exposure; and safe injection practices. Also, equipment or items in the patient environment likely to have been contaminated with infectious body fluids must be handled in a manner to prevent transmission of infectious agents (e.g. wear gloves for direct contact, contain heavily soiled equipment, properly clean and disinfect or sterilize reusable equipment before use on another patient).

The application of Standard Precautions during patient care is determined by the nature of the HCW-patient interaction and the extent of anticipated blood, body fluid, or pathogen exposure. For some interactions (e.g., performing venipuncture), only gloves may be needed; during other interactions (e.g., intubation), use of gloves, gown, and face shield or mask and goggles is necessary. Education and training on the principles and rationale for

recommended practices are critical elements of Standard Precautions because they facilitate appropriate decision-making and promote adherence when HCWs are faced with new circumstances^{655, 681-686}. An example of the importance of the use of Standard Precautions is intubation, especially under emergency circumstances when infectious agents may not be suspected, but later are identified (e.g., SARS-CoV, *N. meningitidis*). The application of Standard Precautions is described below and summarized in Table 4. Guidance on donning and removing gloves, gowns and other PPE is presented in the Figure. Standard Precautions are also intended to protect patients by ensuring that healthcare personnel do not carry infectious agents to patients on their hands or via equipment used during patient care.

III.A.1. New Elements of Standard Precautions Infection control problems that are identified in the course of outbreak investigations often indicate the need for new recommendations or reinforcement of existing infection control recommendations to protect patients. Because such recommendations are considered a standard of care and may not be included in other guidelines, they are added here to Standard Precautions. Three such areas of practice that have been added are: Respiratory Hygiene/Cough Etiquette, safe injection practices, and use of masks for insertion of catheters or injection of material into spinal or epidural spaces via lumbar puncture procedures (e.g., myelogram, spinal or epidural anesthesia). While most elements of Standard Precautions evolved from Universal Precautions that were developed for protection of healthcare personnel, these new elements of Standard Precautions focus on protection of patients.

III.A.1.a. Respiratory Hygiene/Cough Etiquette The transmission of SARS-CoV in emergency departments by patients and their family members during the widespread SARS outbreaks in 2003 highlighted the need for vigilance and prompt implementation of infection control measures at the first point of encounter within a healthcare setting (e.g., reception and triage areas in emergency departments, outpatient clinics, and physician offices)^{21, 254, 897}. The strategy proposed has been termed Respiratory Hygiene/Cough Etiquette^{9, 828} and is intended to be incorporated into infection control practices as a new component of Standard Precautions. The strategy is targeted at patients and accompanying family members and friends with undiagnosed transmissible respiratory infections, and applies to any person with signs of illness including cough, congestion, rhinorrhea, or increased production of respiratory secretions when entering a healthcare facility^{40, 41, 43}. The term *cough etiquette* is derived from recommended source control measures for *M. tuberculosis*^{12, 126}. The elements of Respiratory Hygiene/Cough Etiquette include 1) education of healthcare facility staff, patients, and visitors; 2) posted signs, in language(s) appropriate to the population served, with instructions to patients and accompanying family members or friends; 3) source control measures (e.g., covering the mouth/nose with a tissue when coughing and prompt disposal of used tissues, using surgical masks on the coughing person when tolerated and

appropriate); 4) hand hygiene after contact with respiratory secretions; and 5) spatial separation, ideally >3 feet, of persons with respiratory infections in common waiting areas when possible. Covering sneezes and coughs and placing masks on coughing patients are proven means of source containment that prevent infected persons from dispersing respiratory secretions into the air^{107, 145, 898, 899}. Masking may be difficult in some settings, (e.g., pediatrics, in which case, the emphasis by necessity may be on cough etiquette⁹⁰⁰. Physical proximity of <3 feet has been associated with an increased risk for transmission of infections via the droplet route (e.g., *N. meningitidis*¹⁰³ and group A streptococcus¹¹⁴ and therefore supports the practice of distancing infected persons from others who are not infected. The effectiveness of good hygiene practices, especially hand hygiene, in preventing transmission of viruses and reducing the incidence of respiratory infections both within and outside⁹⁰¹⁻⁹⁰³ healthcare settings is summarized in several reviews^{559, 717, 904}.

These measures should be effective in decreasing the risk of transmission of pathogens contained in large respiratory droplets (e.g., influenza virus²³, adenovirus¹¹¹, *B. pertussis*⁸²⁷ and *Mycoplasma pneumoniae*¹¹². Although fever will be present in many respiratory infections, patients with pertussis and mild upper respiratory tract infections are often afebrile. Therefore, the absence of fever does not always exclude a respiratory infection. Patients who have asthma, allergic rhinitis, or chronic obstructive lung disease also may be coughing and sneezing. While these patients often are not infectious, cough etiquette measures are prudent.

Healthcare personnel are advised to observe Droplet Precautions (i.e., wear a mask) and hand hygiene when examining and caring for patients with signs and symptoms of a respiratory infection. Healthcare personnel who have a respiratory infection are advised to avoid direct patient contact, especially with high risk patients. If this is not possible, then a mask should be worn while providing patient care.

III.A.1.b. Safe Injection Practices The investigation of four large outbreaks of HBV and HCV among patients in ambulatory care facilities in the United States identified a need to define and reinforce safe injection practices⁴⁵³. The four outbreaks occurred in a private medical practice, a pain clinic, an endoscopy clinic, and a hematology/oncology clinic. The primary breaches in infection control practice that contributed to these outbreaks were 1) reinsertion of used needles into a multiple-dose vial or solution container (e.g., saline bag) and 2) use of a single needle/syringe to administer intravenous medication to multiple patients. In one of these outbreaks, preparation of medications in the same workspace where used needle/syringes were dismantled also may have been a contributing factor. These and other outbreaks of viral hepatitis could have been prevented by adherence to basic principles of aseptic technique for the preparation and administration of parenteral medications^{453, 454}. These include the use of a sterile, single-use, disposable needle and syringe for each injection given and prevention of contamination of injection equipment and medication.

Whenever possible, use of single-dose vials is preferred over multiple-dose vials, especially when medications will be administered to multiple patients. Outbreaks related to unsafe injection practices indicate that some healthcare personnel are unaware of, do not understand, or do not adhere to basic principles of infection control and aseptic technique. A survey of US healthcare workers who provide medication through injection found that 1% to 3% reused the same needle and/or syringe on multiple patients⁹⁰⁵. Among the deficiencies identified in recent outbreaks were a lack of oversight of personnel and failure to follow-up on reported breaches in infection control practices in ambulatory settings. Therefore, to ensure that all healthcare workers understand and adhere to recommended practices, principles of infection control and aseptic technique need to be reinforced in training programs and incorporated into institutional policies that are monitored for adherence⁴⁵⁴.

III.A.1.c. Infection Control Practices for Special Lumbar Puncture

Procedures In 2004, CDC investigated eight cases of post-myelography meningitis that either were reported to CDC or identified through a survey of the Emerging Infections Network of the Infectious Disease Society of America. Blood and/or cerebrospinal fluid of all eight cases yielded streptococcal species consistent with oropharyngeal flora and there were changes in the CSF indices and clinical status indicative of bacterial meningitis. Equipment and products used during these procedures (e.g., contrast media) were excluded as probable sources of contamination. Procedural details available for seven cases determined that antiseptic skin preparations and sterile gloves had been used. However, none of the clinicians wore a face mask, giving rise to the speculation that droplet transmission of oropharyngeal flora was the most likely explanation for these infections. Bacterial meningitis following myelogram and other spinal procedures (e.g., lumbar puncture, spinal and epidural anesthesia, intrathecal chemotherapy) has been reported previously⁹⁰⁶⁻⁹¹⁵. As a result, the question of whether face masks should be worn to prevent droplet spread of oral flora during spinal procedures (e.g., myelogram, lumbar puncture, spinal anesthesia) has been debated^{916,917}. Face masks are effective in limiting the dispersal of oropharyngeal droplets⁹¹⁸ and are recommended for the placement of central venous catheters⁹¹⁹. In October 2005, the Healthcare Infection Control Practices Advisory Committee (HICPAC) reviewed the evidence and concluded that there is sufficient experience to warrant the additional protection of a face mask for the individual placing a catheter or injecting material into the spinal or epidural space.

III.B. Transmission-Based Precautions There are three categories of Transmission-Based Precautions: Contact Precautions, Droplet Precautions, and Airborne Precautions. Transmission-Based Precautions are used when the route(s) of transmission is (are) not completely interrupted using Standard Precautions alone. For some diseases that have multiple routes of transmission (e.g., SARS), more than one Transmission-Based Precautions category may be used. When used either singly or in combination, they are always used in

addition to Standard Precautions. See Appendix A for recommended precautions for specific infections. When Transmission-Based Precautions are indicated, efforts must be made to counteract possible adverse effects on patients (i.e., anxiety, depression and other mood disturbances⁹²⁰⁻⁹²², perceptions of stigma⁹²³, reduced contact with clinical staff⁹²⁴⁻⁹²⁶, and increases in preventable adverse events⁵⁶⁵ in order to improve acceptance by the patients and adherence by HCWs.

III.B.1. Contact Precautions Contact Precautions are intended to prevent transmission of infectious agents, including epidemiologically important microorganisms, which are spread by direct or indirect contact with the patient or the patient's environment as described in I.B.3.a. The specific agents and circumstance for which Contact Precautions are indicated are found in Appendix A. The application of Contact Precautions for patients infected or colonized with MDROs is described in the 2006 HICPAC/CDC MDRO guideline⁹²⁷. Contact Precautions also apply where the presence of excessive wound drainage, fecal incontinence, or other discharges from the body suggest an increased potential for extensive environmental contamination and risk of transmission. A single-patient room is preferred for patients who require Contact Precautions. When a single-patient room is not available, consultation with infection control personnel is recommended to assess the various risks associated with other patient placement options (e.g., cohorting, keeping the patient with an existing roommate). In multi-patient rooms, ≥ 3 feet spatial separation between beds is advised to reduce the opportunities for inadvertent sharing of items between the infected/colonized patient and other patients. Healthcare personnel caring for patients on Contact Precautions wear a gown and gloves for all interactions that may involve contact with the patient or potentially contaminated areas in the patient's environment. Donning PPE upon room entry and discarding before exiting the patient room is done to contain pathogens, especially those that have been implicated in transmission through environmental contamination (e.g., VRE, *C. difficile*, noroviruses and other intestinal tract pathogens; RSV)^{54, 72, 73, 78, 274, 275, 740}.

III.B.2. Droplet Precautions Droplet Precautions are intended to prevent transmission of pathogens spread through close respiratory or mucous membrane contact with respiratory secretions as described in I.B.3.b. Because these pathogens do not remain infectious over long distances in a healthcare facility, special air handling and ventilation are not required to prevent droplet transmission. Infectious agents for which Droplet Precautions are indicated are found in Appendix A and include *B. pertussis*, influenza virus, adenovirus, rhinovirus, *N. meningitides*, and group A streptococcus (for the first 24 hours of antimicrobial therapy). A single patient room is preferred for patients who require Droplet Precautions. When a single-patient room is not available, consultation with infection control personnel is recommended to assess the various risks associated with other patient placement options (e.g., cohorting, keeping the patient with an existing roommate). Spatial separation of ≥ 3 feet and drawing

the curtain between patient beds is especially important for patients in multi-bed rooms with infections transmitted by the droplet route. Healthcare personnel wear a mask (a respirator is not necessary) for close contact with infectious patient; the mask is generally donned upon room entry. Patients on Droplet Precautions who must be transported outside of the room should wear a mask if tolerated and follow Respiratory Hygiene/Cough Etiquette.

III.B.3. Airborne Precautions Airborne Precautions prevent transmission of infectious agents that remain infectious over long distances when suspended in the air (e.g., rubeola virus [measles], varicella virus [chickenpox], *M. tuberculosis*, and possibly SARS-CoV) as described in I.B.3.c and Appendix A. The preferred placement for patients who require Airborne Precautions is in an airborne infection isolation room (AIIR). An AIIR is a single-patient room that is equipped with special air handling and ventilation capacity that meet the American Institute of Architects/Facility Guidelines Institute (AIA/FGI) standards for AIIRs (i.e., monitored negative pressure relative to the surrounding area, 12 air exchanges per hour for new construction and renovation and 6 air exchanges per hour for existing facilities, air exhausted directly to the outside or recirculated through HEPA filtration before return)^{12, 13}. Some states require the availability of such rooms in hospitals, emergency departments, and nursing homes that care for patients with *M. tuberculosis*. A respiratory protection program that includes education about use of respirators, fit-testing, and user seal checks is required in any facility with AIIRs. In settings where Airborne Precautions cannot be implemented due to limited engineering resources (e.g., physician offices), masking the patient, placing the patient in a private room (e.g., office examination room) with the door closed, and providing N95 or higher level respirators or masks if respirators are not available for healthcare personnel will reduce the likelihood of airborne transmission until the patient is either transferred to a facility with an AIIR or returned to the home environment, as deemed medically appropriate. Healthcare personnel caring for patients on Airborne Precautions wear a mask or respirator, depending on the disease-specific recommendations (Respiratory Protection II.E.4, Table 2, and Appendix A), that is donned prior to room entry. Whenever possible, non-immune HCWs should not care for patients with vaccine-preventable airborne diseases (e.g., measles, chickenpox, and smallpox).

III.C. Syndromic and empiric applications of Transmission-Based Precautions Diagnosis of many infections requires laboratory confirmation. Since laboratory tests, especially those that depend on culture techniques, often require two or more days for completion, Transmission-Based Precautions must be implemented while test results are pending based on the clinical presentation and likely pathogens. Use of appropriate Transmission-Based Precautions at the time a patient develops symptoms or signs of transmissible infection, or arrives at a healthcare facility for care, reduces transmission opportunities. While it is not possible to identify prospectively all patients needing Transmission-Based Precautions, certain clinical syndromes and conditions carry a sufficiently high

risk to warrant their use empirically while confirmatory tests are pending (Table 2). Infection control professionals are encouraged to modify or adapt this table according to local conditions.

III.D. Discontinuation of Transmission-Based Precautions Transmission-Based Precautions remain in effect for limited periods of time (i.e., while the risk for transmission of the infectious agent persists or for the duration of the illness (Appendix A). For most infectious diseases, this duration reflects known patterns of persistence and shedding of infectious agents associated with the natural history of the infectious process and its treatment. For some diseases (e.g., pharyngeal or cutaneous diphtheria, RSV), Transmission-Based Precautions remain in effect until culture or antigen-detection test results document eradication of the pathogen and, for RSV, symptomatic disease is resolved. For other diseases, (e.g., *M. tuberculosis*) state laws and regulations, and healthcare facility policies, may dictate the duration of precautions¹²). In immunocompromised patients, viral shedding can persist for prolonged periods of time (many weeks to months) and transmission to others may occur during that time; therefore, the duration of contact and/or droplet precautions may be prolonged for many weeks^{500, 928-933}.

The duration of Contact Precautions for patients who are colonized or infected with MDROs remains undefined. MRSA is the only MDRO for which effective decolonization regimens are available⁸⁶⁷. However, carriers of MRSA who have negative nasal cultures after a course of systemic or topical therapy may resume shedding MRSA in the weeks that follow therapy^{934, 935}. Although early guidelines for VRE suggested discontinuation of Contact Precautions after three stool cultures obtained at weekly intervals proved negative⁷⁴⁰, subsequent experiences have indicated that such screening may fail to detect colonization that can persist for >1 year^{27, 936-938}. Likewise, available data indicate that colonization with VRE, MRSA⁹³⁹, and possibly MDR-GNB, can persist for many months, especially in the presence of severe underlying disease, invasive devices, and recurrent courses of antimicrobial agents.

It may be prudent to assume that MDRO carriers are colonized permanently and manage them accordingly. Alternatively, an interval free of hospitalizations, antimicrobial therapy, and invasive devices (e.g., 6 or 12 months) before reculturing patients to document clearance of carriage may be used. Determination of the best strategy awaits the results of additional studies. See the 2006 HICPAC/CDC MDRO guideline⁹²⁷ for discussion of possible criteria to discontinue Contact Precautions for patients colonized or infected with MDROs.

III.E. Application of Transmission-Based Precautions in ambulatory and home care settings Although Transmission-Based Precautions generally apply in all healthcare settings, exceptions exist. For example, in home care, AIIRs are not available. Furthermore, family members already exposed to diseases such as varicella and tuberculosis would not use masks or respiratory protection, but visiting HCWs would need to use such protection. Similarly, management of patients colonized or infected with MDROs may necessitate

Contact Precautions in acute care hospitals and in some LTCFs when there is continued transmission, but the risk of transmission in ambulatory care and home care, has not been defined. Consistent use of Standard Precautions may suffice in these settings, but more information is needed.

III.F. Protective Environment A Protective Environment is designed for allogeneic HSCT patients to minimize fungal spore counts in the air and reduce the risk of invasive environmental fungal infections (see Table 5 for specifications)^{11, 13-15}. The need for such controls has been demonstrated in studies of aspergillus outbreaks associated with construction^{11, 14, 15, 157, 158}. As defined by the American Institute of Architecture¹³ and presented in detail in the Guideline for Environmental Infection Control 2003^{11, 861}, air quality for HSCT patients is improved through a combination of environmental controls that include 1) HEPA filtration of incoming air; 2) directed room air flow; 3) positive room air pressure relative to the corridor; 4) well-sealed rooms (including sealed walls, floors, ceilings, windows, electrical outlets) to prevent flow of air from the outside; 5) ventilation to provide ≥ 12 air changes per hour; 6) strategies to minimize dust (e.g., scrubbable surfaces rather than upholstery⁹⁴⁰ and carpet⁹⁴¹, and routinely cleaning crevices and sprinkler heads); and 7) prohibiting dried and fresh flowers and potted plants in the rooms of HSCT patients. The latter is based on molecular typing studies that have found indistinguishable strains of *Aspergillus terreus* in patients with hematologic malignancies and in potted plants in the vicinity of the patients⁹⁴²⁻⁹⁴⁴. The desired quality of air may be achieved without incurring the inconvenience or expense of laminar airflow^{15, 157}. To prevent inhalation of fungal spores during periods when construction, renovation, or other dust-generating activities that may be ongoing in and around the health-care facility, it has been advised that severely immunocompromised patients wear a high-efficiency respiratory-protection device (e.g., an N95 respirator) when they leave the Protective Environment^{11, 14, 945}). The use of masks or respirators by HSCT patients when they are outside of the Protective Environment for prevention of environmental fungal infections in the absence of construction has not been evaluated. A Protective Environment does not include the use of barrier precautions beyond those indicated for Standard and Transmission-Based Precautions. No published reports support the benefit of placing solid organ transplants or other immunocompromised patients in a Protective Environment.

Part IV:

Recommendations

These recommendations are designed to prevent transmission of infectious agents among patients and healthcare personnel in all settings where healthcare is delivered. As in other CDC/HICPAC guidelines, each recommendation is categorized on the basis of existing scientific data, theoretical rationale, applicability, and when possible, economic impact. The CDC/HICPAC system for categorizing recommendations is as follows:

Category IA Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiologic studies.

Category IB Strongly recommended for implementation and supported by some experimental, clinical, or epidemiologic studies and a strong theoretical rationale.

Category IC Required for implementation, as mandated by federal and/or state regulation or standard.

Category II Suggested for implementation and supported by suggestive clinical or epidemiologic studies or a theoretical rationale.

No recommendation; unresolved issue. Practices for which insufficient evidence or no consensus regarding efficacy exists.

I. Administrative Responsibilities

Healthcare organization administrators should ensure the implementation of recommendations in this section.

- I.A. Incorporate preventing transmission of infectious agents into the objectives of the organization's patient and occupational safety programs^{543-546, 561, 620, 626, 946}. *Category IB/IC*
- I.B. Make preventing transmission of infectious agents a priority for the healthcare organization. Provide administrative support, including fiscal and human resources for maintaining infection control programs^{434, 548, 549, 559, 561, 566, 662 552, 562-564, 946}. *Category IB/IC*
 - I.B.1. Assure that individuals with training in infection control are employed by or are available by contract to all healthcare facilities so that the infection control program is managed by one or more qualified individuals^{552, 566 316, 575, 947 573, 576, 946}. *Category IB/IC*
 - I.B.1.a. Determine the specific infection control full-time equivalents (FTEs) according to the scope of the infection control program, the complexity of the healthcare facility or system, the characteristics of the patient population, the unique or urgent needs of the facility and community, and proposed staffing levels based on survey results and recommendations from professional organizations^{434, 549 552, 566 316, 569, 573, 575 948 949}. *Category IB*
 - I.B.2. Include prevention of healthcare-associated infections (HAI) as one determinant of bedside nurse staffing levels and composition,

- especially in high-risk units^{585-589 590 592 593 551, 594, 595 418, 596, 597 583}.
Category IB
- I.B.3. Delegate authority to infection control personnel or their designees (e.g., patient care unit charge nurses) for making infection control decisions concerning patient placement and assignment of Transmission-Based Precautions^{549 434, 857, 946}. *Category IC*
- I.B.4. Involve infection control personnel in decisions on facility construction and design, determination of AIIR and Protective Environment capacity needs and environmental assessments^{11, 13, 950 951 12}. *Category IB/IC*
- I.B.4.a. Provide ventilation systems required for a sufficient number of AIIRs (as determined by a risk assessment) and Protective Environments in healthcare facilities that provide care to patients for whom such rooms are indicated, according to published recommendations^{11-13, 15}. *Category IB/IC*
- I.B.5. Involve infection control personnel in the selection and post-implementation evaluation of medical equipment and supplies and changes in practice that could affect the risk of HAI^{952, 953}. *Category IC*
- I.B.6. Ensure availability of human and fiscal resources to provide clinical microbiology laboratory support, including a sufficient number of medical technologists trained in microbiology, appropriate to the healthcare setting, for monitoring transmission of microorganisms, planning and conducting epidemiologic investigations, and detecting emerging pathogens. Identify resources for performing surveillance cultures, rapid diagnostic testing for viral and other selected pathogens, preparation of antimicrobial susceptibility summary reports, trend analysis, and molecular typing of clustered isolates (performed either on-site or in a reference laboratory) and use these resources according to facility-specific epidemiologic needs, in consultation with clinical microbiologists^{553, 609, 610, 612, 617, 954 614 603, 615, 616 605 599 554 598, 606, 607}. *Category IB*
- I.B.7. Provide human and fiscal resources to meet occupational health needs related to infection control (e.g., healthcare personnel immunization, post-exposure evaluation and care, evaluation and management of healthcare personnel with communicable infections^{739 12 17, 879-881, 955 134 690}). *Category IB/IC*
- I.B.8. In all areas where healthcare is delivered, provide supplies and equipment necessary for the consistent observance of Standard Precautions, including hand hygiene products and personal protective equipment (e.g., gloves, gowns, face and eye protection)^{739 559 946}. *Category IB/IC*
- I.B.9. Develop and implement policies and procedures to ensure that reusable patient care equipment is cleaned and reprocessed appropriately before use on another patient^{11, 956 957, 958 959 836 87 11, 960 961}. *Category IA/IC*

- I.C. Develop and implement processes to ensure oversight of infection control activities appropriate to the healthcare setting and assign responsibility for oversight of infection control activities to an individual or group within the healthcare organization that is knowledgeable about infection control ^{434, 549, 566}. *Category II*
- I.D. Develop and implement systems for early detection and management (e.g., use of appropriate infection control measures, including isolation precautions, PPE) of potentially infectious persons at initial points of patient encounter in outpatient settings (e.g., triage areas, emergency departments, outpatient clinics, physician offices) and at the time of admission to hospitals and long-term care facilities (LTCF) ^{9, 122, 134, 253, 827}. *Category IB*
- I.E. Develop and implement policies and procedures to limit patient visitation by persons with signs or symptoms of a communicable infection. Screen visitors to high-risk patient care areas (e.g., oncology units, hematopoietic stem cell transplant [HSCT] units, intensive care units, other severely immunocompromised patients) for possible infection ^{43 24, 41, 962, 963}. *Category IB*
- I.F. Identify performance indicators of the effectiveness of organization-specific measures to prevent transmission of infectious agents (Standard and Transmission-Based Precautions), establish processes to monitor adherence to those performance measures and provide feedback to staff members ^{704 739 705 708 666, 964 667 668 555}. *Category IB*

II. Education and Training

- II.A. Provide job- or task-specific education and training on preventing transmission of infectious agents associated with healthcare during orientation to the healthcare facility; update information periodically during ongoing education programs. Target all healthcare personnel for education and training, including but not limited to medical, nursing, clinical technicians, laboratory staff; property service (housekeeping), laundry, maintenance and dietary workers; students, contract staff and volunteers. Document competency initially and repeatedly, as appropriate, for the specific staff positions. Develop a system to ensure that healthcare personnel employed by outside agencies meet these education and training requirements through programs offered by the agencies or by participation in the healthcare facility's program designed for full-time personnel ^{126, 559, 561, 562, 655, 681-684, 686, 688, 689, 702, 893, 919, 965}. *Category IB*
 - II.A.1. Include in education and training programs, information concerning use of vaccines as an adjunctive infection control measure ^{17, 611, 690, 874}. *Category IB*
 - II.A.2. Enhance education and training by applying principles of adult learning, using reading level and language appropriate material for the target audience, and using online educational tools available to the institution ^{658, 694, 695, 697, 698, 700, 966}. *Category IB*

- II.B. Provide instructional materials for patients and visitors on recommended hand hygiene and Respiratory Hygiene/Cough Etiquette practices and the application of Transmission-Based Precautions^{9, 709, 710, 963}. *Category II*

III. Surveillance

- III.A. Monitor the incidence of epidemiologically-important organisms and targeted HAIs that have substantial impact on outcome and for which effective preventive interventions are available; use information collected through surveillance of high-risk populations, procedures, devices and highly transmissible infectious agents to detect transmission of infectious agents in the healthcare facility^{566, 671, 672, 675, 687, 919, 967, 968 673 969 970}.
Category IA
- III.B. Apply the following epidemiologic principles of infection surveillance^{671, 967 673 969 663 664}. *Category IB*
- y Use standardized definitions of infection
 - y Use laboratory-based data (when available)
 - y Collect epidemiologically-important variables (e.g., patient locations and/or clinical service in hospitals and other large multi-unit facilities, population-specific risk factors [e.g., low birth-weight neonates], underlying conditions that predispose to serious adverse outcomes)
 - y Analyze data to identify trends that may indicated increased rates of transmission
 - y Feedback information on trends in the incidence and prevalence of HAIs, probable risk factors, and prevention strategies and their impact to the appropriate healthcare providers, organization administrators, and as required by local and state health authorities
- III.C. Develop and implement strategies to reduce risks for transmission and evaluate effectiveness^{566, 673, 684, 970 963 971}. *Category IB*
- III.D. When transmission of epidemiologically-important organisms continues despite implementation and documented adherence to infection prevention and control strategies, obtain consultation from persons knowledgeable in infection control and healthcare epidemiology to review the situation and recommend additional measures for control^{566 247 687}.
Category IB
- III.E. Review periodically information on community or regional trends in the incidence and prevalence of epidemiologically-important organisms (e.g., influenza, RSV, pertussis, invasive group A streptococcal disease, MRSA, VRE) (including in other healthcare facilities) that may impact transmission of organisms within the facility^{398, 687, 972, 973 974}. *Category II*

IV. Standard Precautions

Assume that every person is potentially infected or colonized with an organism that could be transmitted in the healthcare setting and apply the following infection control practices during the delivery of health care.

- IV.A. Hand Hygiene

- IV.A.1. During the delivery of healthcare, avoid unnecessary touching of surfaces in close proximity to the patient to prevent both contamination of clean hands from environmental surfaces and transmission of pathogens from contaminated hands to surfaces^{72, 73, 739, 800, 975}(CDC, 2001 #970). *Category IB/IC*
- IV.A.2. When hands are visibly dirty, contaminated with proteinaceous material, or visibly soiled with blood or body fluids, wash hands with either a nonantimicrobial soap and water or an antimicrobial soap and water⁵⁵⁹. *Category IA*
- IV.A.3. If hands are not visibly soiled, or after removing visible material with nonantimicrobial soap and water, decontaminate hands in the clinical situations described in IV.A.2.a-f. The preferred method of hand decontamination is with an alcohol-based hand rub^{562, 978}. Alternatively, hands may be washed with an antimicrobial soap and water. Frequent use of alcohol-based hand rub immediately following handwashing with nonantimicrobial soap may increase the frequency of dermatitis⁵⁵⁹. *Category IB*
Perform hand hygiene:
 - IV.A.3.a. Before having direct contact with patients^{664, 979}. *Category IB*
 - IV.A.3.b. After contact with blood, body fluids or excretions, mucous membranes, nonintact skin, or wound dressings⁶⁶⁴. *Category IA*
 - IV.A.3.c. After contact with a patient's intact skin (e.g., when taking a pulse or blood pressure or lifting a patient)^{167, 976, 979, 980}. *Category IB*
 - IV.A.3.d. If hands will be moving from a contaminated-body site to a clean-body site during patient care. *Category II*
 - IV.A.3.e. After contact with inanimate objects (including medical equipment) in the immediate vicinity of the patient^{72, 73, 88, 800, 981, 982}. *Category II*
 - IV.A.3.f. After removing gloves^{728, 741, 742}. *Category IB*
- IV.A.4. Wash hands with non-antimicrobial soap and water or with antimicrobial soap and water if contact with spores (e.g., *C. difficile* or *Bacillus anthracis*) is likely to have occurred. The physical action of washing and rinsing hands under such circumstances is recommended because alcohols, chlorhexidine, iodophors, and other antiseptic agents have poor activity against spores^{559, 956, 983}. *Category II*
- IV.A.5. Do not wear artificial fingernails or extenders if duties include direct contact with patients at high risk for infection and associated adverse outcomes (e.g., those in ICUs or operating rooms)^{30, 31, 559, 722-724}. *Category IA*
 - IV.A.5.a. Develop an organizational policy on the wearing of non-natural nails by healthcare personnel who have direct contact with patients outside of the groups specified above⁹⁸⁴. *Category II*
- IV.B. Personal protective equipment (PPE) (see Figure)
 - IV.B.1. Observe the following principles of use:

- IV.B.1.a. Wear PPE, as described in IV.B.2-4, when the nature of the anticipated patient interaction indicates that contact with blood or body fluids may occur^{739, 780, 896}. *Category IB/IC*
- IV.B.1.b. Prevent contamination of clothing and skin during the process of removing PPE (see Figure). *Category II*
- IV.B.1.c. Before leaving the patient's room or cubicle, remove and discard PPE^{18, 739}. *Category IB/IC*
- IV.B.2. Gloves
 - IV.B.2.a. Wear gloves when it can be reasonably anticipated that contact with blood or other potentially infectious materials, mucous membranes, nonintact skin, or potentially contaminated intact skin (e.g., of a patient incontinent of stool or urine) could occur^{18, 728, 739, 741, 780, 985}. *Category IB/IC*
 - IV.B.2.b. Wear gloves with fit and durability appropriate to the task^{559, 731, 732, 739, 986, 987}. *Category IB*
 - IV.B.2.b.i. Wear disposable medical examination gloves for providing direct patient care.
 - IV.B.2.b.ii. Wear disposable medical examination gloves or reusable utility gloves for cleaning the environment or medical equipment.
 - IV.B.2.c. Remove gloves after contact with a patient and/or the surrounding environment (including medical equipment) using proper technique to prevent hand contamination (see Figure). Do not wear the same pair of gloves for the care of more than one patient. Do not wash gloves for the purpose of reuse since this practice has been associated with transmission of pathogens^{559, 728, 741-743, 988}. *Category IB*
 - IV.B.2.d. Change gloves during patient care if the hands will move from a contaminated body-site (e.g., perineal area) to a clean body-site (e.g., face). *Category II*
- IV.B.3. Gowns
 - IV.B.3.a. Wear a gown, that is appropriate to the task, to protect skin and prevent soiling or contamination of clothing during procedures and patient-care activities when contact with blood, body fluids, secretions, or excretions is anticipated^{739, 780, 896}. *Category IB/IC*
 - IV.B.3.a.i. Wear a gown for direct patient contact if the patient has uncontained secretions or excretions^{24, 88, 89, 739, 744}. *Category IB/IC*
 - IV.B.3.a.ii. Remove gown and perform hand hygiene before leaving the patient's environment^{24, 88, 89, 739, 744}. *Category IB/IC*
 - IV.B.3.b. Do not reuse gowns, even for repeated contacts with the same patient. *Category II*
 - IV.B.3.c. Routine donning of gowns upon entrance into a high risk unit (e.g., ICU, NICU, HSCT unit) is not indicated^{365, 747-750}. *Category IB*
- IV.B.4. Mouth, nose, eye protection

- IV.B.4.a. Use PPE to protect the mucous membranes of the eyes, nose and mouth during procedures and patient-care activities that are likely to generate splashes or sprays of blood, body fluids, secretions and excretions. Select masks, goggles, face shields, and combinations of each according to the need anticipated by the task performed^{113, 739, 780, 896}. *Category IB/IC*
- IV.B.5. During aerosol-generating procedures (e.g., bronchoscopy, suctioning of the respiratory tract [if not using in-line suction catheters], endotracheal intubation) in patients who are not suspected of being infected with an agent for which respiratory protection is otherwise recommended (e.g., *M. tuberculosis*, SARS or hemorrhagic fever viruses), wear one of the following: a face shield that fully covers the front and sides of the face, a mask with attached shield, or a mask and goggles (in addition to gloves and gown)^{95, 96, 113, 126 93 94, 134}. *Category IB*
- IV.C. Respiratory Hygiene/Cough Etiquette
 - IV.C.1. Educate healthcare personnel on the importance of source control measures to contain respiratory secretions to prevent droplet and fomite transmission of respiratory pathogens, especially during seasonal outbreaks of viral respiratory tract infections (e.g., influenza, RSV, adenovirus, parainfluenza virus) in communities^{14, 24, 684 10, 262}. *Category IB*
 - IV.C.2. Implement the following measures to contain respiratory secretions in patients and accompanying individuals who have signs and symptoms of a respiratory infection, beginning at the point of initial encounter in a healthcare setting (e.g., triage, reception and waiting areas in emergency departments, outpatient clinics and physician offices)^{20, 24, 145, 902, 989}.
 - IV.C.2.a. Post signs at entrances and in strategic places (e.g., elevators, cafeterias) within ambulatory and inpatient settings with instructions to patients and other persons with symptoms of a respiratory infection to cover their mouths/noses when coughing or sneezing, use and dispose of tissues, and perform hand hygiene after hands have been in contact with respiratory secretions. *Category II*
 - IV.C.2.b. Provide tissues and no-touch receptacles (e.g., foot-pedal-operated lid or open, plastic-lined waste basket) for disposal of tissues²⁰. *Category II*
 - IV.C.2.c. Provide resources and instructions for performing hand hygiene in or near waiting areas in ambulatory and inpatient settings; provide conveniently-located dispensers of alcohol-based hand rubs and, where sinks are available, supplies for handwashing^{559, 903}. *Category IB*
 - IV.C.2.d. During periods of increased prevalence of respiratory infections in the community (e.g., as indicated by increased school absenteeism, increased number of patients seeking care for a

respiratory infection), offer masks to coughing patients and other symptomatic persons (e.g., persons who accompany ill patients) upon entry into the facility or medical office^{126, 899, 898} and encourage them to maintain special separation, ideally a distance of at least 3 feet, from others in common waiting areas^{23, 103, 111, 114, 20, 134}. *Category IB*

IV.C.2.d.i. Some facilities may find it logistically easier to institute this recommendation year-round as a standard of practice. *Category II*

IV.D. Patient placement

IV.D.1. Include the potential for transmission of infectious agents in patient-placement decisions. Place patients who pose a risk for transmission to others (e.g., uncontained secretions, excretions or wound drainage; infants with suspected viral respiratory or gastrointestinal infections) in a single-patient room when available^{24, 430, 435, 796, 797, 806, 990, 410, 793}. *Category IB*

IV.D.2. Determine patient placement based on the following principles:

- y Route(s) of transmission of the known or suspected infectious agent
- y Risk factors for transmission in the infected patient
- y Risk factors for adverse outcomes resulting from an HAI in other patients in the area or room being considered for patient-placement
- y Availability of single-patient rooms
- y Patient options for room-sharing (e.g., cohorting patients with the same infection) *Category II*

IV.E. Patient-care equipment and instruments/devices⁹⁵⁶

IV.E.1. Establish policies and procedures for containing, transporting, and handling patient-care equipment and instruments/devices that may be contaminated with blood or body fluids^{18, 739, 975}. *Category IB/IC*

IV.E.2. Remove organic material from critical and semi-critical instrument/devices, using recommended cleaning agents before high level disinfection and sterilization to enable effective disinfection and sterilization processes^{836, 991, 992}. *Category IA*

IV.E.3. Wear PPE (e.g., gloves, gown), according to the level of anticipated contamination, when handling patient-care equipment and instruments/devices that is visibly soiled or may have been in contact with blood or body fluids^{18, 739, 975}. *Category IB/IC*

IV.F. Care of the environment¹¹

IV.F.1. Establish policies and procedures for routine and targeted cleaning of environmental surfaces as indicated by the level of patient contact and degree of soiling¹¹. *Category II*

IV.F.2. Clean and disinfect surfaces that are likely to be contaminated with pathogens, including those that are in close proximity to the patient (e.g., bed rails, over bed tables) and frequently-touched surfaces in the patient care environment (e.g., door knobs, surfaces in and

- surrounding toilets in patients' rooms) on a more frequent schedule compared to that for other surfaces (e.g., horizontal surfaces in waiting rooms) ^{11 73, 740, 746, 993, 994 72, 800, 835 995}. *Category IB*
- IV.F.3. Use EPA-registered disinfectants that have microbiocidal (i.e., killing) activity against the pathogens most likely to contaminate the patient-care environment. Use in accordance with manufacturer's instructions ^{842-844, 956, 996}. *Category IB/IC*
- IV.F.3.a. Review the efficacy of in-use disinfectants when evidence of continuing transmission of an infectious agent (e.g., rotavirus, *C. difficile*, norovirus) may indicate resistance to the in-use product and change to a more effective disinfectant as indicated ^{275, 842, 847}. *Category II*
- IV.F.4. In facilities that provide health care to pediatric patients or have waiting areas with child play toys (e.g., obstetric/gynecology offices and clinics), establish policies and procedures for cleaning and disinfecting toys at regular intervals ^{379 80}. *Category IB*
- Use the following principles in developing this policy and procedures: *Category II*
 - y Select play toys that can be easily cleaned and disinfected
 - y Do not permit use of stuffed furry toys if they will be shared
 - y Clean and disinfect large stationary toys (e.g., climbing equipment) at least weekly and whenever visibly soiled
 - y If toys are likely to be mouthed, rinse with water after disinfection; alternatively wash in a dishwasher
 - y When a toy requires cleaning and disinfection, do so immediately or store in a designated labeled container separate from toys that are clean and ready for use
- IV.F.5. Include multi-use electronic equipment in policies and procedures for preventing contamination and for cleaning and disinfection, especially those items that are used by patients, those used during delivery of patient care, and mobile devices that are moved in and out of patient rooms frequently (e.g., daily) ^{850 851, 852, 997}. *Category IB*
- IV.F.5.a. No recommendation for use of removable protective covers or washable keyboards. *Unresolved issue*
- IV.G. Textiles and laundry
- IV.G.1. Handle used textiles and fabrics with minimum agitation to avoid contamination of air, surfaces and persons ^{739, 998, 999}. *Category IB/IC*
- IV.G.2. If laundry chutes are used, ensure that they are properly designed, maintained, and used in a manner to minimize dispersion of aerosols from contaminated laundry ^{11, 13, 1000, 1001}. *Category IB/IC*
- IV.H. Safe injection practices
- The following recommendations apply to the use of needles, cannulas that replace needles, and, where applicable intravenous delivery systems ⁴⁵⁴

- IV.H.1. Use aseptic technique to avoid contamination of sterile injection equipment ^{1002, 1003}. *Category IA*
- IV.H.2. Do not administer medications from a syringe to multiple patients, even if the needle or cannula on the syringe is changed. Needles, cannulae and syringes are sterile, single-use items; they should not be reused for another patient nor to access a medication or solution that might be used for a subsequent patient ^{453, 919, 1004, 1005}.
Category IA
- IV.H.3. Use fluid infusion and administration sets (i.e., intravenous bags, tubing and connectors) for one patient only and dispose appropriately after use. Consider a syringe or needle/cannula contaminated once it has been used to enter or connect to a patient's intravenous infusion bag or administration set ⁴⁵³.
Category IB
- IV.H.4. Use single-dose vials for parenteral medications whenever possible ⁴⁵³. *Category IA*
- IV.H.5. Do not administer medications from single-dose vials or ampules to multiple patients or combine leftover contents for later use ^{369 453, 1005}. *Category IA*
- IV.H.6. If multidose vials must be used, both the needle or cannula and syringe used to access the multidose vial must be sterile ^{453, 1002}.
Category IA
- IV.H.7. Do not keep multidose vials in the immediate patient treatment area and store in accordance with the manufacturer's recommendations; discard if sterility is compromised or questionable ^{453, 1003}. *Category IA*
- IV.H.8. Do not use bags or bottles of intravenous solution as a common source of supply for multiple patients ^{453, 1006}. *Category IB*
- IV.I. Infection control practices for special lumbar puncture procedures
Wear a surgical mask when placing a catheter or injecting material into the spinal canal or subdural space (i.e., during myelograms, lumbar puncture and spinal or epidural anesthesia ^{906 907-909 910, 911 912-914, 918 1007}). *Category IB*
- IV.J. Worker safety
Adhere to federal and state requirements for protection of healthcare personnel from exposure to bloodborne pathogens ⁷³⁹. *Category IC*

V. Transmission-Based Precautions

V.A. General principles

- V.A.1. In addition to Standard Precautions, use Transmission-Based Precautions for patients with documented or suspected infection or colonization with highly transmissible or epidemiologically-important pathogens for which additional precautions are needed to prevent transmission (see Appendix A) ^{24, 93, 126, 141, 306, 806, 1008}. *Category IA*
- V.A.2. Extend duration of Transmission-Based Precautions, (e.g., Droplet, Contact) for immunosuppressed patients with viral infections due to

prolonged shedding of viral agents that may be transmitted to others^{928, 931-933, 1009-1011}.

Category IA

V.B. Contact Precautions

V.B.1. Use Contact Precautions as recommended in Appendix A for patients with known or suspected infections or evidence of syndromes that represent an increased risk for contact transmission. For specific recommendations for use of Contact Precautions for colonization or infection with MDROs, go to the MDRO guideline:

www.cdc.gov/ncidod/dhqp/pdf/ar/mdroGuideline2006.pdf⁸⁷⁰.

V.B.2. Patient placement

V.B.2.a. In *acute care hospitals*, place patients who require Contact Precautions in a single-patient room when available^{24, 687, 793, 796, 797, 806, 837, 893, 1012, 1013} *Category IB*

When single-patient rooms are in short supply, apply the following principles for making decisions on patient placement:

- y Prioritize patients with conditions that may facilitate transmission (e.g., uncontained drainage, stool incontinence) for single-patient room placement. *Category II*
- y Place together in the same room (cohort) patients who are infected or colonized with the same pathogen and are suitable roommates^{29, 638, 808, 811-813, 815, 818, 819} *Category IB*
- y If it becomes necessary to place a patient who requires Contact Precautions in a room with a patient who is not infected or colonized with the same infectious agent:
 - o Avoid placing patients on Contact Precautions in the same room with patients who have conditions that may increase the risk of adverse outcome from infection or that may facilitate transmission (e.g., those who are immunocompromised, have open wounds, or have anticipated prolonged lengths of stay). *Category II*
 - o Ensure that patients are physically separated (i.e., >3 feet apart) from each other. Draw the privacy curtain between beds to minimize opportunities for direct contact.) *Category II*
 - o Change protective attire and perform hand hygiene between contact with patients in the same room, regardless of whether one or both patients are on Contact Precautions^{728, 741, 742, 988, 1014, 1015}. *Category IB*

V.B.2.b. In *long-term care and other residential settings*, make decisions regarding patient placement on a case-by-case basis, balancing infection risks to other patients in the room, the presence of risk factors that increase the likelihood of transmission, and the

- potential adverse psychological impact on the infected or colonized patient^{920, 921}. *Category II*
- V.B.2.c. In *ambulatory settings*, place patients who require Contact Precautions in an examination room or cubicle as soon as possible²⁰. *Category II*
- V.B.3. Use of personal protective equipment
- V.B.3.a. Gloves
Wear gloves whenever touching the patient's intact skin^{24, 89, 134, 559, 746, 837} or surfaces and articles in close proximity to the patient (e.g., medical equipment, bed rails)^{72, 73, 88, 837}. Don gloves upon entry into the room or cubicle. *Category IB*
- V.B.3.b. Gowns
- V.B.3.b.i. Wear a gown whenever anticipating that clothing will have direct contact with the patient or potentially contaminated environmental surfaces or equipment in close proximity to the patient. Don gown upon entry into the room or cubicle. Remove gown and observe hand hygiene before leaving the patient-care environment^{24, 88, 134, 745, 837}. *Category IB*
- V.B.3.b.ii. After gown removal, ensure that clothing and skin do not contact potentially contaminated environmental surfaces that could result in possible transfer of microorganism to other patients or environmental surfaces^{72, 73}. *Category II*
- V.B.4. Patient transport
- V.B.4.a. In *acute care hospitals and long-term care and other residential settings*, limit transport and movement of patients outside of the room to medically-necessary purposes. *Category II*
- V.B.4.b. When transport or movement in any healthcare setting is necessary, ensure that infected or colonized areas of the patient's body are contained and covered. *Category II*
- V.B.4.c. Remove and dispose of contaminated PPE and perform hand hygiene prior to transporting patients on Contact Precautions. *Category II*
- V.B.4.d. Don clean PPE to handle the patient at the transport destination. *Category II*
- V.B.5. Patient-care equipment and instruments/devices
- V.B.5.a. Handle patient-care equipment and instruments/devices according to Standard Precautions^{739, 836}. *Category IB/IC*
- V.B.5.b. In *acute care hospitals and long-term care and other residential settings*, use disposable noncritical patient-care equipment (e.g., blood pressure cuffs) or implement patient-dedicated use of such equipment. If common use of equipment for multiple patients is unavoidable, clean and disinfect such equipment before use on another patient^{24, 88, 796, 836, 837, 854, 1016}. *Category IB*
- V.B.5.c. In *home care settings*

- V.B.5.c.i. Limit the amount of non-disposable patient-care equipment brought into the home of patients on Contact Precautions. Whenever possible, leave patient-care equipment in the home until discharge from home care services. *Category II*
- V.B.5.c.ii. If noncritical patient-care equipment (e.g., stethoscope) cannot remain in the home, clean and disinfect items before taking them from the home using a low- to intermediate-level disinfectant. Alternatively, place contaminated reusable items in a plastic bag for transport and subsequent cleaning and disinfection. *Category II*
- V.B.5.d. In *ambulatory settings*, place contaminated reusable noncritical patient-care equipment in a plastic bag for transport to a soiled utility area for reprocessing. *Category II*
- V.B.6. Environmental measures
 - Ensure that rooms of patients on Contact Precautions are prioritized for frequent cleaning and disinfection (e.g., at least daily) with a focus on frequently-touched surfaces (e.g., bed rails, overbed table, bedside commode, lavatory surfaces in patient bathrooms, doorknobs) and equipment in the immediate vicinity of the patient^{11, 24, 88, 746, 837}. *Category IB*
- V.B.7. Discontinue Contact Precautions after signs and symptoms of the infection have resolved or according to pathogen-specific recommendations in Appendix A. *Category IB*
- V.C. Droplet Precautions
 - V.C.1. Use Droplet Precautions as recommended in Appendix A for patients known or suspected to be infected with pathogens transmitted by respiratory droplets (i.e., large-particle droplets >5 μ in size) that are generated by a patient who is coughing, sneezing or talking^{14, 23, Steinberg, 1969 #1708, 41, 95, 103, 111, 112, 755, 756, 989, 1017}. *Category IB*
 - V.C.2. Patient placement
 - V.C.2.a. In acute care hospitals, place patients who require Droplet Precautions in a single-patient room when available *Category II*
 - When single-patient rooms are in short supply, apply the following principles for making decisions on patient placement:
 - y Prioritize patients who have excessive cough and sputum production for single-patient room placement *Category II*
 - y Place together in the same room (cohort) patients who are infected the same pathogen and are suitable roommates⁸¹⁴
⁸¹⁶. *Category IB*
 - y If it becomes necessary to place patients who require Droplet Precautions in a room with a patient who does not have the same infection:
 - y Avoid placing patients on Droplet Precautions in the same room with patients who have conditions that may increase

- the risk of adverse outcome from infection or that may facilitate transmission (e.g., those who are immunocompromised, have or have anticipated prolonged lengths of stay). *Category II*
- y Ensure that patients are physically separated (i.e., >3 feet apart) from each other. Draw the privacy curtain between beds to minimize opportunities for close contact ^{103, 104 410}. *Category IB*
 - y Change protective attire and perform hand hygiene between contact with patients in the same room, regardless of whether one patient or both patients are on Droplet Precautions ^{741-743, 988, 1014, 1015}. *Category IB*
- V.C.2.b. In *long-term care and other residential settings*, make decisions regarding patient placement on a case-by-case basis after considering infection risks to other patients in the room and available alternatives ⁴¹⁰. *Category II*
- V.C.2.c. In *ambulatory settings*, place patients who require Droplet Precautions in an examination room or cubicle as soon as possible. Instruct patients to follow recommendations for Respiratory Hygiene/Cough Etiquette ^{447, 448 9, 828}. *Category II*
- V.C.3. Use of personal protective equipment
- V.C.3.a. Don a mask upon entry into the patient room or cubicle ^{14, 23, 41, 103, 111, 113, 115, 827}. *Category IB*
 - V.C.3.b. No recommendation for routinely wearing eye protection (e.g., goggle or face shield), in addition to a mask, for close contact with patients who require Droplet Precautions. *Unresolved issue*
 - V.C.3.c. For patients with suspected or proven SARS, avian influenza or pandemic influenza, refer to the following websites for the most current recommendations (www.cdc.gov/ncidod/sars/ ; www.cdc.gov/flu/avian/ ; www.pandemicflu.gov/) ^{134, 1018, 1019}
- V.C.4. Patient transport
- V.C.4.a. In *acute care hospitals and long-term care and other residential settings*, limit transport and movement of patients outside of the room to medically-necessary purposes. *Category II*
 - V.C.4.b. If transport or movement in any healthcare setting is necessary, instruct patient to wear a mask and follow Respiratory Hygiene/Cough Etiquette (www.cdc.gov/flu/professionals/infectioncontrol/resphygiene.htm) . *Category IB*
 - V.C.4.c. No mask is required for persons transporting patients on Droplet Precautions. *Category II*
 - V.C.4.d. Discontinue Droplet Precautions after signs and symptoms have resolved or according to pathogen-specific recommendations in Appendix A. *Category IB*
- V.D. Airborne Precautions

- V.D.1. Use Airborne Precautions as recommended in Appendix A for patients known or suspected to be infected with infectious agents transmitted person-to-person by the airborne route (e.g., *M tuberculosis*¹², measles^{34, 122, 1020}, chickenpox^{123, 773, 1021}, disseminated herpes zoster¹⁰²²). *Category IA/IC*
- V.D.2. Patient placement
- V.D.2.a. In *acute care hospitals and long-term care settings*, place patients who require Airborne Precautions in an AIIR that has been constructed in accordance with current guidelines¹¹⁻¹³. *Category IA/IC*
- V.D.2.a.i. Provide at least six (existing facility) or 12 (new construction/renovation) air changes per hour.
- V.D.2.a.ii. Direct exhaust of air to the outside. If it is not possible to exhaust air from an AIIR directly to the outside, the air may be returned to the air-handling system or adjacent spaces if all air is directed through HEPA filters.
- V.D.2.a.iii. Whenever an AIIR is in use for a patient on Airborne Precautions, monitor air pressure daily with visual indicators (e.g., smoke tubes, flutter strips), regardless of the presence of differential pressure sensing devices (e.g., manometers)^{11, 12, 1023, 1024}.
- V.D.2.a.iv. Keep the AIIR door closed when not required for entry and exit.
- V.D.2.b. When an AIIR is not available, transfer the patient to a facility that has an available AIIR¹². *Category II*
- V.D.2.c. In the event of an outbreak or exposure involving large numbers of patients who require Airborne Precautions:
- y Consult infection control professionals before patient placement to determine the safety of alternative room that do not meet engineering requirements for an AIIR.
 - y Place together (cohort) patients who are presumed to have the same infection(based on clinical presentation and diagnosis when known) in areas of the facility that are away from other patients, especially patients who are at increased risk for infection (e.g., immunocompromised patients).
 - y Use temporary portable solutions (e.g., exhaust fan) to create a negative pressure environment in the converted area of the facility. Discharge air directly to the outside, away from people and air intakes, or direct all the air through HEPA filters before it is introduced to other air spaces¹². *Category II*
- V.D.2.d. In *ambulatory settings*:
- V.D.2.d.i. Develop systems (e.g., triage, signage) to identify patients with known or suspected infections that require Airborne Precautions upon entry into ambulatory settings^{9, 12, 34, 127, 134}. *Category IA*

- V.D.2.d.ii. Place the patient in an AIIR as soon as possible. If an AIIR is not available, place a surgical mask on the patient and place him/her in an examination room. Once the patient leaves, the room should remain vacant for the appropriate time, generally one hour, to allow for a full exchange of air ^{11, 12, 122}. *Category IB/IC*
- V.D.2.d.iii. Instruct patients with a known or suspected airborne infection to wear a surgical mask and observe Respiratory Hygiene/Cough Etiquette. Once in an AIIR, the mask may be removed; the mask should remain on if the patient is not in an AIIR ^{12, 107, 145, 899}. *Category IB/IC*
- V.D.3. Personnel restrictions
Restrict susceptible healthcare personnel from entering the rooms of patients known or suspected to have measles (rubeola), varicella (chickenpox), disseminated zoster, or smallpox if other immune healthcare personnel are available ^{17, 775}. *Category IB*
- V.D.4. Use of PPE
 - V.D.4.a. Wear a fit-tested NIOSH-approved N95 or higher level respirator for respiratory protection when entering the room or home of a patient when the following diseases are suspected or confirmed:
 - y Infectious pulmonary or laryngeal tuberculosis or when infectious tuberculosis skin lesions are present and procedures that would aerosolize viable organisms (e.g., irrigation, incision and drainage, whirlpool treatments) are performed ^{12, 1025, 1026}. *Category IB*
 - y Smallpox (vaccinated and unvaccinated). Respiratory protection is recommended for all healthcare personnel, including those with a documented “take” after smallpox vaccination due to the risk of a genetically engineered virus against which the vaccine may not provide protection, or of exposure to a very large viral load (e.g., from high-risk aerosol-generating procedures, immunocompromised patients, hemorrhagic or flat smallpox ^{108, 129}. *Category II*
 - V.D.4.b. No recommendation is made regarding the use of PPE by healthcare personnel who are presumed to be immune to measles (rubeola) or varicella-zoster based on history of disease, vaccine, or serologic testing when caring for an individual with known or suspected measles, chickenpox or disseminated zoster, due to difficulties in establishing definite immunity ^{1027, 1028}. *Unresolved issue*
 - V.D.4.c. No recommendation is made regarding the type of personal protective equipment (i.e., surgical mask or respiratory protection with a N95 or higher respirator) to be worn by susceptible healthcare personnel who must have contact with patients with known or suspected measles, chickenpox or disseminated herpes zoster. *Unresolved issue*

- V.D.5. Patient transport
- V.D.5.a. In *acute care hospitals and long-term care and other residential settings*, limit transport and movement of patients outside of the room to medically-necessary purposes. *Category II*
 - V.D.5.b. If transport or movement outside an AIIR is necessary, instruct patients to wear a surgical mask, if possible, and observe Respiratory Hygiene/Cough Etiquette ¹². *Category II*
 - V.D.5.c. For patients with skin lesions associated with varicella or smallpox or draining skin lesions caused by *M. tuberculosis*, cover the affected areas to prevent aerosolization or contact with the infectious agent in skin lesions ^{108, 1025, 1026, 1029-1031}. *Category IB*
 - V.D.5.d. Healthcare personnel transporting patients who are on Airborne Precautions do not need to wear a mask or respirator during transport if the patient is wearing a mask and infectious skin lesions are covered. *Category II*
- V.D.6. Exposure management
 Immunize or provide the appropriate immune globulin to susceptible persons as soon as possible following unprotected contact (i.e., exposed) to a patient with measles, varicella or smallpox: *Category IA*
- y Administer measles vaccine to exposed susceptible persons within 72 hours after the exposure or administer immune globulin within six days of the exposure event for high-risk persons in whom vaccine is contraindicated ^{17, 1032-1035}.
 - y Administer varicella vaccine to exposed susceptible persons within 120 hours after the exposure or administer varicella immune globulin (VZIG or alternative product), when available, within 96 hours for high-risk persons in whom vaccine is contraindicated (e.g., immunocompromised patients, pregnant women, newborns whose mother's varicella onset was <5 days before or within 48 hours after delivery ^{888, 1035-1037}).
 - y Administer smallpox vaccine to exposed susceptible persons within 4 days after exposure ^{108, 1038-1040}.
- V.D.7. Discontinue Airborne Precautions according to pathogen-specific recommendations in Appendix A. *Category IB*
- V.D.8. Consult CDC's "Guidelines for Preventing the Transmission of *Mycobacterium tuberculosis* in Health-Care Settings, 2005" ¹² and the "Guideline for Environmental Infection Control in Health-Care Facilities" ¹¹ for additional guidance on environment strategies for preventing transmission of tuberculosis in healthcare settings. The environmental recommendations in these guidelines may be applied to patients with other infections that require Airborne Precautions.

- VI. Protective Environment (Table 4)**
- VI.A. Place allogeneic hematopoietic stem cell transplant (HSCT) patients in a Protective Environment as described in the “Guideline to Prevent Opportunistic Infections in HSCT Patients”¹⁵, the “Guideline for Environmental Infection Control in Health-Care Facilities”¹¹, and the “Guidelines for Preventing Health-Care-Associated Pneumonia, 2003”¹⁴ to reduce exposure to environmental fungi (e.g., *Aspergillus* sp)^{157, 158}.
Category IB
- VI.B. No recommendation for placing patients with other medical conditions that are associated with increased risk for environmental fungal infections (e.g., aspergillosis) in a Protective Environment¹¹. *Unresolved issue*
- VI.C. For patients who require a Protective Environment, implement the following (see Table 5)^{11, 15}
- VI.C.1. Environmental controls
- VI.C.1.a. Filtered incoming air using central or point-of-use high efficiency particulate (HEPA) filters capable of removing 99.97% of particles $\geq 0.3 \mu\text{m}$ in diameter¹³. *Category IB*
- VI.C.1.b. Directed room airflow with the air supply on one side of the room that moves air across the patient bed and out through an exhaust on the opposite side of the room¹³. *Category IB*
- VI.C.1.c. Positive air pressure in room relative to the corridor (pressure differential of $\geq 12.5 \text{ Pa}$ [0.01-in water gauge])¹³. *Category IB*
- VI.C.1.c.i. Monitor air pressure daily with visual indicators (e.g., smoke tubes, flutter strips)^{11, 1024}. *Category IA*
- VI.C.1.d. Well-sealed rooms that prevent infiltration of outside air¹³.
Category IB
- VI.C.1.e. At least 12 air changes per hour¹³. *Category IB*
- VI.C.2. Lower dust levels by using smooth, nonporous surfaces and finishes that can be scrubbed, rather than textured material (e.g., upholstery). Wet dust horizontal surfaces whenever dust detected and routinely clean crevices and sprinkler heads where dust may accumulate^{940, 941}. *Category II*
- VI.C.3. Avoid carpeting in hallways and patient rooms in areas⁹⁴¹.
Category IB
- VI.C.4. Prohibit dried and fresh flowers and potted plants⁹⁴²⁻⁹⁴⁴. *Category II*
- VI.D. Minimize the length of time that patients who require a Protective Environment are outside their rooms for diagnostic procedures and other activities^{11, 158, 945}. *Category IB*
- VI.E. During periods of construction, to prevent inhalation of respirable particles that could contain infectious spores, provide respiratory protection (e.g., N95 respirator) to patients who are medically fit to tolerate a respirator when they are required to leave the Protective Environment^{945 158}.
Category II
- VI.E.1.a. No recommendation for fit-testing of patients who are using respirators. *Unresolved issue*

- VI.E.1.b. No recommendation for use of particulate respirators when leaving the Protective Environment in the absence of construction. *Unresolved issue*
- VI.F. Use of Standard and Transmission-Based Precautions in a Protective Environment.
 - VI.F.1. Use Standard Precautions as recommended for all patient interactions. *Category IA*
 - VI.F.2. Implement Droplet and Contact Precautions as recommended for diseases listed in Appendix A. Transmission-Based precautions for viral infections may need to be prolonged because of the patient's immunocompromised state and prolonged shedding of viruses^{930 1010 928, 932 1011}. *Category IB*
 - VI.F.3. Barrier precautions, (e.g., masks, gowns, gloves) are not required for healthcare personnel in the absence of suspected or confirmed infection in the patient or if they are not indicated according to Standard Precautions¹⁵. *Category II*
 - VI.F.4. Implement Airborne Precautions for patients who require a Protective Environment room and who also have an airborne infectious disease (e.g., pulmonary or laryngeal tuberculosis, acute varicella-zoster). *Category IA*
 - VI.F.4.a. Ensure that the Protective Environment is designed to maintain positive pressure¹³. *Category IB*
 - VI.F.4.b. Use an anteroom to further support the appropriate air-balance relative to the corridor and the Protective Environment; provide independent exhaust of contaminated air to the outside or place a HEPA filter in the exhaust duct if the return air must be recirculated^{13, 1041}. *Category IB*
 - VI.F.4.c. If an anteroom is not available, place the patient in an AIIR and use portable, industrial-grade HEPA filters in the room to enhance filtration of spores¹⁰⁴². *Category II*

Note: The recommendations in this guideline for Ebola Virus Disease has been superseded by CDC's Infection Prevention and Control Recommendations for Hospitalized Patients with Known or Suspected Ebola Virus Disease in U.S. Hospitals.

This information is in Appendix A.

[Click here for current information on how Ebola virus is transmitted.](#)

Appendix A:

Preamble The mode(s) and risk of transmission for each specific disease agent included in Appendix A were reviewed. Principle sources consulted for the development of disease-specific recommendations for Appendix A included infectious disease manuals and textbooks^{833, 1043, 1044}. The published literature was searched for evidence of person-to-person transmission in healthcare and non-healthcare settings with a focus on reported outbreaks that would assist in developing recommendations for all settings where healthcare is delivered. Criteria used to assign Transmission-Based Precautions categories follow:

- A Transmission-Based Precautions category was assigned if there was strong evidence for person-to-person transmission via droplet, contact, or airborne routes in healthcare or non-healthcare settings and/or if patient factors (e.g., diapered infants, diarrhea, draining wounds) increased the risk of transmission
- Transmission-Based Precautions category assignments reflect the predominant mode(s) of transmission
- If there was no evidence for person-to-person transmission by droplet, contact or airborne routes, Standard Precautions were assigned
- If there was a low risk for person-to-person transmission and no evidence of healthcare-associated transmission, Standard Precautions were assigned
- Standard Precautions were assigned for bloodborne pathogens (e.g., hepatitis B and C viruses, human immunodeficiency virus) as per CDC recommendations for Universal Precautions issued in 1988⁷⁸⁰. Subsequent experience has confirmed the efficacy of Standard Precautions to prevent exposure to infected blood and body fluid^{778, 779, 866}.

Additional information relevant to use of precautions was added in the comments column to assist the caregiver in decision-making. Citations were added as needed to support a change in or provide additional evidence for recommendations for a specific disease and for new infectious agents (e.g., SARS-CoV, avian influenza) that have been added to Appendix A. The reader may refer to more detailed discussion concerning modes of transmission and emerging pathogens in the background text and for MDRO control in Appendix B.

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
Abscess			
Draining, major	C	DI	No dressing or containment of drainage; until drainage stops or can be contained by dressing
Draining, minor or limited	S		Dressing covers and contains drainage
Acquired human immunodeficiency syndrome (HIV)	S		Post-exposure chemoprophylaxis for some blood exposures ⁸⁶⁶ .
Actinomycosis	S		Not transmitted from person to person
Adenovirus infection (see agent-specific guidance under gastroenteritis, conjunctivitis, pneumonia)			
Amebiasis	S		Person to person transmission is rare. Transmission in settings for the mentally challenged and in a family group has been reported ¹⁰⁴⁵ . Use care when handling diapered infants and mentally challenged persons ¹⁰⁴⁶ .
Anthrax	S		Infected patients do not generally pose a transmission risk.
Cutaneous	S		Transmission through non-intact skin contact with draining lesions possible, therefore use Contact Precautions if large amount of uncontained drainage. Handwashing with soap and water preferable to use of waterless alcohol based antiseptics since alcohol does not

¹ Type of Precautions: A, Airborne Precautions; C, Contact; D, Droplet; S, Standard; when A, C, and D are specified, also use S.

[†] Duration of precautions: CN, until off antimicrobial treatment and culture-negative; DI, duration of illness (with wound lesions, DI means until wounds stop draining); DE, until environment completely decontaminated; U, until time specified in hours (hrs) after initiation of effective therapy; Unknown: criteria for establishing eradication of pathogen has not been determined

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
			have sporocidal activity ⁹⁸³ .
Pulmonary	S		Not transmitted from person to person
Environmental: aerosolizable spore-containing powder or other substance		DE	Until decontamination of environment complete ²⁰³ . Wear respirator (N95 mask or PAPRs), protective clothing; decontaminate persons with powder on them (http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5135a3.htm) Hand hygiene: Handwashing for 30-60 seconds with soap and water or 2% chlorhexidine gluconate after spore contact (alcohol handrubs inactive against spores ⁹⁸³ . Post-exposure prophylaxis following environmental exposure: 60 days of antimicrobials (either doxycycline, ciprofloxacin, or levofloxacin) and post-exposure vaccine under IND
Antibiotic-associated colitis (see <i>Clostridium difficile</i>)			
Arthropod-borne viral encephalitides (eastern, western, Venezuelan equine encephalomyelitis; St Louis, California encephalitis; West Nile Virus) and viral fevers (dengue, yellow fever, Colorado tick fever)	S		Not transmitted from person to person except rarely by transfusion, and for West Nile virus by organ transplant, breastmilk or transplacentally ^{530, 1047} . Install screens in windows and doors in endemic areas Use DEET-containing mosquito repellants and clothing to cover extremities
Ascariasis	S		Not transmitted from person to person
Aspergillosis	S		Contact Precautions and Airborne Precautions if massive soft tissue infection with copious drainage and repeated irrigations required ¹⁵⁴ .
Avian influenza (see influenza, avian below)			
Babesiosis	S		Not transmitted from person to person except rarely by transfusion,
Blastomycosis, North American, cutaneous or pulmonary	S		Not transmitted from person to person
Botulism	S		Not transmitted from person to person
Bronchiolitis (see respiratory infections in infants and young children)	C	DI	Use mask according to Standard Precautions.

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
Brucellosis (undulant, Malta, Mediterranean fever)	S		Not transmitted from person to person except rarely via banked spermatozoa and sexual contact ^{1048, 1049} . Provide antimicrobial prophylaxis following laboratory exposure ¹⁰⁵⁰ .
<i>Campylobacter</i> gastroenteritis (see gastroenteritis)			
Candidiasis, all forms including mucocutaneous	S		
Cat-scratch fever (benign inoculation lymphoreticulosis)	S		Not transmitted from person to person
Cellulitis	S		
Chancroid (soft chancre) (<i>H. ducreyi</i>)	S		Transmitted sexually from person to person
Chickenpox (see varicella)			
<i>Chlamydia trachomatis</i>			
Conjunctivitis	S		
Genital (lymphogranuloma venereum)	S		
Pneumonia (infants \leq 3 mos. of age))	S		
<i>Chlamydia pneumoniae</i>	S		Outbreaks in institutionalized populations reported, rarely ^{1051, 1052}
Cholera (see gastroenteritis)			
Closed-cavity infection			
Open drain in place; limited or minor drainage	S		Contact Precautions if there is copious uncontained drainage
No drain or closed drainage system in place	S		
<i>Clostridium</i>			
<i>C. botulinum</i>	S		Not transmitted from person to person
<i>C. difficile</i> (see Gastroenteritis, <i>C. difficile</i>)	C	DI	
<i>C. perfringens</i>			
Food poisoning	S		Not transmitted from person to person
Gas gangrene	S		Transmission from person to person rare; one outbreak in a surgical setting reported ¹⁰⁵³ . Use Contact Precautions if wound drainage is

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
			extensive.
Coccidioidomycosis (valley fever)			
Draining lesions	S		Not transmitted from person to person except under extraordinary circumstances because the infectious arthroconidial form of <i>Coccidioides immitis</i> is not produced in humans ¹⁰⁵⁴ .
Pneumonia	S		Not transmitted from person to person except under extraordinary circumstances, (e.g., inhalation of aerosolized tissue phase endospores during necropsy, transplantation of infected lung) because the infectious arthroconidial form of <i>Coccidioides immitis</i> is not produced in humans ^{1054, 1055} .
Colorado tick fever	S		Not transmitted from person to person
Congenital rubella	C	Until 1 yr of age	Standard Precautions if nasopharyngeal and urine cultures repeatedly neg. after 3 mos. of age
Conjunctivitis			
Acute bacterial	S		
<i>Chlamydia</i>	S		
Gonococcal	S		
Acute viral (acute hemorrhagic)	C	DI	Adenovirus most common; enterovirus 70 ¹⁰⁵⁶ , Coxsackie virus A24 ¹⁰⁵⁷) also associated with community outbreaks. Highly contagious; outbreaks in eye clinics, pediatric and neonatal settings, institutional settings reported. Eye clinics should follow Standard Precautions when handling patients with conjunctivitis. Routine use of infection control measures in the handling of instruments and equipment will prevent the occurrence of outbreaks in this and other settings. ^{460, 814, 1058, 1059 461, 1060} .
Corona virus associated with SARS (SARS-CoV) (see severe acute respiratory syndrome)			

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
Coxsackie virus disease (see enteroviral infection)			
Creutzfeldt-Jakob disease CJD, vCJD	S		Use disposable instruments or special sterilization/disinfection for surfaces, objects contaminated with neural tissue if CJD or vCJD suspected and has not been R/O; No special burial procedures ¹⁰⁶¹
Croup (see respiratory infections in infants and young children)			
Crimean-Congo Fever (see Viral Hemorrhagic Fever)	S		
Cryptococcosis	S		Not transmitted from person to person, except rarely via tissue and corneal transplant ^{1062, 1063}
Cryptosporidiosis (see gastroenteritis)			
Cysticercosis	S		Not transmitted from person to person
Cytomegalovirus infection, including in neonates and immunosuppressed patients	S		No additional precautions for pregnant HCWs
Decubitus ulcer (see Pressure ulcer)			
Dengue fever	S		Not transmitted from person to person
Diarrhea, acute-infective etiology suspected (see gastroenteritis)			
Diphtheria			
Cutaneous	C	CN	Until 2 cultures taken 24 hrs. apart negative
Pharyngeal	D	CN	Until 2 cultures taken 24 hrs. apart negative
Ebola virus (see viral hemorrhagic fevers)			
Echinococcosis (hydatidosis)	S		Not transmitted from person to person
Echovirus (see enteroviral infection)			
Encephalitis or encephalomyelitis (see specific etiologic agents)			
Endometritis (endomyometritis)	S		
Enterobiasis (pinworm disease, oxyuriasis)	S		
<i>Enterococcus</i> species (see multidrug-resistant organisms if			

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
epidemiologically significant or vancomycin resistant)			
Enterocolitis, <i>C. difficile</i> (see <i>C. difficile</i> , gastroenteritis)			
Enteroviral infections (i.e., Group A and B Coxsackie viruses and Echo viruses) (excludes polio virus)	S		Use Contact Precautions for diapered or incontinent children for duration of illness and to control institutional outbreaks
Epiglottitis, due to <i>Haemophilus influenzae</i> type b	D	U 24 hrs	See specific disease agents for epiglottitis due to other etiologies)
Epstein-Barr virus infection, including infectious mononucleosis	S		
Erythema infectiosum (also see Parvovirus B19)			
<i>Escherichia coli</i> gastroenteritis (see gastroenteritis)			
Food poisoning			
Botulism	S		Not transmitted from person to person
<i>C. perfringens</i> or <i>welchii</i>	S		Not transmitted from person to person
Staphylococcal	S		Not transmitted from person to person
Furunculosis, staphylococcal	S		Contact if drainage not controlled. Follow institutional policies if MRSA
Infants and young children	C	DI	
Gangrene (gas gangrene)	S		Not transmitted from person to person
Gastroenteritis	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks for gastroenteritis caused by all of the agents below
Adenovirus	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
<i>Campylobacter</i> species	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
Cholera (<i>Vibrio cholerae</i>)	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
<i>C. difficile</i>	C	DI	Discontinue antibiotics if appropriate. Do not share electronic thermometers ^{853, 854} ; ensure consistent environmental cleaning and

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
			disinfection. Hypochlorite solutions may be required for cleaning if transmission continues ⁸⁴⁷ . Handwashing with soap and water preferred because of the absence of sporicidal activity of alcohol in waterless antiseptic handrubs ⁹⁸³ .
<i>Cryptosporidium species</i>	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
<i>E. coli</i>			
Enteropathogenic O157:H7 and other shiga toxin-producing Strains	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
Other species	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
<i>Giardia lamblia</i>	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
Noroviruses	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks. Persons who clean areas heavily contaminated with feces or vomitus may benefit from wearing masks since virus can be aerosolized from these body substances ^{142, 147 148} ; ensure consistent environmental cleaning and disinfection with focus on restrooms even when apparently unsoiled ^{273, 1064}). Hypochlorite solutions may be required when there is continued transmission ²⁹⁰⁻²⁹² . Alcohol is less active, but there is no evidence that alcohol antiseptic handrubs are not effective for hand decontamination ²⁹⁴ . Cohorting of affected patients to separate airspaces and toilet facilities may help interrupt transmission during outbreaks.
Rotavirus	C	DI	Ensure consistent environmental cleaning and disinfection and frequent removal of soiled diapers. Prolonged shedding may occur in

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
			both immunocompetent and immunocompromised children and the elderly ^{932, 933}
<i>Salmonella</i> species (including <i>S. typhi</i>)	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
<i>Shigella</i> species (Bacillary dysentery)	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
<i>Vibrio parahaemolyticus</i>	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
Viral (if not covered elsewhere)	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
<i>Yersinia enterocolitica</i>	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
German measles (see rubella; see congenital rubella)			
Giardiasis (see gastroenteritis)			
Gonococcal ophthalmia neonatorum (gonorrhoeal ophthalmia, acute conjunctivitis of newborn)	S		
Gonorrhea	S		
Granuloma inguinale (Donovanosis, granuloma venereum)	S		
Guillain-Barré' syndrome	S		Not an infectious condition
<i>Haemophilus influenzae</i> (see disease-specific recommendations)			
Hand, foot, and mouth disease (see enteroviral infection)			
Hansen's Disease (see Leprosy)			
Hantavirus pulmonary syndrome	S		Not transmitted from person to person
<i>Helicobacter pylori</i>	S		
Hepatitis, viral			
Type A	S		Provide hepatitis A vaccine post-exposure as recommended ¹⁰⁶⁵
Diapered or incontinent patients	C		Maintain Contact Precautions in infants and children <3 years of age

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
			for duration of hospitalization; for children 3-14 yrs. of age for 2 weeks after onset of symptoms; >14 yrs. of age for 1 week after onset of symptoms ^{833, 1066, 1067} .
Type B-HBsAg positive; acute or chronic	S		See specific recommendations for care of patients in hemodialysis centers ⁷⁷⁸
Type C and other unspecified non-A, non-B	S		See specific recommendations for care of patients in hemodialysis centers ⁷⁷⁸
Type D (seen only with hepatitis B)	S		
Type E	S		Use Contact Precautions for diapered or incontinent individuals for the duration of illness ¹⁰⁶⁸
Type G	S		
Herpangina (see enteroviral infection)			
Hookworm	S		
Herpes simplex (<i>Herpesvirus hominis</i>)			
Encephalitis	S		
Mucocutaneous, disseminated or primary, severe	C	Until lesions dry and crusted	
Mucocutaneous, recurrent (skin, oral, genital)	S		
Neonatal	C	Until lesions dry and crusted	Also, for asymptomatic, exposed infants delivered vaginally or by C-section and if mother has active infection and membranes have been ruptured for more than 4 to 6 hrs until infant surface cultures obtained at 24-36 hrs. of age negative after 48 hrs incubation ^{1069, 1070}
Herpes zoster (varicella-zoster) (shingles)			
Disseminated disease in any patient Localized disease in immunocompromised patient until disseminated infection ruled out	A,C	DI	Susceptible HCWs should not enter room if immune caregivers are available; no recommendation for protection of immune HCWs; no recommendation for type of protection, i.e. surgical mask or respirator; for susceptible HCWs.

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
Localized in patient with intact immune system with lesions that can be contained/covered	S	DI	Susceptible HCWs should not provide direct patient care when other immune caregivers are available.
Histoplasmosis	S		Not transmitted from person to person
Human immunodeficiency virus (HIV)	S		Post-exposure chemoprophylaxis for some blood exposures ⁸⁶⁶ .
Human metapneumovirus	C	DI	HAI reported ¹⁰⁷¹ , but route of transmission not established ⁸²³ . Assumed to be Contact transmission as for RSV since the viruses are closely related and have similar clinical manifestations and epidemiology. Wear masks according to Standard Precautions..
Impetigo	C	U 24 hrs	
Infectious mononucleosis	S		
Influenza			
Human (seasonal influenza)			See www.cdc.gov/flu/professionals/infectioncontrol/healthcaresettings.htm for current seasonal influenza guidance.
Avian (e.g., H5N1, H7, H9 strains))			See www.cdc.gov/flu/avian/professional/infect-control.htm for current avian influenza guidance.
Pandemic influenza (also a human influenza virus)	D	5 days from onset of symptoms	See http://www.pandemicflu.gov for current pandemic influenza guidance.
Kawasaki syndrome	S		Not an infectious condition
Lassa fever (see viral hemorrhagic fevers)			

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
Legionnaires' disease	S		Not transmitted from person to person
Leprosy	S		
Leptospirosis	S		Not transmitted from person to person
Lice			http://www.cdc.gov/ncidod/dpd/parasites/lice/default.htm
Head (pediculosis)	C	U 24 hrs	
Body	S		Transmitted person to person through infested clothing. Wear gown and gloves when removing clothing; bag and wash clothes according to CDC guidance above
Pubic	S		Transmitted person to person through sexual contact
Listeriosis (<i>listeria monocytogenes</i>)	S		Person-to-person transmission rare; cross-transmission in neonatal settings reported ^{1072, 1073 1074, 1075}
Lyme disease	S		Not transmitted from person to person
Lymphocytic choriomeningitis	S		Not transmitted from person to person
Lymphogranuloma venereum	S		
Malaria	S		Not transmitted from person to person except through transfusion rarely and through a failure to follow Standard Precautions during patient care ¹⁰⁷⁶⁻¹⁰⁷⁹ . Install screens in windows and doors in endemic areas. Use DEET-containing mosquito repellants and clothing to cover extremities
Marburg virus disease (see viral hemorrhagic fevers)			
Measles (rubeola)	A	4 days after onset of rash; DI in immune compromised	Susceptible HCWs should not enter room if immune care providers are available; no recommendation for face protection for immune HCW; no recommendation for type of face protection for susceptible HCWs, i.e., mask or respirator ^{1027, 1028} . For exposed susceptibles, post-exposure vaccine within 72 hrs. or immune globulin within 6 days when available ^{17, 1032, 1034} . Place exposed susceptible patients on Airborne Precautions and exclude susceptible healthcare personnel

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
			from duty from day 5 after first exposure to day 21 after last exposure, regardless of post-exposure vaccine ¹⁷ .
Melioidosis, all forms	S		Not transmitted from person to person
Meningitis			
Aseptic (nonbacterial or viral; also see enteroviral infections)	S		Contact for infants and young children
Bacterial, gram-negative enteric, in neonates	S		
Fungal	S		
<i>Haemophilus influenzae</i> , type b known or suspected	D	U 24 hrs	
<i>Listeria monocytogenes</i> (See Listeriosis)	S		
<i>Neisseria meningitidis</i> (meningococcal) known or suspected	D	U 24 hrs	See meningococcal disease below
<i>Streptococcus pneumoniae</i>	S		
<i>M. tuberculosis</i>	S		Concurrent, active pulmonary disease or draining cutaneous lesions may necessitate addition of Contact and/or Airborne Precautions; For children, airborne precautions until active tuberculosis ruled out in visiting family members (see tuberculosis below) ⁴²
Other diagnosed bacterial	S		
Meningococcal disease: sepsis, pneumonia, meningitis	D	U 24 hrs	Postexposure chemoprophylaxis for household contacts, HCWs exposed to respiratory secretions; postexposure vaccine only to control outbreaks ^{15, 17} .
<i>Molluscum contagiosum</i>	S		
Monkeypox	A,C	A-Until monkeypox confirmed and smallpox excluded C-Until lesions crusted	Use See www.cdc.gov/ncidod/monkeypox for most current recommendations. Transmission in hospital settings unlikely ²⁶⁹ . Pre- and post-exposure smallpox vaccine recommended for exposed HCWs

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
Mucormycosis	S		
Multidrug-resistant organisms (MDROs), infection or colonization (e.g., MRSA, VRE, VISA/VRSA, ESBLs, resistant <i>S. pneumoniae</i>)	S/C		MDROs judged by the infection control program, based on local, state, regional, or national recommendations, to be of clinical and epidemiologic significance. Contact Precautions recommended in settings with evidence of ongoing transmission, acute care settings with increased risk for transmission or wounds that cannot be contained by dressings. See recommendations for management options in Management of Multidrug-Resistant Organisms In Healthcare Settings, 2006 ⁸⁷⁰ . Contact state health department for guidance regarding new or emerging MDRO.
Mumps (infectious parotitis)	D	U 9 days	After onset of swelling; susceptible HCWs should not provide care if immune caregivers are available. Note: (Recent assessment of outbreaks in healthy 18-24 year olds has indicated that salivary viral shedding occurred early in the course of illness and that 5 days of isolation after onset of parotitis may be appropriate in community settings; however the implications for healthcare personnel and high-risk patient populations remain to be clarified.)
Mycobacteria, nontuberculosis (atypical)			Not transmitted person-to-person
Pulmonary	S		
Wound	S		
<i>Mycoplasma pneumoniae</i>	D	DI	
Necrotizing enterocolitis	S		Contact Precautions when cases clustered temporally ¹⁰⁸⁰⁻¹⁰⁸³ .
Nocardiosis, draining lesions, or other presentations	S		Not transmitted person-to-person
Norovirus (see gastroenteritis)			
Norwalk agent gastroenteritis (see gastroenteritis)			
Orf	S		

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
Parainfluenza virus infection, respiratory in infants and young children	C	DI	Viral shedding may be prolonged in immunosuppressed patients ^{1009, 1010} . Reliability of antigen testing to determine when to remove patients with prolonged hospitalizations from Contact Precautions uncertain.
Parvovirus B19 (Erythema infectiosum)	D		Maintain precautions for duration of hospitalization when chronic disease occurs in an immunocompromised patient. For patients with transient aplastic crisis or red-cell crisis, maintain precautions for 7 days. Duration of precautions for immunosuppressed patients with persistently positive PCR not defined, but transmission has occurred ⁹²⁹ .
Pediculosis (lice)	C	U 24 hrs after treatment	
Pertussis (whooping cough)	D	U 5 days	Single patient room preferred. Cohorting an option. Post-exposure chemoprophylaxis for household contacts and HCWs with prolonged exposure to respiratory secretions ⁸⁶³ . Recommendations for Tdap vaccine in adults under development.
Pinworm infection (Enterobiasis)	S		
Plague (<i>Yersinia pestis</i>)			
Bubonic	S		
Pneumonic	D	U 48 hrs	Antimicrobial prophylaxis for exposed HCW ²⁰⁷ .
Pneumonia			
Adenovirus	D, C	DI	Outbreaks in pediatric and institutional settings reported ^{376, 1084-1086} . In immunocompromised hosts, extend duration of Droplet and Contact Precautions due to prolonged shedding of virus ⁹³¹ .
Bacterial not listed elsewhere (including gram-negative bacterial)	S		
<i>B. cepacia</i> in patients with CF, including respiratory tract colonization	C	Unknown	Avoid exposure to other persons with CF; private room preferred. Criteria for D/C precautions not established. See CF Foundation guideline ²⁰ .

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
<i>B. cepacia</i> in patients without CF(see Multidrug-resistant organisms)			
<i>Chlamydia</i>	S		
Fungal	S		
<i>Haemophilus influenzae</i> , type b			
Adults	S		
Infants and children	D	U 24 hrs	
<i>Legionella spp.</i>	S		
Meningococcal	D	U 24 hrs	See meningococcal disease above
Multidrug-resistant bacterial (see multidrug-resistant organisms)			
<i>Mycoplasma</i> (primary atypical pneumonia)	D	DI	
Pneumococcal pneumonia	S		Use Droplet Precautions if evidence of transmission within a patient care unit or facility ^{196-198, 1087}
<i>Pneumocystis jiroveci</i> (<i>Pneumocystis carinii</i>)	S		Avoid placement in the same room with an immunocompromised patient.
<i>Staphylococcus aureus</i>	S		For MRSA, see MDROs
<i>Streptococcus</i> , group A			
Adults	D	U 24 hrs	See streptococcal disease (group A streptococcus) below
Infants and young children	D	U 24 hrs	Contact precautions if skin lesions present
Varicella-zoster (See Varicella-Zoster)			Contact Precautions if skin lesions present
Viral			
Adults	S		
Infants and young children (see respiratory infectious disease, acute, or specific viral agent)			
Poliomyelitis	C	DI	
Pressure ulcer (decubitus ulcer, pressure sore) infected			

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
Major	C	DI	If no dressing or containment of drainage; until drainage stops or can be contained by dressing
Minor or limited	S		If dressing covers and contains drainage
Prion disease (See Creutzfeld-Jacob Disease)			
Psittacosis (ornithosis) (<i>Chlamydia psittaci</i>)	S		Not transmitted from person to person
Q fever	S		
Rabies	S		Person to person transmission rare; transmission via corneal, tissue and organ transplants has been reported ^{539, 1088} . If patient has bitten another individual or saliva has contaminated an open wound or mucous membrane, wash exposed area thoroughly and administer postexposure prophylaxis. ¹⁰⁸⁹
Rat-bite fever (<i>Streptobacillus moniliformis</i> disease, <i>Spirillum minus</i> disease)	S		Not transmitted from person to person
Relapsing fever	S		Not transmitted from person to person
Resistant bacterial infection or colonization (see multidrug-resistant organisms)			
Respiratory infectious disease, acute (if not covered elsewhere)			
Adults	S		
Infants and young children	C	DI	Also see syndromes or conditions listed in Table 2
Respiratory syncytial virus infection, in infants, young children and immunocompromised adults	C	DI	Wear mask according to Standard Precautions ²⁴ CB ^{116, 117} . In immunocompromised patients, extend the duration of Contact Precautions due to prolonged shedding ⁹²⁸). Reliability of antigen testing to determine when to remove patients with prolonged hospitalizations from Contact Precautions uncertain.
Reye's syndrome	S		Not an infectious condition
Rheumatic fever	S		Not an infectious condition
Rhinovirus	D	DI	Droplet most important route of transmission ^{104 1090} . Outbreaks have

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
			occurred in NICUs and LTCFs ^{413, 1091, 1092} . Add Contact Precautions if copious moist secretions and close contact likely to occur (e.g., young infants) ^{111, 833} .
Rickettsial fevers, tickborne (Rocky Mountain spotted fever, tickborne typhus fever)	S		Not transmitted from person to person except through transfusion, rarely
Rickettsialpox (vesicular rickettsiosis)	S		Not transmitted from person to person
Ringworm (dermatophytosis, dermatomycosis, tinea)	S		Rarely, outbreaks have occurred in healthcare settings, (e.g., NICU ¹⁰⁹³ , rehabilitation hospital ¹⁰⁹⁴). Use Contact Precautions for outbreak.
Ritter's disease (staphylococcal scalded skin syndrome)	C	DI	See staphylococcal disease, scalded skin syndrome below
Rocky Mountain spotted fever	S		Not transmitted from person to person except through transfusion, rarely
Roseola infantum (exanthem subitum; caused by HHV-6)	S		
Rotavirus infection (see gastroenteritis)			
Rubella (German measles) (also see congenital rubella)	D	U 7 days after onset of rash	Susceptible HCWs should not enter room if immune caregivers are available. No recommendation for wearing face protection (e.g., a surgical mask) if immune. Pregnant women who are not immune should not care for these patients ^{17, 33} . Administer vaccine within three days of exposure to non-pregnant susceptible individuals. Place exposed susceptible patients on Droplet Precautions; exclude susceptible healthcare personnel from duty from day 5 after first exposure to day 21 after last exposure, regardless of post-exposure vaccine.
Rubeola (see measles)			
Salmonellosis (see gastroenteritis)			
Scabies	C	U 24	
Scalded skin syndrome, staphylococcal	C	DI	See staphylococcal disease, scalded skin syndrome below)
Schistosomiasis (bilharziasis)	S		

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
Severe acute respiratory syndrome (SARS)	A, D,C	DI plus 10 days after resolution of fever, provided respiratory symptoms are absent or improving	Airborne Precautions preferred; D if AIIR unavailable. N95 or higher respiratory protection; surgical mask if N95 unavailable; eye protection (goggles, face shield); aerosol-generating procedures and “supershedders” highest risk for transmission via small droplet nuclei and large droplets ^{93, 94, 96} . Vigilant environmental disinfection (see www.cdc.gov/ncidod/sars)
Shigellosis (see gastroenteritis)			
Smallpox (variola; see vaccinia for management of vaccinated persons)	A,C	DI	Until all scabs have crusted and separated (3-4 weeks). Non-vaccinated HCWs should not provide care when immune HCWs are available; N95 or higher respiratory protection for susceptible and successfully vaccinated individuals; postexposure vaccine within 4 days of exposure protective ^{108, 129, 1038-1040} .
Sporotrichosis	S		
<i>Spirillum minor</i> disease (rat-bite fever)	S		Not transmitted from person to person
Staphylococcal disease (<i>S aureus</i>)			
Skin, wound, or burn			
Major	C	DI	No dressing or dressing does not contain drainage adequately
Minor or limited	S		Dressing covers and contains drainage adequately
Enterocolitis	S		Use Contact Precautions for diapered or incontinent children for duration of illness
Multidrug-resistant (see multidrug-resistant organisms)			
Pneumonia	S		
Scalded skin syndrome	C	DI	Consider healthcare personnel as potential source of nursery, NICU outbreak ¹⁰⁹⁵ .
Toxic shock syndrome	S		
<i>Streptobacillus moniliformis</i> disease (rat-bite fever)	S		Not transmitted from person to person

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
Streptococcal disease (group A streptococcus)			
Skin, wound, or burn			
Major	C,D	U 24 hrs	No dressing or dressing does not contain drainage adequately
Minor or limited	S		Dressing covers and contains drainage adequately
Endometritis (puerperal sepsis)	S		
Pharyngitis in infants and young children	D	U 24 hrs	
Pneumonia	D	U 24 hrs	
Scarlet fever in infants and young children	D	U 24 hrs	
Serious invasive disease	D	U24 hrs	Outbreaks of serious invasive disease have occurred secondary to transmission among patients and healthcare personnel ^{162, 972, 1096-1098} . Contact Precautions for draining wound as above; follow rec. for antimicrobial prophylaxis in selected conditions ¹⁶⁰ .
Streptococcal disease (group B streptococcus), neonatal	S		
Streptococcal disease (not group A or B) unless covered elsewhere	S		
Multidrug-resistant (see multidrug-resistant organisms)			
Strongyloidiasis	S		
Syphilis			
Latent (tertiary) and seropositivity without lesions	S		
Skin and mucous membrane, including congenital, primary, Secondary	S		
Tapeworm disease			
<i>Hymenolepis nana</i>	S		Not transmitted from person to person
<i>Taenia solium</i> (pork)	S		
Other	S		
Tetanus	S		Not transmitted from person to person
Tinea (e.g., dermatophytosis, dermatomycosis, ringworm)	S		Rare episodes of person-to-person transmission

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
Toxoplasmosis	S		Transmission from person to person is rare; vertical transmission from mother to child, transmission through organs and blood transfusion rare
Toxic shock syndrome (staphylococcal disease, streptococcal disease)	S		Droplet Precautions for the first 24 hours after implementation of antibiotic therapy if Group A streptococcus is a likely etiology
Trachoma, acute	S		
Transmissible spongiform encephalopathy (see Creutzfeld-Jacob disease, CJD, vCJD)			
Trench mouth (Vincent's angina)	S		
Trichinosis	S		
Trichomoniasis	S		
Trichuriasis (whipworm disease)	S		
Tuberculosis (<i>M. tuberculosis</i>)			
Extrapulmonary, draining lesion)	A,C		Discontinue precautions only when patient is improving clinically, and drainage has ceased or there are three consecutive negative cultures of continued drainage ^{1025, 1026} . Examine for evidence of active pulmonary tuberculosis.
Extrapulmonary, no draining lesion, meningitis	S		Examine for evidence of pulmonary tuberculosis. For infants and children, use Airborne Precautions until active pulmonary tuberculosis in visiting family members ruled out ⁴²
Pulmonary or laryngeal disease, confirmed	A		Discontinue precautions only when patient on effective therapy is improving clinically and has three consecutive sputum smears negative for acid-fast bacilli collected on separate days (MMWR 2005; 54: RR-17 http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5417a1.htm?s_cid=rr5417a1_e) ¹² .
Pulmonary or laryngeal disease, suspected	A		Discontinue precautions only when the likelihood of infectious TB

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
			disease is deemed negligible, and either 1) there is another diagnosis that explains the clinical syndrome or 2) the results of three sputum smears for AFB are negative. Each of the three sputum specimens should be collected 8-24 hours apart, and at least one should be an early morning specimen
Skin-test positive with no evidence of current active disease	S		
Tularemia			
Draining lesion	S		Not transmitted from person to person
Pulmonary	S		Not transmitted from person to person
Typhoid (<i>Salmonella typhi</i>) fever (see gastroenteritis)			
Typhus			
<i>Rickettsia prowazekii</i> (Epidemic or Louse-borne typhus)	S		Transmitted from person to person through close personal or clothing contact
<i>Rickettsia typhi</i>	S		Not transmitted from person to person
Urinary tract infection (including pyelonephritis), with or without urinary catheter	S		
Vaccinia (vaccination site, adverse events following vaccination) *			Only vaccinated HCWs have contact with active vaccination sites and care for persons with adverse vaccinia events; if unvaccinated, only HCWs without contraindications to vaccine may provide care.
Vaccination site care (including autoinoculated areas)	S		Vaccination recommended for vaccinators; for newly vaccinated HCWs: semi-permeable dressing over gauze until scab separates, with dressing change as fluid accumulates, ~3-5 days; gloves, hand hygiene for dressing change; vaccinated HCW or HCW without contraindication to vaccine for dressing changes ^{205, 221, 225} .
Eczema vaccinatum	C	Until lesions dry and crusted, scabs separated	For contact with virus-containing lesions and exudative material
Fetal vaccinia	C		
Generalized vaccinia	C		

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
Progressive vaccinia	C		
Postvaccinia encephalitis	S		
Blepharitis or conjunctivitis	S/C		Use Contact Precautions if there is copious drainage
Iritis or keratitis	S		
Vaccinia-associated erythema multiforme (Stevens Johnson Syndrome)	S		Not an infectious condition
Secondary bacterial infection (e.g., <i>S. aureus</i> , group A beta hemolytic streptococcus)	S/C		Follow organism-specific (strep, staph most frequent) recommendations and consider magnitude of drainage
Varicella Zoster	A,C	Until lesions dry and crusted	Susceptible HCWs should not enter room if immune caregivers are available; no recommendation for face protection of immune HCWs; no recommendation for type of protection, i.e. surgical mask or respirator for susceptible HCWs. In immunocompromised host with varicella pneumonia, prolong duration of precautions for duration of illness. Post-exposure prophylaxis: provide post-exposure vaccine ASAP but within 120 hours; for susceptible exposed persons for whom vaccine is contraindicated (immunocompromised persons, pregnant women, newborns whose mother's varicella onset is ≤5days before delivery or within 48 hrs after delivery) provide VZIG, when available, within 96 hours; if unavailable, use IVIG, Use Airborne Precautions for exposed susceptible persons and exclude exposed susceptible healthcare workers beginning 8 days after first exposure until 21 days after last exposure or 28 if received VZIG, regardless of postexposure vaccination. ¹⁰³⁶
Variola (see smallpox)			
<i>Vibrio parahaemolyticus</i> (see gastroenteritis)			
Vincent's angina (trench mouth)	S		
Viral hemorrhagic fevers	S, D, C	DI	Single-patient room preferred. Emphasize: 1) use of sharps safety

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
due to Lassa, Ebola, Marburg, Crimean-Congo fever viruses			devices and safe work practices, 2) hand hygiene; 3) barrier protection against blood and body fluids upon entry into room (single gloves and fluid-resistant or impermeable gown, face/eye protection with masks, goggles or face shields); and 4) appropriate waste handling. Use N95 or higher respirators when performing aerosol-generating procedures. Largest viral load in final stages of illness when hemorrhage may occur; additional PPE, including double gloves, leg and shoe coverings may be used, especially in resource-limited settings where options for cleaning and laundry are limited. Notify public health officials immediately if Ebola is suspected ^{212, 314, 740, 772} Also see Table 3 for Ebola as a bioterrorism agent
Viral respiratory diseases (not covered elsewhere)			
Adults	S		
Infants and young children (see respiratory infectious disease, acute)			
Whooping cough (see pertussis)			
Wound infections			
Major	C	DI	No dressing or dressing does not contain drainage adequately
Minor or limited	S		Dressing covers and contains drainage adequately
<i>Yersinia enterocolitica</i> gastroenteritis (see gastroenteritis)			
Zoster (varicella-zoster) (see herpes zoster)			
Zygomycosis (phycomycosis, mucormycosis)	S		Not transmitted person-to-person

TABLE 1. HISTORY OF GUIDELINES FOR ISOLATION PRECAUTIONS IN HOSPITALS*

YEAR (Ref)	DOCUMENT ISSUED	COMMENT
1970 1099	Isolation Techniques for Use in Hospitals, 1 st ed.	<ul style="list-style-type: none"> - Introduced seven isolation precaution categories with color-coded cards: Strict, Respiratory, Protective, Enteric, Wound and Skin, Discharge, and Blood - No user decision-making required - Simplicity a strength; over isolation prescribed for some infections
1975 1100	Isolation Techniques for Use in Hospitals, 2 nd ed.	<ul style="list-style-type: none"> - Same conceptual framework as 1st edition
1983 1101	CDC Guideline for Isolation Precautions in Hospitals	<ul style="list-style-type: none"> - Provided two systems for isolation: category-specific and disease-specific - Protective Isolation eliminated; Blood Precautions expanded to include Body Fluids - Categories included Strict, Contact, Respiratory, AFB, Enteric, Drainage/Secretion, Blood and Body Fluids - Emphasized decision-making by users
1985-88 780, 896	Universal Precautions	<ul style="list-style-type: none"> - Developed in response to HIV/AIDS epidemic - Dictated application of Blood and Body Fluid precautions to all patients, regardless of infection status - Did not apply to feces, nasal secretions, sputum, sweat, tears, urine, or vomitus unless contaminated by visible blood - Added personal protective equipment to protect HCWs from mucous membrane exposures - Handwashing recommended immediately after glove removal - Added specific recommendations for handling needles and other sharp devices; concept became integral to OSHA's 1991 rule on occupational exposure to blood-borne pathogens in healthcare settings

<p>1987 1102</p>	<p>Body Substance Isolation</p>	<ul style="list-style-type: none"> - Emphasized avoiding contact with all moist and potentially infectious body substances except sweat even if blood not present - Shared some features with Universal Precautions - Weak on infections transmitted by large droplets or by contact with dry surfaces - Did not emphasize need for special ventilation to contain airborne infections - Handwashing after glove removal not specified in the absence of visible soiling
<p>1996 1</p>	<p>Guideline for Isolation Precautions in Hospitals</p>	<ul style="list-style-type: none"> - Prepared by the Healthcare Infection Control Practices Advisory Committee (HICPAC) - Melded major features of Universal Precautions and Body Substance Isolation into Standard Precautions to be used with all patients at all times - Included three transmission-based precaution categories: airborne, droplet, and contact - Listed clinical syndromes that should dictate use of empiric isolation until an etiological diagnosis is established

* Derived from Garner ICHE 1996

TABLE 2. CLINICAL SYNDROMES OR CONDITIONS WARRANTING EMPIRIC TRANSMISSION-BASED PRECAUTIONS IN ADDITION TO STANDARD PRECAUTIONS PENDING CONFIRMATION OF DIAGNOSIS*

Clinical Syndrome or Condition†	Potential Pathogens‡	Empiric Precautions (Always includes Standard Precautions)
DIARRHEA		
Acute diarrhea with a likely infectious cause in an incontinent or diapered patient	Enteric pathogens§	Contact Precautions (pediatrics and adult)
MENINGITIS		
	<i>Neisseria meningitidis</i>	Droplet Precautions for first 24 hrs of antimicrobial therapy; mask and face protection for intubation
	Enteroviruses	Contact Precautions for infants and children
	<i>M. tuberculosis</i>	Airborne Precautions if pulmonary infiltrate Airborne Precautions plus Contact Precautions if potentially infectious draining body fluid present
RASH OR EXANTHEMS, GENERALIZED, ETIOLOGY UNKNOWN		
Petechial/ecchymotic with fever (general) - If positive history of travel to an area with an ongoing outbreak of VHF in the 10 days before onset of fever	<i>Neisseria meningitides</i> Ebola, Lassa, Marburg viruses	Droplet Precautions for first 24 hrs of antimicrobial therapy Droplet Precautions plus Contact Precautions, with face/eye protection, emphasizing safety sharps and barrier precautions when blood exposure likely. Use N95 or higher respiratory protection when aerosol-generating procedure performed

Vesicular	Varicella-zoster, <i>herpes simplex</i> , variola (smallpox), vaccinia viruses Vaccinia virus	Airborne plus Contact Precautions; Contact Precautions only if <i>herpes simplex</i> , localized zoster in an immunocompetent host or vaccinia viruses most likely
Maculopapular with cough, coryza and fever	Rubeola (measles) virus	Airborne Precautions

Clinical Syndrome or Condition†	Potential Pathogens‡	Empiric Precautions (Always includes Standard Precautions)
RESPIRATORY INFECTIONS		
Cough/fever/upper lobe pulmonary infiltrate in an HIV-negative patient or a patient at low risk for human immunodeficiency virus (HIV) infection	<i>M. tuberculosis</i> , Respiratory viruses, <i>S. pneumoniae</i> , <i>S. aureus</i> (MSSA or MRSA)	Airborne Precautions plus Contact precautions
Cough/fever/pulmonary infiltrate in any lung location in an HIV-infected patient or a patient at high risk for HIV infection	<i>M. tuberculosis</i> , Respiratory viruses, <i>S. pneumoniae</i> , <i>S. aureus</i> (MSSA or MRSA)	Airborne Precautions plus Contact Precautions Use eye/face protection if aerosol-generating procedure performed or contact with respiratory secretions anticipated. If tuberculosis is unlikely and there are no AIIRs and/or respirators available, use Droplet Precautions instead of Airborne Precautions Tuberculosis more likely in HIV-infected individual than in HIV negative individual
Cough/fever/pulmonary infiltrate in any lung location in a patient with a history of recent travel (10-21 days) to countries with active outbreaks of SARS, avian influenza	<i>M. tuberculosis</i> , severe acute respiratory syndrome virus (SARS-CoV), avian influenza	Airborne plus Contact Precautions plus eye protection. If SARS and tuberculosis unlikely, use Droplet Precautions instead of Airborne Precautions.
Respiratory infections, particularly bronchiolitis and pneumonia, in infants and young children	Respiratory syncytial virus, parainfluenza virus, adenovirus, influenza virus, Human metapneumovirus	Contact plus Droplet Precautions; Droplet Precautions may be discontinued when adenovirus and influenza have been ruled out

Skin or Wound Infection

Abscess or draining wound that cannot be covered	<i>Staphylococcus aureus</i> (MSSA or MRSA), group A streptococcus	Contact Precautions Add Droplet Precautions for the first 24 hours of appropriate antimicrobial therapy if invasive Group A streptococcal disease is suspected
--------------------------------------------------	--------------------------------------------------------------------	-------------------------------------------------------------------------------------------------------------------------------------------------------------------

- * Infection control professionals should modify or adapt this table according to local conditions. To ensure that appropriate empiric precautions are implemented always, hospitals must have systems in place to evaluate patients routinely according to these criteria as part of their preadmission and admission care.
- † Patients with the syndromes or conditions listed below may present with atypical signs or symptoms (e.g. neonates and adults with pertussis may not have paroxysmal or severe cough). The clinician's index of suspicion should be guided by the prevalence of specific conditions in the community, as well as clinical judgment.
- ‡ The organisms listed under the column "Potential Pathogens" are not intended to represent the complete, or even most likely, diagnoses, but rather possible etiologic agents that require additional precautions beyond Standard Precautions until they can be ruled out.
- § These pathogens include enterohemorrhagic *Escherichia coli* O157:H7, *Shigella spp*, hepatitis A virus, noroviruses, rotavirus, *C. difficile*.

TABLE 3.
INFECTION CONTROL CONSIDERATIONS FOR HIGH-PRIORITY (CDC CATEGORY A) DISEASES THAT MAY RESULT FROM BIOTERRORIST ATTACKS OR ARE CONSIDERED TO BE BIOTERRORIST THREATS

(www.bt.cdc.gov)^a

^a Abbreviations used in this table: RT = respiratory tract; GIT = gastrointestinal tract; CXR = chest x-ray; CT = computerized axial tomography; CSF = cerebrospinal fluid; and LD₅₀ – lethal dose for 50% of experimental animals; HCWs = healthcare worker; BSL = biosafety level; PAPR = powered air purifying respirator; PCR = polymerase chain reaction; IHC = immunohistochemistry

Disease	Anthrax
Site(s) of Infection; Transmission Mode Cutaneous and inhalation disease have occurred in past bioterrorist incidents	Cutaneous (contact with spores); RT (inhalation of spores); GIT (ingestion of spores - rare) Comment: Spores can be inhaled into the lower respiratory tract. The infectious dose of <i>B. anthracis</i> in humans by any route is not precisely known. In primates, the LD ₅₀ (i.e., the dose required to kill 50% of animals) for an aerosol challenge with <i>B. anthracis</i> is estimated to be 8,000–50,000 spores; the infectious dose may be as low as 1-3 spores
Incubation Period	Cutaneous: 1 to 12 days; RT: Usually 1 to 7 days but up to 43 days reported; GIT: 15-72 hours
Clinical Features	Cutaneous: Painless, reddish papule, which develops a central vesicle or bulla in 1-2 days; over next 3-7 days lesion becomes pustular, and then necrotic, with black eschar; extensive surrounding edema. RT: initial flu-like illness for 1-3 days with headache, fever, malaise, cough; by day 4 severe dyspnea and shock, and is usually fatal (85%-90% if untreated; meningitis in 50% of RT cases). GIT: ; if intestinal form, necrotic, ulcerated edematous lesions develop in intestines with fever, nausea and vomiting, progression to hematemesis and bloody diarrhea; 25-60% fatal
Diagnosis	Cutaneous: Swabs of lesion (under eschar) for IHC, PCR and culture; punch biopsy for IHC, PCR and culture; vesicular fluid aspirate for Gram stain and culture; blood culture if systemic symptoms; acute and

	<p>convalescent sera for ELISA serology</p> <p>RT: CXR or CT demonstrating wide mediastinal widening and/or pleural effusion, hilar abnormalities; blood for culture and PCR; pleural effusion for culture, PCR and IHC; CSF if meningeal signs present for IHC, PCR and culture; acute and convalescent sera for ELISA serology; pleural and/or bronchial biopsies IHC.</p> <p>GIT: blood and ascites fluid, stool samples, rectal swabs, and swabs of oropharyngeal lesions if present for culture, PCR and IHC</p>
Infectivity	<p>Cutaneous: Person-to-person transmission from contact with lesion of untreated patient possible, but extremely rare.</p> <p>RT and GIT: Person-to-person transmission does not occur.</p> <p>Aerosolized powder, environmental exposures: Highly infectious if aerosolized</p>
Recommended Precautions	<p>Cutaneous: Standard Precautions; Contact Precautions if uncontained copious drainage.</p> <p>RT and GIT: Standard Precautions.</p> <p>Aerosolized powder, environmental exposures: Respirator (N95 mask or PAPRs), protective clothing; decontamination of persons with powder on them (http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5135a3.htm)</p> <p>Hand hygiene: Handwashing for 30-60 seconds with soap and water or 2% chlorhexidine gluconate after spore contact (alcohol handrubs inactive against spores [Weber DJ JAMA 2003; 289:1274]).</p> <p>Post-exposure prophylaxis following environmental exposure: 60 days of antimicrobials (either doxycycline, ciprofloxacin, or levofloxacin) and post-exposure vaccine under IND</p>

Disease	Botulism
Site(s) of Infection; Transmission Mode	<p>GIT: Ingestion of toxin-containing food, RT: Inhalation of toxin containing aerosol cause disease.</p> <p>Comment: Toxin ingested or potentially delivered by aerosol in bioterrorist incidents. LD₅₀ for type A is 0.001 µg/ml/kg.</p>
Incubation Period	1-5 days.
Clinical Features	Ptosis, generalized weakness, dizziness, dry mouth and throat, blurred vision, diplopia, dysarthria, dysphonia, and dysphagia followed by symmetrical descending paralysis and respiratory failure.

Diagnosis	Clinical diagnosis; identification of toxin in stool, serology unless toxin-containing material available for toxin neutralization bioassays.
Infectivity	Not transmitted from person to person. Exposure to toxin necessary for disease.
Recommended Precautions	Standard Precautions.
Disease	Ebola Hemorrhagic Fever
Site(s) of Infection; Transmission Mode	As a rule infection develops after exposure of mucous membranes or RT, or through broken skin or percutaneous injury.
Incubation Period	2-19 days, usually 5-10 days
Clinical Features	Febrile illnesses with malaise, myalgias, headache, vomiting and diarrhea that are rapidly complicated by hypotension, shock, and hemorrhagic features. Massive hemorrhage in < 50% pts.
Diagnosis	Etiologic diagnosis can be made using RT-PCR, serologic detection of antibody and antigen, pathologic assessment with immunohistochemistry and viral culture with EM confirmation of morphology,
Infectivity	Person-to-person transmission primarily occurs through unprotected contact with blood and body fluids; percutaneous injuries (e.g., needlestick) associated with a high rate of transmission; transmission in healthcare settings has been reported but is prevented by use of barrier precautions.
Recommended Precautions	Hemorrhagic fever specific barrier precautions: If disease is believed to be related to intentional release of a bioweapon, epidemiology of transmission is unpredictable pending observation of disease transmission. Until the nature of the pathogen is understood and its transmission pattern confirmed, Standard, Contact and Airborne Precautions should be used. Once the pathogen is characterized, if the epidemiology of transmission is consistent with natural disease, Droplet Precautions can be substituted for Airborne Precautions. Emphasize: 1) use of sharps safety devices and safe work practices, 2) hand hygiene; 3) barrier protection against blood and body fluids upon entry into room (single gloves and fluid-resistant or impermeable gown, face/eye protection with masks, goggles or face shields); and 4) appropriate waste handling. Use N95 or higher respirators when performing aerosol-generating procedures. In settings where AIIRs are unavailable or the large numbers of patients cannot be accommodated by existing AIIRs, observe Droplet Precautions (plus Standard Precautions and Contact Precautions) and segregate patients from those not suspected of VHF infection. Limit blood draws to those essential to care. See text for discussion and Appendix A for recommendations for naturally

	occurring VHF.
--	----------------

Disease	Plague ²
Site(s) of Infection; Transmission Mode	RT: Inhalation of respiratory droplets. Comment: Pneumonic plague most likely to occur if used as a biological weapon, but some cases of bubonic and primary septicemia may also occur. Infective dose 100 to 500 bacteria
Incubation Period	1 to 6, usually 2 to 3 days.
Clinical Features	Pneumonic: fever, chills, headache, cough, dyspnea, rapid progression of weakness, and in a later stage hemoptysis, circulatory collapse, and bleeding diathesis
Diagnosis	Presumptive diagnosis from Gram stain or Wayson stain of sputum, blood, or lymph node aspirate; definitive diagnosis from cultures of same material, or paired acute/convalescent serology.
Infectivity	Person-to-person transmission occurs via respiratory droplets risk of transmission is low during first 20-24 hours of illness and requires close contact. Respiratory secretions probably are not infectious within a few hours after initiation of appropriate therapy.
Recommended Precautions	Standard Precautions, Droplet Precautions until patients have received 48 hours of appropriate therapy. Chemoprophylaxis: Consider antibiotic prophylaxis for HCWs with close contact exposure.

² Pneumonic plague is not as contagious as is often thought. Historical accounts and contemporary evidence indicate that persons with plague usually only transmit the infection when the disease is in the end stage. These persons cough copious amounts of bloody sputum that contains many plague bacteria. Patients in the early stage of primary pneumonic plague (approximately the first 20–24 h) apparently pose little risk [1, 2]. Antibiotic medication rapidly clears the sputum of plague bacilli, so that a patient generally is not infective within hours after initiation of effective antibiotic treatment [3]. This means that in modern times many patients will never reach a stage where they pose a significant risk to others. Even in the end stage of disease, transmission only occurs after close contact. Simple protective measures, such as wearing masks, good hygiene, and avoiding close contact, have been effective to interrupt transmission during many pneumonic plague outbreaks [2]. In the United States, the last known cases of person to person transmission of pneumonic plague occurred in 1925 [2].

1. Wu L-T. A treatise on pneumonic plague. Geneva: League of Nations, 1926. III. Health.
2. Kool JL. Risk of person to person transmission of pneumonic plague. *Clinical Infectious Diseases*, 2005; 40 (8): 1166-1172
3. Butler TC. Plague and other Yersinia infections. In: Greenough WB, ed. *Current topics in infectious disease*. New York: Plenum Medical Book Company, 1983.

Disease	Smallpox
Site(s) of Infection; Transmission Mode	RT Inhalation of droplet or, rarely, aerosols; and skin lesions (contact with virus). Comment: If used as a biological weapon, natural disease, which has not occurred since 1977, will likely result.
Incubation Period	7 to 19 days (mean 12 days)
Clinical Features	Fever, malaise, backache, headache, and often vomiting for 2-3 days; then generalized papular or maculopapular rash (more on face and extremities), which becomes vesicular (on day 4 or 5) and then pustular; lesions all in same stage.
Diagnosis	Electron microscopy of vesicular fluid or culture of vesicular fluid by WHO approved laboratory (CDC); detection by PCR available only in select LRN labs, CDC and USAMRID
Infectivity	Secondary attack rates up to 50% in unvaccinated persons; infected persons may transmit disease from time rash appears until all lesions have crusted over (about 3 weeks); greatest infectivity during first 10 days of rash.
Recommended Precautions	Combined use of Standard, Contact, and Airborne Precautions ^b until all scabs have separated (3-4 weeks). Only immune HCWs to care for pts; post-exposure vaccine within 4 days. Vaccinia: HCWs cover vaccination site with gauze and semi-permeable dressing until scab separates (≥ 21 days). Observe hand hygiene. Adverse events with virus-containing lesions: Standard plus Contact Precautions until all lesions crusted

^b Transmission by the airborne route is a rare event; Airborne Precautions is recommended when possible, but in the event of mass exposures, barrier precautions and containment within a designated area are most important^{204, 212}.

^c Vaccinia adverse events with lesions containing infectious virus include inadvertent autoinoculation, ocular lesions (blepharitis, conjunctivitis), generalized vaccinia, progressive vaccinia, eczema vaccinatum; bacterial superinfection also requires addition of contact precautions if exudates cannot be contained^{216, 217}.

Disease	Tularemia
Site(s) of Infection; Transmission Mode	RT: Inhalation of aerosolized bacteria. GIT: Ingestion of food or drink contaminated with aerosolized bacteria. Comment: Pneumonic or typhoidal disease likely to occur after bioterrorist event using aerosol delivery. Infective dose 10-50 bacteria
Incubation Period	2 to 10 days, usually 3 to 5 days
Clinical Features	Pneumonic: malaise, cough, sputum production, dyspnea; Typhoidal: fever, prostration, weight loss and frequently an associated pneumonia.
Diagnosis	Diagnosis usually made with serology on acute and convalescent serum specimens; bacterium can be detected by PCR (LRN) or isolated from blood and other body fluids on cysteine-enriched media or mouse inoculation.
Infectivity	Person-to-person spread is rare. Laboratory workers who encounter/handle cultures of this organism are at high risk for disease if exposed.
Recommended Precautions	Standard Precautions

TABLE 4.
RECOMMENDATIONS FOR APPLICATION OF STANDARD PRECAUTIONS FOR THE CARE OF ALL PATIENTS IN ALL HEALTHCARE SETTINGS
(See Sections II.D.-II.J. and III.A.1)

COMPONENT	RECOMMENDATIONS
Hand hygiene	After touching blood, body fluids, secretions, excretions, contaminated items; immediately after removing gloves; between patient contacts.
Personal protective equipment (PPE)	
Gloves	For touching blood, body fluids, secretions, excretions, contaminated items; for touching mucous membranes and nonintact skin
Gown	During procedures and patient-care activities when contact of clothing/exposed skin with blood/body fluids, secretions, and excretions is anticipated..
Mask, eye protection (goggles), face shield*	During procedures and patient-care activities likely to generate splashes or sprays of blood, body fluids, secretions, especially suctioning, endotracheal intubation
Soiled patient-care equipment	Handle in a manner that prevents transfer of microorganisms to others and to the environment; wear gloves if visibly contaminated; perform hand hygiene.
Environmental control	Develop procedures for routine care, cleaning, and disinfection of environmental surfaces, especially frequently touched surfaces in patient-care areas.
Textiles and laundry	Handle in a manner that prevents transfer of microorganisms to others and to the environment
Needles and other sharps	Do not recap, bend, break, or hand-manipulate used needles; if recapping is required, use a one-handed scoop technique only; use safety features when available; place used sharps in puncture-resistant container
Patient resuscitation	Use mouthpiece, resuscitation bag, other ventilation devices to prevent contact with mouth and oral secretions

Patient placement	Prioritize for single-patient room if patient is at increased risk of transmission, is likely to contaminate the environment, does not maintain appropriate hygiene, or is at increased risk of acquiring infection or developing adverse outcome following infection.
Respiratory hygiene/cough etiquette (source containment of infectious respiratory secretions in symptomatic patients, beginning at initial point of encounter e.g., triage and reception areas in emergency departments and physician offices)	Instruct symptomatic persons to cover mouth/nose when sneezing/coughing; use tissues and dispose in no-touch receptacle; observe hand hygiene after soiling of hands with respiratory secretions; wear surgical mask if tolerated or maintain spatial separation, >3 feet if possible.

* * During aerosol-generating procedures on patients with suspected or proven infections transmitted by respiratory aerosols (e.g., SARS), wear a fit-tested N95 or higher respirator in addition to gloves, gown, and face/eye protection.

TABLE 5. COMPONENTS OF A PROTECTIVE ENVIRONMENT

(Adapted from MMWR 2003; 52 [RR-10])

I. Patients: allogeneic hematopoietic stem cell transplant (HSCT) only

- Maintain in PE room except for required diagnostic or therapeutic procedures that cannot be performed in the room, e.g. radiology, operating room
- Respiratory protection e.g., N95 respirator, for the patient when leaving PE during periods of construction

II. Standard and Expanded Precautions

- Hand hygiene observed before and after patient contact
- Gown, gloves, mask NOT required for HCWs or visitors for routine entry into the room
- Use of gown, gloves, mask by HCWs and visitors according to Standard Precautions and as indicated for suspected or proven infections for which Transmission-Based Precautions are recommended

III. Engineering

- Central or point-of-use HEPA (99.97% efficiency) filters capable of removing particles 0.3 μm in diameter for supply (incoming) air
- Well-sealed rooms
 - Proper construction of windows, doors, and intake and exhaust ports
 - Ceilings: smooth, free of fissures, open joints, crevices
 - Walls sealed above and below the ceiling
 - If leakage detected, locate source and make necessary repairs
- Ventilation to maintain ≥ 12 ACH
- Directed air flow: air supply and exhaust grills located so that clean, filtered air enters from one side of the room, flows across the patient's bed, exits on opposite side of the room
- Positive room air pressure in relation to the corridor
 - Pressure differential of >2.5 Pa [0.01" water gauge]
- Monitor and document results of air flow patterns daily using visual methods (e.g., flutter strips, smoke tubes) or a hand held pressure gauge
- Self-closing door on all room exits
- Maintain back-up ventilation equipment (e.g., portable units for fans or filters) for emergency provision of ventilation requirements for PE areas and take immediate steps to restore the fixed ventilation system
- For patients who require both a PE and Airborne Infection Isolation, use an anteroom to ensure proper air balance relationships and provide independent exhaust of contaminated air to the outside or place a HEPA filter in the exhaust duct. If an anteroom is not available, place patient in an AIIR and use portable ventilation units, industrial-grade HEPA filters to enhance filtration of spores.

IV. Surfaces

- Daily wet-dusting of horizontal surfaces using cloths moistened with EPA-registered hospital disinfectant/detergent
- Avoid dusting methods that disperse dust
- No carpeting in patient rooms or hallways
- No upholstered furniture and furnishings

V. Other

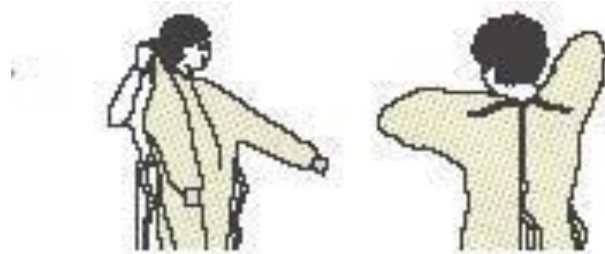
- No flowers (fresh or dried) or potted plants in PE rooms or areas
- Use vacuum cleaner equipped with HEPA filters when vacuum cleaning is necessary

Figure.
Example of Safe Donning and Removal of Personal
Protective Equipment (PPE)

DONNING PPE

GOWN

- Fully cover torso from neck to knees, arms to end of wrist, and wrap around the back
- Fasten in back at neck and waist



MASK OR RESPIRATOR

- Secure ties or elastic band at middle of head and neck
- Fit flexible band to nose bridge
- Fit snug to face and below chin
- Fit-check respirator



GOGGLES/FACE SHIELD

- Put on face and adjust to fit



GLOVES

- Use non-sterile for isolation
- Select according to hand size
- Extend to cover wrist of isolation gown



SAFE WORK PRACTICES

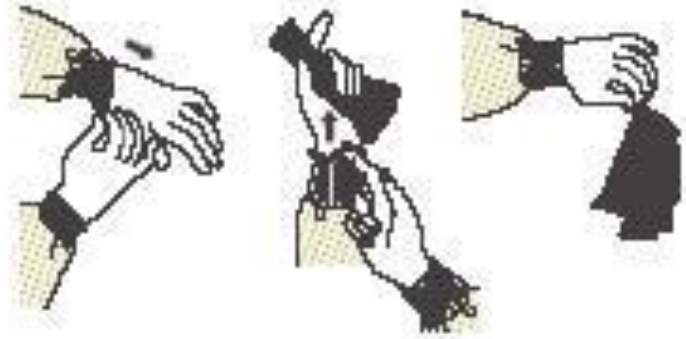
- Keep hands away from face
- Work from clean to dirty
- Limit surfaces touched
- Change when torn or heavily contaminated
- Perform hand hygiene

REMOVING PPE

Remove PPE at doorway before leaving patient room or in anteroom

GLOVES

- Outside of gloves are contaminated!
- Grasp outside of glove with opposite gloved hand; peel off
- Hold removed glove in gloved hand
- Slide fingers of ungloved hand under remaining glove at wrist



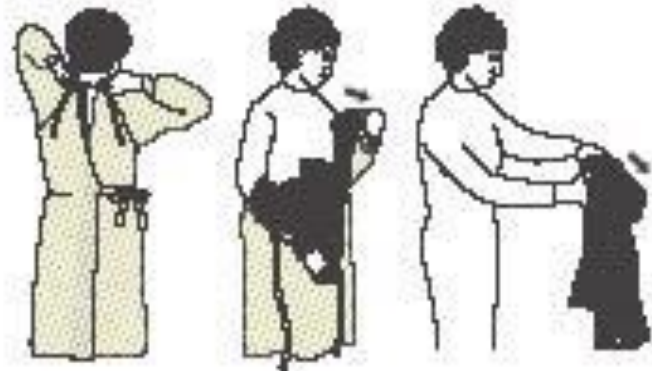
GOGGLES/FACE SHIELD

- Outside of goggles or face shield are contaminated!
- To remove, handle by “clean” head band or ear pieces
- Place in designated receptacle for reprocessing or in waste container



GOWN

- Gown front and sleeves are contaminated!
- Unfasten neck, then waist ties
- Remove gown using a peeling motion; pull gown from each shoulder toward the same hand
- Gown will turn inside out
- Hold removed gown away from body, roll into a bundle and discard into waste or linen receptacle



MASK OR RESPIRATOR

- Front of mask/respirator is contaminated – DO NOT TOUCH!
- Grasp ONLY bottom then top ties/elastics and remove
- Discard in waste container



HAND HYGIENE

Perform hand hygiene immediately after removing all PPE!

GLOSSARY

Airborne infection isolation room (AIIR). Formerly, negative pressure isolation room, an AIIR is a single-occupancy patient-care room used to isolate persons with a suspected or confirmed airborne infectious disease. Environmental factors are controlled in AIIRs to minimize the transmission of infectious agents that are usually transmitted from person to person by droplet nuclei associated with coughing or aerosolization of contaminated fluids. AIIRs should provide negative pressure in the room (so that air flows under the door gap into the room); **and** an air flow rate of 6-12 ACH (6 ACH for existing structures, 12 ACH for new construction or renovation); **and** direct exhaust of air from the room to the outside of the building or recirculation of air through a HEPA filter before retruning to circulation (MMWR 2005; 54 [RR-17]).

American Institute of Architects (AIA). A professional organization that develops standards for building ventilation, The “2001 Guidelines for Design and Construction of Hospital and Health Care Facilities”, the development of which was supported by the AIA, Academy of Architecture for Health, Facilities Guideline Institute, with assistance from the U.S. Department of Health and Human Services and the National Institutes of Health, is the primary source of guidance for creating airborne infection isolation rooms (AIIRs) and protective environments (www.aia.org/aah).

Ambulatory care settings. Facilities that provide health care to patients who do not remain overnight (e.g., hospital-based outpatient clinics, nonhospital-based clinics and physician offices, urgent care centers, surgicenters, free-standing dialysis centers, public health clinics, imaging centers, ambulatory behavioral health and substance abuse clinics, physical therapy and rehabilitation centers, and dental practices).

Bioaerosols. An airborne dispersion of particles containing whole or parts of biological entities, such as bacteria, viruses, dust mites, fungal hyphae, or fungal spores. Such aerosols usually consist of a mixture of mono-dispersed and aggregate cells, spores or viruses, carried by other materials, such as respiratory secretions and/or inert particles. Infectious bioaerosols (i.e., those that contain biological agents capable of causing an infectious disease) can be generated from human sources (e.g., expulsion from the respiratory tract during coughing, sneezing, talking or singing; during suctioning or wound irrigation), wet environmental sources (e.g. HVAC and cooling tower water with Legionella) or dry sources (e.g., construction dust with spores produced by *Aspergillus* spp.). Bioaerosols include large respiratory droplets and small droplet nuclei (Cole EC. AJIC 1998;26: 453-64).

Caregivers. All persons who are not employees of an organization, are not paid, and provide or assist in providing healthcare to a patient (e.g., family member, friend) and acquire technical training as needed based on the tasks that must be performed.

Cohorting. In the context of this guideline, this term applies to the practice of grouping patients infected or colonized with the same infectious agent together to confine their care to one area and prevent contact with susceptible patients (cohorting patients). During outbreaks, healthcare personnel may be assigned to a cohort of patients to further limit opportunities for transmission (cohorting staff).

Colonization. Proliferation of microorganisms on or within body sites without detectable host immune response, cellular damage, or clinical expression. The presence of a microorganism within a host may occur with varying duration, but may become a source of potential transmission. In many instances, colonization and carriage are synonymous.

Droplet nuclei. Microscopic particles < 5 µm in size that are the residue of evaporated droplets and are produced when a person coughs, sneezes, shouts, or sings. These particles can remain suspended in the air for prolonged periods of time and can be carried on normal air currents in a room or beyond, to adjacent spaces or areas receiving exhaust air.

Engineering controls. Removal or isolation of a workplace hazard through technology. AllRs, a Protective Environment, engineered sharps injury prevention devices and sharps containers are examples of engineering controls.

Epidemiologically important pathogens . Infectious agents that have one or more of the following characteristics: 1) are readily transmissible; 2) have a proclivity toward causing outbreaks; 3) may be associated with a severe outcome; or 4) are difficult to treat. Examples include *Acinetobacter sp.*, *Aspergillus sp.*, *Burkholderia cepacia*, *Clostridium difficile*, *Klebsiella* or *Enterobacter sp.*, extended-spectrum-beta-lactamase producing gram negative bacilli [ESBLs], methicillin-resistant *Staphylococcus aureus* [MRSA], *Pseudomonas aeruginosa*, vancomycin-resistant enterococci [VRE], methicillin resistant *Staphylococcus aureus* [MRSA], vancomycin resistant *Staphylococcus aureus* [VRSA] influenza virus, respiratory syncytial virus [RSV], rotavirus, SARS-CoV, noroviruses and the hemorrhagic fever viruses).

Hand hygiene. A general term that applies to any one of the following: 1) handwashing with plain (nonantimicrobial) soap and water); 2) antiseptic handwash (soap containing antiseptic agents and water); 3) antiseptic handrub (waterless antiseptic product, most often alcohol-based, rubbed on all surfaces of hands); or 4) surgical hand antisepsis (antiseptic handwash or antiseptic handrub performed preoperatively by surgical personnel to eliminate transient hand flora and reduce resident hand flora)⁵⁵⁹.

Healthcare-associated infection (HAI). An infection that develops in a patient who is cared for in any setting where healthcare is delivered (e.g., acute care hospital, chronic care facility, ambulatory clinic, dialysis center, surgicenter, home) and is related to receiving health care (i.e., was not incubating or present at the time healthcare was provided). In ambulatory and home settings, HAI would apply to any infection that is associated with a medical or surgical intervention. Since the geographic location of infection acquisition is often uncertain, the preferred term is considered to be *healthcare-associated* rather than *healthcare-acquired*.

Healthcare epidemiologist. A person whose primary training is medical (M.D., D.O.) and/or masters or doctorate-level epidemiology who has received advanced training in healthcare epidemiology. Typically these professionals direct or provide consultation to an infection control program in a hospital, long term care facility (LTCF), or healthcare delivery system (also see infection control professional).

Healthcare personnel, healthcare worker (HCW). All paid and unpaid persons who work in a healthcare setting (e.g. any person who has professional or technical training in a healthcare-related field and provides patient care in a healthcare setting or any person who provides services that support the delivery of healthcare such as dietary, housekeeping, engineering, maintenance personnel).

Hematopoietic stem cell transplantation (HSCT). Any transplantation of blood- or bone marrow-derived hematopoietic stem cells, regardless of donor type (e.g., allogeneic or autologous) or cell source (e.g., bone marrow, peripheral blood, or placental/umbilical cord blood); associated with periods of severe immunosuppression that vary with the source of the cells, the intensity of chemotherapy required, and the presence of graft versus host disease (MMWR 2000; 49: RR-10).

High-efficiency particulate air (HEPA) filter. An air filter that removes >99.97% of particles $\geq 0.3\mu\text{m}$ (the most penetrating particle size) at a specified flow rate of air. HEPA filters may be integrated into the central air handling systems, installed at the point of use above the ceiling of a room, or used as portable units (MMWR 2003; 52: RR-10).

Home care. A wide-range of medical, nursing, rehabilitation, hospice and social services delivered to patients in their place of residence (e.g., private residence, senior living center, assisted living facility). Home health-care services include care provided by home health aides and skilled nurses, respiratory therapists, dietitians, physicians, chaplains, and volunteers; provision of durable medical equipment; home infusion therapy; and physical, speech, and occupational therapy.

Immunocompromised patients. Those patients whose immune mechanisms are deficient because of congenital or acquired immunologic disorders (e.g., human immunodeficiency virus [HIV] infection, congenital immune deficiency syndromes), chronic diseases such as diabetes mellitus, cancer, emphysema, or cardiac failure, ICU care, malnutrition, and immunosuppressive therapy of another disease process [e.g., radiation, cytotoxic chemotherapy, anti-graft• rejection medication, corticosteroids, monoclonal antibodies directed against a specific component of the immune system]). The type of infections for which an immunocompromised patient has increased susceptibility is determined by the severity of immunosuppression and the specific component(s) of the immune system that is affected. Patients undergoing allogeneic HSCT and those with chronic graft versus host disease are considered the most vulnerable to HAIs. Immunocompromised states also make it more difficult to diagnose certain infections (e.g., tuberculosis) and are associated with more severe clinical disease states than persons with the same infection and a normal immune system.

Infection. The transmission of microorganisms into a host after evading or overcoming defense mechanisms, resulting in the organism's proliferation and invasion within host tissue(s). Host responses to infection may include clinical symptoms or may be subclinical, with manifestations of disease mediated by direct organisms pathogenesis and/or a function of cell-mediated or antibody responses that result in the destruction of host tissues.

Infection control and prevention professional (ICP). A person whose primary training is in either nursing, medical technology, microbiology, or epidemiology and who has acquired special training in infection control. Responsibilities may include collection, analysis, and feedback of infection data and trends to healthcare providers; consultation on infection risk assessment, prevention and control strategies; performance of education and training activities; implementation of evidence-based infection control practices or those mandated by regulatory and licensing agencies; application of epidemiologic principles to improve patient outcomes; participation in planning renovation and construction projects (e.g., to ensure appropriate containment of construction dust); evaluation of new products or procedures on patient outcomes; oversight of employee health services related to infection prevention; implementation of preparedness plans; communication within the healthcare setting, with local and state health departments, and with the community at large concerning infection control issues; and participation in research. Certification in infection control (CIC) is available through the Certification Board of Infection Control and Epidemiology.

Infection control and prevention program. A multidisciplinary program that includes a group of activities to ensure that recommended practices for the prevention of healthcare-associated infections are implemented and followed by HCWs, making the healthcare setting safe from infection for patients and

healthcare personnel. The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) requires the following five components of an infection control program for accreditation: 1) *surveillance*: monitoring patients and healthcare personnel for acquisition of infection and/or colonization; 2) *investigation*: identification and analysis of infection problems or undesirable trends; 3) *prevention*: implementation of measures to prevent transmission of infectious agents and to reduce risks for device- and procedure-related infections; 4) *control*: evaluation and management of outbreaks; and 5) *reporting*: provision of information to external agencies as required by state and federal law and regulation (www.jcaho.org). The infection control program staff has the ultimate authority to determine infection control policies for a healthcare organization with the approval of the organization's governing body.

Long-term care facilities (LTCFs). An array of residential and outpatient facilities designed to meet the bio-psychosocial needs of persons with sustained self-care deficits. These include skilled nursing facilities, chronic disease hospitals, nursing homes, foster and group homes, institutions for the developmentally disabled, residential care facilities, assisted living facilities, retirement homes, adult day health care facilities, rehabilitation centers, and long-term psychiatric hospitals.

Mask. A term that applies collectively to items used to cover the nose and mouth and includes both procedure masks and surgical masks (www.fda.gov/cdrh/ode/guidance/094.html#4).

Multidrug-resistant organisms (MDROs). In general, bacteria that are resistant to one or more classes of antimicrobial agents and usually are resistant to all but one or two commercially available antimicrobial agents (e.g., MRSA, VRE, extended spectrum beta-lactamase [ESBL]-producing or intrinsically resistant gram-negative bacilli) ¹⁷⁶.

Nosocomial infection. A term that is derived from two Greek words “nosos” (disease) and “komeion” (to take care of) and refers to any infection that develops during or as a result of an admission to an acute care facility (hospital) and was not incubating at the time of admission.

Personal protective equipment (PPE). A variety of barriers used alone or in combination to protect mucous membranes, skin, and clothing from contact with infectious agents. PPE includes gloves, masks, respirators, goggles, face shields, and gowns.

Procedure Mask. A covering for the nose and mouth that is intended for use in general patient care situations. These masks generally attach to the face with ear loops rather than ties or elastic. Unlike surgical masks, procedure masks are not regulated by the Food and Drug Administration.

Protective Environment. A specialized patient-care area, usually in a hospital, that has a positive air flow relative to the corridor (i.e., air flows from the room to the outside adjacent space). The combination of high-efficiency particulate air (HEPA) filtration, high numbers (≥ 12) of air changes per hour (ACH), and minimal leakage of air into the room creates an environment that can safely accommodate patients with a severely compromised immune system (e.g., those who have received allogeneic hemopoietic stem-cell transplant [HSCT]) and decrease the risk of exposure to spores produced by environmental fungi. Other components include use of scrubbable surfaces instead of materials such as upholstery or carpeting, cleaning to prevent dust accumulation, and prohibition of fresh flowers or potted plants.

Quasi-experimental studies. Studies to evaluate interventions but do not use randomization as part of the study design. These studies are also referred to as nonrandomized, pre-post-intervention study designs. These studies aim to demonstrate causality between an intervention and an outcome but cannot achieve the level of confidence concerning attributable benefit obtained through a randomized, controlled trial. In hospitals and public health settings, randomized control trials often cannot be implemented due to ethical, practical and urgency reasons; therefore, quasi-experimental design studies are used commonly. However, even if an intervention appears to be effective statistically, the question can be raised as to the possibility of alternative explanations for the result.. Such study design is used when it is not logistically feasible or ethically possible to conduct a randomized, controlled trial, (e.g., during outbreaks). Within the classification of quasi-experimental study designs, there is a hierarchy of design features that may contribute to validity of results (Harris et al. CID 2004:38: 1586).

Residential care setting. A facility in which people live, minimal medical care is delivered, and the psychosocial needs of the residents are provided for.

Respirator. A personal protective device worn by healthcare personnel to protect them from inhalation exposure to airborne infectious agents that are $< 5 \mu\text{m}$ in size. These include infectious droplet nuclei from patients with *M. tuberculosis*, variola virus [smallpox], SARS-CoV), and dust particles that contain infectious particles, such as spores of environmental fungi (e.g., *Aspergillus* sp.). The CDC's National Institute for Occupational Safety and Health (NIOSH) certifies respirators used in healthcare settings (www.cdc.gov/niosh/topics/respirators/). The N95 disposable particulate, air purifying, respirator is the type used most commonly by healthcare personnel. Other respirators used include N-99 and N-100 particulate respirators, powered air-purifying respirators (PAPRS) with high efficiency filters; and non-powered full-facepiece elastomeric negative pressure respirators. A listing of NIOSH-approved respirators can be found at www.cdc.gov/niosh/npptl/respirators/disp_part/particlist.html. Respirators must be used in conjunction with a complete Respiratory Protection Program, as

required by the Occupational Safety and Health Administration (OSHA), that includes fit testing, training, proper selection of respirators, medical clearance and respirator maintenance.

Respiratory Hygiene/ Cough Etiquette. A combination of measures designed to minimize the transmission of respiratory pathogens via droplet or airborne routes in healthcare settings. The components of Respiratory Hygiene/Cough Etiquette are 1) covering the mouth and nose during coughing and sneezing, 2) using tissues to contain respiratory secretions with prompt disposal into a no-touch receptacle, 3) offering a surgical mask to persons who are coughing to decrease contamination of the surrounding environment, and 4) turning the head away from others and maintaining spatial separation, ideally >3 feet, when coughing. These measures are targeted to all patients with symptoms of respiratory infection and their accompanying family members or friends beginning at the point of initial encounter with a healthcare setting (e.g., reception/triage in emergency departments, ambulatory clinics, healthcare provider offices) ¹²⁶ (Srinivasin A ICHE 2004; 25: 1020; www.cdc.gov/flu/professionals/infectioncontrol/resphygiene.htm).

Safety culture/climate. The shared perceptions of workers and management regarding the expectations of safety in the work environment. A hospital safety climate includes the following six organizational components: 1) senior management support for safety programs; 2) absence of workplace barriers to safe work practices; 3) cleanliness and orderliness of the worksite; 4) minimal conflict and good communication among staff members; 5) frequent safety-related feedback/training by supervisors; and 6) availability of PPE and engineering controls ⁶²⁰.

Source Control. The process of containing an infectious agent either at the portal of exit from the body or within a confined space. The term is applied most frequently to containment of infectious agents transmitted by the respiratory route but could apply to other routes of transmission, (e.g., a draining wound, vesicular or bullous skin lesions). Respiratory Hygiene/Cough Etiquette that encourages individuals to “cover your cough” and/or wear a mask is a source control measure. The use of enclosing devices for local exhaust ventilation (e.g., booths for sputum induction or administration of aerosolized medication) is another example of source control.

Standard Precautions. A group of infection prevention practices that apply to all patients, regardless of suspected or confirmed diagnosis or presumed infection status. Standard Precautions is a combination and expansion of Universal Precautions ⁷⁸⁰ and Body Substance Isolation ¹¹⁰². Standard Precautions is based on the principle that all blood, body fluids, secretions, excretions except sweat, nonintact skin, and mucous membranes may contain transmissible infectious agents. Standard Precautions includes hand hygiene, and depending on the anticipated exposure, use of gloves, gown, mask, eye protection, or face shield. Also, equipment or items in the patient environment

likely to have been contaminated with infectious fluids must be handled in a manner to prevent transmission of infectious agents, (e.g. wear gloves for handling, contain heavily soiled equipment, properly clean and disinfect or sterilize reusable equipment before use on another patient).

Surgical mask. A device worn over the mouth and nose by operating room personnel during surgical procedures to protect both surgical patients and operating room personnel from transfer of microorganisms and body fluids. Surgical masks also are used to protect healthcare personnel from contact with large infectious droplets (>5 μm in size). According to draft guidance issued by the Food and Drug Administration on May 15, 2003, surgical masks are evaluated using standardized testing procedures for fluid resistance, bacterial filtration efficiency, differential pressure (air exchange), and flammability in order to mitigate the risks to health associated with the use of surgical masks. These specifications apply to any masks that are labeled surgical, laser, isolation, or dental or medical procedure_ (www.fda.gov/cdrh/ode/guidance/094.html#4). Surgical masks do not protect against inhalation of small particles or droplet nuclei and should not be confused with particulate respirators that are recommended for protection against selected airborne infectious agents, (e.g., *Mycobacterium tuberculosis*).

References:

1. Garner JS. Guideline for isolation precautions in hospitals. The Hospital Infection Control Practices Advisory Committee. *Infect Control Hosp Epidemiol* 1996;17(1):53-80.(s).
2. Harris AD, Bradham DD, Baumgarten M, Zuckerman IH, Fink JC, Perencevich EN. The use and interpretation of quasi-experimental studies in infectious diseases. *Clin Infect Dis* 2004;38(11):1586-91.
3. Morton V, Torgerson DJ. Effect of regression to the mean on decision making in health care. *Bmj* 2003;326(7398):1083-4.
4. Pocock SJ, Elbourne DR. Randomized trials or observational tribulations? *N Engl J Med* 2000;342(25):1907-9.
5. Ioannidis JP, Haidich AB, Pappa M, et al. Comparison of evidence of treatment effects in randomized and nonrandomized studies. *Jama* 2001;286(7):821-30.
6. Bent S, Shojania KG, Saint S. The use of systematic reviews and meta-analyses in infection control and hospital epidemiology. *Am J Infect Control* 2004;32(4):246-54.
7. Harris AD, Lautenbach E, Perencevich E. A systematic review of quasi-experimental study designs in the fields of infection control and antibiotic resistance. *Clin Infect Dis* 2005;41(1):77-82.
8. Evans R, Lloyd JF, Abouzelof RH, Taylor CW, Anderson VR, Samore MH. System-wide Surveillance for Clinical Encounters by Patients Previously Identified with MRSA and VRE. *Medinfo* 2004;2004:212-6.
9. Srinivasan A, McDonald LC, Jernigan D, et al. Foundations of the severe acute respiratory syndrome preparedness and response plan for healthcare facilities. *Infect Control Hosp Epidemiol* 2004;25(12):1020-5.
10. www.cdc.gov/flu/avian/professional/infect-control.htm
11. CDC. Guidelines for Environmental Infection Control in Health-Care Facilities. Recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). *MMWR* 2003;52(RR10):1-42.
12. CDC. Guidelines for preventing the transmission of Mycobacterium tuberculosis in health-care settings, 2005. *MMWR Recomm Rep* 2005;54(17):1-141.
13. AIA. Guidelines for Design and Construction of Hospital and Health Care Facilities. In: American Institute of Architects. Washington, DC: American Institute of Architects Press; 2006.
14. CDC. Guidelines for Preventing Health-Care-Associated Pneumonia, 2003. Recommendations of CDC and the Healthcare Infection Control

- Practices Advisory Committee. MMWR Recomm Rep 2004;53(RR-3):1-40.
15. CDC. Guidelines for preventing opportunistic infections among hematopoietic stem cell transplant recipients. Recommendations of CDC, the Infectious Disease Society of America, and the American Society of Blood and Marrow Transplantation. MMWR - Morbidity & Mortality Weekly Report 2000;49(RR-10):1-125.
 16. Boyce JM, Pittet D. Guideline for Hand Hygiene in Health-Care Settings. Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force. Society for Healthcare Epidemiology of America/Association for Professionals in Infection Control/Infectious Diseases Society of America. MMWR Recomm Rep 2002;51(RR-16):1-45, quiz CE1-4.
 17. Bolyard EA, Tablan OC, Williams WW, Pearson ML, Shapiro CN, Deitchmann SD. Guideline for infection control in healthcare personnel, 1998. Hospital Infection Control Practices Advisory Committee. Infect Control Hosp Epidemiol 1998;19(6):407-63.
 18. CDC. Recommendations for Preventing Transmission of Infections Among Chronic Hemodialysis Patients. MMWR 2001;50(RR05):1-43.
 19. Kohn WG, Collins AS, Cleveland JL, Harte JA, Eklund KJ, Malvitz DM. Guidelines for infection control in dental health-care settings--2003. MMWR Recomm Rep 2003;52(RR-17):1-61.
 20. Saiman L, Siegel J. Infection control recommendations for patients with cystic fibrosis: microbiology, important pathogens, and infection control practices to prevent patient-to-patient transmission. Infect Control Hosp Epidemiol 2003;24(5 Suppl):S6-52.
 21. Varia M, Wilson S, Sarwal S, et al. Investigation of a nosocomial outbreak of severe acute respiratory syndrome (SARS) in Toronto, Canada. Cmaj 2003;169(4):285-92.
 22. Haley RW, Cushion NB, Tenover FC, et al. Eradication of endemic methicillin-resistant Staphylococcus aureus infections from a neonatal intensive care unit. J Infect Dis 1995;171(3):614-24.
 23. Bridges CB, Kuehnert MJ, Hall CB. Transmission of influenza: implications for control in health care settings. Clin Infect Dis 2003;37(8):1094-101.
 24. Hall CB. Nosocomial respiratory syncytial virus infections: the "Cold War" has not ended. Clin Infect Dis 2000;31(2):590-6.
 25. Campbell JR, Zaccaria E, Mason EO, Jr., Baker CJ. Epidemiological analysis defining concurrent outbreaks of Serratia marcescens and methicillin-resistant Staphylococcus aureus in a neonatal intensive-care unit. Infect Control Hosp Epidemiol 1998;19(12):924-8.

26. Pena C, Pujol M, Ardanuy C, et al. Epidemiology and successful control of a large outbreak due to *Klebsiella pneumoniae* producing extended-spectrum beta-lactamases. *Antimicrob Agents Chemother* 1998;42(1):53-8.
27. Bonten MJ, Slaughter S, Ambergen AW, et al. The role of "colonization pressure" in the spread of vancomycin-resistant enterococci: an important infection control variable. *Arch Intern Med* 1998;158(10):1127-32.
28. Jensenius M, Ringertz SH, Berild D, Bell H, Espinoza R, Grinde B. Prolonged nosocomial outbreak of hepatitis A arising from an alcoholic with pneumonia. *Scand J Infect Dis* 1998;30(2):119-23.
29. Zawacki A, O'Rourke E, Potter-Bynoe G, Maccone A, Harbarth S, Goldmann D. An outbreak of *Pseudomonas aeruginosa* pneumonia and bloodstream infection associated with intermittent otitis externa in a healthcare worker. *Infect Control Hosp Epidemiol* 2004;25(12):1083-9.
30. Foca M, Jakob K, Whittier S, et al. Endemic *Pseudomonas aeruginosa* infection in a neonatal intensive care unit. *N Engl J Med* 2000;343(10):695-700.
31. Gupta A, Della-Latta P, Todd B, et al. Outbreak of extended-spectrum beta-lactamase-producing *Klebsiella pneumoniae* in a neonatal intensive care unit linked to artificial nails. *Infect Control Hosp Epidemiol* 2004;25(3):210-5.
32. Boyce JM, Opal SM, Potter-Bynoe G, Medeiros AA. Spread of methicillin-resistant *Staphylococcus aureus* in a hospital after exposure to a health care worker with chronic sinusitis. *Clin Infect Dis* 1993;17(3):496-504.
33. Fliegel PE, Weinstein WM. Rubella outbreak in a prenatal clinic: management and prevention. *Am J Infect Control* 1982;10(1):29-33.
34. Atkinson WL, Markowitz LE, Adams NC, Seastrom GR. Transmission of measles in medical settings--United States, 1985-1989. *Am J Med* 1991;91(3B):320S-4S.
35. Carman WF, Elder AG, Wallace LA, et al. Effects of influenza vaccination of health-care workers on mortality of elderly people in long-term care: a randomised controlled trial. *Lancet* 2000;355(9198):93-7.
36. CDC. Outbreaks of pertussis associated with hospitals--Kentucky, Pennsylvania, and Oregon, 2003. *MMWR Morb Mortal Wkly Rep* 2005;54(3):67-71.
37. Mermel LA, McKay M, Dempsey J, Parenteau S. *Pseudomonas* surgical-site infections linked to a healthcare worker with onychomycosis. *Infect Control Hosp Epidemiol* 2003;24(10):749-52.
38. Barnes GL, Callaghan SL, Kirkwood CD, Bogdanovic-Sakran N, Johnston LJ, Bishop RF. Excretion of serotype G1 rotavirus strains by

- asymptomatic staff: a possible source of nosocomial infection. *J Pediatr* 2003;142(6):722-5.
39. Wang JT, Chang SC, Ko WJ, et al. A hospital-acquired outbreak of methicillin-resistant *Staphylococcus aureus* infection initiated by a surgeon carrier. *J Hosp Infect* 2001;47(2):104-9.
 40. Valenti WM, Pincus PH, Messner MK. Nosocomial pertussis: possible spread by a hospital visitor. *Am J Dis Child* 1980;134(5):520-1.
 41. Christie CD, Glover AM, Willke MJ, Marx ML, Reising SF, Hutchinson NM. Containment of pertussis in the regional pediatric hospital during the Greater Cincinnati epidemic of 1993. *Infect Control Hosp Epidemiol* 1995;16(10):556-63.
 42. Munoz FM, Ong LT, Seavy D, Medina D, Correa A, Starke JR. Tuberculosis among adult visitors of children with suspected tuberculosis and employees at a children's hospital. *Infect Control Hosp Epidemiol* 2002;23(10):568-72.
 43. Garcia R, Raad I, Abi-Said D, et al. Nosocomial respiratory syncytial virus infections: prevention and control in bone marrow transplant patients. *Infect Control Hosp Epidemiol* 1997;18(6):412-6.
 44. Whimbey E, Champlin RE, Couch RB, et al. Community respiratory virus infections among hospitalized adult bone marrow transplant recipients. *Clin Infect Dis* 1996;22(5):778-82.
 45. Saiman L, O'keefe M, Graham PL, et al. Hospital transmission of community-acquired methicillin-resistant *Staphylococcus aureus* among postpartum women. *Clin Infect Dis* 2003;37(10):1313-9.
 46. Bonten MJ, Slaughter S, Hayden MK, Nathan C, van Voorhis J, Weinstein RA. External sources of vancomycin-resistant enterococci for intensive care units. *Crit Care Med* 1998;26(12):2001-4.
 47. Flynn DM, Weinstein RA, Nathan C, Gaston MA, Kabins SA. Patients' endogenous flora as the source of "nosocomial" *Enterobacter* in cardiac surgery. *J Infect Dis* 1987;156(2):363-8.
 48. Olson B, Weinstein RA, Nathan C, Chamberlin W, Kabins SA. Epidemiology of endemic *Pseudomonas aeruginosa*: why infection control efforts have failed. *J Infect Dis* 1984;150(6):808-16.
 49. Perl TM, Cullen JJ, Wenzel RP, et al. Intranasal mupirocin to prevent postoperative *Staphylococcus aureus* infections. *N Engl J Med* 2002;346(24):1871-7.
 50. Toltzis P, Hoyen C, et al. Factors that predict preexisting colonization with antibiotic-resistant gram-negative bacilli in patients admitted to a pediatric intensive care unit. *Pediatrics* 1999;103 (4 Pt1):719-23.

51. Sarginson RE, Taylor N, Reilly N, Baines PB, Van Saene HK. Infection in prolonged pediatric critical illness: A prospective four-year study based on knowledge of the carrier state. *Crit Care Med* 2004;32(3):839-47.
52. Silvestri L, Monti Bragadin C, Milanese M, et al. Are most ICU infections really nosocomial? A prospective observational cohort study in mechanically ventilated patients. *J Hosp Infect* 1999;42(2):125-33.
53. Heggens JP, Phillips LG, Boertman JA, et al. The epidemiology of methicillin-resistant *Staphylococcus aureus* in a burn center. *J Burn Care Rehabil* 1988;9(6):610-2.
54. Donskey CJ. The role of the intestinal tract as a reservoir and source for transmission of nosocomial pathogens. *Clin Infect Dis* 2004;39(2):219-26.
55. Osterholm MT, Hedberg CW, Moore KA. The epidemiology of infectious diseases. . In: G.L. M, Jr DRG, J.E. B, eds. *Principles and Practice of Infectious Diseases*. 5th ed ed. Philadelphia: Churchill Livingstone; 2000:161-3.
56. Thomsen RW, Hundborg HH, Lervang HH, Johnsen SP, Schonheyder HC, Sorensen HT. Risk of community-acquired pneumococcal bacteremia in patients with diabetes: a population-based case-control study. *Diabetes Care* 2004;27(5):1143-7.
57. Carton JA, Maradona JA, Nuno FJ, Fernandez-Alvarez R, Perez-Gonzalez F, Asensi V. Diabetes mellitus and bacteraemia: a comparative study between diabetic and non-diabetic patients. *Eur J Med* 1992;1(5):281-7.
58. Hirschtick RE, Glassroth J, Jordan MC, et al. Bacterial pneumonia in persons infected with the human immunodeficiency virus. Pulmonary Complications of HIV Infection Study Group. *N Engl J Med* 1995;333(13):845-51.
59. Rosenberg AL, Seneff MG, Atiyeh L, Wagner R, Bojanowski L, Zimmerman JE. The importance of bacterial sepsis in intensive care unit patients with acquired immunodeficiency syndrome: implications for future care in the age of increasing antiretroviral resistance. *Crit Care Med* 2001;29(3):548-56.
60. Malone JL, Ijaz K, Lambert L, et al. Investigation of healthcare-associated transmission of *Mycobacterium tuberculosis* among patients with malignancies at three hospitals and at a residential facility. *Cancer* 2004;101(12):2713-21.
61. Fishman JA, Rubin RH. Infection in organ-transplant recipients. *N Engl J Med* 1998;338(24):1741-51.
62. Safdar N, Kluger DM, Maki DG. A review of risk factors for catheter-related bloodstream infection caused by percutaneously inserted, noncuffed central venous catheters: implications for preventive strategies. *Medicine (Baltimore)* 2002;81(6):466-79.

63. Jarvis WR, Robles B. Nosocomial infections in pediatric patients. *Adv Pediatr Infect Dis* 1996;12:243-959(js).
64. Yogaraj JS, Elward AM, Fraser VJ. Rate, risk factors, and outcomes of nosocomial primary bloodstream infection in pediatric intensive care unit patients. *Pediatrics* 2002;110(3):481-5.
65. Donlan RM. Biofilms: microbial life on surfaces. *Emerg Infect Dis* 2002;8(9):881-90.
66. Rosen HR. Acquisition of hepatitis C by a conjunctival splash. *Am J Infect Control* 1997;25(3):242-7.
67. Beltrami EM, Kozak A, Williams IT, et al. Transmission of HIV and hepatitis C virus from a nursing home patient to a health care worker. *Am J Infect Control* 2003;31(3):168-75.
68. Obasanjo OO, Wu P, Conlon M, et al. An outbreak of scabies in a teaching hospital: lessons learned. *Infect Control Hosp Epidemiol* 2001;22(1):13-8.
69. Andersen BM, Haugen H, Rasch M, Heldal Haugen A, Tageson A. Outbreak of scabies in Norwegian nursing homes and home care patients: control and prevention. *J Hosp Infect* 2000;45(2):160-4.
70. Avitzur Y, Amir J. Herpetic whitlow infection in a general pediatrician--an occupational hazard. *Infection* 2002;30(4):234-6.
71. Adams G, Stover BH, Keenlyside RA, et al. Nosocomial herpetic infections in a pediatric intensive care unit. *Am J Epidemiol* 1981;113(2):126-32.
72. Bhalla A, Pultz NJ, Gries DM, et al. Acquisition of nosocomial pathogens on hands after contact with environmental surfaces near hospitalized patients. *Infect Control Hosp Epidemiol* 2004;25(2):164-7.
73. Duckro AN, Blom DW, Lyle EA, Weinstein RA, Hayden MK. Transfer of vancomycin-resistant enterococci via health care worker hands. *Arch Intern Med* 2005;165(3):302-7.
74. Brooks SE, Veal RO, Kramer M, Dore L, Schupf N, Adachi M. Reduction in the incidence of *Clostridium difficile*-associated diarrhea in an acute care hospital and a skilled nursing facility following replacement of electronic thermometers with single-use disposables. *Infect Control Hosp Epidemiol* 1992;13(2):98-103.
75. CDC. Nosocomial hepatitis B virus infection associated with reusable fingerstick blood sampling devices--Ohio and New York City, 1996. *MMWR Morb Mortal Wkly Rep* 1997;46 (10):217-21.
76. Desenclos JC, Bourdiol-Razes M, Rolin B, et al. Hepatitis C in a ward for cystic fibrosis and diabetic patients: possible transmission by spring-loaded finger-stick devices for self-monitoring of capillary blood glucose. *Infect Control Hosp Epidemiol* 2001;22(11):701-7.

77. CDC. Transmission of hepatitis B virus among persons undergoing blood glucose monitoring in long-term-care facilities--Mississippi, North Carolina, and Los Angeles County, California, 2003-2004. *MMWR Morb Mortal Wkly Rep* 2005;54(9):220-3.
78. Hall CB, Douglas RG, Jr. Modes of transmission of respiratory syncytial virus. *J Pediatr* 1981;99(1):100-3.
79. Hall CB, Douglas RG, Jr., Geiman JM. Possible transmission by fomites of respiratory syncytial virus. *J Infect Dis* 1980;141(1):98-102.
80. Buttery JP, Alabaster SJ, Heine RG, et al. Multiresistant *Pseudomonas aeruginosa* outbreak in a pediatric oncology ward related to bath toys. *Pediatr Infect Dis J* 1998;17(6):509-13.
81. Agerton T, Valway S, Gore B, et al. Transmission of a highly drug-resistant strain (strain W1) of *Mycobacterium tuberculosis*. Community outbreak and nosocomial transmission via a contaminated bronchoscope. *JAMA* 1997;278(13):1073-7.
82. Bronowicki JP, Venard V, Botte C, et al. Patient-to-patient transmission of hepatitis C virus during colonoscopy. *N Engl J Med* 1997;337(4):237-40.
83. Michele TM, Cronin WA, Graham NM, et al. Transmission of *Mycobacterium tuberculosis* by a fiberoptic bronchoscope. Identification by DNA fingerprinting. *JAMA* 1997;278(13):1093-5.
84. Schelenz S, French G. An outbreak of multidrug-resistant *Pseudomonas aeruginosa* infection associated with contamination of bronchoscopes and an endoscope washer-disinfector. *J Hosp Infect* 2000;46(1):23-30.
85. Weber DJ, Rutala WA. Lessons from outbreaks associated with bronchoscopy. *Infect Control Hosp Epidemiol* 2001;22(7):403-8.
86. Kirschke DL, Jones TF, Craig AS, et al. *Pseudomonas aeruginosa* and *Serratia marcescens* contamination associated with a manufacturing defect in bronchoscopes. *N Engl J Med* 2003;348(3):214-20.
87. Srinivasan A, Wolfenden LL, Song X, et al. An outbreak of *Pseudomonas aeruginosa* infections associated with flexible bronchoscopes. *N Engl J Med* 2003;348(3):221-7.
88. Boyce JM, Potter-Bynoe G, Chenevert C, King T. Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. *Infect Control Hosp Epidemiol* 1997;18(9):622-7.(mj).
89. Zachary KC, Bayne PS, Morrison VJ, Ford DS, Silver LC, Hooper DC. Contamination of gowns, gloves, and stethoscopes with vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 2001;22(9):560-4.
90. Perry C, Marshall R, Jones E. Bacterial contamination of uniforms. *J Hosp Infect* 2001;48(3):238-41.

91. Papineni RS, Rosenthal FS. The size distribution of droplets in the exhaled breath of healthy human subjects. *J Aerosol Med* 1997;10(2):105-16.
92. Wells WF. On airborne infection: Study II. Droplets and droplet nuclei. *Am J Hygiene* 1934;20:611-18.
93. Loeb M, McGeer A, Henry B, et al. SARS among critical care nurses, Toronto. *Emerg Infect Dis* 2004;10(2):251-5.
94. Fowler RA, Guest CB, Lapinsky SE, et al. Transmission of severe acute respiratory syndrome during intubation and mechanical ventilation. *Am J Respir Crit Care Med* 2004;169(11):1198-202.
95. Gehanno JF, Kohen-Couderc L, Lemeland JF, Leroy J. Nosocomial meningococemia in a physician. *Infect Control Hosp Epidemiol* 1999;20(8):564-5.
96. Scales D, et al. Illness in intensive-care staff after brief exposure to severe acute respiratory syndrome. *Emerg Infect Dis* 2003;9(10):1205-10.
97. Ensor E, Humphreys H, Peckham D, Webster C, Knox AJ. Is Burkholderia (Pseudomonas) cepacia disseminated from cystic fibrosis patients during physiotherapy? *J Hosp Infect* 1996;32(1):9-15.
98. Christian MD, Loutfy M, McDonald LC, et al. Possible SARS coronavirus transmission during cardiopulmonary resuscitation. *Emerg Infect Dis* 2004;10(2):287-93.
99. Valenzuela TD, Hooton TM, Kaplan EL, Schlievert P. Transmission of 'toxic strep' syndrome from an infected child to a firefighter during CPR. *Ann Emerg Med* 1991;20(1):90-2.
100. Bassinet L, Matrat M, Njamkepo E, Aberrane S, Housset B, Guiso N. Nosocomial pertussis outbreak among adult patients and healthcare workers. *Infect Control Hosp Epidemiol* 2004;25(11):995-7.
101. Wong TW, Lee CK, Tam W, et al. Cluster of SARS among medical students exposed to single patient, Hong Kong. *Emerg Infect Dis* 2004;10(2):269-76.
102. Pachucki CT, Pappas SA, Fuller GF, Krause SL, Lentino JR, Schaaff DM. Influenza A among hospital personnel and patients. Implications for recognition, prevention, and control. *Arch Intern Med* 1989;149(1):77-80.
103. Feigin RD, Baker CJ, Herwaldt LA, Lampe RM, Mason EO, Whitney SE. Epidemic meningococcal disease in an elementary-school classroom. *N Engl J Med* 1982;307(20):1255-7.
104. Dick EC, Jennings LC, Mink KA, Wartgow CD, Inhorn SL. Aerosol transmission of rhinovirus colds. *J Infect Dis* 1987;156(3):442-8.
105. Duguid JP. The size and duration of air-carriage of respiratory droplets and droplet nuclei. *J Hyg (Lond)* 1946;44:471-9.

106. Hall CB, Douglas RG, Jr., Schnabel KC, Geiman JM. Infectivity of respiratory syncytial virus by various routes of inoculation. *Infect Immun* 1981;33(3):779-83.
107. Downie AW, Meiklejohn M, St Vincent L, Rao AR, Sundara Babu BV, Kempe CH. The recovery of smallpox virus from patients and their environment in a smallpox hospital. *Bull World Health Organ* 1965;33(5):615-22.
108. Fenner F, Henderson DA, Arita I, Jezek Z, Ladnyi ID. The epidemiology of smallpox. In: *Smallpox and its eradication*. Switzerland: World Health Organization; 1988.
109. Cole EC, Cook CE. Characterization of infectious aerosols in health care facilities: an aid to effective engineering controls and preventive strategies. *Am J Infect Control* 1998;26(4):453-64.
110. Christie C, Mazon D, Hierholzer W, Jr., Patterson JE. Molecular heterogeneity of *Acinetobacter baumannii* isolates during seasonal increase in prevalence. *Infect Control Hosp Epidemiol* 1995;16(10):590-4.
111. Musher DM. How contagious are common respiratory tract infections? *N Engl J Med* 2003;348(13):1256-66.
112. Steinberg P, White RJ, Fuld SL, Gutekunst RR, Chanock RM, Senterfit LB. Ecology of *Mycoplasma pneumoniae* infections in marine recruits at Parris Island, South Carolina. *Am J Epidemiol* 1969;89(1):62-73.
113. Seto WH, Tsang D, Yung RW, et al. Effectiveness of precautions against droplets and contact in prevention of nosocomial transmission of severe acute respiratory syndrome (SARS). *Lancet* 2003;361(9368):1519-20.
114. Hamburger M, Robertson OH. Expulsion of group A haemolytic streptococci in droplets and droplet nuclei by sneezing, coughing and talking. *Am J Med* 1948;4:690.
115. CDC. Nosocomial meningococemia. *MMWR Morb Mortal Wkly Rep* 1978;27:358.
116. LeClair JM, Freeman J, Sullivan BF, Crowley CM, Goldmann DA. Prevention of nosocomial respiratory syncytial virus infections through compliance with glove and gown isolation precautions. *N Engl J Med* 1987;317(6):329-34.
117. Madge P, Paton JY, McColl JH, Mackie PL. Prospective controlled study of four infection-control procedures to prevent nosocomial infection with respiratory syncytial virus. *Lancet* 1992;340(8827):1079-83.
118. Bassetti S, Bischoff WE, Walter M, et al. Dispersal of *Staphylococcus aureus* Into the Air Associated With a Rhinovirus Infection. *Infect Control Hosp Epidemiol* 2005;26(2):196-203.
119. Eichenwald HF, Kotsevalov O, Fasso LA. The "cloud baby": an example of bacterial-viral interaction. *Am J Dis Child* 1960;100:161-73.

120. Sheretz RJ, Reagan DR, Hampton KD, et al. A cloud adult: the Staphylococcus aureus-virus interaction revisited. *Ann Intern Med* 1996;124(6):539-47.
121. Coronado VG, Beck-Sague CM, Hutton MD, et al. Transmission of multidrug-resistant Mycobacterium tuberculosis among persons with human immunodeficiency virus infection in an urban hospital: epidemiologic and restriction fragment length polymorphism analysis. *J Infect Dis* 1993;168(4):1052-5.
122. Bloch AB, Orenstein WA, Ewing WM, et al. Measles outbreak in a pediatric practice: airborne transmission in an office setting. *Pediatrics* 1985;75(4):676-83.
123. LeClair JM, Zaia JA, Levin MJ, Congdon RG, Goldmann DA. Airborne transmission of chickenpox in a hospital. *N Engl J Med* 1980;302(8):450-3.
124. Riley RL, Mills CC, Nyka W, et al. Aerial dissemination of pulmonary tuberculosis. A two-year study of contagion in a tuberculosis ward. 1959. *Am J Hyg* 1959;70:185-96.
125. Beck-Sague C, Dooley SW, Hutton MD, et al. Hospital outbreak of multidrug-resistant Mycobacterium tuberculosis infections. Factors in transmission to staff and HIV-infected patients. *JAMA* 1992;268(10):1280-6.
126. CDC. Guidelines for preventing the transmission of Mycobacterium tuberculosis in health-care facilities, 1994. Centers for Disease Control and Prevention. *MMWR Recomm Rep* 1994;43(RR-13):1-132.
127. Haley CE, McDonald RC, Rossi L, Jones WD, Jr., Haley RW, Luby JP. Tuberculosis epidemic among hospital personnel. *Infect Control Hosp Epidemiol* 1989;10(5):204-10.
128. Wehrle PF, Posch J, Richter KH, Henderson DA. An airborne outbreak of smallpox in a German hospital and its significance with respect to other recent outbreaks in Europe. *Bull World Health Organ* 1970;43(5):669-79.
129. Gelfand HM, Posch J. The recent outbreak of smallpox in Meschede, West Germany. *Am J Epidemiol* 1971;93(4):234-7.
130. Moser MR, Bender TR, Margolis HS, Noble GR, Kendal AP, Ritter DG. An outbreak of influenza aboard a commercial airliner. *Am J Epidemiol* 1979;110(1):1-6.
131. Alford RH, Kasel JA, Gerone PJ, Knight V. Human influenza resulting from aerosol inhalation. *Proc Soc Exp Biol Med* 1966;122(3):800-4.
132. Chadwick PR, McCann R. Transmission of a small round structured virus by vomiting during a hospital outbreak of gastroenteritis. *J Hosp Infect* 1994;26(4):251-9.

133. Prince DS, Astry C, Vonderfecht S, Jakab G, Shen FM, Yolken RH. Aerosol transmission of experimental rotavirus infection. *Pediatr Infect Dis* 1986;5(2):218-22.
134. www.cdc.gov/ncidod/sars.
135. Peiris JS, Yuen KY, Osterhaus AD, Stohr K. The severe acute respiratory syndrome. *N Engl J Med* 2003;349(25):2431-41.
136. Olsen SJ, Chang HL, Cheung TY, et al. Transmission of the severe acute respiratory syndrome on aircraft. *N Engl J Med* 2003;349(25):2416-22.
137. Wilder-Smith A, Leong HN, Villacian JS. In-flight transmission of Severe Acute Respiratory Syndrome (SARS): A Case Report. *J Travel Med* 2003;10(5):299-300.
138. Booth TF, Kournikakis B, Bastien N, et al. Detection of airborne severe acute respiratory syndrome (SARS) coronavirus and environmental contamination in SARS outbreak units. *J Infect Dis* 2005;191(9):1472-7.
139. Yu IT, Li Y, Wong TW, et al. Evidence of airborne transmission of the severe acute respiratory syndrome virus. *N Engl J Med* 2004;350(17):1731-9.
140. CDC. Update: Outbreak of severe acute respiratory syndrome--worldwide. *MMWR Morb Mortal Wkly Rep* 2003;52 (12):241-6, 8.
141. CDC. Cluster of severe acute respiratory syndrome cases among protected health-care workers--Toronto, Canada, April 2003. *MMWR Morb Mortal Wkly Rep* 2003;52(19):433-6.
142. Sawyer LA, Murphy JJ, Kaplan JE, et al. 25- to 30-nm virus particle associated with a hospital outbreak of acute gastroenteritis with evidence for airborne transmission. *Am J Epidemiol* 1988;127(6):1261-71.
143. Marks PJ, Vipond IB, Carlisle D, Deakin D, Fey RE, Caul EO. Evidence for airborne transmission of Norwalk-like virus (NLV) in a hotel restaurant. *Epidemiol Infect* 2000;124(3):481-7.
144. Salgado CD, Farr BM, Hall KK, Hayden FG. Influenza in the acute hospital setting. *Lancet Infect Dis* 2002;2(3):145-55.
145. Riley RL. Airborne infection. *Am J Med* 1974;57(3):466-75.
146. McLean R. General discussion. . *Am Rev Respir Dis* 1961;83 36-8.
147. Cheesbrough JS, Green J, Gallimore CI, Wright PA, Brown DW. Widespread environmental contamination with Norwalk-like viruses (NLV) detected in a prolonged hotel outbreak of gastroenteritis. *Epidemiol Infect* 2000;125(1):93-8.
148. Marks PJ, Vipond IB, Regan FM, Wedgwood K, Fey RE, Caul EO. A school outbreak of Norwalk-like virus: evidence for airborne transmission. *Epidemiol Infect* 2003;131(1):727-36.

149. Roy CJ, Milton DK. Airborne transmission of communicable infection--the elusive pathway. *N Engl J Med* 2004;350(17):1710-2.
150. Dull PM, Wilson KE, Kournikakis B, et al. Bacillus anthracis aerosolization associated with a contaminated mail sorting machine. *Emerg Infect Dis* 2002;8(10):1044-7.
151. Weis CP, Intrepido AJ, Miller AK, et al. Secondary aerosolization of viable Bacillus anthracis spores in a contaminated US Senate Office. *JAMA* 2002;288(22):2853-8.
152. Patterson JE, Zidouh A, Minitier P, Andriole VT, Patterson TF. Hospital epidemiologic surveillance for invasive aspergillosis: patient demographics and the utility of antigen detection. *Infect Control Hosp Epidemiol* 1997;18(2):104-8.
153. Arnow PM, Andersen RL, Mainous PD, Smith EJ. Pulmonary aspergillosis during hospital renovation. *Am Rev Respir Dis* 1978;118(1):49-53.
154. Pegues DA, Lasker BA, McNeil MM, Hamm PM, Lundal JL, Kubak BM. Cluster of cases of invasive aspergillosis in a transplant intensive care unit: evidence of person-to-person airborne transmission. *Clin Infect Dis* 2002;34(3):412-6.
155. Buffington J, Reporter R, Lasker BA, et al. Investigation of an epidemic of invasive aspergillosis: utility of molecular typing with the use of random amplified polymorphic DNA probes. *Pediatr Infect Dis J* 1994;13(5):386-93.
156. Krasinski K, Holzman RS, Hanna B, Greco MA, Graff M, Bhogal M. Nosocomial fungal infection during hospital renovation. *Infect Control* 1985;6(7):278-82.
157. Humphreys H. Positive-pressure isolation and the prevention of invasive aspergillosis. What is the evidence? *J Hosp Infect* 2004;56(2):93-100.
158. Thio CL, Smith D, Merz WG, et al. Refinements of environmental assessment during an outbreak investigation of invasive aspergillosis in a leukemia and bone marrow transplant unit. *Infect Control Hosp Epidemiol* 2000;21(1):18-23.
159. Anaissie EJ, Stratton SL, Dignani MC, et al. Pathogenic Aspergillus species recovered from a hospital water system: a 3-year prospective study. *Clin Infect Dis* 2002;34(6):780-9.
160. CDC. Prevention of invasive group A streptococcal disease among household contacts of case patients and among postpartum and postsurgical patients: recommendations from the Centers for Disease Control and Prevention. *Clin Infect Dis* 2002;35 (8):950-9.
161. Gruteke P, van Belkum A, Schouls LM, et al. Outbreak of group A streptococci in a burn center: use of pheno- and genotypic procedures for strain tracking. *J Clin Microbiol* 1996;34(1):114-8.

162. Greene CM, Van Beneden CA, Javadi M, et al. Cluster of deaths from group A streptococcus in a long-term care facility--Georgia, 2001. *Am J Infect Control* 2005;33(2):108-13.
163. Sabria M, Campins M. Legionnaires' disease: update on epidemiology and management options. *Am J Respir Med* 2003;2(3):235-43.
164. Bille J, Marchetti O, Calandra T. Changing face of health-care associated fungal infections. *Curr Opin Infect Dis* 2005;18(4):314-9.
165. Hall IC, O'Toole E. Intestinal flora in newborn infants with a description of a new pathogenic anaerobe, *Bacillus difficilis*. *Am J Dis Child* 1935;49:390-402.
166. George WL, Sutter VL, Finegold SM. Antimicrobial agent-induced diarrhea--a bacterial disease. *J Infect Dis* 1977;136(6):822-8.
167. McFarland LV, Mulligan ME, Kwok RY, Stamm WE. Nosocomial acquisition of *Clostridium difficile* infection. *N Engl J Med* 1989;320(4):204-10.
168. Pepin J, Valiquette L, Alary ME, et al. *Clostridium difficile*-associated diarrhea in a region of Quebec from 1991 to 2003: a changing pattern of disease severity. *Cmaj* 2004;171(5):466-72.
169. 169. Agency HP. Outbreak of *Clostridium difficile* infection in a hospital in south east England. *Communicable Disease Report Weekly* 2005;31(24).
170. Koopmans M, Wilbrink B, Conyn M, et al. Transmission of H7N7 avian influenza A virus to human beings during a large outbreak in commercial poultry farms in the Netherlands. *Lancet* 2004;363(9409):587-93.
171. McDonald LC, Killgore GE, Thompson A, et al. An epidemic, toxin gene-variant strain of *Clostridium difficile*. *N Engl J Med* 2005;353(23):2433-41.
172. Loo VG, Poirier L, Miller MA, et al. A predominantly clonal multi-institutional outbreak of *Clostridium difficile*-associated diarrhea with high morbidity and mortality. *N Engl J Med* 2005;353(23):2442-9.
173. Warny M, Pepin J, Fang A, et al. Toxin production by an emerging strain of *Clostridium difficile* associated with outbreaks of severe disease in North America and Europe. *Lancet* 2005;366(9491):1079-84.
174. Layton BA ML, Gerding DN, Liedtke LA, Strausbaugh LJ. Perceived increases in the incidence and severity of *Clostridium difficile* disease: an emerging threat that continues to unfold. In: 15th Annual Scientific Meeting of the Society for Healthcare Epidemiology of America;. Los Angeles, CA.; 2005.
175. Sohn S, Climo M, Diekema D, et al. Varying rates of *Clostridium difficile*-associated diarrhea at prevention epicenter hospitals. *Infect Control Hosp Epidemiol* 2005;26(8):676-9.

176. IOM. Antimicrobial Resistance: Issues and Options. Workshop report. In: Harrison PF, Lederberg J, eds. Washington, DC: National Academy Press; 1998:8-74.
177. Shlaes DM, Gerding DN, John JF, Jr., et al. Society for Healthcare Epidemiology of America and Infectious Diseases Society of America Joint Committee on the Prevention of Antimicrobial Resistance: guidelines for the prevention of antimicrobial resistance in hospitals. *Infect Control Hosp Epidemiol* 1997;18(4):275-91.
178. Whitener CJ, Park SY, Browne FA, et al. Vancomycin-resistant *Staphylococcus aureus* in the absence of vancomycin exposure. *Clin Infect Dis* 2004;38(8):1049-55.
179. CDC. *Staphylococcus aureus* with reduced susceptibility to vancomycin-- United States. *MMWR Morb Mortal Wkly Rep* 1997;46 (33):765-6.
180. CDC. *Staphylococcus aureus* resistant to vancomycin--United States, 2002. *MMWR Morb Mortal Wkly Rep* 2002;51 (26):565-7.
181. CDC. Public Health Dispatch: Vancomycin-Resistant *Staphylococcus aureus* --- Pennsylvania, 2002. *MMWR - Morbidity & Mortality Weekly Report* 2002;51(40):902.
182. CDC. Vancomycin-resistant *Staphylococcus aureus*--New York, 2004. *MMWR Morb Mortal Wkly Rep* 2004;53(15):322-3.
183. Chang S, Sievert DM, Hageman JC, et al. Infection with vancomycin-resistant *Staphylococcus aureus* containing the vanA resistance gene. *N Engl J Med* 2003;348(14):1342-7.
184. Fridkin SK, Hageman J, McDougal LK, et al. Epidemiological and microbiological characterization of infections caused by *Staphylococcus aureus* with reduced susceptibility to vancomycin, United States, 1997-2001. *Clin Infect Dis* 2003;36(4):429-39.
185. Gold HS, Moellering RC, Jr. Antimicrobial-drug resistance. *N Engl J Med* 1996;335(19):1445-53.
186. Hageman JC, Pegues DA, Jepson C, et al. Vancomycin-intermediate *Staphylococcus aureus* in a home health-care patient. *Emerg Infect Dis* 2001;7(6):1023-5.
187. Harwell JI, Brown RB. The drug-resistant pneumococcus: clinical relevance, therapy, and prevention. *Chest* 2000;117(2):530-41.
188. Jones RN. Resistance patterns among nosocomial pathogens: trends over the past few years. *Chest* 2001;119(2 Suppl):397S-404S.
189. Murray BE. Vancomycin-resistant enterococcal infections. *N Engl J Med* 2000;342(10):710-21.
190. Neuhauser MM, Weinstein RA, Rydman R, Danziger LH, Karam G, Quinn JP. Antibiotic resistance among gram-negative bacilli in US

- intensive care units: implications for fluoroquinolone use. *JAMA* 2003;289(7):885-8.
191. Pitout JD, Sanders CC, Sanders WE, Jr. Antimicrobial resistance with focus on beta-lactam resistance in gram-negative bacilli. *Am J Med* 1997;103(1):51-9.
 192. Rotun SS, McMath V, Schoonmaker DJ, et al. *Staphylococcus aureus* with reduced susceptibility to vancomycin isolated from a patient with fatal bacteremia. *Emerg Infect Dis* 1999;5(1):147-9.
 193. Smith TL, Pearson ML, Wilcox KR, et al. Emergence of vancomycin resistance in *Staphylococcus aureus*. Glycopeptide-Intermediate *Staphylococcus aureus* Working Group. *N Engl J Med* 1999;340(7):493-501.
 194. Srinivasan A, Dick JD, Perl TM. Vancomycin resistance in staphylococci. *Clin Microbiol Rev* 2002;15(3):430-8.
 195. Hyle EP, Lipworth AD, Zaoutis TE, et al. Risk Factors for Increasing Multidrug Resistance among Extended-Spectrum β -lactamase-Lactamase-Producing *Escheria coli* and *Klebsiella* species *Clin Infect Dis* 2005;40(9):1317-24.
 196. Gleich S, Morad Y, Echague R, et al. *Streptococcus pneumoniae* serotype 4 outbreak in a home for the aged: report and review of recent outbreaks. *Infect Control Hosp Epidemiol* 2000;21:711.
 197. Fry AM, Udeagu CC, Soriano-Gabarro M, et al. Persistence of fluoroquinolone-resistant, multidrug-resistant *Streptococcus pneumoniae* in a long-term-care facility: efforts to reduce intrafacility transmission. *Infect Control Hosp Epidemiol* 2005;26(3):239-47.
 198. Carter RJ, Sorenson G, Heffernan R, et al. Failure to control an outbreak of multidrug-resistant *Streptococcus pneumoniae* in a long-term-care facility: emergence and ongoing transmission of a fluoroquinolone-resistant strain. *Infect Control Hosp Epidemiol* 2005;26(3):248-55.
 199. Blok HE, Troelstra A, Kamp-Hopmans TE, et al. Role of healthcare workers in outbreaks of methicillin-resistant *Staphylococcus aureus*: a 10-year evaluation from a Dutch university hospital. *Infect Control Hosp Epidemiol* 2003;24(9):679-85.
 200. Muto CA, Jernigan JA, Ostrowsky BE, et al. SHEA guideline for preventing nosocomial transmission of multidrug-resistant strains of *Staphylococcus aureus* and enterococcus. *Infect Control Hosp Epidemiol* 2003;24(5):362-86.
 201. Tammelin A, Klotz F, Hambraeus A, Stahle E, Ransjo U. Nasal and hand carriage of *Staphylococcus aureus* in staff at a Department for Thoracic and Cardiovascular Surgery: endogenous or exogenous source? *Infect Control Hosp Epidemiol* 2003;24(9):686-9.

202. CDC. Biological and chemical terrorism: strategic plan for preparedness and response. Recommendations of the CDC Strategic Planning Workgroup. MMWR Recomm Rep 2000;49(RR-4):1-14.
203. Inglesby TV, O'Toole T, Henderson DA, et al. Anthrax as a biological weapon, 2002: updated recommendations for management. JAMA 2002;287(17):2236-52.
204. Henderson DA, Inglesby TV, Bartlett JG, et al. Smallpox as a biological weapon: medical and public health management. Working Group on Civilian Biodefense. JAMA 1999;281(22):2127-37.
205. www.bt.cdc.gov/agent/smallpox/
206. www.who.int/csr/disease/smallpox/en/
207. Kool JL. Risk of person-to-person transmission of pneumonic plague. Clin Infect Dis 2005;40(8):1166-72.
208. Inglesby TV, Dennis DT, Henderson DA, et al. Plague as a biological weapon: medical and public health management. Working Group on Civilian Biodefense. JAMA 2000;283(17):2281-90.
209. Arnon SS, Schechter R, Inglesby TV, et al. Botulinum toxin as a biological weapon: medical and public health management. JAMA 2001;285(8):1059-70.
210. Dennis DT, Inglesby TV, Henderson DA, et al. Tularemia as a biological weapon: medical and public health management. JAMA 2001;285(21):2763-73.
211. CDC. Notice to Readers Update: Management of Patients with Suspected Viral Hemorrhagic Fever -- United States. MMWR Recomm Rep 1995;44(25):475-9.
212. Borio L, Inglesby T, Peters CJ, et al. Hemorrhagic fever viruses as biological weapons: medical and public health management. JAMA 2002;287(18):2391-405.
213. Neff JM, Lane JM, Fulginiti VA, Henderson DA. Contact vaccinia--transmission of vaccinia from smallpox vaccination. JAMA 2002;288(15):1901-5.
214. Sepkowitz KA. How contagious is vaccinia? N Engl J Med 2003;348(5):439-46.
215. Lane JM, Fulginiti VA. Transmission of vaccinia virus and rationale for measures for prevention. Clin Infect Dis 2003;37(2):281-4.
216. CDC. Smallpox Vaccination and Adverse Reactions: Guidance for Clinicians. MMWR - Morbidity & Mortality Weekly Report 2003;52(RR04):1-28.

217. Fulginiti VA, Papier A, Lane JM, Neff JM, Henderson DA. Smallpox vaccination: a review, part II. Adverse events. *Clin Infect Dis* 2003;37(2):251-71.
218. www.smallpox.mil./event/SPSafetySum.asp
219. CDC. Update: adverse events following civilian smallpox vaccination--United States, 2003. *MMWR Morb Mortal Wkly Rep* 2003;52(18):419-20.
220. CDC. Secondary and tertiary transfer of vaccinia virus among U.U. military personnel--United States and worldwide, 2002-2004. *MMWR Morb Mortal Wkly Rep* 2004;53(5):103-5.
221. Talbot TR, Ziel E, Doersam JK, LaFleur B, Tollefson S, Edwards KM. Risk of vaccinia transfer to the hands of vaccinated persons after smallpox immunization. *Clin Infect Dis* 2004;38(4):536-41.
222. Hepburn MJ, Dooley DP, Murray CK, et al. Frequency of vaccinia virus isolation on semipermeable versus nonocclusive dressings covering smallpox vaccination sites in hospital personnel. *Am J Infect Control* 2004;32(3):126-30.
223. Waibel KH, Ager EP, Topolski RL, Walsh DS. Randomized trial comparing vaccinia on the external surfaces of 3 conventional bandages applied to smallpox vaccination sites in primary vaccinees. *Clin Infect Dis* 2004;39(7):1004-7.
224. Tenorio AR, Peoples M, Patri M, et al. Quantitative Vaccinia Cultures and Evolution of Vaccinia-Specific CD8+ Cytotoxic T-lymphocytes (CTL) Responses in Revaccinees. Abstract #823, 41st Annual Meeting IDSA, October 2003, San Diego 2003.
225. Wharton M, Strikas RA, Harpaz R, et al. Recommendations for using smallpox vaccine in a pre-event vaccination program. Supplemental recommendations of the Advisory Committee on Immunization Practices (ACIP) and the Healthcare Infection Control Practices Advisory Committee (HICPAC). *MMWR Recomm Rep* 2003;52(RR-7):1-16.
226. CDC. Surveillance for Creutzfeldt-Jakob Disease -- United States. *MMWR* 1996;45(31):665-8.
227. Johnson RT, Gibbs CJ, Jr. Creutzfeldt-Jakob disease and related transmissible spongiform encephalopathies. *N Engl J Med* 1998;339(27):1994-2004.
228. Brown P, Gajdusek DC, Gibbs CJ, Jr., Asher DM. Potential epidemic of Creutzfeldt-Jakob disease from human growth hormone therapy. *N Engl J Med* 1985;313(12):728-31.
229. Frasier SD, Foley TP, Jr. Clinical review 58: Creutzfeldt-Jakob disease in recipients of pituitary hormones. *J Clin Endocrinol Metab* 1994;78(6):1277-9.

230. CDC. Update: Creutzfeldt-Jakob disease associated with cadaveric dura mater grafts--Japan, 1979-2003. *MMWR Morb Mortal Wkly Rep* 2003;52(48):1179-81.
231. Lang CJ, Heckmann JG, Neundorfer B. Creutzfeldt-Jakob disease via dural and corneal transplants. *J Neurol Sci* 1998;160(2):128-39.
232. el Hachimi KH, Chaunu MP, Cervenakova L, Brown P, Foncin JF. Putative neurosurgical transmission of Creutzfeldt-Jakob disease with analysis of donor and recipient: agent strains. *C R Acad Sci III* 1997;320(4):319-28.
233. Will RG, Matthews WB. Evidence for case-to-case transmission of Creutzfeldt-Jakob disease. *J Neurol Neurosurg Psychiatry* 1982;45(3):235-8.
234. Bernoulli C, Siegfried J, Baumgartner G, et al. Danger of accidental person-to-person transmission of Creutzfeldt-Jakob disease by surgery. *Lancet* 1977;1(8009):478-9.
235. Rutala WA, Weber DJ. Creutzfeldt-Jakob disease: recommendations for disinfection and sterilization. *Clin Infect Dis* 2001;32(9):1348-56.
236. Belay ED, Maddox RA, Williams ES, Miller MW, Gambetti P, Schonberger LB. Chronic wasting disease and potential transmission to humans. *Emerg Infect Dis* 2004;10(6):977-84.
237. Collinge J, Sidle KC, Meads J, Ironside J, Hill AF. Molecular analysis of prion strain variation and the aetiology of 'new variant' CJD. *Nature* 1996;383(6602):685-90.
238. Belay ED, Schonberger LB. The public health impact of prion diseases. *Annu Rev Public Health* 2005;26:191-212.
239. Belay ED, Schonberger LB. Variant Creutzfeldt-Jakob disease and bovine spongiform encephalopathy. *Clin Lab Med* 2002;22(4):849-62, v-vi.
240. Hill AF, Butterworth RJ, Joiner S, et al. Investigation of variant Creutzfeldt-Jakob disease and other human prion diseases with tonsil biopsy samples. *Lancet* 1999;353(9148):183-9.
241. Evatt B. Creutzfeldt-Jakob disease and haemophilia: assessment of risk. *Haemophilia* 2000;6 Suppl 1:94-9.
242. Chamberland ME. Emerging infectious agents: do they pose a risk to the safety of transfused blood and blood products? *Clin Infect Dis* 2002;34(6):797-805.
243. www.fda.gov/cber/gdlns/cjdvcjd.htm
244. Llewelyn CA, Hewitt PE, Knight RS, et al. Possible transmission of variant Creutzfeldt-Jakob disease by blood transfusion. *Lancet* 2004;363(9407):417-21.

245. Peden AH, Head MW, Ritchie DL, Bell JE, Ironside JW. Preclinical vCJD after blood transfusion in a PRNP codon 129 heterozygous patient. *Lancet* 2004;364(9433):527-9.
246. Brown P. Guidelines for high risk autopsy cases: special precautions for Creutzfeldt-Jakob Disease. In: Hutchins G, ed. *Autopsy Performance and Reporting*, Northfield, Ill.: College of American Pathologists 1990:68-74.
247. Drosten C, Gunther S, Preiser W, et al. Identification of a novel coronavirus in patients with severe acute respiratory syndrome. *N Engl J Med* 2003;348(20):1967-76.
248. Ksiazek TG, Erdman D, Goldsmith CS, et al. A novel coronavirus associated with severe acute respiratory syndrome. *N Engl J Med* 2003;348(20):1953-66.
249. Chan WM, Kwan YW, Wan HS, Leung CW, Chiu MC. Epidemiologic linkage and public health implication of a cluster of severe acute respiratory syndrome in an extended family. *Pediatr Infect Dis J* 2004;23(12):1156-9.
250. Leung CW, Kwan YW, Ko PW, et al. Severe acute respiratory syndrome among children. *Pediatrics* 2004;113(6):e535-43.
251. Bitnun A, Allen U, Heurter H, et al. Children hospitalized with severe acute respiratory syndrome-related illness in Toronto. *Pediatrics* 2003;112(4):e261.
252. Chow KY, Lee CE, Ling ML, Heng DM, Yap SG. Outbreak of severe acute respiratory syndrome in a tertiary hospital in Singapore, linked to an index patient with atypical presentation: epidemiological study. *Bmj* 2004;328(7433):195.
253. Shen Z, Ning F, Zhou W, et al. Superspreading SARS events, Beijing, 2003. *Emerg Infect Dis* 2004;10(2):256-60.
254. Chen Y-C, Huang L-M, Chan C-C, et al. SARS in Hospital Emergency Room. *Emerg Infect Dis* 2004;10:782-8.
255. Chung Y-C, Huang L-M, Chan C-C, et al. SARS in Hospital Emergency Room. *Emerg Infect Dis* 2004;10:782-8.
256. Gamage B, Moore D, Copes R, Yassi A, Bryce E. Protecting health care workers from SARS and other respiratory pathogens: a review of the infection control literature. *Am J Infect Control* 2005;33(2):114-21.
257. Moore D, Gamage B, Bryce E, Copes R, Yassi A. Protecting health care workers from SARS and other respiratory pathogens: organizational and individual factors that affect adherence to infection control guidelines. *Am J Infect Control* 2005;33(2):88-96.
258. Dowell SF, Simmerman JM, Erdman DD, et al. Severe acute respiratory syndrome coronavirus on hospital surfaces. *Clin Infect Dis* 2004;39(5):652-7.

259. Public Health Guidance for Community-Level Preparedness and Response to Severe Acute Respiratory Syndrome (SARS). 2004. (Accessed at <http://www.cdc.gov/ncidod/sars/guidance/I/occupational.htm>.)
260. Le DH, Bloom SA, Nguyen QH, et al. Lack of SARS transmission among public hospital workers, Vietnam. *Emerg Infect Dis* 2004;10(2):265-8.
261. Lim PL, Kurup A, Gopalakrishna G, et al. Laboratory-acquired severe acute respiratory syndrome. *N Engl J Med* 2004;350(17):1740-5.
262. CDC. www.cdc.gov/ncidod/sars. 2003.
263. Reed KD, Melski JW, Graham MB, et al. The detection of monkeypox in humans in the Western Hemisphere. *N Engl J Med* 2004;350(4):342-50.
264. Anderson MG, Frenkel LD, Homann S, Guffey J. A case of severe monkeypox virus disease in an American child: emerging infections and changing professional values. *Pediatr Infect Dis J* 2003;22:1093-6.
265. Jezek Z, Fenner F. Human monkey pox. In: Melnick JL ed. *Monographs in virology*. Vol. 17. Basel, Switzerland: S Karger AG. 1988:81-102.
266. Marennikova SS, Jezek Z, Szczeniowski M, Mbudi PM, Vernette M. [Contagiousness of monkey pox for humans: results of an investigation of 2 outbreaks of the infection in Zaire]. *Zh Mikrobiol Epidemiol Immunobiol* 1985(8):38-43.
267. Jezek Z, Arita I, Mutombo M, Dunn C, Nakano JH, Szczeniowski M. Four generations of probable person-to-person transmission of human monkeypox. *Am J Epidemiol* 1986;123(6):1004-12.
268. Learned LA, Reynolds MG, Wasswa DW, et al. Extended interhuman transmission of monkeypox in a hospital community in the Republic of the Congo, 2003. *Am J Trop Med Hyg* 2005;73(2):428-34.
269. Fleischauer AT, Kile JC, Davidson M, et al. Evaluation of human-to-human transmission of monkeypox from infected patients to health care workers. *Clin Infect Dis* 2005;40(5):689-94.
270. Likos AM, Sammons SA, Olson VA, et al. A tale of two clades: monkeypox viruses. *J Gen Virol* 2005;86(Pt 10):2661-72.
271. Fine PE, Jezek Z, Grab B, Dixon H. The transmission potential of monkeypox virus in human populations. *Int J Epidemiol* 1988;17(3):643-50.
272. Jezek Z, Grab B, Paluku KM, Szczeniowski MV. Human monkeypox: disease pattern, incidence and attack rates in a rural area of northern Zaire. *Trop Geogr Med* 1988;40(2):73-83.
273. CDC. "Norwalk-Like Viruses": Public Health Consequences and Outbreak Management. *MMWR - Morbidity & Mortality Weekly Report* 2001;50 (RR-09)(June):1-18.

274. Evans MR, Meldrum R, Lane W, et al. An outbreak of viral gastroenteritis following environmental contamination at a concert hall. *Epidemiol Infect* 2002;129(2):355-60.
275. Wu HM, Fornek M, Kellogg JS, et al. A Norovirus Outbreak at a Long-Term-Care Facility: The Role of Environmental Surface Contamination. *Infect Control Hosp Epidemiol* 2005;26(10):802-10.
276. Duizer E, Schwab KJ, Neill FH, Atmar RL, Koopmans MP, Estes MK. Laboratory efforts to cultivate noroviruses. *J Gen Virol* 2004;85(Pt 1):79-87.
277. Zingg W, Colombo C, Jucker T, Bossart W, Ruef C. Impact of an outbreak of norovirus infection on hospital resources. *Infect Control Hosp Epidemiol* 2005;26:263-7.
278. Calderon-Margalit R, Sheffer R, Halperin T, Orr N, Cohen D, Shohat T. A large-scale gastroenteritis outbreak associated with Norovirus in nursing homes. *Epidemiol Infect* 2005;133(1):35-40.
279. Marx A, Shay DK, Noel JS, et al. An outbreak of acute gastroenteritis in a geriatric long-term-care facility: combined application of epidemiological and molecular diagnostic methods. *Infect Control Hosp Epidemiol* 1999;20(5):306-11.
280. Gellert GA, Waterman SH, Ewert D, et al. An outbreak of acute gastroenteritis caused by a small round structured virus in a geriatric convalescent facility. *Infect Control Hosp Epidemiol* 1990;11(9):459-64.
281. Cooper E, Blamey S. A Norovirus Gastroenteritis Epidemic in a Long-Term-Care Facility. *Infect Control Hosp Epidemiol* 2005;26(3):256.
282. Navarro G, Sala RM, Segura F, et al. An Outbreak of Norovirus Infection in a Long-Term-Care. *Infect Control Hosp Epidemiol* 2005;26(3):259.
283. Green KY, Belliot G, Taylor JL, et al. A predominant role for Norwalk-like viruses as agents of epidemic gastroenteritis in Maryland nursing homes for the elderly. *J Infect Dis* 2002;185(2):133-46.
284. Widdowson MA, Cramer EH, Hadley L, et al. Outbreaks of acute gastroenteritis on cruise ships and on land: identification of a predominant circulating strain of norovirus--United States, 2002. *J Infect Dis* 2004;190(1):27-36.
285. CDC. Outbreaks of gastroenteritis associated with noroviruses on cruise ships--United States, 2002. *MMWR Morb Mortal Wkly Rep* 2002;51(49):1112-5.
286. CDC. Norovirus outbreak among evacuees from hurricane Katrina--Houston, Texas, September 2005. *MMWR Morb Mortal Wkly Rep* 2005;54(40):1016-8

287. Mattner F, Mattner L, Borck HU, Gastmeier P. Evaluation of the Impact of the Source (Patient Versus Staff) on Nosocomial Norovirus Outbreak Severity. *Infect Control Hosp Epidemiol* 2005;26(3):268-72.
288. Isakbaeva ET, Bulens SN, Beard RS, et al. Norovirus and child care: challenges in outbreak control. *Pediatr Infect Dis J* 2005;24(6):561-3.
289. Kapikian AZ, Estes MK, Chanock RM. Norwalk group of viruses. In: Fields BN, Knipe DM, Howley PM, eds. *Fields virology*. 3rd ed. Philadelphia, PA: Lippincott-Raven. 1996:783-810.
290. Duizer E, Bijkerk P, Rockx B, De Groot A, Twisk F, Koopmans M. Inactivation of caliciviruses. *Appl Environ Microbiol* 2004;70(8):4538-43.
291. Doultree JC, Druce JD, Birch CJ, Bowden DS, Marshall JA. Inactivation of feline calicivirus, a Norwalk virus surrogate. *J Hosp Infect* 1999;41(1):51-7.
292. Barker J, Vipond IB, Bloomfield SF. Effects of cleaning and disinfection in reducing the spread of Norovirus contamination via environmental surfaces. *J Hosp Infect* 2004;58(1):42-9.
293. Gulati BR, Allwood PB, Hedberg CW, Goyal SM. Efficacy of commonly used disinfectants for the inactivation of calicivirus on strawberry, lettuce, and a food-contact surface. *J Food Prot* 2001;64(9):1430-4.
294. Gehrke C, Steinmann J, Goroncy-Bermes P. Inactivation of feline calicivirus, a surrogate of norovirus (formerly Norwalk-like viruses), by different types of alcohol in vitro and in vivo. *J Hosp Infect* 2004;56(1):49-55.
295. Hutson AM, Atmar RL, Graham DY, Estes MK. Norwalk virus infection and disease is associated with ABO histo-blood group type. *J Infect Dis* 2002;185(9):1335-7.
296. National Center for Infectious Diseases - Division of Viral and Rickettsial Diseases. (www.cdc.gov/ncidod/dvrd/index.htm)
297. LeDuc JW. Epidemiology of hemorrhagic fever viruses. *Rev Infect Dis* 1989;11 Suppl 4:S730-5.
298. Roels TH, Bloom AS, Buffington J, et al. Ebola hemorrhagic fever, Kikwit, Democratic Republic of the Congo, 1995: risk factors for patients without a reported exposure. *J Infect Dis* 1999 Feb;179 Suppl 1:S92-7.
299. Suleiman MN, Muscat-Baron JM, Harries JR, et al. Congo/Crimean haemorrhagic fever in Dubai. An outbreak at the Rashid Hospital. *Lancet* 1980;2(8201):939-41.
300. Monath TP, Mertens PE, Patton R, et al. A hospital epidemic of Lassa fever in Zorzor, Liberia, March-April 1972. *Am J Trop Med Hyg* 2005;54(40):1016-9

301. Dowell SF, Mukunu R, Ksiazek TG, Khan AS, Rollin PE, Peters CJ. Transmission of Ebola hemorrhagic fever: a study of risk factors in family members, Kikwit, Democratic Republic of the Congo, 1995. *J Infect Dis* 1999 Feb;179 Suppl 1:S87-91.
302. Peters CJ. Marburg and Ebola--arming ourselves against the deadly filoviruses. *N Engl J Med* 2005;352(25):2571-3.
303. Ebola haemorrhagic fever in Zaire, 1976. *Bull World Health Organ* 1978;56(2):271-93.
304. Emond RT, Evans B, Bowen ET, Lloyd G. A case of Ebola virus infection. *Br Med J* 1977;2(6086):541-4.
305. Zaki SR, Shieh WJ, Greer PW, et al. A novel immunohistochemical assay for the detection of Ebola virus in skin: implications for diagnosis, spread, and surveillance of Ebola hemorrhagic fever. Commission de Lutte contre les Epidemies a Kikwit. *J Infect Dis* 1999;179 Suppl 1:S36-47.
306. Khan AS, Tshioko FK, Heymann DL, et al. The reemergence of Ebola hemorrhagic fever, Democratic Republic of the Congo, 1995. Commission de Lutte contre les Epidemies a Kikwit. *J Infect Dis* 1999;179 Suppl 1:S76-86.
307. Muyembe-Tamfum JJ, Kipasa M, Kiyungu C, Colebunders R. Ebola outbreak in Kikwit, Democratic Republic of the Congo: discovery and control measures. *J Infect Dis* 1999;179 Suppl 1:S259-62.
308. Haas WH, Breuer T, Pfaff G, et al. Imported Lassa fever in Germany: surveillance and management of contact persons. *Clin Infect Dis* 2003;36(10):1254-8.
309. Simpson DI. Marburg agent disease: in monkeys. *Trans R Soc Trop Med Hyg* 1969;63(3):303-9.
310. Jaax NK, Davis KJ, Geisbert TJ, et al. Lethal experimental infection of rhesus monkeys with Ebola-Zaire (Mayinga) virus by the oral and conjunctival route of exposure. *Arch Pathol Lab Med* 1996;120(2):140-55.
311. Stephenson EH, Larson EW, Dominik JW. Effect of environmental factors on aerosol-induced Lassa virus infection. *J Med Virol* 1984;14(4):295-303.
312. Johnson E, Jaax N, White J, Jahrling P. Lethal experimental infections of rhesus monkeys by aerosolized Ebola virus. *Int J Exp Pathol* 1995;76(4):227-36.
313. Jaax N, Jahrling P, Geisbert T, et al. Transmission of Ebola virus (Zaire strain) to uninfected control monkeys in a biocontainment laboratory. *Lancet* 1995;346(8991-8992):1669-71.
314. www.bt.cdc.gov/agent/vhf/.

315. Nguyen GT, Proctor SE, Sinkowitz-Cochran RL, Garrett DO, Jarvis WR. Status of infection surveillance and control programs in the United States, 1992-1996. Association for Professionals in Infection Control and Epidemiology, Inc. *Am J Infect Control* 2000;28(6):392-400.
316. Richards C, Emori TG, Edwards J, Fridkin S, Tolson J, Gaynes R. Characteristics of hospitals and infection control professionals participating in the National Nosocomial Infections Surveillance System 1999. *Am J Infect Control* 2001;29(6):400-3.
317. Wenzel RP, Thompson RL, Landry SM, et al. Hospital-acquired infections in intensive care unit patients: an overview with emphasis on epidemics. *Infect Control* 1983;4(5):371-5.
318. Wenzel RP, Gennings C. Bloodstream infections due to *Candida* species in the intensive care unit: identifying especially high-risk patients to determine prevention strategies. *Clin Infect Dis* 2005;41 Suppl 6:S389-93.
319. San Miguel LG, Cobo J, Otheo E, Sanchez-Sousa A, Abaira V, Moreno S. Secular trends of candidemia in a large tertiary-care hospital from 1988 to 2000: emergence of *Candida parapsilosis*. *Infect Control Hosp Epidemiol* 2005;26(6):548-52.
320. NNIS. National Nosocomial Infections Surveillance (NNIS) System Report, data summary from January 1992 through June 2004, issued October 2004. *Am J Infect Control* 2004;32(8):470-85.
321. Richards MJ, Edwards JR, Culver DH, Gaynes RP. Nosocomial infections in medical intensive care units in the United States. National Nosocomial Infections Surveillance System. *Crit Care Med* 1999;27(5):887-92.
322. Richards MJ, Edwards JR, Culver DH, Gaynes RP. Nosocomial infections in combined medical-surgical intensive care units in the United States. *Infect Control Hosp Epidemiol* 2000;21(8):510-5.
323. Hugonnet S, Eggimann P, Borst F, Maricot P, Chevrolet JC, Pittet D. Impact of ventilator-associated pneumonia on resource utilization and patient outcome. *Infect Control Hosp Epidemiol* 2004;25(12):1090-6.
324. O'Neill JM, Schutze GE, Heulitt MJ, Simpson PM, Taylor BJ. Nosocomial infections during extracorporeal membrane oxygenation. *Intensive Care Med* 2001;27(8):1247-53.
325. Villegas MV, Hartstein AI. *Acinetobacter* Outbreaks, 1977–2000. *Infect Control Hosp Epidemiol* 2003;24(4):284-95.
326. Gordon SM, Schmitt SK, Jacobs M, et al. Nosocomial bloodstream infections in patients with implantable left ventricular assist devices. *Ann Thorac Surg* 2001;72(3):725-30.
327. Giamarellou H. Nosocomial cardiac infections. *J Hosp Infect* 2002;50(2):91-105.

328. Fridkin SK. Increasing prevalence of antimicrobial resistance in intensive care units. *Crit Care Med* 2001;29(4 Suppl):N64-8.
329. Kollef MH, Fraser VJ. Antibiotic resistance in the intensive care unit. *Ann Intern Med* 2001;134(4):298-314.
330. Fridkin SK, Edwards JR, Courval JM, et al. The effect of vancomycin and third-generation cephalosporins on prevalence of vancomycin-resistant enterococci in 126 U.S. adult intensive care units. *Ann Intern Med* 2001;135(3):175-83.
331. Crnich CJ, Safdar N, Maki DG. The role of the intensive care unit environment in the pathogenesis and prevention of ventilator-associated pneumonia. *Respir Care* 2005;50(6):813-36; discussion 36-8.
332. Knaus WA, Wagner DP, Zimmerman JE, Draper EA. Variations in mortality and length of stay in intensive care units. *Ann Intern Med* 1993;118(10):753-61.
333. Villarino ME, Stevens LE, Schable B, et al. Risk factors for epidemic *Xanthomonas maltophilia* infection/colonization in intensive care unit patients. *Infect Control Hosp Epidemiol* 1992;13(4):201-6.
334. Sanchez V, Vazquez JA, Barth-Jones D, Dembry L, Sobel JD, Zervos MJ. Nosocomial acquisition of *Candida parapsilosis*: an epidemiologic study. *Am J Med* 1993;94(6):577-82.
335. Husni RN, Goldstein LS, Arroliga AC, et al. Risk factors for an outbreak of multi-drug-resistant *Acinetobacter* nosocomial pneumonia among intubated patients. *Chest* 1999;115(5):1378-82.
336. McDonald LC, Walker M, Carson L, et al. Outbreak of *Acinetobacter* spp. bloodstream infections in a nursery associated with contaminated aerosols and air conditioners. *Pediatr Infect Dis J* 1998;17(8):716-22.
337. Trick WE, Kioski CM, Howard KM, et al. Outbreak of *Pseudomonas aeruginosa* ventriculitis among patients in a neurosurgical intensive care unit. *Infect Control Hosp Epidemiol* 2000;21(3):204-8.
338. Guidry GG, Black-Payne CA, Payne DK, Jamison RM, George RB, Bocchini JA, Jr. Respiratory syncytial virus infection among intubated adults in a university medical intensive care unit. *Chest* 1991;100(5):1377-84.
339. Wurtz R, Karajovic M, Dacumos E, Jovanovic B, Hanumadass M. Nosocomial infections in a burn intensive care unit. *Burns* 1995;21(3):181-4.
340. Rodgers GL, Mortensen J, Fisher MC, Lo A, Cresswell A, Long SS. Predictors of infectious complications after burn injuries in children. *Pediatr Infect Dis J* 2000;19(10):990-5.
341. Pruitt BA, Jr., McManus AT, Kim SH, Goodwin CW. Burn wound infections: current status. *World J Surg* 1998;22(2):135-45.

342. Weber J, ed. *Epidemiology of Infections and Strategies for Control in Burn Care and Therapy*. St. Louis: Mosby, Inc.; 1998.
343. Heggors JP, McCoy L, Reisner B, Smith M, Edgar P, Ramirez RJ. Alternate antimicrobial therapy for vancomycin-resistant enterococci burn wound infections. *J Burn Care Rehabil* 1998;19(5):399-403.
344. Sheridan RL, Weber J, Benjamin J, Pasternack MS, Tompkins RG. Control of methicillin-resistant *Staphylococcus aureus* in a pediatric burn unit. *Am J Infect Control* 1994;22(6):340-5.
345. Matsumura H, Yoshizawa N, Narumi A, Harunari N, Sugamata A, Watanabe K. Effective control of methicillin-resistant *Staphylococcus aureus* in a burn unit. *Burns* 1996;22(4):283-6.
346. McGregor JC. Profile of the first four years of the Regional Burn Unit based at St. John's Hospital, West Lothian (1992-1996). *J R Coll Surg Edinb* 1998;43(1):45-8.
347. Desai MH, Rutan RL, Heggors JP, Herndon DN. Candida infection with and without nystatin prophylaxis. A 11-year experience with patients with burn injury. *Arch Surg* 1992;127(2):159-62.
348. Ekenna O, Sherertz RJ, Bingham H. Natural history of bloodstream infections in a burn patient population: the importance of candidemia. *Am J Infect Control* 1993;21(4):189-95.
349. Bowser-Wallace BH, Graves DB, Caldwell FT. An epidemiological profile and trend analysis of wound flora in burned children: 7 years' experience. *Burns Incl Therm Inj* 1984;11(1):16-25.
350. Tredget EE, Shankowsky HA, Rennie R, Burrell RE, Logsetty S. *Pseudomonas* infections in the thermally injured patient. *Burns* 2004;30(1):3-26.
351. Edgar P, Mlcak R, Desai M, Linares HA, Phillips LG, Heggors JP. Containment of a multiresistant *Serratia marcescens* outbreak. *Burns* 1997;23(1):15-8.
352. Embil JM, McLeod JA, Al-Barrak AM, et al. An outbreak of methicillin resistant *Staphylococcus aureus* on a burn unit: potential role of contaminated hydrotherapy equipment. *Burns* 2001;27(7):681-8.
353. Meier PA, Carter CD, Wallace SE, Hollis RJ, Pfaller MA, Herwaldt LA. A prolonged outbreak of methicillin-resistant *Staphylococcus aureus* in the burn unit of a tertiary medical center. *Infect Control Hosp Epidemiol* 1996;17(12):798-802.
354. Snyder LL, Wiebelhaus P, Boon SE, Morin RA, Goering R. Methicillin-resistant *Staphylococcus aureus* eradication in a burn center. *J Burn Care Rehabil* 1993;14(2 Pt 1):164-8.
355. May AK, Melton SM, McGwin G, Cross JM, Moser SA, Rue LW. Reduction of vancomycin-resistant enterococcal infections by limitation of

- broad-spectrum cephalosporin use in a trauma and burn intensive care unit. *Shock* 2000;14(3):259-64.
356. Sheridan RL, Weber JM, Budkevich LG, Tompkins RG. Candidemia in the pediatric patient with burns. *J Burn Care Rehabil* 1995;16(4):440-3.
357. Mayhall CG. The epidemiology of burn wound infections: then and now. *Clin Infect Dis* 2003;37(4):543-50.
358. McManus AT, Mason AD, Jr., McManus WF, Pruitt BA, Jr. A decade of reduced gram-negative infections and mortality associated with improved isolation of burned patients. *Arch Surg* 1994;129(12):1306-9.
359. Bryce EA, Walker M, Scharf S, et al. An outbreak of cutaneous aspergillosis in a tertiary-care hospital. *Infect Control Hosp Epidemiol* 1996;17(3):170-2.
360. Levenson C, Wohlford P, Djou J, Evans S, Zawacki B. Preventing postoperative burn wound aspergillosis. *J Burn Care Rehabil* 1991;12(2):132-5.
361. Tredget EE, Shankowsky HA, Joffe AM, et al. Epidemiology of infections with *Pseudomonas aeruginosa* in burn patients: the role of hydrotherapy. *Clin Infect Dis* 1992;15(6):941-9.
362. Wisplinghoff H, Perbix W, Seifert H. Risk factors for nosocomial bloodstream infections due to *Acinetobacter baumannii*: a case-control study of adult burn patients. *Clin Infect Dis* 1999;28(1):59-66.
363. Weber JM, Sheridan RL, Schulz JT, Tompkins RG, Ryan CM. Effectiveness of bacteria-controlled nursing units in preventing cross-colonization with resistant bacteria in severely burned children. *Infect Control Hosp Epidemiol* 2002;23(9):549-51.
364. Bayat A, Shaaban H, Dodgson A, Dunn KW. Implications for Burns Unit design following outbreak of multi-resistant *Acinetobacter* infection in ICU and Burns Unit. *Burns* 2003;29(4):303-6.
365. Lee JJ, Marvin JA, Heimbach DM, Grube BJ, Engrav LH. Infection control in a burn center. *J Burn Care Rehabil* 1990;11(6):575-80.
366. Raymond J, Aujard Y. Nosocomial infections in pediatric patients: a European, multicenter prospective study. European Study Group. *Infect Control Hosp Epidemiol* 2000;21(4):260-3.
367. Campins M, Vaque J, Rossello J, et al. Nosocomial infections in pediatric patients: a prevalence study in Spanish hospitals. EPINE Working Group. *Am J Infect Control* 1993;21(2):58-63.
368. Allen U, Ford-Jones EL. Nosocomial infections in the pediatric patient: an update. *Am J Infect Control* 1990;18(3):176-93.

369. Grohskopf LA, Sinkowitz-Cochran RL, Garrett DO, et al. A national point-prevalence survey of pediatric intensive care unit-acquired infections in the United States. *J Pediatr* 2002;140(4):432-8.
370. Sohn AH, Garrett DO, Sinkowitz-Cochran RL, et al. Prevalence of nosocomial infections in neonatal intensive care unit patients: Results from the first national point-prevalence survey. *J Pediatr* 2001;139(6):821-7.
371. Gaynes RP, Edwards JR, Jarvis WR, Culver DH, Tolson JS, Martone WJ. Nosocomial infections among neonates in high-risk nurseries in the United States. National Nosocomial Infections Surveillance System. *Pediatrics* 1996;98(3 Pt 1):357-61.
372. Richards MJ, Edwards JR, Culver DH, Gaynes RP. Nosocomial infections in pediatric intensive care units in the United States. National Nosocomial Infections Surveillance System. *Pediatrics* 1999;103(4):e39.
373. Maltezou HC, Drancourt M. Nosocomial influenza in children. *J Hosp Infect* 2003;55(2):83-91.
374. Moisiuk SE, Robson D, Klass L, et al. Outbreak of parainfluenza virus type 3 in an intermediate care neonatal nursery. *Pediatr Infect Dis J* 1998;17(1):49-53.(mj).
375. Mullins JA, Erdman DD, Weinberg GA, et al. Human metapneumovirus infection among children hospitalized with acute respiratory illness. *Emerg Infect Dis* 2004;10(4):700-5.
376. Hatherill M, Levin M, Lawrenson J, Hsiao NY, Reynolds L, Argent A. Evolution of an adenovirus outbreak in a multidisciplinary children's hospital. *J Paediatr Child Health* 2004;40(8):449-54.
377. Langley JM, Hanakowski M. Variation in risk for nosocomial chickenpox after inadvertent exposure. *J Hosp Infect* 2000;44(3):224-6.
378. Ratner AJ, Neu N, Jakob K, et al. Nosocomial rotavirus in a pediatric hospital. *Infect Control Hosp Epidemiol* 2001;22(5):299-301.
379. Avila-Aguero ML, German G, Paris MM, Herrera JF. Toys in a pediatric hospital: are they a bacterial source? *Am J Infect Control* 2004;32(5):287-90.
380. Nyqvist KH, Lutes LM. Co-bedding twins: a developmentally supportive care strategy. *J Obstet Gynecol Neonatal Nurs* 1998;27(4):450-6.
381. Feldman R, Eidelman AI, Sirota L, Weller A. Comparison of skin-to-skin (kangaroo) and traditional care: parenting outcomes and preterm infant development. *Pediatrics* 2002;110(1 Pt 1):16-26.
382. Conde-Agudelo A, Diaz-Rossello JL, Belizan JM. Kangaroo mother care to reduce morbidity and mortality in low birthweight infants. *Cochrane Database Syst Rev* 2003(2):CD002771.

383. Adcock PM, Pastor P, Medley F, Patterson JE, Murphy TV. Methicillin-resistant *Staphylococcus aureus* in two child care centers. *J Infect Dis* 1998;178(2):577-80.
384. Shahin R, Johnson IL, Jamieson F, McGeer A, Tolkin J, Ford-Jones EL. Methicillin-resistant *Staphylococcus aureus* carriage in a child care center following a case of disease. Toronto Child Care Center Study Group. *Arch Pediatr Adolesc Med* 1999;153(8):864-8.
385. Stover BH, Duff A, Adams G, Buck G, Hancock G, Rabalais G. Emergence and control of methicillin-resistant *Staphylococcus aureus* in a children's hospital and pediatric long-term care facility. *Am J Infect Control* 1992;20(5):248-55.
386. Herold BC, Immergluck LC, Maranan MC, et al. Community-acquired methicillin-resistant *Staphylococcus aureus* in children with no identified predisposing risk. *JAMA* 1998;279(8):593-8.
387. CDC. Four pediatric deaths from community-acquired methicillin-resistant *Staphylococcus aureus*--Minnesota and North Dakota, 1997-1999. *MMWR - Morbidity & Mortality Weekly Report* 1999;48:707-10.
388. Abi-Hanna P, Frank AL, Quinn JP, et al. Clonal features of community-acquired methicillin-resistant *Staphylococcus aureus* in children. *Clin Infect Dis* 2000;30(3):630-1.
389. Fergie JE, Purcell K. Community-acquired methicillin-resistant *Staphylococcus aureus* infections in south Texas children. *Pediatr Infect Dis J* 2001;20(9):860-3.
390. Sattler CA, Mason EO, Jr., Kaplan SL. Prospective comparison of risk factors and demographic and clinical characteristics of community-acquired, methicillin-resistant versus methicillin-susceptible *Staphylococcus aureus* infection in children. *Pediatr Infect Dis J* 2002;21(10):910-7.
391. Kaplan SL, Hulten KG, Gonzalez BE, et al. Three-year surveillance of community-acquired *Staphylococcus aureus* infections in children. *Clin Infect Dis* 2005;40(12):1785-91.
392. Jarvis WR. Infection control and changing health-care delivery systems. *Emerg Infect Dis* 2001;7(2):170-3.
393. Garibaldi RA. Residential care and the elderly: the burden of infection. *J Hosp Infect* 1999;43 Suppl:S9-18.
394. Hsu K, Harris JA. Control of infections in nonacute care pediatric settings. *Seminars Pedi Infect Dis* 2001;12(2):92-9.
395. Strausbaugh LJ, Joseph CL, et al. The 2000-2001 outbreak of methicillin-resistant *Staphylococcus aureus* in long-term care. *Clin Infect Dis* 2002;21(10):1744-9.

396. Centers for Medicare and Medicaid Services. Healthcare Industry Market Update. http://www.cms.hhs.gov/reports/hcimu/hcimu_05202003pdf May 2003.
397. Lee YL, Thrupp LD, Friis RH, Fine M, Maleki P, Cesario TC. Nosocomial infection and antibiotic utilization in geriatric patients: a pilot prospective surveillance program in skilled nursing facilities. *Gerontology* 1992;38(4):223-32.
398. Stevenson KB. Regional data set of infection rates for long-term care facilities: description of a valuable benchmarking tool. *Am J Infect Control* 1999;27(1):20-6.
399. Jackson MM, Fierer J, Barrett-Connor E, et al. Intensive surveillance for infections in a three-year study of nursing home patients. *Am J Epidemiol* 1992;135(6):685-96.
400. Darnowski SB, Gordon M, Simor AE. Two years of infection surveillance in a geriatric long-term care facility. *Am J Infect Control* 1991;19(4):185-90.
401. Hoffman N, Jenkins R, Putney K. Nosocomial infection rates during a one-year period in a nursing home care unit of a Veterans Administration hospital. *Am J Infect Control* 1990;18(2):55-63.
402. Tsan L, Hojlo C, Kearns MA, et al. Infection surveillance and control programs in the Department of Veterans Affairs nursing home care units: a preliminary assessment. *Am J Infect Control* 2006;34(2):80-3.
403. Kane RA, Caplan AL, Urv-Wong EK, Freeman IC, Aroskar MA, Finch M. Everyday matters in the lives of nursing home residents: wish for and perception of choice and control. *J Am Geriatr Soc* 1997;45:1086-93.
404. Libow LS, Starer P. Care of the nursing home patient. *N Engl J Med* 1989;321(2):93-6.
405. Perls TT, Herget M. Higher respiratory infection rates on an Alzheimer's special care unit and successful intervention. *J Am Geriatr Soc* 1995;43(12):1341-4.
406. Bradley SF. Issues in the management of resistant bacteria in long-term-care facilities. *Infect Control Hosp Epidemiol* 1999;20(5):362-6.
407. Crossley K. Vancomycin-resistant enterococci in long-term-care facilities. *Infect Control Hosp Epidemiol* 1998;19(7):521-5.
408. Strausbaugh LJ, Crossley KB, Nurse BA, Thrupp LD. Antimicrobial resistance in long-term-care facilities. *Infect Control Hosp Epidemiol* 1996;17(2):129-40.
409. Richards CL. Infections in Long-Term-Care Facilities: Screen or Clean? *Infect Control Hosp Epidemiol* 2005;26(10):800-01.

410. Drinka PJ, Krause P, Nest L, Goodman BM, Gravenstein S. Risk of acquiring influenza A in a nursing home from a culture-positive roommate. *Infect Control Hosp Epidemiol* 2003;24(11):872-4.
411. Falsey AR, Treanor JJ, Betts RF, Walsh EE. Viral respiratory infections in the institutionalized elderly: clinical and epidemiologic findings. *J Am Geriatr Soc* 1992;40(2):115-9.
412. Ellis SE, Coffey CS, Mitchel EF, Jr., Dittus RS, Griffin MR. Influenza- and respiratory syncytial virus-associated morbidity and mortality in the nursing home population. *J Am Geriatr Soc* 2003;51(6):761-7.
413. Louie JK, Yagi S, Nelson FA, et al. Rhinovirus outbreak in a long term care facility for elderly persons associated with unusually high mortality. *Clin Infect Dis* 2005;41(2):262-5.
414. Piednoir E, Bureau-Chalot F, Merle C, Gotzamanis A, Wuibout J, Bajolet O. Direct costs associated with a nosocomial outbreak of adenoviral conjunctivitis infection in a long-term care institution. *Am J Infect Control* 2002;30(7):407-10.
415. Addiss DG, Davis JP, Meade BD, et al. A pertussis outbreak in a Wisconsin nursing home. *J Infect Dis* 1991;164(4):704-10.
416. Gaynes R, Rimland D, Killum E, et al. Outbreak of *Clostridium difficile* infection in a long-term care facility: association with gatifloxacin use. *Clin Infect Dis* 2004;38(5):640-5.
417. High KP, Bradley S, Loeb M, Palmer R, Quagliarello V, Yoshikawa T. A new paradigm for clinical investigation of infectious syndromes in older adults: assessment of functional status as a risk factor and outcome measure. *Clin Infect Dis* 2005;40(1):114-22.
418. Loeb MB, Craven S, McGeer AJ, et al. Risk factors for resistance to antimicrobial agents among nursing home residents. *Am J Epidemiol* 2003;157(1):40-7.
419. Vergis EN, Brennen C, Wagener M, Muder RR. Pneumonia in long-term care: a prospective case-control study of risk factors and impact on survival. *Arch Intern Med* 2001;161(19):2378-81.
420. Loeb M, McGeer A, McArthur M, Walter S, Simor AE. Risk factors for pneumonia and other lower respiratory tract infections in elderly residents of long-term care facilities. *Arch Intern Med* 1999;159(17):2058-64.
421. Brandeis GH, Ooi WL, Hossain M, Morris JN, Lipsitz LA. A longitudinal study of risk factors associated with the formation of pressure ulcers in nursing homes. *J Am Geriatr Soc* 1994;42(4):388-93.
422. Allard JP, Aghdassi E, McArthur M, et al. Nutrition risk factors for survival in the elderly living in Canadian long-term care facilities. *J Am Geriatr Soc* 2004;52(1):59-65.

423. Pick N, McDonald A, Bennett N, et al. Pulmonary aspiration in a long-term care setting: clinical and laboratory observations and an analysis of risk factors. *J Am Geriatr Soc* 1996;44(7):763-8.
424. Nicolle LE. The chronic indwelling catheter and urinary infection in long-term-care facility residents. *Infect Control Hosp Epidemiol* 2001;22(5):316-21.
425. Pien EC, Hume KE, Pien FD. Gastrostomy tube infections in a community hospital. *Am J Infect Control* 1996;24(5):353-8.
426. Gomes GF, Pisani JC, Macedo ED, Campos AC. The nasogastric feeding tube as a risk factor for aspiration and aspiration pneumonia. *Curr Opin Clin Nutr Metab Care* 2003;6(3):327-33.
427. Bula CJ, Ghilardi G, Wietlisbach V, Petignat C, Francioli P. Infections and functional impairment in nursing home residents: a reciprocal relationship. *J Am Geriatr Soc* 2004;52(5):700-6.
428. Bradley SF, Terpenning MS, Ramsey MA, et al. Methicillin-resistant *Staphylococcus aureus*: colonization and infection in a long-term care facility. *Ann Intern Med* 1991;115(6):417-22.
429. Washio M, Nishisaka S, Kishikawa K, et al. Incidence of methicillin-resistant *Staphylococcus aureus* (MRSA) isolation in a skilled nursing home: a third report on the risk factors for the occurrence of MRSA infection in the elderly. *J Epidemiol* 1996;6(2):69-73.
430. Trick WE, Weinstein RA, DeMarais PL, et al. Colonization of skilled-care facility residents with antimicrobial-resistant pathogens. *J Am Geriatr Soc* 2001;49(3):270-6.
431. Nicolle LE, Garibaldi RA. Infection control in long-term-care facilities. *Infect Control Hosp Epidemiol* 1995;16(6):348-53.(s).
432. Crossley K. Long-term care facilities as sources of antibiotic-resistant nosocomial pathogens. *Curr Opin Infect Dis* 2001;14(4):455-9.
433. Smith PW, Rusnak PG. Infection prevention and control in the long-term-care facility. SHEA Long-Term-Care Committee and APIC Guidelines Committee. *Infect Control Hosp Epidemiol* 1997;18(12):831-49.(s).
434. Friedman C, Barnette M, Buck AS, et al. Requirements for infrastructure and essential activities of infection control and epidemiology in out-of-hospital settings: a consensus panel report. Association for Professionals in Infection Control and Epidemiology and Society for Healthcare Epidemiology of America. *Infect Control Hosp Epidemiol* 1999;20(10):695-705.
435. Nicolle LE. Infection control in long-term care facilities. *Clin Infect Dis* 2000;31(3):752-6.
436. Bradley SF. Methicillin-resistant *Staphylococcus aureus*: long-term care concerns. *Am J Med* 1999;106(5A):2S-10S; discussion 48S-52S.

437. www.cms.hhs.gov/manuals/Downloads/som107ap_pp_guidelines_ltcf
438. Mylotte JM, Goodnough S, Tayara A. Antibiotic-resistant organisms among long-term care facility residents on admission to an inpatient geriatrics unit: Retrospective and prospective surveillance. *Am J Infect Control* 2001;29(3):139-44.
439. Strausbaugh LJ, Jacobson C, Yost T. Methicillin-resistant *Staphylococcus aureus* in a nursing home and affiliated hospital: a four-year perspective. *Infect Control Hosp Epidemiol* 1993;14(6):331-6.
440. Wiener J, Quinn JP, Bradford PA, et al. Multiple antibiotic-resistant *Klebsiella* and *Escherichia coli* in nursing homes. *Jama* 1999;281(6):517-23.
441. Pop-Vicas AE, D'Agata EM. The rising influx of multidrug-resistant gram-negative bacilli into a tertiary care hospital. *Clin Infect Dis* 2005;40(12):1792-8.
442. Ly N, McCaig LF. National Hospital Ambulatory Medical Care Survey: 2000 outpatient department summary. *Adv Data* 2002(327):1-27.
443. Cherry DK, Woodwell DA. National Ambulatory Medical Care Survey: 2000 summary. *Adv Data* 2002(328):1-32.
444. Finelli L, Miller JT, Tokars JI, Alter MJ, Arduino MJ. National surveillance of dialysis-associated diseases in the United States, 2002. *Semin Dial* 2005;18(1):52-61.
445. D'Agata EM. Antimicrobial-resistant, Gram-positive bacteria among patients undergoing chronic hemodialysis. *Clin Infect Dis* 2002;35(10):1212-8.
446. Goodman RA, Solomon SL. Transmission of infectious diseases in outpatient health care settings. *JAMA* 1991;265(18):2377-81.
447. Nafziger DA, Lundstrom T, Chandra S, Massanari RM. Infection control in ambulatory care. *Infect Dis Clin North Am* 1997;11(2):279-96.
448. Herwaldt LA, Smith SD, Carter CD. Infection control in the outpatient setting. *Infect Control Hosp Epidemiol* 1998;19(1):41-74.
449. Hlady WG, Hopkins RS, Ogilby TE, Allen ST. Patient-to-patient transmission of hepatitis B in a dermatology practice. *Am J Public Health* 1993;83(12):1689-93.
450. Birnie GG, Quigley EM, Clements GB, Follet EA, Watkinson G. Endoscopic transmission of hepatitis B virus. *Gut* 1983;24(2):171-4.
451. Chant K, Lowe D, Rubin G, et al. Patient-to-patient transmission of HIV in private surgical consulting rooms. *Lancet* 1993;342(8886-8887):1548-9.
452. Chant K, Kociuba K, Munro R, et al. Investigation of Possible Patient-to-Patient Transmission of Hepatitis C in a Hospital. *NSW Public Health Bull* 1994;5(5):47-51.

453. CDC. Transmission of hepatitis B and C viruses in outpatient settings-- New York, Oklahoma, and Nebraska, 2000-2002. *MMWR Morb Mortal Wkly Rep* 2003;52(38):901-6.
454. Williams IT, Perz JF, Bell BP. Viral hepatitis transmission in ambulatory health care settings. *Clin Infect Dis* 2004;38(11):1592-8.
455. Couldwell DL, Dore GJ, Harkness JL, et al. Nosocomial outbreak of tuberculosis in an outpatient HIV treatment room. *Aids* 1996;10(5):521-5.
456. CDC. Mycobacterium tuberculosis Transmission in a Health Clinic -- Florida. *MMWR* 1989;38(15):256-8; 63-64.
457. Calder RA, Duclos P, Wilder MH, Pryor VL, Scheel WJ. Mycobacterium tuberculosis transmission in a health clinic. *Bull Int Union Tuberc Lung Dis* 1991;66(2-3):103-6.
458. Istre GR, McKee PA, West GR, et al. Measles spread in medical settings: an important focus of disease transmission? *Pediatrics* 1987;79(3):356-8.
459. Dawson C, Darrell R. Infections due to adenovirus type 8 in the United States. I. An outbreak of epidemic keratoconjunctivitis originating in a physician's office. *N Engl J Med* 1963;268:1031-4.
460. Montessori V, Scharf S, Holland S, Werker DH, Roberts FJ, Bryce E. Epidemic keratoconjunctivitis outbreak at a tertiary referral eye care clinic. *Am J Infect Control* 1998;26(4):399-405.
461. Jernigan JA, Lowry BS, Hayden FG, et al. Adenovirus type 8 epidemic keratoconjunctivitis in an eye clinic: risk factors and control. *J Infect Dis* 1993;167(6):1307-13.
462. Buehler JW, Finton RJ, Goodman RA, et al. Epidemic keratoconjunctivitis: report of an outbreak in an ophthalmology practice and recommendations for prevention. *Infect Control* 1984;5(8):390-4.
463. Johnston CP, Cooper L, Ruby W, Teeter T, al. E. Community-associated methicillin resistant *Staphylococcus aureus* skin infections among outpatient healthcare workers and its isolation in the clinic environment. Presented at the 15th Annual Scientific Meeting of the Society for Healthcare Epidemiology of America (SHEA), Los Angeles, California, 4/10/05. Abstract #132 2005.
464. Biddick R, Spilker T, Martin A, LiPuma JJ. Evidence of transmission of *Burkholderia cepacia*, *Burkholderia multivorans* and *Burkholderia dolosa* among persons with cystic fibrosis. *FEMS Microbiol Lett* 2003;228(1):57-62.
465. Griffiths AL, Jansen K, Carlin JB, et al. Effects of segregation on an epidemic *Pseudomonas aeruginosa* strain in a cystic fibrosis clinic. *Am J Respir Crit Care Med* 2005;171(9):1020-5.

466. Danzig LE, Short LJ, Collins K, et al. Bloodstream infections associated with a needleless intravenous infusion system in patients receiving home infusion therapy. *JAMA* 1995;273(23):1862-4.
467. Kellerman S, Shay DK, Howard J, et al. Bloodstream infections in home infusion patients: the influence of race and needleless intravascular access devices. *J Pediatr* 1996;129(5):711-7.
468. Do AN, Ray BJ, Banerjee SN, et al. Bloodstream infection associated with needleless device use and the importance of infection-control practices in the home health care setting. *J Infect Dis* 1999;179(2):442-8.
469. Tokars JI, Cookson ST, McArthur MA, Boyer CL, McGeer AJ, Jarvis WR. Prospective evaluation of risk factors for bloodstream infection in patients receiving home infusion therapy. *Ann Intern Med* 1999;131(5):340-7.
470. Manangan LP, Pearson ML, Tokars JI, Miller E, Jarvis WR. Feasibility of national surveillance of health-care-associated infections in home-care settings. *Emerg Infect Dis* 2002;8(3):233-6.
471. Shah SS, Manning ML, Leahy E, Magnusson M, Rheingold SR, Bell LM. Central venous catheter-associated bloodstream infections in pediatric oncology home care. *Infect Control Hosp Epidemiol* 2002;23(2):99-101.
472. Gorski LA. Central venous access device outcomes in a homecare agency: a 7-year study. *J Infus Nurs* 2004;27(2):104-11.
473. Rosenheimer L, Embry FC, Sanford J, Silver SR. Infection surveillance in home care: device-related incidence rates. *Am J Infect Control* 1998;26(3):359-63.
474. White MC, Ragland KE. Surveillance of intravenous catheter-related infections among home care clients. *Am J Infect Control* 1994;22(4):231-5.
475. Beltrami EM, McArthur MA, McGeer A, et al. The nature and frequency of blood contacts among home healthcare workers. *Infect Control Hosp Epidemiol* 2000;21(12):765-70.
476. Embry FC, Chinnes LF. Draft definitions for surveillance of infections in home health care. *Am J Infect Control* 2000;28(6):449-53.
477. Fraser TG, Stosor V, Wang Q, Allen A, Zembower TR. Vancomycin and home health care. *Emerg Infect Dis* 2005;11(10):1558-64.
478. Carrico RM, Niner S. Multidrug resistant organisms--VRE and MRSA: practical home care tips. *Home Healthc Nurse* 2002;20(1):23-8; quiz 8-9.
479. Friedman MM, Rhinehart E. Improving infection control in home care: from ritual to science-based practice. *Home Healthc Nurse* 2000;18(2):99-105; quiz 6.

480. Friedman MM, Rhinehart E. Putting infection control principles into practice in home care. *Nurs Clin North Am* 1999;34(2):463-82.
481. Davis PL, Madigan EA. Evidence-based practice and the home care nurse's bag. *Home Healthc Nurse* 1999;17(5):295-9.
482. Sitzman KL, Pett MA, Blosswick DS. An exploratory study of nurse bag use by home visiting nurses. *Home Healthc Nurse* 2002;20(4):237-43.
483. Anderson MA, Madigan EA, Helms LB. Nursing research in home health care: endangered species? *Home Care Provid* 2001;6(6):200-4.
484. White MC. Identifying infectious diseases in prisons: surveillance, protection, and intervention. *West J Med* 1999;170(3):177.
485. Puisis M. Update on public health in correctional facilities. *West J Med* 1998;169(6):374.
486. Levy MH, Lerwitworapong J. Issues facing TB control (3.1). Tuberculosis in prisons. *Scott Med J* 2000;45(5 Suppl):30-2; discussion 3.
487. Parece MS, Herrera GA, Voigt RF, Middlekauff SL, Irwin KL. STD testing policies and practices in U.S. city and county jails. *Sex Transm Dis* 1999;26(8):431-7.
488. Cieslak PR, Curtis MB, Coulombier DM, Hathcock AL, Bean NH, Tauxe RV. Preventable disease in correctional facilities. Desmoteric foodborne outbreaks in the United States, 1974-1991. *Arch Intern Med* 1996;156(16):1883-8.
489. CDC. Public health dispatch: tuberculosis outbreak in a homeless population--Portland, Maine, 2002-2003. *MMWR Morb Mortal Wkly Rep* 2003;52(48):1184.
490. CDC. Public health dispatch: tuberculosis outbreak among homeless persons--King County, Washington, 2002-2003. *MMWR Morb Mortal Wkly Rep* 2003;52(49):1209-10.
491. CDC. Tuberculosis transmission in a homeless shelter population--New York, 2000-2003. *MMWR Morb Mortal Wkly Rep* 2005;54(6):149-52.
492. Baillargeon J, Kelley MF, Leach CT, Baillargeon G, Pollock BH. Methicillin-resistant *Staphylococcus aureus* infection in the Texas prison system. *Clin Infect Dis* 2004;38(9):e92-5.
493. Young DM, Harris HW, Charlebois ED, et al. An epidemic of methicillin-resistant *Staphylococcus aureus* soft tissue infections among medically underserved patients. *Arch Surg* 2004;139(9):947-51; discussion 51-3.
494. Pan ES, Diep BA, Carleton HA, et al. Increasing prevalence of methicillin-resistant *Staphylococcus aureus* infection in California jails. *Clin Infect Dis* 2003;37(10):1384-8.

495. CDC. Drug-susceptible tuberculosis outbreak in a state correctional facility housing HIV-infected inmates--South Carolina, 1999-2000. *MMWR Morb Mortal Wkly Rep* 2000;49(46):1041-4.
496. CDC. Methicillin-resistant *Staphylococcus aureus* skin or soft tissue infections in a state prison--Mississippi, 2000. *MMWR Morb Mortal Wkly Rep* 2001;50(42):919-22.
497. Mohle-Boetani JC, Miguelino V, Dewsnup DH, et al. Tuberculosis outbreak in a housing unit for human immunodeficiency virus-infected patients in a correctional facility: transmission risk factors and effective outbreak control. *Clin Infect Dis* 2002;34(5):668-76.
498. CDC. Prevention and Control of Tuberculosis in Correctional Facilities. Recommendations of the Advisory Council for the Elimination of Tuberculosis. *MMWR Recomm Rep* 1996;45(RR-8):1-37.
499. Whimbey E, Englund JA, Couch RB. Community respiratory virus infections in immunocompromised patients with cancer. *Am J Med* 1997;102(3A):10-8; discussion 25-6.
500. Zambon M, Bull T, Sadler CJ, Goldman JM, Ward KN. Molecular epidemiology of two consecutive outbreaks of parainfluenza 3 in a bone marrow transplant unit. *J Clin Microbiol* 1998;36(8):2289-93.
501. Gams AS, Howells WB, DeSwarte-Wallace J, Feusner JH, Buckley JD, Woods WG. Alpha hemolytic streptococcal infection during intensive treatment for acute myeloid leukemia: a report from the Children's cancer group study CCG-2891. *J Clin Oncol* 2000;18(9):1845-55.
502. Ek T, Mellander L, Andersson B, Abrahamsson J. Immune reconstitution after childhood acute lymphoblastic leukemia is most severely affected in the high risk group. *Pediatr Blood Cancer* 2005;44(5):461-8.
503. Pascual V, Allantaz F, Arce E, Punaro M, Banchereau J. Role of interleukin-1 (IL-1) in the pathogenesis of systemic onset juvenile idiopathic arthritis and clinical response to IL-1 blockade. *J Exp Med* 2005;201(9):1479-86.
504. Marchesoni A, Puttini PS, Gorla R, et al. Cyclosporine in addition to infliximab and methotrexate in refractory rheumatoid arthritis. *Clin Exp Rheumatol* 2005;23(6):916-7.
505. Isaacs KL, Lewis JD, Sandborn WJ, Sands BE, Targan SR. State of the art: IBD therapy and clinical trials in IBD. *Inflamm Bowel Dis* 2005;11 Suppl 1:S3-12.
506. CDC. Guidelines for preventing opportunistic infections among HIV-infected persons. *MMWR Morb Mortal Wkly Rep* 2002;51 (RR-8):1-52.
507. Kusne S, and Krystofak S. Infection control issues after solid organ transplantation in transplant infections (Second Edition), ed. Bowden RA,

Ljungman P, Paya CV. Lippincott, Williams and Wilkins. Philadelphia; 2003.

508. Anderson D, DeFor T, Burns L, et al. A comparison of related donor peripheral blood and bone marrow transplants: importance of late-onset chronic graft-versus-host disease and infections. *Biol Blood Marrow Transplant* 2003;9(1):52-9.
509. Pitchford KC, Corey M, Highsmith AK, et al. *Pseudomonas* species contamination of cystic fibrosis patients' home inhalation equipment. *J Pediatr* 1987;111(2):212-6.
510. Hamill RJ, Houston ED, Georghiou PR, et al. An outbreak of *Burkholderia* (formerly *Pseudomonas*) *cepacia* respiratory tract colonization and infection associated with nebulized albuterol therapy. *Ann Intern Med* 1995;122(10):762-6.
511. Hutchinson GR, Parker S, Pryor JA, et al. Home-use nebulizers: a potential primary source of *Burkholderia cepacia* and other colistin-resistant, gram-negative bacteria in patients with cystic fibrosis. *J Clin Microbiol* 1996;34(3):584-7.
512. Jakobsson BM, Onnered AB, Hjelte L, Nystrom B. Low bacterial contamination of nebulizers in home treatment of cystic fibrosis patients. *J Hosp Infect* 1997;36(3):201-7.
513. Rosenfeld M, Joy P, Nguyen CD, Krzewinski J, Burns JL. Cleaning home nebulizers used by patients with cystic fibrosis: is rinsing with tap water enough? *J Hosp Infect* 2001;49(3):229-30.
514. Govan JR. Infection control in cystic fibrosis: methicillin-resistant *Staphylococcus aureus*, *Pseudomonas aeruginosa* and the *Burkholderia cepacia* complex. *J R Soc Med* 2000;93(Suppl 38):40-5.
515. Frederiksen B, Koch C, Hoiby N. Changing epidemiology of *Pseudomonas aeruginosa* infection in Danish cystic fibrosis patients (1974-1995). *Pediatr Pulmonol* 1999;28(3):159-66.
516. Isles A, Maclusky I, Corey M, et al. *Pseudomonas cepacia* infection in cystic fibrosis: an emerging problem. *J Pediatr* 1984;104(2):206-10.
517. LiPuma JJ. *Burkholderia cepacia*. Management issues and new insights. *Clin Chest Med* 1998;19(3):473-86, vi.
518. Tablan OC, Chorba TL, Schidlow DV, et al. *Pseudomonas cepacia* colonization in patients with cystic fibrosis: risk factors and clinical outcome. *J Pediatr* 1985;107(3):382-7.
519. Hudson VL, Wielinski CL, Regelman WE. Prognostic implications of initial oropharyngeal bacterial flora in patients with cystic fibrosis diagnosed before the age of two years. *J Pediatr* 1993;122(6):854-60.

520. Farrell PM, Li Z, Kosorok MR, et al. Bronchopulmonary disease in children with cystic fibrosis after early or delayed diagnosis. *Am J Respir Crit Care Med* 2003;168(9):1100-8.
521. Smith DL, Gumery LB, Smith EG, Stableforth DE, Kaufmann ME, Pitt TL. Epidemic of *Pseudomonas cepacia* in an adult cystic fibrosis unit: evidence of person-to-person transmission. *J Clin Microbiol* 1993;31(11):3017-22.js.
522. Pegues DA, Schidlow DV, Tablan OC, Carson LA, Clark NC, Jarvis WR. Possible nosocomial transmission of *Pseudomonas cepacia* in patients with cystic fibrosis. *Arch Pediatr Adolesc Med* 1994;148(8):805-12.
523. Govan JR, Brown PH, Maddison J, et al. Evidence for transmission of *Pseudomonas cepacia* by social contact in cystic fibrosis. *Lancet* 1993;342(8862):15-9.
524. Pegues DA, Carson LA, Tablan OC, et al. Acquisition of *Pseudomonas cepacia* at summer camps for patients with cystic fibrosis. Summer Camp Study Group. *J Pediatr* 1994;124(5 Pt 1):694-702.
525. Tablan OC, Martone WJ, Doershuk CF, et al. Colonization of the respiratory tract with *Pseudomonas cepacia* in cystic fibrosis. Risk factors and outcomes. *Chest* 1987;91(4):527-32.
526. Thomassen MJ, Demko CA, Doershuk CF, Stern RC, Klinger JD. *Pseudomonas cepacia*: decrease in colonization in patients with cystic fibrosis. *Am Rev Respir Dis* 1986;134(4):669-71.js.
527. Weber DJ, Rutala WA. Gene therapy: a new challenge for infection control. *Infect Control Hosp Epidemiol* 1999;20(8):530-2.
528. Evans ME, Lesnaw JA. Infection control for gene therapy: a busy physician's primer. *Clin Infect Dis* 2002;35(5):597-605.
529. Strausbaugh LJ. Gene therapy and infection control: more light on the way. *Infect Control Hosp Epidemiol* 2000;21(10):630-2.
530. CDC. West Nile virus infections in organ transplant recipients--New York and Pennsylvania, August-September, 2005. *MMWR Morb Mortal Wkly Rep* 2005;54(40):1021-3.
531. Lawson CA. Cytomegalovirus after kidney transplantation: a case review. *Prog Transplant* 2005;15(2):157-60.
532. Tugwell BD, Patel PR, Williams IT, et al. Transmission of hepatitis C virus to several organ and tissue recipients from an antibody-negative donor. *Ann Intern Med* 2005;143(9):648-54.
533. Kainer MA, Linden JV, Whaley DN, et al. Clostridium infections associated with musculoskeletal-tissue allografts. *N Engl J Med* 2004;350(25):2564-71.

534. CDC. Invasive *Streptococcus pyogenes* after allograft implantation-- Colorado, 2003. *MMWR Morb Mortal Wkly Rep* 2003;52(48):1174-6.
535. Mungai M, Tegtmeier G, Chamberland M, Parise M. Transfusion-transmitted malaria in the United States from 1963 through 1999. *N Engl J Med* 2001;344(26):1973-8.
536. Lux JZ, Weiss D, Linden JV, et al. Transfusion-associated babesiosis after heart transplant. *Emerg Infect Dis* 2003;9(1):116-9.
537. CDC. Chagas Disease After Organ Transplantation --- United States, 2001. *MMWR* 2002;51(10):210-2.
538. CDC. Lymphocytic choriomeningitis virus infection in organ transplant recipients--Massachusetts, Rhode Island, 2005. *MMWR Morb Mortal Wkly Rep* 2005;54(21):537-9.
539. Srinivasan A, Burton EC, Kuehnert MJ, et al. Transmission of rabies virus from an organ donor to four transplant recipients. *N Engl J Med* 2005;352(11):1103-11.
540. Gottesdiener KM. Transplanted infections: donor-to-host transmission with the allograft. *Ann Intern Med* 1989;110(12):1001-16.
541. Borie DC, Cramer DV, Phan-Thanh L, et al. Microbiological hazards related to xenotransplantation of porcine organs into man. *Infect Control Hosp Epidemiol* 1998;19(5):355-65.
542. CDC. U.S. Public Health Service Guideline on Infectious Disease Issues in Xenotransplantation. Centers for Disease Control and Prevention. *MMWR - Morbidity & Mortality Weekly Report* 2001;50 (RR-15)(August):1-46.
543. IOM. Institute of Medicine. To err is human: building a safer health system. Washington DC National Academy Press; 1999;<http://www.iom.edu/report.asp?id=5575>.
544. Gerberding JL. Hospital-onset infections: a patient safety issue. *Ann Intern Med* 2002;137(8):665-70.
545. Leape LL, Berwick DM, Bates DW. What practices will most improve safety? Evidence-based medicine meets patient safety. *JAMA* 2002;288(4):501-7.
546. Burke JP. Patient safety: infection control - a problem for patient safety. *N Engl J Med* 2003;348(7):651-6.
547. Shulman L, Ost D. Managing infection in the critical care unit: how can infection control make the ICU safe? *Crit Care Clin* 2005;21(1):111-28, ix.
548. Goldmann DA, Weinstein RA, Wenzel RP, et al. Strategies to Prevent and Control the Emergence and Spread of Antimicrobial-Resistant

- Microorganisms in Hospitals. A challenge to hospital leadership. *JAMA* 1996;275(3):234-40.
549. Scheckler WE, Brimhall D, Buck AS, et al. Requirements for infrastructure and essential activities of infection control and epidemiology in hospitals: a consensus panel report. Society for Healthcare Epidemiology of America. *Infect Control Hosp Epidemiol* 1998;19(2):114-24.
550. www.jointcommission.org/PatientSafety/NationalPatientSafetyGoals/
551. Jackson M, Chiarello LA, Gaynes RP, Gerberding JL. Nurse staffing and health care-associated infections: Proceedings from a working group meeting. *Am J Infect Control* 2002;30(4):199-206.
552. O'Boyle C, Jackson M, Henly SJ. Staffing requirements for infection control programs in US health care facilities: Delphi project. *Am J Infect Control* 2002;30(6):321-33.
553. Peterson LR, Hamilton JD, Baron EJ, et al. Role of clinical microbiology laboratories in the management and control of infectious diseases and the delivery of health care. *Clin Infect Dis* 2001;32(4):605-11.
554. McGowan JE, Jr., Tenover FC. Confronting bacterial resistance in healthcare settings: a crucial role for microbiologists. *Nat Rev Microbiol* 2004;2(3):251-8.
555. . (Accessed at
556. Curtis JR, Cook DJ, Wall RJ, et al. Intensive care unit quality improvement: a "how-to" guide for the interdisciplinary team. *Crit Care Med* 2006;34(1):211-8.
557. Pronovost PJ, Nolan T, Zeger S, Miller M, Rubin H. How can clinicians measure safety and quality in acute care? *Lancet* 2004;363(9414):1061-7.
558. Goldrick BA, Dingle DA, Gilmore GK, Curchoe RM, Plackner CL, Fabrey LJ. Practice analysis for infection control and epidemiology in the new millennium. *Am J Infect Control* 2002;30(8):437-48.
559. CDC. Guideline for Hand Hygiene in Health-Care Settings: Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force. *MMWR* 2002;51(16)(RR-16):1-44.
560. Bonomo RA, Rice LB. Emerging issues in antibiotic resistant infections in long-term care facilities. *J Gerontol A Biol Sci Med Sci* 1999;54(6):B260-7.
561. Larson EL, Early E, Cloonan P, Sugrue S, Parides M. An organizational climate intervention associated with increased handwashing and decreased nosocomial infections. *Behav Med* 2000;26(1):14-22.

562. Pittet D, Hugonnet S, Harbarth S, et al. Effectiveness of a hospital-wide programme to improve compliance with hand hygiene. *Infection Control Programme. Lancet* 2000;356(9238):1307-12.
563. Murthy R. Implementation of strategies to control antimicrobial resistance. *Chest* 2001;119(2 Suppl):405S-11S.
564. Rondeau KV, Wagar TH. Organizational learning and continuous quality improvement: examining the impact on nursing home performance. *Health Manage Forum* 2002;15(2):17-23.
565. Stelfox HT, Bates DW, Redelmeier DA. Safety of patients isolated for infection control. *JAMA* 2003;290(14):1899-905.
566. Haley RW, Culver DH, White JW, et al. The efficacy of infection surveillance and control programs in preventing nosocomial infections in US hospitals. *Am J Epidemiol* 1985;121(2):182-205.
567. McArthur BJ, Pugliese G, Weinstein S, et al. A national task analysis of infection control practitioners, 1982. Part One: methodology and demography. *Am J Infect Control* 1984;12(2):88-95.
568. Shannon R, McArthur BJ, Weinstein S, et al. A national task analysis of infection control practitioners, 1982. Part Two: Tasks, knowledge, and abilities for practice. *Am J Infect Control* 1984;12(3):187-96.
569. Pugliese G, McArthur BJ, Weinstein S, et al. A national task analysis of infection control practitioners, 1982. Part Three: The relationship between hospital size and tasks performed. *Am J Infect Control* 1984;12(4):221-7.
570. Larson E, Eisenberg R, Soule BM. Validating the certification process for infection control practice. *Am J Infect Control* 1988;16(5):198-205.
571. Bjerke NB, Fabrey LJ, Johnson CB, et al. Job analysis 1992: infection control practitioner. *Am J Infect Control* 1993;21(2):51-7.
572. Turner JG, Kolenc KM, Docken L. Job analysis 1996: Infection control professional. Certification Board in Infection Control and Epidemiology, Inc, 1996 Job Analysis Committee. *Am J Infect Control* 1999;27(2):145-57.
573. Health Canada. Nosocomial and Occupational Infections Section. Development of a resource model for infection prevention and control programs in acute, long term, and home care settings: conference proceedings of the Infection Prevention and Control Alliance. *AJIC* 2004;32:2-6.
574. Lee TH, Meyer GS, Brennan TA. A middle ground on public accountability. *N Engl J Med* 2004;350(23):2409-12.
575. Stevenson KB, Murphy CL, Samore MH, et al. Assessing the status of infection control programs in small rural hospitals in the western United States. *Am J Infect Control* 2004;32(5):255-61.

576. Simonds DN, Horan TC, Kelley R, Jarvis WR. Detecting pediatric nosocomial infections: how do infection control and quality assurance personnel compare? *Am J Infect Control* 1997;25(3):202-8.
577. Dawson SJ. The role of the infection control link nurse. *J Hosp Infect* 2003;54(4):251-7; quiz 320.
578. Wright J, Stover BH, Wilkerson S, Bratcher D. Expanding the infection control team: development of the infection control liaison position for the neonatal intensive care unit. *Am J Infect Control* 2002;30(3):174-8.
579. Teare EL, Peacock A. The development of an infection control link-nurse programme in a district general hospital. *J Hosp Infect* 1996;34(4):267-78.
580. Ching TY, Seto WH. Evaluating the efficacy of the infection control liaison nurse in the hospital. *J Adv Nurs* 1990;15(10):1128-31.
581. Amundsen J, Drennan DP. An infection control nurse-advisor program. *Am J Infect Control* 1983;11(1):20-3.
582. Ross KA. A program for infection surveillance utilizing an infection control liaison nurse. *Am J Infect Control* 1982;10(1):24-8.
583. Needleman J, Buerhaus P, Mattke S, Stewart M, Zelevinsky K. Nurse-staffing levels and the quality of care in hospitals. *N Engl J Med* 2002;346(22):1715-22.
584. Dimick JB, Swoboda SM, Pronovost PJ, Lipsett PA. Effect of nurse-to-patient ratio in the intensive care unit on pulmonary complications and resource use after hepatectomy. *Am J Crit Care* 2001;10(6):376-82.
585. Mayhall CG, Lamb VA, Gayle WE, Jr., Haynes BW, Jr. *Enterobacter cloacae* septicemia in a burn center: epidemiology and control of an outbreak. *J Infect Dis* 1979;139(2):166-71.
586. Goldmann DA, Durbin WA, Jr., Freeman J. Nosocomial infections in a neonatal intensive care unit. *J Infect Dis* 1981;144(5):449-59.(mj).
587. Arnow P, Allyn PA, Nichols EM, Hill DL, Pezzlo M, Bartlett RH. Control of methicillin-resistant *Staphylococcus aureus* in a burn unit: role of nurse staffing. *J Trauma* 1982;22(11):954-9.
588. Haley RW, Bregman DA. The role of understaffing and overcrowding in recurrent outbreaks of staphylococcal infection in a neonatal special-care unit. *J Infect Dis* 1982;145(6):875-85.
589. Fridkin SK, Pear SM, Williamson TH, Galgiani JN, Jarvis WR. The role of understaffing in central venous catheter-associated bloodstream infections. *Infect Control Hosp Epidemiol* 1996;17(3):150-8.
590. Robert J, Fridkin SK, Blumberg HM, et al. The influence of the composition of the nursing staff on primary bloodstream infection rates in a surgical intensive care unit. *Infect Control Hosp Epidemiol* 2000;21(1):12-7.(mj).

591. Li J, Birkhead GS, Strogatz DS, Coles FB. Impact of institution size, staffing patterns, and infection control practices on communicable disease outbreaks in New York State nursing homes. *Am J Epidemiol* 1996;143(10):1042-9.
592. Archibald LK, Manning ML, Bell LM, Banerjee S, Jarvis WR. Patient density, nurse-to-patient ratio and nosocomial infection risk in a pediatric cardiac intensive care unit. *Pediatr Infect Dis J* 1997;16(11):1045-8.
593. Harbarth S, Sudre P, Dharan S, Cadenas M, Pittet D. Outbreak of *Enterobacter cloacae* related to understaffing, overcrowding, and poor hygiene practices. *Infect Control Hosp Epidemiol* 1999;20(9):598-603.
594. Vicca AF. Nursing staff workload as a determinant of methicillin-resistant *Staphylococcus aureus* spread in an adult intensive therapy unit. *J Hosp Infect* 1999;43(2):109-13.
595. Stegenga J, Bell E, Matlow A. The role of nurse understaffing in nosocomial viral gastrointestinal infections on a general pediatrics ward. *Infect Control Hosp Epidemiol* 2002;23(3):133-6.
596. Alonso-Echanove J, Edwards JR, Richards MJ, et al. Effect of nurse staffing and antimicrobial-impregnated central venous catheters on the risk for bloodstream infections in intensive care units. *Infect Control Hosp Epidemiol* 2003;24(12):916-25.
597. Petrosillo N, Gilli P, Serraino D, et al. Prevalence of infected patients and understaffing have a role in hepatitis C virus transmission in dialysis. *Am J Kidney Dis* 2001;37(5):1004-10.
598. Pfaller MA, Herwaldt LA. The clinical microbiology laboratory and infection control: emerging pathogens, antimicrobial resistance, and new technology. *Clin Infect Dis* 1997;25(4):858-70.
599. Simor AE. The role of the laboratory in infection prevention and control programs in long-term-care facilities for the elderly. *Infect Control Hosp Epidemiol* 2001;22(7):459-63.
600. Weinstein RA, Mallison GF. The role of the microbiology laboratory in surveillance and control of nosocomial infections. *Am J Clin Pathol* 1978;69(2):130-6.
601. Kolmos HJ. Interaction between the microbiology laboratory and clinician: what the microbiologist can provide. *J Hosp Infect* 1999;43 Suppl:S285-91.
602. Clinical and Laboratory Standards Institute. www.clsi.org/
603. Ginocchio CC. Role of NCCLS in antimicrobial susceptibility testing and monitoring. *Am J Health Syst Pharm* 2002;59(8 Suppl 3):S7-11.
604. National Committee for Clinical Laboratory Standards. Performance standards for antimicrobial susceptibility testing; twelfth informational supplement. Document M100-S12. NCCLS, Wayne (PA) 2002.

605. NCCLS. (2002). Analysis and Presentation of Cumulative Antimicrobial Susceptibility Test Data. Approved Guideline. NCCLS document M39-A (ISBN 1-56238-422-9). Wayne: PA, NCCLS. 2002.
606. Halstead DC, Gomez N, McCarter YS. Reality of developing a community-wide antibiogram. *J Clin Microbiol* 2004;42(1):1-6.
607. Ernst EJ, Diekema DJ, BootsMiller BJ, et al. Are United States hospitals following national guidelines for the analysis and presentation of cumulative antimicrobial susceptibility data? *Diagn Microbiol Infect Dis* 2004;49(2):141-5.
608. Bergeron MG, Ouellette M. Preventing antibiotic resistance through rapid genotypic identification of bacteria and of their antibiotic resistance genes in the clinical microbiology laboratory. *J Clin Microbiol* 1998;36(8):2169-72.
609. Hacek DM, Suriano T, Noskin GA, Kruszynski J, Reisberg B, Peterson LR. Medical and economic benefit of a comprehensive infection control program that includes routine determination of microbial clonality. *Am J Clin Pathol* 1999;111(5):647-54.
610. Rodriguez WJ, Schwartz RH, Thorne MM. Evaluation of diagnostic tests for influenza in a pediatric practice. *Pediatr Infect Dis J* 2002;21(3):193-6.
611. CDC. Prevention and control of influenza. Recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep* 2005;54(RR-8):1-40.
612. Uyeki TM. Influenza diagnosis and treatment in children: a review of studies on clinically useful tests and antiviral treatment for influenza. *Pediatr Infect Dis J* 2003;22(2):164-77.
613. Chan EL, Antonishyn N, McDonald R, et al. The use of TaqMan PCR assay for detection of *Bordetella pertussis* infection from clinical specimens. *Arch Pathol Lab Med* 2002;126(2):173-6.
614. Barenfanger J, Drake C, Kacich G. Clinical and financial benefits of rapid bacterial identification and antimicrobial susceptibility testing. *J Clin Microbiol* 1999;37(5):1415-8.
615. Barenfanger J, Drake C, Leon N, Mueller T, Troutt T. Clinical and financial benefits of rapid detection of respiratory viruses: an outcomes study. *J Clin Microbiol* 2000;38(8):2824-8.
616. Ramers C, Billman G, Hartin M, Ho S, Sawyer MH. Impact of a diagnostic cerebrospinal fluid enterovirus polymerase chain reaction test on patient management. *JAMA* 2000;283(20):2680-5.
617. Mackie PL, Joannidis PA, Beattie J. Evaluation of an acute point-of-care system screening for respiratory syncytial virus infection. *J Hosp Infect* 2001;48(1):66-71.

618. Guillemot D, Courvalin P. Better control of antibiotic resistance. *Clin Infect Dis* 2001;33(4):542-7.
619. Paterson DL. The role of antimicrobial management programs in optimizing antibiotic prescribing within hospitals. *Clin Infect Dis* 2006;42 Suppl 2:S90-5.
620. Lundstrom T, Pugliese G, Bartley J, Cox J, Guither C. Organizational and environmental factors that affect worker health and safety and patient outcomes. *Am J Infect Control* 2002;30(2):93-106.
621. www.patientsafety.com/vision.html
622. Pronovost PJ, Jenckes MW, Dorman T, et al. Organizational characteristics of intensive care units related to outcomes of abdominal aortic surgery. *JAMA* 1999;281(14):1310-7.
623. Pronovost PJ, Angus DC, Dorman T, Robinson KA, Dremsizov TT, Young TL. Physician staffing patterns and clinical outcomes in critically ill patients: a systematic review. *JAMA* 2002;288(17):2151-62.
624. Pronovost PJ, Weast B, Holzmueller CG, et al. Evaluation of the culture of safety: survey of clinicians and managers in an academic medical center. *Qual Saf Health Care* 2003;12(6):405-10.
625. Nieva VF, Sorra J. Safety culture assessment: a tool for improving patient safety in healthcare organizations. *Qual Saf Health Care* 2003;12 Suppl 2:ii17-23.
626. Clarke SP, Rockett JL, Sloane DM, Aiken LH. Organizational climate, staffing, and safety equipment as predictors of needlestick injuries and near-misses in hospital nurses. *Am J Infect Control* 2002;30(4):207-16.
627. Rivers DL, Aday LA, Frankowski RF, Felknor S, White D, Nichols B. Predictors of nurses' acceptance of an intravenous catheter safety device. *Nurs Res* 2003;52(4):249-55.
628. Gershon RR, Karkashian CD, Grosch JW, et al. Hospital safety climate and its relationship with safe work practices and workplace exposure incidents. *Am J Infect Control* 2000;28(3):211-21.
629. Gershon RR, Vlahov D, Felknor SA, et al. Compliance with universal precautions among health care workers at three regional hospitals. *Am J Infect Control* 1995;23(4):225-36.
630. Michalsen A, Delclos GL, Felknor SA, et al. Compliance with universal precautions among physicians. *J Occup Environ Med* 1997;39(2):130-7.
631. Vaughn TE, McCoy KD, Beekmann SE, Woolson RE, Torner JC, Doebbeling BN. Factors promoting consistent adherence to safe needle precautions among hospital workers. *Infect Control Hosp Epidemiol* 2004;25(7):548-55.

632. Grosch JW, Gershon RR, Murphy LR, DeJoy DM. Safety climate dimensions associated with occupational exposure to blood-borne pathogens in nurses. *Am J Ind Med* 1999;Suppl 1:122-4.
633. Piotrowski MM, Hinshaw DB. The safety checklist program: creating a culture of safety in intensive care units. *Jt Comm J Qual Improv* 2002;28(6):306-15.
634. Weeks WB, Bagian JP. Developing a culture of safety in the Veterans Health Administration. *Eff Clin Pract* 2000;3(6):270-6.
635. Bagian JP, Gosbee JW. Developing a culture of patient safety at the VA. *Ambul Outreach* 2000:25-9.
636. Tokars JJ, McKinley GF, Otten J, et al. Use and efficacy of tuberculosis infection control practices at hospitals with previous outbreaks of multidrug-resistant tuberculosis. *Infect Control Hosp Epidemiol* 2001;22(7):449-55.
637. Maloney SA, Pearson ML, Gordon MT, Del Castillo R, Boyle JF, Jarvis WR. Efficacy of control measures in preventing nosocomial transmission of multidrug-resistant tuberculosis to patients and health care workers. *Ann Intern Med* 1995;122(2):90-5.
638. Montecalvo MA, Jarvis WR, Uman J, et al. Infection-control measures reduce transmission of vancomycin-resistant enterococci in an endemic setting. *Ann Intern Med* 1999;131(4):269-72.
639. Sherertz RJ, Ely EW, Westbrook DM, et al. Education of physicians-in-training can decrease the risk for vascular catheter infection. *Ann Intern Med* 2000;132(8):641-8.
640. Lynch P, Cummings MJ, Roberts PL, Herriott MJ, Yates B, Stamm WE. Implementing and evaluating a system of generic infection precautions: body substance isolation. *Am J Infect Control* 1990;18(1):1-12.
641. Kelen GD, DiGiovanna TA, Celentano DD, et al. Adherence to Universal (barrier) Precautions during interventions on critically ill and injured emergency department patients. *J Acquir Immune Defic Syndr* 1990;3(10):987-94.
642. Courington KR, Patterson SL, Howard RJ. Universal precautions are not universally followed. *Arch Surg* 1991;126(1):93-6.
643. Kaczmarek RG, Moore RM, Jr., McCrohan J, et al. Glove use by health care workers: results of a tristate investigation. *Am J Infect Control* 1991;19(5):228-32.
644. Freeman SW, Chambers CV. Compliance with universal precautions in a medical practice with a high rate of HIV infection. *J Am Board Fam Pract* 1992;5(3):313-8.

645. Friedland LR, Joffe M, Wiley JF, 2nd, Schapire A, Moore DF. Effect of educational program on compliance with glove use in a pediatric emergency department. *Am J Dis Child* 1992;146(11):1355-8.
646. Henry K, Campbell S, Maki M. A comparison of observed and self-reported compliance with universal precautions among emergency department personnel at a Minnesota public teaching hospital: implications for assessing infection control programs. *Ann Emerg Med* 1992;21(8):940-6.
647. Henry K, Campbell S, Collier P, Williams CO. Compliance with universal precautions and needle handling and disposal practices among emergency department staff at two community hospitals. *Am J Infect Control* 1994;22(3):129-37.
648. Eustis TC, Wright SW, Wrenn KD, Fowlie EJ, Slovis CM. Compliance with recommendations for universal precautions among prehospital providers. *Ann Emerg Med* 1995;25(4):512-5.
649. DiGiacomo JC, Hoff WS, Rotondo MF, et al. Barrier precautions in trauma resuscitation: real-time analysis utilizing videotape review. *Am J Emerg Med* 1997;15(1):34-9.
650. Thompson BL, Dwyer DM, Ussery XT, Denman S, Vacek P, Schwartz B. Handwashing and glove use in a long-term-care facility. *Infect Control Hosp Epidemiol* 1997;18(2):97-103.(s).
651. Helfgott AW, Taylor-Burton J, Garcini FJ, Eriksen NL, Grimes R. Compliance with universal precautions: knowledge and behavior of residents and students in a department of obstetrics and gynecology. *Infect Dis Obstet Gynecol* 1998;6(3):123-8.
652. Moore S, Goodwin H, Grossberg R, Toltzis P. Compliance with universal precautions among pediatric residents. *Arch Pediatr Adolesc Med* 1998;152(6):554-7.
653. Akduman D, Kim LE, Parks RL, et al. Use of personal protective equipment and operating room behaviors in four surgical subspecialties: personal protective equipment and behaviors in surgery. *Infect Control Hosp Epidemiol* 1999;20(2):110-4.
654. Brooks AJ, Phipson M, Potgieter A, Koertzen H, Boffard KD. Education of the trauma team: video evaluation of the compliance with universal barrier precautions in resuscitation. *Eur J Surg* 1999;165(12):1125-8.
655. Kidd F, Heitkemper P, Kressel AB. A comprehensive educational approach to improving patient isolation practice. *Clin Perform Qual Health Care* 1999;7(2):74-6.
656. Madan AK, Rentz DE, Wahle MJ, Flint LM. Noncompliance of health care workers with universal precautions during trauma resuscitations. *South Med J* 2001;94(3):277-80.

657. Madan AK, Raafat A, Hunt JP, Rentz D, Wahle MJ, Flint LM. Barrier precautions in trauma: is knowledge enough? *J Trauma* 2002;52(3):540-3.
658. Jeffe DB, Mutha S, Kim LE, Evanoff BA, Fraser VJ. Evaluation of a preclinical, educational and skills-training program to improve students' use of blood and body fluid precautions: one-year follow-up. *Prev Med* 1999;29(5):365-73.
659. Williams CO, Campbell S, Henry K, Collier P. Variables influencing worker compliance with universal precautions in the emergency department. *Am J Infect Control* 1994;22(3):138-48.
660. Larson E, McGeer A, Quraishi ZA, et al. Effect of an automated sink on handwashing practices and attitudes in high-risk units. *Infect Control Hosp Epidemiol* 1991;12(7):422-8.
661. Swoboda SM, Earsing K, Strauss K, Lane S, Lipsett PA. Electronic monitoring and voice prompts improve hand hygiene and decrease nosocomial infections in an intermediate care unit. *Crit Care Med* 2004;32(2):358-63.
662. Kretzer EK, Larson EL. Behavioral interventions to improve infection control practices. *Am J Infect Control* 1998;26(3):245-53.
663. CDC. Updated Guidelines for Evaluating Public Health Surveillance Systems. Recommendations from the Guidelines Working Group. *MMWR Recomm Rep* 2001;50(RR-13):1-35.
664. Semmelweis IP. Die aetiologie, der begriff und die prophylaxis des kindbettfiebers. Pest, Wein, und Leipzig: CA Harleben's Verlags-Expedition 1861.
665. Bratzler DW, Houck PM. Antimicrobial prophylaxis for surgery: an advisory statement from the National Surgical Infection Prevention Project. *Clin Infect Dis* 2004 Jun 15;38:1706-15.
666. Bloom BT, Craddock A, Delmore PM, et al. Reducing acquired infections in the NICU: observing and implementing meaningful differences in process between high and low acquired infection rate centers. *J Perinatol* 2003;23:489-92.
667. Braun BI, Kritchevsky SB, Wong ES, et al. Preventing central venous catheter-associated primary bloodstream infections: characteristics of practices among hospitals participating in the Evaluation of Processes and Indicators in Infection Control (EPIC) study. *Infect Control Hosp Epidemiol* 2003;24(12):926-35.
668. Baker OG. Process surveillance: an epidemiologic challenge for all health care organizations. *AJIC* 1997;25:96-101.
669. Loeb M, McGeer A, McArthur M, Peeling RW, Petric M, Simor AE. Surveillance for outbreaks of respiratory tract infections in nursing homes. *Cmaj* 2000;162(8):1133-7.

670. Nicolle LE. Preventing infections in non-hospital settings: long-term care. *Emerg Infect Dis* 2001;7(2):205-7.
671. Pottinger JM, Herwaldt LA, Perl TM. Basics of surveillance--an overview. *Infect Control Hosp Epidemiol* 1997;18(7):513-27.
672. Lee TB, Baker OG, Lee JT, Scheckler WE, Steele L, Laxton CE. Recommended practices for surveillance. Association for Professionals in Infection Control and Epidemiology, Inc. Surveillance Initiative working Group. *Am J Infect Control* 1998;26(3):277-88.
673. Haley RW. The scientific basis for using surveillance and risk factor data to reduce nosocomial infection rates. *J Hosp Infect* 1995;30 Suppl:3-14.
674. Benneyan JC, Lloyd RC, Plsek PE. Statistical process control as a tool for research and healthcare improvement. *Qual Saf Health Care* 2003;12(6):458-64.
675. Lemmen SW, Zolldann D, Gastmeier P, Lutticken R. Implementing and evaluating a rotating surveillance system and infection control guidelines in 4 intensive care units. *Am J Infect Control* 2001;29(2):89-93.
676. Gaynes R, Richards C, Edwards J, et al. Feeding back surveillance data to prevent hospital-acquired infections. *Emerg Infect Dis* 2001;7(2):295-8.
677. Tokars JI, Richards C, Andrus M, et al. The changing face of surveillance for health care-associated infections. *Clin Infect Dis* 2004;39:1347-52.
678. Sands KE, Yokoe DS, Hooper DC, et al. Detection of postoperative surgical-site infections: comparison of health plan-based surveillance with hospital-based programs. *Infect Control Hosp Epidemiol* 2003;24(10):741-3.
679. Jodra VM, Rodela AR, Martinez EM, Fresnena NL. Standardized infection ratios for three general surgery procedures: a comparison between Spanish hospitals and U.S. centers participating in the National Nosocomial Infections Surveillance System. *Infect Control Hosp Epidemiol* 2003;24(10):744-8.
680. McKibben L, Horan T, Tokars JI, et al. Guidance on public reporting of healthcare-associated infections: recommendations of the Healthcare Infection Control Practices Advisory Committee. *Am J Infect Control* 2005;33(4):217-26.
681. Gould D, Chamberlain A. The use of a ward-based educational teaching package to enhance nurses' compliance with infection control procedures. *J Clin Nurs* 1997;6(1):55-67.
682. Calabro K, Weltge A, Parnell S, Kouzekanani K, Ramirez E. Intervention for medical students: effective infection control. *Am J Infect Control* 1998;26(4):431-6.

683. Haiduven DJ, Hench CP, Simpkins SM, Stevens DA. Standardized management of patients and employees exposed to pertussis. *Infect Control Hosp Epidemiol* 1998;19(11):861-4.
684. Macartney KK, Gorelick MH, Manning ML, Hodinka RL, Bell LM. Nosocomial respiratory syncytial virus infections: the cost- effectiveness and cost-benefit of infection control. *Pediatrics* 2000;106(3):520-6.
685. Beekmann SE, Vaughn TE, McCoy KD, et al. Hospital bloodborne pathogens programs: program characteristics and blood and body fluid exposure rates. *Infect Control Hosp Epidemiol* 2001;22(2):73-82.
686. Sokas RK, Simmens S, Scott J. A training program in universal precautions for second-year medical students. *Acad Med* 1993;68(5):374-6.
687. Ostrowsky BE, Trick WE, Sohn AH, et al. Control of vancomycin-resistant enterococcus in health care facilities in a region. *N Engl J Med* 2001;344(19):1427-33.
688. Bonten MJ, Kollef MH, Hall JB. Risk factors for ventilator-associated pneumonia: from epidemiology to patient management. *Clin Infect Dis* 2004;38(8):1141-9.
689. Lau JT, Fung KS, Wong TW, et al. SARS transmission among hospital workers in Hong Kong. *Emerg Infect Dis* 2004;10(2):280-6.
690. Talbot TR, Bradley SE, Cosgrove SE, Ruef C, Siegel JD, Weber DJ. Influenza vaccination of healthcare workers and vaccine allocation for healthcare workers during vaccine shortages. *Infect Control Hosp Epidemiol* 2005;26(11):882-90.
691. Harbarth S, Siegrist CA, Schira JC, Wunderli W, Pittet D. Influenza immunization: improving compliance of healthcare workers. *Infect Control Hosp Epidemiol* 1998;19(5):337-42.
692. Bryant KA, Stover B, Cain L, Levine GL, Siegel J, Jarvis WR. Improving influenza immunization rates among healthcare workers caring for high-risk pediatric patients. *Infect Control Hosp Epidemiol* 2004;25(11):912-7.
693. Martinello RA, Jones L, Topal JE. Correlation between healthcare workers' knowledge of influenza vaccine and vaccine receipt. *Infect Control Hosp Epidemiol* 2003;24(11):845-7.
694. Goldrick B, Gruendemann B, Larson E. Learning styles and teaching/learning strategy preferences: implications for educating nurses in critical care, the operating room, and infection control. *Heart Lung* 1993;22(2):176-82.
695. Davis D, O'Brien MA, Freemantle N, Wolf FM, Mazmanian P, Taylor-Vaisey A. Impact of formal continuing medical education: do conferences, workshops, rounds, and other traditional continuing education activities

change physician behavior or health care outcomes? JAMA
1999;282(9):867-74.

696. Carr H, and Hinson P. Education and Training. ed. APIC Text of Infection Control and Epidemiology. 2nd edition. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc. (APIC); pp. 11-1; 2005.
697. Caffarella RS. Planning Programs for Adult Learners: A Practical Guide for Educators, Trainers, and Staff Developers, Second Edition. In. San Francisco: Jossey-Bass; 2001.
698. Sargeant J, Curran V, Jarvis-Selinger S, et al. Interactive on-line continuing medical education: physicians' perceptions and experiences. J Contin Educ Health Prof 2004;24(4):227-36.
699. Van Harrison R. Systems-based framework for continuing medical education and improvements in translating new knowledge into physicians' practices. J Contin Educ Health Prof 2004;24 Suppl 1:S50-62.
700. Cole TB, Glass RM. Learning associated with participation in journal-based continuing medical education. J Contin Educ Health Prof 2004;24(4):205-12.
701. Diekema DJ, Albanese MA, Schuldt SS, Doebbeling BN. Blood and body fluid exposures during clinical training: relation to knowledge of universal precautions. J Gen Intern Med 1996;11(2):109-11.
702. Diekema DJ, Schuldt SS, Albanese MA, Doebbeling BN. Universal precautions training of preclinical students: impact on knowledge, attitudes, and compliance. Prev Med 1995;24(6):580-5.
703. Warren DK, Zack JE, Cox MJ, Cohen MM, Fraser VJ. An educational intervention to prevent catheter-associated bloodstream infections in a nonteaching, community medical center. Crit Care Med 2003;31(7):1959-63.
704. Dubbert PM, Dolce J, Richter W, Miller M, Chapman SW. Increasing ICU staff handwashing: effects of education and group feedback. Infect Control Hosp Epidemiol 1990;11(4):191-3.
705. Avila-Aguero ML, Umana MA, Jimenez AL, Faingezicht I, Paris MM. Handwashing practices in a tertiary-care, pediatric hospital and the effect on an educational program. Clin Perform Qual Health Care 1998;6(2):70-2.
706. Lai KK, Fontecchio SA, Kelley AL, Melvin ZS. Knowledge of the transmission of tuberculosis and infection control measures for tuberculosis among healthcare workers. Infect Control Hosp Epidemiol 1996;17(3):168-70.
707. Koenig S, Chu J. Senior medical students' knowledge of universal precautions. Acad Med 1993;68(5):372-4.

708. Babcock HM, Zack JE, Garrison T, et al. An educational intervention to reduce ventilator-associated pneumonia in an integrated health system: a comparison of effects. *Chest* 2004;125(6):2224-31.
709. McGuckin M, Taylor A, Martin V, Porten L, Salcido R. Evaluation of a patient education model for increasing hand hygiene compliance in an inpatient rehabilitation unit. *Am J Infect Control* 2004;32(4):235-8.
710. Cirone N. Patient-education handbook. *Nursing* 1997;27(8):44-5.
711. Chase TM. Learning styles and teaching strategies: enhancing the patient education experience. *SCI Nurse* 2001;18:138-41.
712. Jarvis WR. Handwashing--the Semmelweis lesson forgotten? *Lancet* 1994;344(8933):1311-2.
713. Daniels IR, Rees BI. Handwashing: simple, but effective. *Ann R Coll Surg Engl* 1999;81:117-8.
714. Webster J, Faoagali JL, Cartwright D. Elimination of methicillin-resistant *Staphylococcus aureus* from a neonatal intensive care unit after hand washing with triclosan. *J Paediatr Child Health* 1994;30(1):59-64.
715. Zafar AB, Butler RC, Reese DJ, Gaydos LA, Mennonna PA. Use of 0.3% triclosan (Bacti-Stat) to eradicate an outbreak of methicillin-resistant *Staphylococcus aureus* in a neonatal nursery. *Am J Infect Control* 1995;23(3):200-8.
716. Malik RK, Montecalvo MA, Reale MR, et al. Epidemiology and control of vancomycin-resistant enterococci in a regional neonatal intensive care unit. *Pediatr Infect Dis J* 1999;18(4):352-6.
717. Pittet D, Boyce JM. Hand hygiene and patient care: pursuing the Semmelweis legacy. *Lancet Infect Dis* 2001:9-20.
718. Lin CM, Wu FM, Kim HK, Doyle MP, Michael BS, Williams LK. A comparison of hand washing techniques to remove *Escherichia coli* and caliciviruses under natural or artificial fingernails. *J Food Prot* 2003;66(12):2296-301.
719. Edel E, Houston S, Kennedy V, LaRocco M. Impact of a 5-minute scrub on the microbial flora found on artificial, polished, or natural fingernails of operating room personnel. *Nurs Res* 1998;47(1):54-9.
720. Pottinger J, Burns S, Manske C. Bacterial carriage by artificial versus natural nails. *Am J Infect Control* 1989;17(6):340-4.
721. Hedderwick SA, McNeil SA, Lyons MJ, Kauffman CA. Pathogenic organisms associated with artificial fingernails worn by healthcare workers. *Infect Control Hosp Epidemiol* 2000;21(8):505-9.
722. Passaro DJ, Waring L, Armstrong R, et al. Postoperative *Serratia marcescens* wound infections traced to an out-of-hospital source. *J Infect Dis* 1997;175(4):992-5.

723. Moolenaar RL, Crutcher JM, San Joaquin VH, et al. A prolonged outbreak of *Pseudomonas aeruginosa* in a neonatal intensive care unit: did staff fingernails play a role in disease transmission? *Infect Control Hosp Epidemiol* 2000;21(2):80-5.
724. Parry MF, Grant B, Yukna M, et al. *Candida* osteomyelitis and diskitis after spinal surgery: an outbreak that implicates artificial nail use. *Clin Infect Dis* 2001;32(3):352-7.
725. Boszczowski I, Nicoletti C, Puccini DM, et al. Outbreak of extended spectrum beta-lactamase-producing *Klebsiella pneumoniae* infection in a neonatal intensive care unit related to onychomycosis in a health care worker. *Pediatr Infect Dis J* 2005;24(7):648-50.
726. Trick WE, Vernon MO, Hayes RA, et al. Impact of ring wearing on hand contamination and comparison of hand hygiene agents in a hospital. *Clin Infect Dis* 2003;36(11):1383-90.
727. Pittet D, Dharan S, Touveneau S, Sauvan V, Perneger TV. Bacterial contamination of the hands of hospital staff during routine patient care. *Arch Intern Med* 1999;159(8):821-6.
728. Tenorio AR, Badri SM, Sahgal NB, et al. Effectiveness of gloves in the prevention of hand carriage of vancomycin-resistant enterococcus species by health care workers after patient care. *Clin Infect Dis* 2001;32(5):826-9.(s).
729. Mast ST, Woolwine JD, Gerberding JL. Efficacy of gloves in reducing blood volumes transferred during simulated needlestick injury. *J Infect Dis* 1993;168(6):1589-92.
730. Medical Glove Guidance Manual.
www.fda.gov/cdrh/dsma/gloveman/gloveman99.pdf
731. Korniewicz DM, El-Masri M, Broyles JM, Martin CD, O'Connell K P. Performance of latex and nonlatex medical examination gloves during simulated use. *Am J Infect Control* 2002;30(2):133-8.
732. Korniewicz DM, McLeskey SW. Latex allergy and gloving standards. *Semin Perioper Nurs* 1998;7(4):216-21.
733. Ranta PM, Ownby DR. A review of natural-rubber latex allergy in health care workers. *Clin Infect Dis* 2004;38(2):252-6.
734. Korniewicz DM, Kirwin M, Cresci K, et al. Barrier protection with examination gloves: double versus single. *Am J Infect Control* 1994;22(1):12-5.
735. Korniewicz DM, Kirwin M, Cresci K, Larson E. Leakage of latex and vinyl exam gloves in high and low risk clinical settings. *Am Ind Hyg Assoc J* 1993;54(1):22-6.
736. Rego A, Roley L. In-use barrier integrity of gloves: latex and nitrile superior to vinyl. *Am J Infect Control* 1999;27(5):405-10.

737. Kotilainen HR, Brinker JP, Avato JL, Gantz NM. Latex and vinyl examination gloves. Quality control procedures and implications for health care workers. *Arch Intern Med* 1989;149(12):2749-53.
738. Korniewicz DM, Laughon BE, Butz A, Larson E. Integrity of vinyl and latex procedure gloves. *Nurs Res* 1989;38(3):144-6.
739. OSHA. OSHA. Department of Labor: Occupational Safety and Health Administration. Occupational exposure to bloodborne pathogens: Final rule. 29 CFR Part 1910:1030 *Federal Register* 1991;56:64003-64182 Revised 2001 CFR 66 2001:5317-25.
740. CDC. Recommendations for preventing the spread of vancomycin resistance. Recommendations of the Hospital Infection Control Practices Advisory Committee (HICPAC). *MMWR Recomm Rep* 1995;44 (RR-12):1-13.
741. Olsen RJ, Lynch P, Coyle MB, Cummings J, Bokete T, Stamm WE. Examination gloves as barriers to hand contamination in clinical practice. *JAMA* 1993;270(3):350-3.
742. Doebbeling BN, Pfaller MA, Houston AK, Wenzel RP. Removal of nosocomial pathogens from the contaminated glove. Implications for glove reuse and handwashing. *Ann Intern Med* 1988;109(5):394-8.
743. Maki DG, McCormick RD, Zilz MA, et al. A MRSA outbreak in an SICU during universal precautions: new epidemiology for nosocomial MRSA. Abstract # 473 Presented at the 30th Annual Meeting of the Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC), Chicago, Illinois October 21-24, 1990.
744. Boyce JM, Jackson MM, Pugliese G, et al. Methicillin-resistant *Staphylococcus aureus* (MRSA): a briefing for acute care hospitals and nursing facilities. The AHA Technical Panel on Infections Within Hospitals. *Infect Control Hosp Epidemiol* 1994;15(2):105-15.
745. Boyce JM, Mermel LA, Zervos MJ, et al. Controlling vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 1995;16(11):634-7.
746. Gerding DN, Johnson S, Peterson LR, Mulligan ME, Silva J, Jr. *Clostridium difficile*-associated diarrhea and colitis. *Infect Control Hosp Epidemiol* 1995;16(8):459-77.
747. Cloney DL, Donowitz LG. Overgrown use for infection control in nurseries and neonatal intensive care units. *Am J Dis Child* 1986;140(7):680-3.
748. Pelke S, Ching D, Easa D, Melish ME. Gowning does not affect colonization or infection rates in a neonatal intensive care unit. *Arch Pediatr Adolesc Med* 1994;148(10):1016-20.
749. Slaughter S, Hayden MK, Nathan C, et al. A comparison of the effect of universal use of gloves and gowns with that of glove use alone on

- acquisition of vancomycin-resistant enterococci in a medical intensive care unit. *Ann Intern Med* 1996;125(6):448-56.
750. Duquette-Petersen L, Francis ME, Dohnalek L, Skinner R, Dudas P. The role of protective clothing in infection prevention in patients undergoing autologous bone marrow transplantation. *Oncol Nurs Forum* 1999;26(8):1319-24. <http://www.ons.org>.
751. Sartori M, La Terra G, Aglietta M, Manzin A, Navino C, Verzetti G. Transmission of hepatitis C via blood splash into conjunctiva. *Scand J Infect Dis* 1993;25(2):270-1.
752. Hosoglu S, Celen MK, Akalin S, Geyik MF, Soyoral Y, Kara IH. Transmission of hepatitis C by blood splash into conjunctiva in a nurse. *Am J Infect Control* 2003;31(8):502-4.
753. CDC. Update: human immunodeficiency virus infections in health-care workers exposed to blood of infected patients. *MMWR Morb Mortal Wkly Rep* 1987;36(19):285-9.
754. Keijman J, Tjhie J, Olde Damink S, Alink M. Unusual nosocomial transmission of *Mycobacterium tuberculosis*. *Eur J Clin Microbiol Infect Dis* 2001;20(11):808-9.
755. Weaver GH. Value of the face mask and other measures. *JAMA* 1918;70:76.
756. Weaver GH. Droplet infection and its prevention by the face mask. *J Infect Dis* 1919;24:218-30.
757. Davidson IR, Crisp AJ, Hinwood DC, Whitaker SC, Gregson RH. Eye splashes during invasive vascular procedures. *Br J Radiol* 1995;68(805):39-41.
758. Guidance for Industry and FDA Staff - Surgical Masks - Premarket Notification [510(k)] Submissions; Guidance for Industry and FDA. <http://www.fda.gov/cdrh/ode/guidance/094.html>
759. National Institute for Occupational Health and Safety - Eye Protection for Infection Control. <http://www.cdc.gov/niosh/topics/eye/eye-infectious.html>
760. Gala CL, Hall CB, Schnabel KC, et al. The use of eye-nose goggles to control nosocomial respiratory syncytial virus infection. *Jama* 1986;256(19):2706-8.
761. Agah R, Cherry JD, Garakian AJ, Chapin M. Respiratory syncytial virus (RSV) infection rate in personnel caring for children with RSV infections. Routine isolation procedure vs routine procedure supplemented by use of masks and goggles. *Am J Dis Child* 1987;141(6):695-7.
762. Thorburn K, Kerr S, Taylor N, van Saene HK. RSV outbreak in a paediatric intensive care unit. *J Hosp Infect* 2004;57(3):194-201.

763. <http://a257.g.akamaitech.net/7/257/2422/06jun20041800/edocket.access.gpo.gov/2004/04-25183.htm>
764. Occupational Safety & Health Administration - Respiratory Protection. www.osha.gov/dcsp/ote/trng-materials/respirators/respirators.html
765. Campbell DL, Coffey CC, Lenhart SW. Respiratory protection as a function of respirator fitting characteristics and fit-test accuracy. *Aihaj* 2001;62(1):36-44.
766. Lee K, Slavcev A, Nicas M. Respiratory protection against *Mycobacterium tuberculosis*: quantitative fit test outcomes for five type N95 filtering-facepiece respirators. *J Occup Environ Hyg* 2004;1(1):22-8.
767. Coffey CC, Campbell DL, Zhuang Z. Simulated workplace performance of N95 respirators. *Am Ind Hyg Assoc J* 1999;60(5):618-24.
768. Coffey CC, Lawrence RB, Zhuang Z, Campbell DL, Jensen PA, Myers WR. Comparison of five methods for fit-testing N95 filtering-facepiece respirators. *Appl Occup Environ Hyg* 2002;17(10):723-30.
769. National Personal Protective Technology Laboratory. www.cdc.gov/niosh/npptl/.
770. McGowan JE, Jr. Nosocomial tuberculosis: new progress in control and prevention. *Clin Infect Dis* 1995;21(3):489-505.
771. Jarvis WR. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*. *Am J Infect Control* 1995;23(2):146-51.
772. CDC. Emergency Preparedness & Response. www.btcdcgov 2003.
773. Anderson JD, Bonner M, Scheifele DW, Schneider BC. Lack of nosocomial spread of Varicella in a pediatric hospital with negative pressure ventilated patient rooms. *Infect Control* 1985;6(3):120-1.
774. Brunell PA, Wood D. Varicella serological status of healthcare workers as a guide to whom to test or immunize. *Infect Control Hosp Epidemiol* 1999;20(5):355-7.
775. Saiman L, LaRussa P, Steinberg SP, et al. Persistence of immunity to varicella-zoster virus after vaccination of healthcare workers. *Infect Control Hosp Epidemiol* 2001;22(5):279-83.
776. Willy ME, Koziol DE, Fleisher T, et al. Measles immunity in a population of healthcare workers. *Infect Control Hosp Epidemiol* 1994;15(1):12-7.
777. Wright LJ, Carlquist JF. Measles immunity in employees of a multihospital healthcare provider. *Infect Control Hosp Epidemiol* 1994;15(1):8-11.
778. CDC. Updated U.S. Public Health Service Guidelines for the Management of Occupational Exposures to HBV, HCV, and HIV. *MMWR Recomm Rep* 2001;50 (RR-11):1-52.

779. Do AN, Ciesielski CA, Metler RP, Hammett TA, Li J, Fleming PL. Occupationally acquired human immunodeficiency virus (HIV) infection: national case surveillance data during 20 years of the HIV epidemic in the United States. *Infect Control Hosp Epidemiol* 2003;24(2):86-96.
780. CDC. Update: universal precautions for prevention of transmission of human immunodeficiency virus, hepatitis B virus, and other bloodborne pathogens in health-care settings. *MMWR Morb Mortal Wkly Rep* 1988;37(24):377-82, 87-8.
781. Davis MS. Occupational hazards of operating: opportunities for improvement. *Infect Control Hosp Epidemiol* 1996;17(10):691-3.
782. Gerberding JL. Procedure-specific infection control for preventing intraoperative blood exposures. *Am J Infect Control* 1993;21(6):364-7.
783. Fry DE, Telford GL, Fecteau DL, Sperling RS, Meyer AA. Prevention of blood exposure. Body and facial protection. *Surg Clin North Am* 1995;75(6):1141-57.
784. Hansen ME. Bloodborne pathogens and procedure safety in interventional radiology. *Semin Ultrasound CT MR* 1998;19(2):209-14.
785. Holodnick CL, Barkauskas V. Reducing percutaneous injuries in the OR by educational methods. *Aorn J* 2000;72(3):461-4, 8-72, 75-6.
786. www.osha.gov/SLTC/bloodbornepathogens/index.html
787. www.cdc.gov/niosh/2000-108.html
788. National Institute for Occupational Health and Safety - Safer Medical Device Implementation in Health Care Facilities.
<http://www.cdc.gov/niosh/topics/bbp/safer/>
789. www.cdc.gov/sharpssafety/resources.html.
790. Catanzaro A. Nosocomial tuberculosis. *Am Rev Respir Dis* 1982;125(5):559-62.
791. Cepeda JA, Whitehouse T, Cooper B, et al. Isolation of patients in single rooms or cohorts to reduce spread of MRSA in intensive-care units: prospective two-centre study. *Lancet* 2005;365(9456):295-304.
792. Mulin B, Rouget C, Clement C, et al. Association of private isolation rooms with ventilator-associated *Acinetobacter baumannii* pneumonia in a surgical intensive-care unit. *Infect Control Hosp Epidemiol* 1997;18(7):499-503.
793. www.aia.org/aah_gd_hospcons.
794. Raad I, Abbas J, Whimbey E. Infection control of nosocomial respiratory viral disease in the immunocompromised host. *Am J Med* 1997;102(3A):48-52; discussion 3-4.

795. Isaacs D, Dickson H, O'Callaghan C, Sheaves R, Winter A, Moxon ER. Handwashing and cohorting in prevention of hospital acquired infections with respiratory syncytial virus. *Arch Dis Child* 1991;66(2):227-31.
796. Chang VT, Nelson K. The role of physical proximity in nosocomial diarrhea. *Clin Infect Dis* 2000;31(3):717-22.
797. Byers KE, Anglim AM, Anneski CJ, et al. A hospital epidemic of vancomycin-resistant *Enterococcus*: risk factors and control. *Infect Control Hosp Epidemiol* 2001;22(3):140-7.
798. Dassut B. The implementation of a commode cleaning and identification system. *Nurs Times* 2004;100(8):47.
799. Mayer RA, Geha RC, Helfand MS, Hoyen CK, Salata RA, Donskey CJ. Role of fecal incontinence in contamination of the environment with vancomycin-resistant enterococci. *Am J Infect Control* 2003;31(4):221-5.
800. Samore MH, Venkataraman L, DeGirolami PC, Arbeit RD, Karchmer AW. Clinical and molecular epidemiology of sporadic and clustered cases of nosocomial *Clostridium difficile* diarrhea. *Am J Med* 1996;100(1):32-40.
801. Clabots CR, Johnson S, Olson MM, Peterson LR, Gerding DN. Acquisition of *Clostridium difficile* by hospitalized patients: evidence for colonized new admissions as a source of infection. *J Infect Dis* 1992;166(3):561-7.
802. Samore MH. Epidemiology of nosocomial *clostridium difficile* diarrhoea. *J Hosp Infect* 1999;43 Suppl:S183-90.
803. Tokars JI, Satake S, Rimland D, et al. The prevalence of colonization with vancomycin-resistant *Enterococcus* at a Veterans' Affairs institution. *Infect Control Hosp Epidemiol* 1999;20(3):171-5.
804. Cone R, Mohan K, Thouless M, Corey L. Nosocomial transmission of rotavirus infection. *Pediatr Infect Dis J* 1988;7(2):103-9.
805. Bruce BB, Blass MA, Blumberg HM, Lennox JL, del Rio C, Horsburgh CR, Jr. Risk of *Cryptosporidium parvum* transmission between hospital roommates. *Clin Infect Dis* 2000;31(4):947-50.
806. Ford-Jones EL, Mindorff CM, Gold R, Petric M. The incidence of viral-associated diarrhea after admission to a pediatric hospital. *Am J Epidemiol* 1990;131(4):711-8.
807. Murray-Leisure KA, Geib S, Graceley D, et al. Control of epidemic methicillin-resistant *Staphylococcus aureus*. *Infect Control Hosp Epidemiol* 1990;11(7):343-50.
808. Jochimsen EM, Fish L, Manning K, et al. Control of vancomycin-resistant enterococci at a community hospital: efficacy of patient and staff cohorting. *Infect Control Hosp Epidemiol* 1999;20(2):106-9.

809. Sample ML, Gravel D, Oxley C, Toye B, Garber G, Ramotar K. An outbreak of vancomycin-resistant enterococci in a hematology-oncology unit: control by patient cohorting and terminal cleaning of the environment. *Infect Control Hosp Epidemiol* 2002;23(8):468-70.
810. Podnos YD, Cinat ME, Wilson SE, Cooke J, Gornick W, Thrupp LD. Eradication of multi-drug resistant *Acinetobacter* from an Intensive Care Unit. *Surgical Infections* 2001;2(2):297-301.
811. Graham PL, 3rd, Morel AS, Zhou J, et al. Epidemiology of methicillin-susceptible *Staphylococcus aureus* in the neonatal intensive care unit. *Infect Control Hosp Epidemiol* 2002;23(11):677-82.
812. Doherty JA, Brookfield DS, Gray J, McEwan RA. Cohorting of infants with respiratory syncytial virus. *J Hosp Infect* 1998;38(3):203-6.
813. Hall CB, Geiman JM, Douglas RG, Jr., Meagher MP. Control of nosocomial respiratory syncytial viral infections. *Pediatrics* 1978;62(5):728-32.
814. Buffington J, Chapman LE, Stobierski MG, et al. Epidemic keratoconjunctivitis in a chronic care facility: risk factors and measures for control. *J Am Geriatr Soc* 1993;41(11):1177-81.
815. Grehn M, Kunz J, Sigg P, Slongo R, Zbinden R. Nosocomial rotavirus infections in neonates: means of prevention and control. *J Perinat Med* 1990;18(5):369-74.
816. Tan YM, Chow PK, Tan BH, et al. Management of inpatients exposed to an outbreak of severe acute respiratory syndrome (SARS). *J Hosp Infect* 2004;58(3):210-5.
817. Talon D, Vichard P, Muller A, Bertin M, Jeunet L, Bertrand X. Modelling the usefulness of a dedicated cohort facility to prevent the dissemination of MRSA. *J Hosp Infect* 2003;54(1):57-62.
818. Hotchkiss JR, Strike DG, Simonson DA, Broccard AF, Crooke PS. An agent-based and spatially explicit model of pathogen dissemination in the intensive care unit. *Crit Care Med* 2005;33(1):168-76; discussion 253-4.
819. Austin DJ, Bonten MJ, Weinstein RA, Slaughter S, Anderson RM. Vancomycin-resistant enterococci in intensive-care hospital settings: transmission dynamics, persistence, and the impact of infection control programs. *Proc Natl Acad Sci U S A* 1999;96(12):6908-13.
820. Kovner CT, Harrington C. Counting nurses. Data show many nursing homes to be short staffed. *Am J Nurs* 2000;100(9):53-4.
821. Mueller C. Staffing problems in long-term care. Let's do something about it! *J Gerontol Nurs* 2003;29(3):3-4.
822. Stats & facts. Nursing staff shortages in long-term care facilities. *Manag Care Interface* 2000;13(11):46-7.

823. Mejias A, Chavez-Bueno S, Ramilo O. Human metapneumovirus: a not so new virus. *Pediatr Infect Dis J* 2004;23(1):1-7; quiz 8-10.
824. Iwane MK, Edwards KM, Szilagyi PG, et al. Population-based surveillance for hospitalizations associated with respiratory syncytial virus, influenza virus, and parainfluenza viruses among young children. *Pediatrics* 2004;113(6):1758-64.
825. Ong GM, Wyatt DE, O'Neill HJ, McCaughey C, Coyle PV. A comparison of nested polymerase chain reaction and immunofluorescence for the diagnosis of respiratory infections in children with bronchiolitis, and the implications for a cohorting strategy. *J Hosp Infect* 2001;49(2):122-8.
826. von Linstow ML, Larsen HH, Eugen-Olsen J, et al. Human metapneumovirus and respiratory syncytial virus in hospitalized danish children with acute respiratory tract infection. *Scan J Infect Dis* 2004;36:578-84.
827. Gehanno JF, Pestel-Caron M, Nouvellon M, Caillard JF. Nosocomial pertussis in healthcare workers from a pediatric emergency unit in France. *Infect Control Hosp Epidemiol* 1999;20(8):549-52.
828. www.cdc.gov/flu/professionals/infectioncontrol/resphygiene.htm.
829. Edlin BR, Tokars JI, Grieco MH, et al. An outbreak of multidrug-resistant tuberculosis among hospitalized patients with the acquired immunodeficiency syndrome. *N Engl J Med* 1992;326(23):1514-21.
830. CDC. Update: Severe Acute Respiratory Syndrome --- Toronto, Canada, 2003. *MMWR* 2003;52(23):547-50.
831. Starke JR. Transmission of *Mycobacterium tuberculosis* to and from children and adolescents. *Seminars Pedi Infect Dis* 2001;12:115-23.
832. Saiman L, Macdonald N, Burns JL, Hoiby N, Speert DP, Weber D. Infection control in cystic fibrosis: practical recommendations for the hospital, clinic, and social settings. *Am J Infect Control* 2000;28(5):381-5.(s).
833. COID. 2003 Report of the Committee on Infectious Diseases. In: Redbook. Elk Grove Village, IL: American Academy of Pediatrics; 2003.
834. Lau JT, Lau M, Kim JH, Tsui HY, Tsang T, Wong TW. Probable secondary infections in households of SARS patients in Hong Kong. *Emerg Infect Dis* 2004;10(2):235-43.
835. Hota B. Contamination, disinfection, and cross-colonization: are hospital surfaces reservoirs for nosocomial infection? *Clin Infect Dis* 2004;39(8):1182-9.
836. Rutala WA, Weber DJ. Disinfection and sterilization in health care facilities: what clinicians need to know. *Clin Infect Dis* 2004;39(5):702-9.

837. Boyce JM, Opal SM, Chow JW, et al. Outbreak of multidrug-resistant *Enterococcus faecium* with transferable vanB class vancomycin resistance. *J Clin Microbiol* 1994;32(5):1148-53.
838. Engelhart S, Krizek L, Glasmacher A, Fischnaller E, Marklein G, Exner M. *Pseudomonas aeruginosa* outbreak in a haematology-oncology unit associated with contaminated surface cleaning equipment. *J Hosp Infect* 2002;52(2):93-8.
839. Denton M, Wilcox MH, Parnell P, et al. Role of environmental cleaning in controlling an outbreak of *Acinetobacter baumannii* on a neurosurgical intensive care unit. *J Hosp Infect* 2004;56(2):106-10.
840. Hollyoak V, Allison D, Summers J. *Pseudomonas aeruginosa* wound infection associated with a nursing home's whirlpool bath. *Commun Dis Rep CDR Rev* 1995;5(7):R100-2.
841. Malik RE, Cooper RA, Griffith CJ. Use of audit tools to evaluate the efficacy of cleaning systems in hospitals. *Am J Infect Control* 2003;31(3):181-7.
842. Ansari SA, Springthorpe VS, Sattar SA. Survival and vehicular spread of human rotaviruses: possible relation to seasonality of outbreaks. *Rev Infect Dis* 1991;13(3):448-61.
843. Kaatz GW, Gitlin SD, Schaberg DR, et al. Acquisition of *Clostridium difficile* from the hospital environment. *Am J Epidemiol* 1988;127(6):1289-94.
844. Mayfield JL, Leet T, Miller J, Mundy LM. Environmental control to reduce transmission of *Clostridium difficile*. *Clin Infect Dis* 2000;31(4):995-1000.
845. Dennehy PH. Transmission of rotavirus and other enteric pathogens in the home. *Pediatr Infect Dis J* 2000;19(10 Suppl):S103-5.
846. Dennehy P. Rotavirus infections in infection control reference service. In: Abrutyn E, Goldmann D, Scheckler W, eds. Philadelphia: WE Saunders; 2001:821-3.
847. Wilcox MH, Fawley WN, Wigglesworth N, Parnell P, Verity P, Freeman J. Comparison of the effect of detergent versus hypochlorite cleaning on environmental contamination and incidence of *Clostridium difficile* infection. *J Hosp Infect* 2003;54(2):109-14.
848. Rutala WA, Weber DJ, Committee HICPA. Guideline for Disinfection and Sterilization in Health-Care Facilities 2007 (in press).
849. Bernards AT, Harinck HI, Dijkshoorn L, van der Reijden TJ, van den Broek PJ. Persistent *Acinetobacter baumannii*? Look inside your medical equipment. *Infect Control Hosp Epidemiol* 2004;25(11):1002-4.

850. Neely AN, Weber JM, Daviau P, et al. Computer equipment used in patient care within a multihospital system: recommendations for cleaning and disinfection. *Am J Infect Control* 2005;33(4):233-7.
851. Neely AN, Maley MP, Warden GD. Computer keyboards as reservoirs for *Acinetobacter baumannii* in a burn hospital. *Clin Infect Dis* 1999;29(5):1358-60.
852. Bures S, Fishbain JT, Uyehara CF, Parker JM, Berg BW. Computer keyboards and faucet handles as reservoirs of nosocomial pathogens in the intensive care unit. *Am J Infect Control* 2000;28(6):465-71.
853. Brooks S, Khan A, Stoica D, et al. Reduction in vancomycin-resistant *Enterococcus* and *Clostridium difficile* infections following change to tympanic thermometers. *Infect Control Hosp Epidemiol* 1998;19(5):333-6.
854. Jernigan JA, Siegman-Igra Y, Guerrant RC, Farr BM. A randomized crossover study of disposable thermometers for prevention of *Clostridium difficile* and other nosocomial infections. *Infect Control Hosp Epidemiol* 1998;19(7):494-9.
855. Weinstein SA, Gantz NM, Pelletier C, Hibert D. Bacterial surface contamination of patients' linen: isolation precautions versus standard care. *Am J Infect Control* 1989;17(5):264-7.
856. Pugliese G. Isolating and double-bagging laundry: is it really necessary? *Health Facil Manage* 1989;2(2):16, 8-21.
857. . (Accessed 2007, at
858. Kiehl E, Wallace R, Warren C. Tracking perinatal infection: is it safe to launder your scrubs at home? *MCN Am J Matern Child Nurs* 1997;22(4):195-7.
859. Jurkovich P. Home- versus hospital-laundered scrubs: a pilot study. *MCN Am J Matern Child Nurs* 2004;29(2):106-10.
860. United States Environmental Protection Agency - Medical Waste. www.epa.gov/epaoswer/other/medical/
861. www.cdc.gov/ncidod/dhqp/gl_enviroinfection.html
862. Maki DG, Alvarado C, Hassemer C. Double-bagging of items from isolation rooms is unnecessary as an infection control measure: a comparative study of surface contamination with single- and double-bagging. *Infect Control* 1986;7(11):535-7.
863. CDC. Recommended antimicrobial agents for the treatment and postexposure prophylaxis of pertussis: 2005 CDC Guidelines. *MMWR Recomm Rep* 2005;54(RR-14):1-16.
864. CDC. Prevention and control of meningococcal disease. Recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep* 2000;49(RR-7):1-10.

865. CDC. Notice to Readers: Additional options for preventive treatment for persons exposed to inhalational anthrax. *MMWR Morb Mortal Wkly Rep* 2001;50 (50):1142-51.
866. CDC. Updated U.S. Public Health Service guidelines for the management of occupational exposures to HIV and recommendations for postexposure prophylaxis. *MMWR Recomm Rep* 2005;54(RR-9):1-17.
867. Boyce JM. MRSA patients: proven methods to treat colonization and infection. *J Hosp Infect* 2001;48 Suppl A:S9-14.
868. Barrett FF, Mason EO, Jr., Fleming D. Brief clinical and laboratory observations. *J Pediatr* 1979;94(5):796-800.
869. American Academy of Pediatrics. Guidelines for Perinatal Care. American Academy of Obstetricians and Gynecologists, 2002. Elk Grove Village, IL; 2002.
870. Management of Multidrug-Resistant Organisms In Healthcare Settings, 2006. CDC, 2006. (Accessed 2007, at
871. Kallen AJ, Wilson CT, Larson RJ. Perioperative intranasal mupirocin for the prevention of surgical-site infections: systematic review of the literature and meta-analysis. *Infect Control Hosp Epidemiol* 2005;26(12):916-22.
872. Carrier M, Marchand R, Auger P, et al. Methicillin-resistant *Staphylococcus aureus* infection in a cardiac surgical unit. *J Thorac Cardiovasc Surg* 2002;123(1):40-4.
873. Tacconelli E, Carmeli Y, Aizer A, Ferreira G, Foreman MG, D'Agata EM. Mupirocin prophylaxis to prevent *Staphylococcus aureus* infection in patients undergoing dialysis: a meta-analysis. *Clin Infect Dis* 2003;37(12):1629-38.
874. CDC. Immunization of health-care workers: recommendations of the Advisory Committee on Immunization Practices (ACIP) and the Hospital Infection Control Practices Advisory Committee (HICPAC). *MMWR Recomm Rep* 1997;46(RR18):1-42.
875. Mahoney FJ, Stewart K, Hu H, Coleman P, Alter MJ. Progress toward the elimination of hepatitis B virus transmission among health care workers in the United States. *Arch Intern Med* 1997;157(22):2601-5.
876. Gladstone JL, Millian SJ. Rubella exposure in an obstetric clinic. *Obstet Gynecol* 1981;57(2):182-6.
877. Wilde JA, McMillan JA, Serwint J, Butta J, O'Riordan MA, Steinhoff MC. Effectiveness of influenza vaccine in health care professionals: a randomized trial. *JAMA* 1999;281(10):908-13.
878. Potter J, Stott DJ, Roberts MA, et al. Influenza vaccination of health care workers in long-term-care hospitals reduces the mortality of elderly patients. *J Infect Dis* 1997;175(1):1-6.

879. Pearson ML, Bridges CB, Harper SA. Influenza vaccination of health-care personnel: recommendations of the Healthcare Infection Control Practices Advisory Committee (HICPAC) and the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep* 2006;55(RR-2):1-16.
880. Wright SW, Decker MD, Edwards KM. Incidence of pertussis infection in healthcare workers. *Infect Control Hosp Epidemiol* 1999;20(2):120-3.
881. Calugar A, Ortega-Sanchez IR, Tiwari T, Oakes L, Jahre JA, Murphy TV. Nosocomial pertussis: costs of an outbreak and benefits of vaccinating health care workers. *Clin Infect Dis* 2006;42(7):981-8.
882. www.fda.gov
883. Campins-Marti M, Cheng HK, Forsyth K, et al. Recommendations are needed for adolescent and adult pertussis immunisation: rationale and strategies for consideration. *Vaccine* 2001;20(5-6):641-6.
884. www.cdc.gov/nip/recs/provisional_rec/default.htm
885. CDC. Recommended childhood and adolescent immunization schedule -- United States, 2006. *MMWR Morb Mortal Wkly Rep* 2006;54(Nos. 51 & 52):Q1-Q4.
886. Recommended childhood and adolescent immunization schedule--United States, 2006. *Pediatrics* 2006;117(1):239-40.
887. CDC. Recommended adult immunization schedule -- United States, October 2005-September 2006. *MMWR Morb Mortal Wkly Rep* 2005;54(40):Q1-Q4; October 14.
888. CDC. Prevention of Varicella Updated Recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 1999;48 (RR-6):1-5.
889. McKenney D, Pouliot KL, Wang Y, et al. Broadly protective vaccine for *Staphylococcus aureus* based on an in vivo-expressed antigen. *Science* 1999;284(5419):1523-7.
890. Shinefield H, Black S, Fattom A, et al. Use of a *Staphylococcus aureus* conjugate vaccine in patients receiving hemodialysis. *N Engl J Med* 2002;346(7):491-6.
891. Abadesso C, Almeida HI, Virella D, Carreiro MH, Machado MC. Use of palivizumab to control an outbreak of syncytial respiratory virus in a neonatal intensive care unit. *J Hosp Infect* 2004;58(1):38-41.
892. George RH, Gully PR, Gill ON, Innes JA, Bakhshi SS, Connolly M. An outbreak of tuberculosis in a children's hospital. *J Hosp Infect* 1986;8(2):129-42.

893. Simor AE, Lee M, Vearncombe M, et al. An outbreak due to multiresistant *Acinetobacter baumannii* in a burn unit: risk factors for acquisition and management. *Infect Control Hosp Epidemiol* 2002;23(5):261-7.
894. Puzniak LA, Leet T, Mayfield J, Kollef M, Mundy LM. To gown or not to gown: the effect on acquisition of vancomycin-resistant enterococci. *Clin Infect Dis* 2002;35(1):18-25.
895. Hanna H, Umphrey J, Tarrand J, Mendoza M, Raad I. Management of an outbreak of vancomycin-resistant enterococci in the medical intensive care unit of a cancer center. *Infect Control Hosp Epidemiol* 2001;22(4):217-9.
896. CDC. Recommendations for preventing transmission of infection with human T-lymphotropic virus type III/lymphadenopathy-associated virus in the workplace. *MMWR Morb Mortal Wkly Rep* 1985;34(450):681-6, 91-5.
897. CDC. Severe acute respiratory syndrome--Taiwan, 2003. *MMWR Morb Mortal Wkly Rep* 2003;52(20):461-6.
898. Capps JA. Measures for the prevention and control of respiratory infections in military camps. *JAMA* 1918;71:448-51.
899. Thomas C. Efficiency of surgical masks in use in hospital wards. Report to the Control of Infection Subcommittee. *Guys Hosp Rep* 1961;110:157-67.
900. Beck M, Antle BJ, Berlin D, et al. Wearing masks in a pediatric hospital: developing practical guidelines. *Can J Public Health* 2004;95(4):256-7.
901. Ryan MA, Christian RS, Wohlrabe J. Handwashing and respiratory illness among young adults in military training. *Am J Prev Med* 2001;21(2):79-83.
902. Roberts L, Smith W, Jorm L, Patel M, Douglas RM, McGilchrist C. Effect of infection control measures on the frequency of upper respiratory infection in child care: a randomized, controlled trial. *Pediatrics* 2000;105(4 Pt 1):738-42.js.
903. White C, Kolble R, Carlson R, et al. The effect of hand hygiene on illness rate among students in university residence halls. *Am J Infect Control* 2003;31(6):364-70.
904. Aiello AE, Larson EL. What is the evidence for a causal link between hygiene and infections? *Lancet Infect Dis* 2002;2(2):103-10.
905. www.aana.com/news.aspx?ucNavMenu_TSMMenuTargetID=171&ucNavMenu_TSMMenuTargetType=4&ucNavMenu_TSMMenuID=6&id=1613.
906. Watanakunakorn C, Stahl C. *Streptococcus salivarius* meningitis following myelography. *Infect Control Hosp Epidemiol* 1992;13(8):454.
907. Gelfand MS, Abolnik IZ. Streptococcal meningitis complicating diagnostic myelography: three cases and review. *Clin Infect Dis* 1995;20(3):582-7.

908. Schlesinger JJ, Salit IE, McCormack G. Streptococcal meningitis after myelography. *Arch Neurol* 1982;39(9):576-7.
909. Yaniv LG, Potasman I. Iatrogenic meningitis: an increasing role for resistant viridans streptococci? Case report and review of the last 20 years. *Scand J Infect Dis* 2000;32(6):693-6.
910. Schlegel L, Merlet C, Laroche JM, Fremaux A, Geslin P. Iatrogenic meningitis due to *Abiotrophia defectiva* after myelography. *Clin Infect Dis* 1999;28(1):155-6.
911. Schneeberger PM, Janssen M, Voss A. Alpha-hemolytic streptococci: a major pathogen of iatrogenic meningitis following lumbar puncture. Case reports and a review of the literature. *Infection* 1996;24(1):29-33.
912. Veringa E, van Belkum A, Schellekens H. Iatrogenic meningitis by *Streptococcus salivarius* following lumbar puncture. *J Hosp Infect* 1995;29(4):316-8.
913. Couzigou C, Vuong TK, Botharel AH, Aggoune M, Astagneau P. Iatrogenic *Streptococcus salivarius* meningitis after spinal anaesthesia: need for strict application of standard precautions. *J Hosp Infect* 2003;53(4):313-4.
914. Torres E, Alba D, Frank A, Diez-Tejedor E. Iatrogenic meningitis due to *Streptococcus salivarius* following a spinal tap. *Clin Infect Dis* 1993;17(3):525-6.
915. Trautmann M, Lepper PM, Schmitz FJ. Three cases of bacterial meningitis after spinal and epidural anesthesia. *Eur J Clin Microbiol Infect Dis* 2002;21(1):43-5.
916. Baer ET. Iatrogenic meningitis: the case for face masks. *Clin Infect Dis* 2000;31(2):519-21.
917. Black SR, Weinstein RA. The case for face masks-zorro or zero? *Clin Infect Dis* 2000;31(2):522-3.
918. Philips BJ, Fergusson S, Armstrong P, Anderson FM, Wildsmith JA. Surgical face masks are effective in reducing bacterial contamination caused by dispersal from the upper airway. *Br J Anaesth* 1992;69(4):407-8.
919. CDC. Guidelines for the Prevention of Intravascular Catheter-Related Infections. *MMWR* 2002;51(RR10)(10):1-26.
920. Catalano G, Houston SH, Catalano MC, et al. Anxiety and depression in hospitalized patients in resistant organism isolation. *South Med J* 2003;96(2):141-5.
921. Tarzi S, Kennedy P, Stone S, Evans M. Methicillin-resistant *Staphylococcus aureus*: psychological impact of hospitalization and isolation in an older adult population. *J Hosp Infect* 2001;49(4):250-4.

922. Kelly-Rossini L, Perlman DC, Mason DJ. The experience of respiratory isolation for HIV-infected persons with tuberculosis. *J Assoc Nurses AIDS Care* 1996;Jan-Feb; 7(1):29-36.
923. Knowles HE. The experience of infectious patients in isolation. *Nurs Times* 1993;89(30):53-6.
924. Evans HL, Shaffer MM, Hughes MG, et al. Contact isolation in surgical patients: a barrier to care? *Surgery* 2003;134(2):180-8.
925. Kirkland KB, Weinstein JM. Adverse effects of contact isolation. *Lancet* 1999;354(9185):1177-8.
926. Saint S, Higgins LA, Nallamotheu BK, Chenoweth C. Do physicians examine patients in contact isolation less frequently? A brief report. *Am J Infect Control* 2003;31(6):354-6.
927. Management of Multidrug-Resistant Organisms In Healthcare Settings, 2006. 2006. www.cdc.gov/ncidod/dhqp/pdf/ar/mdroGuideline2006.pdf
928. Hall CB, Powell KR, MacDonald NE, et al. Respiratory syncytial viral infection in children with compromised immune function. *N Engl J Med* 1986;315(2):77-81.
929. Lui SL, Luk WK, Cheung CY, Chan TM, Lai KN, Peiris JS. Nosocomial outbreak of parvovirus B19 infection in a renal transplant unit. *Transplantation* 2001;71(1):59-64.
930. Weinstock DM, Gubareva LV, Zuccotti G. Prolonged shedding of multidrug-resistant influenza A virus in an immunocompromised patient. *N Engl J Med* 2003;348(9):867-8.
931. van Tol MJ, Claas EC, Heemskerk B, et al. Adenovirus infection in children after allogeneic stem cell transplantation: diagnosis, treatment and immunity. *Bone Marrow Transplant* 2005;35 Suppl 1:S73-6.
932. Wood DJ, David TJ, Chrystie IL, Totterdell B. Chronic enteric virus infection in two T-cell immunodeficient children. *J Med Virol* 1988;24(4):435-44.
933. 933. Mori I, Matsumoto K, Sugimoto K, et al. Prolonged shedding of rotavirus in a geriatric inpatient. *J Med Virol* 2002;67(4):613-5.
934. Cederna JE, Terpenning MS, Ensberg M, Bradley SF, Kauffman CA. *Staphylococcus aureus* nasal colonization in a nursing home: eradication with mupirocin. *Infect Control Hosp Epidemiol* 1990;11(1):13-6.
935. Kauffman CA, Terpenning MS, He X, et al. Attempts to eradicate methicillin-resistant *Staphylococcus aureus* from a long-term-care facility with the use of mupirocin ointment. *Am J Med* 1993;94(4):371-8.
936. Montecalvo MA, de Lencastre H, Carraher M, et al. Natural history of colonization with vancomycin-resistant *Enterococcus faecium*. *Infect Control Hosp Epidemiol* 1995;16(12):680-5.

937. D'Agata EM, et al. High rate of false-negative results of the rectal swab culture method in detection of gastrointestinal colonization with vancomycin-resistant enterococci. *Clin Infect Dis* 2002;34(2):167-72.
938. Donskey CJ, Hoyer CK, Das SM, Helfand MS, Hecker MT. Recurrence of vancomycin-resistant *Enterococcus* stool colonization during antibiotic therapy. *Infect Control Hosp Epidemiol* 2002;23(8):436-40.
939. Scanvic A, Denic L, Gaillon S, Giry P, Andremont A, Lucet JC. Duration of colonization by methicillin-resistant *Staphylococcus aureus* after hospital discharge and risk factors for prolonged carriage. *Clin Infect Dis* 2001;32(10):1393-8.
940. Noskin GA, Bednarz P, Suriano T, Reiner S, Peterson LR. Persistent contamination of fabric-covered furniture by vancomycin-resistant enterococci: implications for upholstery selection in hospitals. *Am J Infect Control* 2000;28(4):311-3.
941. Gerson SL, Parker P, Jacobs MR, Creger R, Lazarus HM. Aspergillosis due to carpet contamination. *Infect Control Hosp Epidemiol* 1994;15(4 Pt 1):221-3.
942. Taplin D, Mertz PM. Flower vases in hospitals as reservoirs of pathogens. *Lancet* 1973;2(7841):1279-81.
943. Walsh TJ, Dixon DM. Nosocomial aspergillosis: environmental microbiology, hospital epidemiology, diagnosis and treatment. *Eur J Epidemiol* 1989;5(2):131-42.
944. Lass-Flörl C, Rath P, Niederwieser D, et al. *Aspergillus terreus* infections in haematological malignancies: molecular epidemiology suggests association with in-hospital plants. *J Hosp Infect* 2000;46(1):31-5.
945. Raad I, Hanna H, Osting C, et al. Masking of neutropenic patients on transport from hospital rooms is associated with a decrease in nosocomial aspergillosis during construction. *Infect Control Hosp Epidemiol* 2002;23(1):41-3.
946. www.cms.hhs.gov/CLIA.
947. Emori TG, Haley RW, Stanley RC. The infection control nurse in US hospitals, 1976-1977. Characteristics of the position and its occupant. *Am J Epidemiol* 1980;111(5):592-607.
948. Richet HM, Benbachir M, Brown DE, et al. Are there regional variations in the diagnosis, surveillance, and control of methicillin-resistant *Staphylococcus aureus*? *Infect Control Hosp Epidemiol* 2003;24(5):334-41.
949. Anderson DJ, Kirkland KB, McDonald JR, et al. Results of a survey of work duties of 56 infection control professionals (ICPs): Are new guidelines needed for the staffing of infection control (IC) programs?

- Abstract #146. In: 16th Annual Society for Healthcare Epidemiology of America. Chicago, Ill; 2006.
950. Harvey MA. Critical-care-unit bedside design and furnishing: impact on nosocomial infections. *Infect Control Hosp Epidemiol* 1998;19(8):597-601.
951. Srinivasan A, Beck C, Buckley T, et al. The ability of hospital ventilation systems to filter *Aspergillus* and other fungi following a building implosion. *Infect Control Hosp Epidemiol* 2002;23(9):520-4.
952. Maragakis LL, Bradley KL, Song X, et al. Increased catheter-related bloodstream infection rates after the introduction of a new mechanical valve intravenous access port. *Infect Control Hosp Epidemiol* 2006;27(1):67-70.
953. Organizations JCoAoH. *Comprehensive Accreditation Manual for Hospitals: The Official Handbook*. Oakbrook Terrace: JCAHO; 2007.
954. Peterson LR, Noskin GA. New technology for detecting multidrug-resistant pathogens in the clinical microbiology laboratory. *Emerg Infect Dis* 2001;7(2):306-11.
955. Diekema DJ, Doebbeling BN. Employee health and infection control. *Infect Control Hosp Epidemiol* 1995;16(5):292-301.
956. Rutala WA, Weber DJ, Healthcare Infection Control Practices Advisory Committee (HICPAC). *Guideline for Disinfection and Sterilization in Health-Care Facilities*. In preparation.
957. Weems JJ, Jr. Nosocomial outbreak of *Pseudomonas cepacia* associated with contamination of reusable electronic ventilator temperature probes. *Infect Control Hosp Epidemiol* 1993;14(10):583-6.
958. Berthelot P, Grattard F, Mahul P, et al. Ventilator temperature sensors: an unusual source of *Pseudomonas cepacia* in nosocomial infection. *J Hosp Infect* 1993;25(1):33-43.
959. 959. CDC. Bronchoscopy-related infections and pseudoinfections--New York, 1996 and 1998. *MMWR Morb Mortal Wkly Rep* 1999;48(26):557-60.
960. Heeg P, Roth K, Reichl R, Cogdill CP, Bond WW. Decontaminated single-use devices: an oxymoron that may be placing patients at risk for cross-contamination. *Infect Control Hosp Epidemiol* 2001;22(9):542-9.
961. www.fda.gov/cdrh/reprocessing/
962. CDC. *Prevention and Control of Influenza: Recommendations of the Advisory Committee on Immunization Practices (ACIP)*. *MMWR - Morbidity & Mortality Weekly Report* 2003;52(RR08):1-36.

963. Weinstock DM, Eagan J, Malak SA, et al. Control of influenza A on a bone marrow transplant unit. *Infect Control Hosp Epidemiol* 2000;21(11):730-2.
964. Cromer AL, Hutsell SO, Latham SC, et al. Impact of implementing a method of feedback and accountability related to contact precautions compliance. *Am J Infect Control* 2004;32(8):451-5.
965. Eveillard M, Eb F, Tramier B, et al. Evaluation of the contribution of isolation precautions in prevention and control of multi-resistant bacteria in a teaching hospital. *J Hosp Infect* 2001;47(2):116-24.
966. Pfeiffer J, Gilmore G. The Text as an Orientation Tool. In: Pfeiffer J, ed. *APIC Text of Infection Control and Epidemiology*. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc. (APIC); 2000:7/1 - 7/8.
967. Gaynes RP, Emori TG. Chapter 5: Surveillance for Nosocomial Infections. In: Abrutyn E, Goldmann DA, Scheckler WE, eds. *Saunders Infection Control Reference Service*. Philadelphia, PA: W.B. Saunders Company; 2001:40-4.
968. CDC. Monitoring hospital-acquired infections to promote patient safety - United States, 1990-1999. *MMWR Morb Mortal Wkly Rep* 2000;49(8):149-53.
969. 969. Curran ET, Benneyan JC, Hood J. Controlling methicillin-resistant *Staphylococcus aureus*: a feedback approach using annotated statistical process control charts. *Infect Control Hosp Epidemiol* 2002;23(1):13-8.
970. Lanotte P, Cantagrel S, Mereghetti L, et al. Spread of *Stenotrophomonas maltophilia* colonization in a pediatric intensive care unit detected by monitoring tracheal bacterial carriage and molecular typing. *Clin Microbiol Infect* 2003;9(11):1142-7.
971. Coopersmith CM, Zack JE, Ward MR, et al. The impact of bedside behavior on catheter-related bacteremia in the intensive care unit. *Arch Surg* 2004;139(2):131-6.
972. O'Brien KL, Beall B, Barrett NL, et al. Epidemiology of invasive group A streptococcus disease in the United States, 1995-1999. *Clin Infect Dis* 2002;35(3):268-76.
973. Nicolle LE, Dyck B, Thompson G, et al. Regional dissemination and control of epidemic methicillin-resistant *Staphylococcus aureus*. Manitoba Chapter of CHICA-Canada. *Infect Control Hosp Epidemiol* 1999;20(3):202-5.
974. Seybold U, Kourbatova EV, Johnson JG, et al. Emergence of community-associated methicillin-resistant *Staphylococcus aureus* USA300 genotype as a major cause of health care-associated blood stream infections. *Clin Infect Dis* 2006;42(5):647-56.

975. Bond WW, Favero MS, Petersen NJ, Gravelle CR, Ebert JW, Maynard JE. Survival of hepatitis B virus after drying and storage for one week. *Lancet* 1981;1(8219):550-1.
976. Ehrenkranz NJ, Alfonso BC. Failure of bland soap handwash to prevent hand transfer of patient bacteria to urethral catheters. *Infect Control Hosp Epidemiol* 1991;12(11):654-62.
977. Winnefeld M, Richard MA, Drancourt M, Grob JJ. Skin tolerance and effectiveness of two hand decontamination procedures in everyday hospital use. *Br J Dermatol* 2000;143(3):546-50.
978. Widmer AF. Replace hand washing with use of a waterless alcohol hand rub? *Clin Infect Dis* 2000;31(1):136-43.
979. Mortimer EA, Jr., Lipsitz PJ, Wolinsky E, Gonzaga AJ, Rammelkamp CH, Jr. Transmission of staphylococci between newborns. Importance of the hands to personnel. *Am J Dis Child* 1962;104:289-95.
980. Casewell M, Phillips I. Hands as route of transmission for *Klebsiella* species. *Br Med J* 1977;2(6098):1315-7.
981. Ojajarvi J. Effectiveness of hand washing and disinfection methods in removing transient bacteria after patient nursing. *J Hyg (Lond)* 1980;85(2):193-203.
982. Otter J, Havill N, Adams N, Joyce J. Extensive environmental contamination associated with patients with loose stools and MRSA colonization of the gastrointestinal tract. Abstract #159. In: the 16th annual scientific meeting of the Society for Healthcare Epidemiology of America. In. Chicago, Illinois; 2006.
983. Weber DJ, Sickbert-Bennett E, Gergen MF, Rutala WA. Efficacy of selected hand hygiene agents used to remove *Bacillus atrophaeus* (a surrogate of *Bacillus anthracis*) from contaminated hands. *Jama* 2003;289(10):1274-7.
984. Saiman L, Lerner A, Saal L, et al. Banning artificial nails from health care settings. *Am J Infect Control* 2002;30(4):252-4.
985. Johnson S, Gerding DN, Olson MM, et al. Prospective, controlled study of vinyl glove use to interrupt *Clostridium difficile* nosocomial transmission. *Am J Med* 1990;88(2):137-40.
986. Neal JG, Jackson EM, Suber F, Edlich RF. Latex glove penetration by pathogens: a review of the literature. *J Long Term Eff Med Implants* 1998;8(3-4):233-40.
987. Broyles JM, O'Connell KP, Korniewicz DM. PCR-based method for detecting viral penetration of medical exam gloves. *J Clin Microbiol* 2002;40(8):2725-8.

988. Patterson JE, Vecchio J, Pantelick EL, et al. Association of contaminated gloves with transmission of *Acinetobacter calcoaceticus* var. *anitratus* in an intensive care unit. *Am J Med* 1991;91(5):479-83.
989. Goldmann DA. Epidemiology and prevention of pediatric viral respiratory infections in health-care institutions. *Emerg Infect Dis* 2001;7(2):249-53.
990. Gaggero A, Avendano LF, Fernandez J, Spencer E. Nosocomial transmission of rotavirus from patients admitted with diarrhea. *J Clin Microbiol* 1992;30(12):3294-7.
991. Merritt K, Hitchins VM, Brown SA. Safety and cleaning of medical materials and devices. *J Biomed Mater Res* 2000;53(2):131-6.
992. Kampf G, Bloss R, Martiny H. Surface fixation of dried blood by glutaraldehyde and peracetic acid. *J Hosp Infect* 2004;57(2):139-43.
993. Weber DJ, Rutala WA. Role of environmental contamination in the transmission of vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 1997;18(5):306-9.
994. Byers KE, Durbin LJ, Simonton BM, Anglim AM, Adal KA, Farr BM. Disinfection of hospital rooms contaminated with vancomycin-resistant *Enterococcus faecium*. *Infect Control Hosp Epidemiol* 1998;19(4):261-4.
995. Martinez JA, Ruthazer R, Hansjosten K, Barefoot L, Snyderman DR. Role of environmental contamination as a risk factor for acquisition of vancomycin-resistant enterococci in patients treated in a medical intensive care unit. *Arch Intern Med* 2003;163(16):1905-12.
996. EPA. Federal Insecticide, Fungicide, and Rodenticidal Act 7 U.S.C. 136 et seq. In: Agency EP, ed.
997. Devine J, Cooke RP, Wright EP. Is methicillin-resistant *Staphylococcus aureus* (MRSA) contamination of ward-based computer terminals a surrogate marker for nosocomial MRSA transmission and handwashing compliance? *J Hosp Infect* 2001;48(1):72-5.
998. Sattar SA, Springthorpe S, Mani S, et al. Transfer of bacteria from fabrics to hands and other fabrics: development and application of a quantitative method using *Staphylococcus aureus* as a model. *J Appl Microbiol* 2001;90(6):962-70.
999. Shiomori T, Miyamoto H, Makishima K, et al. Evaluation of bedmaking-related airborne and surface methicillin-resistant *Staphylococcus aureus* contamination. *J Hosp Infect* 2002;50(1):30-5.
1000. Whyte W, Baird G, Annand R. Bacterial contamination on the surface of hospital linen chutes. *J Hyg (Lond)* 1969;67(3):427-35.
1001. Michaelsen GS. Designing Linen Chutes to Reduce Spread of Infectious Organisms. *Hospitals* 1965;39:116-9.

1002. Plott RT, Wagner RF, Jr., Tying SK. Iatrogenic contamination of multidose vials in simulated use. A reassessment of current patient injection technique. *Arch Dermatol* 1990;126(11):1441-4.
1003. Samandari T, Malakmadze N, Balter S, et al. A large outbreak of hepatitis B virus infections associated with frequent injections at a physician's office. *Infect Control Hosp Epidemiol* 2005;26(9):745-50.
1004. Comstock RD, Mallonee S, Fox JL, et al. A large nosocomial outbreak of hepatitis C and hepatitis B among patients receiving pain remediation treatments. *Infect Control Hosp Epidemiol* 2004;25(7):576-83.
1005. Germain JM, Carbonne A, Thiers V, et al. Patient-to-patient transmission of hepatitis C virus through the use of multidose vials during general anesthesia. *Infect Control Hosp Epidemiol* 2005;26(9):789-92.
1006. Macedo de Oliveira A, White KL, Leschinsky DP, et al. An outbreak of hepatitis C virus infections among outpatients at a hematology/oncology clinic. *Ann Intern Med* 2005;142(11):898-902.
1007. Hsu J, Jensen B, Arduino M, et al. Streptococcal Meningitis Following Myelogram Procedures. *Infect Control Hosp Epidemiol* 2007;28(5):614-17.
1008. Srinivasan A, Song X, Ross T, Merz W, Brower R, Perl TM. A prospective study to determine whether cover gowns in addition to gloves decrease nosocomial transmission of vancomycin-resistant enterococci in an intensive care unit. *Infect Control Hosp Epidemiol* 2002;23(8):424-8.
1009. Nichols WG, Corey L, Gooley T, Davis C, Boeckh M. Parainfluenza virus infections after hematopoietic stem cell transplantation: risk factors, response to antiviral therapy, and effect on transplant outcome. *Blood* 2001;98(3):573-8.
1010. Elizaga J, Olavarria E, Apperley J, Goldman J, Ward K. Parainfluenza virus 3 infection after stem cell transplant: relevance to outcome of rapid diagnosis and ribavirin treatment. *Clin Infect Dis* 2001;32(3):413-8.
1011. Oishi I, Kimura T, Murakami T, et al. Serial observations of chronic rotavirus infection in an immunodeficient child. *Microbiology and Immunology* 1991;35(11):953-61.
1012. Fierobe L, Lucet JC, Decre D, et al. An outbreak of imipenem-resistant *Acinetobacter baumannii* in critically ill surgical patients. *Infect Control Hosp Epidemiol* 2001;22(1):35-40.
1013. Montesinos I, Salido E, Delgado T, Lecuona M, Sierra A. Epidemiology of methicillin-resistant *Staphylococcus aureus* at a university hospital in the Canary Islands. *Infect Control Hosp Epidemiol* 2003;24(9):667-72.
1014. Poutanen SM, Vearncombe M, McGeer AJ, Gardam M, Large G, Simor AE. Nosocomial acquisition of methicillin-resistant *Staphylococcus*

- aureus during an outbreak of severe acute respiratory syndrome. *Infect Control Hosp Epidemiol* 2005;26(2):134-7.
1015. Yap FH, Gomersall CD, Fung KS, et al. Increase in methicillin-resistant *Staphylococcus aureus* acquisition rate and change in pathogen pattern associated with an outbreak of severe acute respiratory syndrome. *Clin Infect Dis* 2004;39(4):511-6.
1016. Layton MC, Perez M, Heald P, Patterson JE. An outbreak of mupirocin-resistant *Staphylococcus aureus* on a dermatology ward associated with an environmental reservoir. *Infect Control Hosp Epidemiol* 1993;14(7):369-75.
1017. Gilmore A, Stuart J, Andrews N. Risk of secondary meningococcal disease in health-care workers. *Lancet* 2000;356(9242):1654-5.
1018. [. www.cdc.gov/flu/avian/index.htm](http://www.cdc.gov/flu/avian/index.htm)
1019. www.hhs.gov/pandemicflu/plan/pdf/S04.pdf
1020. Ehresmann KR, Hedberg CW, Grimm MB, Norton CA, MacDonald KL, Osterholm MT. An outbreak of measles at an international sporting event with airborne transmission in a domed stadium. *J Infect Dis* 1995;171(3):679-83.
1021. Gustafson TL, Lavelly GB, Brawner ER, Jr., Hutcheson RH, Jr., Wright PF, Schaffner W. An outbreak of airborne nosocomial varicella. *Pediatrics* 1982;70(4):550-6.
1022. Hyams PJ, Stuewe MC, Heitzer V. Herpes zoster causing varicella (chickenpox) in hospital employees: cost of a casual attitude. *Am J Infect Control* 1984;12(1):2-5.
1023. Pavelchak N, DePersis RP, London M, et al. Identification of factors that disrupt negative air pressurization of respiratory isolation rooms. *Infect Control Hosp Epidemiol* 2000;21(3):191-5.
1024. Rice N, Streifel A, Vesley D. An evaluation of hospital special-ventilation-room pressures. *Infect Control Hosp Epidemiol* 2001;22(1):19-23.
1025. Hutton MD, Stead WW, Cauthen GM, Bloch AB, Ewing WM. Nosocomial transmission of tuberculosis associated with a draining abscess. *J Infect Dis* 1990;161(2):286-95.
1026. Frampton MW. An outbreak of tuberculosis among hospital personnel caring for a patient with a skin ulcer. *Ann Intern Med* 1992;117(4):312-3.
1027. Ammari LK, Bell LM, Hodinka RL. Secondary measles vaccine failure in healthcare workers exposed to infected patients. *Infect Control Hosp Epidemiol* 1993;14(2):81-6.

1028. Behrman A, Schmid DS, Crivaro A, Watson B. A cluster of primary varicella cases among healthcare workers with false-positive varicella zoster virus titers. *Infect Control Hosp Epidemiol* 2003;24(3):202-6.
1029. Josephson A, Gombert ME. Airborne transmission of nosocomial varicella from localized zoster. *J Infect Dis* 1988;158(1):238-41.
1030. Brodtkin RH. Zoster Causing Varicella. Current Dangers Of Contagion Without Isolation. *Arch Dermatol* 1963;88:322-4.
1031. Suzuki K, Yoshikawa T, Tomitaka A, Matsunaga K, Asano Y. Detection of aerosolized varicella-zoster virus DNA in patients with localized herpes zoster. *J Infect Dis* 2004;189(6):1009-12.
1032. Ruuskanen O, Salmi TT, Halonen P. Measles vaccination after exposure to natural measles. *J Pediatr* 1978;93(1):43-6.
1033. Berkovich S, Starr S. Use of live-measles-virus vaccine to abort an expected outbreak of measles within a closed population. *N Engl J Med* 1963;269:75-7.
1034. CDC. Measles, mumps, and rubella--vaccine use and strategies for elimination of measles, rubella, and congenital rubella syndrome and control of mumps: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep* 1998;47(RR-8):1-57.
1035. CDC. General recommendations on immunization: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 2006;55(RR-15):1-48.
1036. Watson B, Seward J, Yang A, et al. Postexposure effectiveness of varicella vaccine. *Pediatrics* 2000;105(1 Pt 1):84-8.
1037. Salzman MB, Garcia C. Postexposure varicella vaccination in siblings of children with active varicella. *Pediatr Infect Dis J* 1998;17(3):256-7.
1038. CDC. Vaccinia (smallpox) vaccine: recommendations of the Advisory Committee on Immunization Practices (ACIP), 2001. *MMWR Recomm Rep* 2001;50(RR-10):1-25; quiz CE1-7.
1039. Fulginiti VA, Papier A, Lane JM, Neff JM, Henderson DA. Smallpox vaccination: a review, part I. Background, vaccination technique, normal vaccination and revaccination, and expected normal reactions. *Clin Infect Dis* 2003;37(2):241-50.
1040. Dixon CW. Smallpox in Tripolitania, 1946: an epidemiological and clinical study of 500 cases, including trials of penicillin treatment. *J Hyg (Lond)* 1948;46:351-77.
1041. Murray WA, Streifel AJ, O'Dea TJ, Rhame FS. Ventilation for protection of immune compromised patients. *ASHRAE Transactions* 1988;94:1185.

1042. Rutala WA, Jones SM, Worthington JM, Reist PC, Weber DJ. Efficacy of portable filtration units in reducing aerosolized particles in the size range of *Mycobacterium tuberculosis*. *Infect Control Hosp Epidemiol* 1995;16(7):391-8.
1043. Mandell GL, Bennett JE, Dolin R. *Mandell, Douglas and Bennett's Principles and Practice of Infectious Diseases*. GL Mandell, JE Bennett, R Dolin, Eds. 5th edition. Churchill Livingstone, Philadelphia, 2000. 2000.
1044. *Control of Communicable Diseases Manual*. DL Heymann Ed. 18th edition, American Public Health Association, Washington, DC 2005. 2005.
1045. Vreden SG, Visser LG, Verweij JJ, et al. Outbreak of amebiasis in a family in The Netherlands. *Clin Infect Dis* 2000;31(4):1101-4.
1046. Thacker SB, Kimball AM, Wolfe M, Choi K, Gilmore L. Parasitic disease control in a residential facility for the mentally retarded: failure of selected isolation procedures. *Am J Public Health* 1981;71(3):303-5.
1047. Sampathkumar P. West Nile virus: epidemiology, clinical presentation, diagnosis, and prevention. *Mayo Clin Proc* 2003;78(9):1137-43; quiz 44.
1048. Ruben B, Band JD, Wong P, Colville J. Person-to-person transmission of *Brucella melitensis*. *Lancet* 1991;337(8732):14-5.
1049. Vandercam B, Zech F, de Cooman S, Bughin C, Gigi J, Wauters G. Isolation of *Brucella melitensis* from human sperm. *Eur J Clin Microbiol Infect Dis* 1990;9(4):303-4.
1050. Robichaud S, Libman M, Behr M, Rubin E. Prevention of laboratory-acquired brucellosis. *Clin Infect Dis* 2004;38(12):e119-22.
1051. Troy CJ, Peeling RW, Ellis AG, et al. *Chlamydia pneumoniae* as a new source of infectious outbreaks in nursing homes. *Jama* 1997;277(15):1214-8.
1052. Ekman MR, Grayston JT, Visakorpi R, Kleemola M, Kuo CC, Saikku P. An epidemic of infections due to *Chlamydia pneumoniae* in military conscripts. *Clin Infect Dis* 1993;17(3):420-5.
1053. Eickhoff TC. An outbreak of surgical wound infections due to *Clostridium perfringens*. *Surg Gynecol Obstet* 1962;114:102-8.
1054. Kohn GJ, Linne SR, Smith CM, Hoeprich PD. Acquisition of coccidioidomycosis at necropsy by inhalation of coccidioidal endospores. *Diagn Microbiol Infect Dis* 1992;15(6):527-30.
1055. Wright PW, Pappagianis D, Wilson M, et al. Donor-related coccidioidomycosis in organ transplant recipients. *Clin Infect Dis* 2003;37(9):1265-9.

1056. Maitreyi RS, Dar L, Muthukumar A, et al. Acute hemorrhagic conjunctivitis due to enterovirus 70 in India. *Emerg Infect Dis* 1999;5(2):267-9.
1057. CDC. Acute hemorrhagic conjunctivitis outbreak caused by Coxsackievirus A24--Puerto Rico, 2003. *MMWR Morb Mortal Wkly Rep* 2004;53(28):632-4.
1058. Faden H, Wynn RJ, Campagna L, Ryan RM. Outbreak of adenovirus type 30 in a neonatal intensive care unit. *J Pediatr* 2005;146(4):523-7.
1059. Chaberny IE, Schnitzler P, Geiss HK, Wendt C. An outbreak of epidemic keratoconjunctivitis in a pediatric unit due to adenovirus type 8. *Infect Control Hosp Epidemiol* 2003;24(7):514-9.
1060. Warren D, Nelson KE, Farrar JA, et al. A large outbreak of epidemic keratoconjunctivitis: problems in controlling nosocomial spread. *J Infect Dis* 1989;160(6):938-43.
1061. www.cdc.gov/ncidod/dvrd/cjd/qa_cjd_infection_control.htm
1062. Wang CY, Wu HD, Hsueh PR. Nosocomial transmission of cryptococcosis. *N Engl J Med* 2005;352(12):1271-2.
1063. Beyt BE, Jr., Waltman SR. Cryptococcal endophthalmitis after corneal transplantation. *N Engl J Med* 1978;298(15):825-6.
1064. Widdowson MA, Glass R, Monroe S, et al. Probable transmission of norovirus on an airplane. *Jama* 2005;293(15):1859-60.
1065. CDC. Prevention of Hepatitis A Through Active or Passive Immunization: Recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep* 1999;48(RR-12):1-37.
1066. Rosenblum LS, Villarino ME, Nainan OV, et al. Hepatitis A outbreak in a neonatal intensive care unit: risk factors for transmission and evidence of prolonged viral excretion among preterm infants. *J Infect Dis* 1991;164(3):476-82.
1067. Carl M, Kantor RJ, Webster HM, Fields HA, Maynard JE. Excretion of hepatitis A virus in the stools of hospitalized hepatitis patients. *J Med Virol* 1982;9(2):125-9.
1068. Robson SC, Adams S, Brink N, Woodruff B, Bradley D. Hospital outbreak of hepatitis E. *Lancet* 1992;339(8806):1424-5.
1069. Arvin A, Whitley R. Herpes Simplex virus infections in *Infectious Diseases of the Fetus and Newborn Infant*, ed. Remington JS and Klein JO. Fifth Edition. WB Saunders Co., Philadelphia, PA. 2001.
1070. Enright AM, Prober CG. Neonatal herpes infection: diagnosis, treatment and prevention. *Semin Neonatol* 2002;7(4):283-91.
1071. Esper F, Boucher D, Weibel C, Martinello RA, Kahn JS. Human metapneumovirus infection in the United States: clinical manifestations

- associated with a newly emerging respiratory infection in children. *Pediatrics* 2003;111(6 Pt 1):1407-10.
1072. Colodner R, Sakran W, Miron D, Teitler N, Khavalevsky E, Kopelowitz J. *Listeria monocytogenes* cross-contamination in a nursery. *Am J Infect Control* 2003;31(5):322-4.
1073. Farber JM, Peterkin PI, Carter AO, Varughese PV, Ashton FE, Ewan EP. Neonatal listeriosis due to cross-infection confirmed by isoenzyme typing and DNA fingerprinting. *J Infect Dis* 1991;163(4):927-8.
1074. Schuchat A, Lizano C, Broome CV, Swaminathan B, Kim C, Winn K. Outbreak of neonatal listeriosis associated with mineral oil. *Pediatr Infect Dis J* 1991;10(3):183-9.
1075. Pejaver RK, Watson AH, Mucklow ES. Neonatal cross-infection with *Listeria monocytogenes*. *J Infect* 1993;26(3):301-3.
1076. Jain SK, Persaud D, Perl TM, et al. Nosocomial malaria and saline flush. *Emerg Infect Dis* 2005;11(7):1097-9.
1077. Abulrahi HA, Bohlega EA, Fontaine RE, al-Seghayer SM, al-Ruwais AA. *Plasmodium falciparum* malaria transmitted in hospital through heparin locks. *Lancet* 1997;349(9044):23-5.
1078. Al-Saigul AM, Fontaine RE, Haddad Q. Nosocomial malaria from contamination of a multidose heparin container with blood. *Infect Control Hosp Epidemiol* 2000;21(5):329-30.
1079. Piro S, Sammud M, Badi S, Al Ssabi L. Hospital-acquired malaria transmitted by contaminated gloves. *J Hosp Infect* 2001;47(2):156-8.
1080. Book LS, Overall JC, Jr., Herbst JJ, Britt MR, Epstein B, Jung AL. Clustering of necrotizing enterocolitis. Interruption by infection-control measures. *N Engl J Med* 1977;297(18):984-6.
1081. Rotbart HA, Levin MJ. How contagious is necrotizing enterocolitis? *Pediatr Infect Dis* 1983;2(5):406-13.
1082. Rotbart HA, Levin MJ, Yolken RH, Manchester DK, Jantzen J. An outbreak of rotavirus-associated neonatal necrotizing enterocolitis. *J Pediatr* 1983;103(3):454-9.
1083. Gerber AR, Hopkins RS, Lauer BA, Curry-Kane AG, Rotbart HA. Increased risk of illness among nursery staff caring for neonates with necrotizing enterocolitis. *Pediatr Infect Dis* 1985;4(3):246-9.
1084. Sanchez MP, Erdman DD, Torok TJ, Freeman CJ, Matyas BT. Outbreak of adenovirus 35 pneumonia among adult residents and staff of a chronic care psychiatric facility. *J Infect Dis* 1997;176(3):760-3.(s).
1085. Singh-Naz N, Brown M, Ganeshanathan M. Nosocomial adenovirus infection: molecular epidemiology of an outbreak. *Pediatr Infect Dis J* 1993;12(11):922-5.

1086. Uemura T, Kawashita T, Ostuka Y, Tanaka Y, Kusubae R, Yoshinaga M. A recent outbreak of adenovirus type 7 infection in a chronic inpatient facility for the severely handicapped. *Infect Control Hosp Epidemiol* 2000;21(9):559-60.
1087. Nuorti JP, Butler JC, Crutcher JM, et al. An outbreak of multidrug-resistant pneumococcal pneumonia and bacteremia among unvaccinated nursing home residents. *N Engl J Med* 1998;338(26):1861-8.
1088. Houff SA, Burton RC, Wilson RW, et al. Human-to-human transmission of rabies virus by corneal transplant. *N Engl J Med* 1979;300(11):603-4.
1089. CDC. Human Rabies Prevention - United States, 1999 Recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR* 1999;48 (RR-1):1-21.
1090. Hayden FG. Rhinovirus and the lower respiratory tract. *Rev Med Virol* 2004;14(1):17-31.
1091. Valenti WM, Clarke TA, Hall CB, Menegus MA, Shapiro DL. Concurrent outbreaks of rhinovirus and respiratory syncytial virus in an intensive care nursery: epidemiology and associated risk factors. *J Pediatr* 1982;100(5):722-6.
1092. Chidekel AS, Rosen CL, Bazy AR. Rhinovirus infection associated with serious lower respiratory illness in patients with bronchopulmonary dysplasia. *Pediatr Infect Dis J* 1997;16(1):43-7.
1093. Drusin LM, Ross BG, Rhodes KH, Krauss AN, Scott RA. Nosocomial ringworm in a neonatal intensive care unit: a nurse and her cat. *Infect Control Hosp Epidemiol* 2000;21(9):605-7.
1094. Lewis SM, Lewis BG. Nosocomial transmission of *Trichophyton tonsurans* tinea corporis in a rehabilitation hospital. *Infect Control Hosp Epidemiol* 1997;18(5):322-5.
1095. Saiman L, Jakob K, Holmes KW, et al. Molecular epidemiology of staphylococcal scalded skin syndrome in premature infants. *Pediatr Infect Dis J* 1998;17(4):329-34.
1096. Ramage L, Green K, Pyskir D, Simor AE. An outbreak of fatal nosocomial infections due to group A streptococcus on a medical ward. *Infect Control Hosp Epidemiol* 1996;17(7):429-31.
1097. Kakis A, Gibbs L, Eguia J, et al. An outbreak of group A Streptococcal infection among health care workers. *Clin Infect Dis* 2002;35(11):1353-9.
1098. Schwartz B, Elliott JA, Butler JC, et al. Clusters of invasive group A streptococcal infections in family, hospital, and nursing home settings. *Clin Infect Dis* 1992;15(2):277-84.
1099. National Communicable Disease Center. Isolation Techniques for Use in Hospitals. 1st ed. Washington, DC: US Government Printing Office;. PHS publication no 2054 1970.

1100. CDC. Isolation Techniques for Use in Hospitals. 2nd ed. Washington, DC: US Government Printing Office;1975. HHS publication no. (CDC) 80-8314. 1975.
1101. Garner JS, Simmons BP. CDC Guideline for Isolation Precautions in Hospitals. Atlanta, GA: US Department of Health and Human Services, Public Health Service, Centers for Disease Control; 1983. HHS publication no. (CDC) 83-8314. Infect Control 1983;4:245-325.
1102. Lynch P, Jackson MM, Cummings MJ, Stamm WE. Rethinking the role of isolation practices in the prevention of nosocomial infections. Ann Intern Med 1987;107(2):243-6.



Norovirus Gastroenteritis:

Management of Outbreaks in Healthcare Settings



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

Norovirus



- ❑ The most common cause of cases of acute gastroenteritis and gastroenteritis outbreaks
- ❑ Can affect nearly everyone in the population (from children to the elderly and everyone in between!) particularly because there is no long term immunity to the virus
- ❑ Causes acute but self-limited diarrhea, often with vomiting, abdominal cramping, fever, and fatigue
 - Most individuals recover from acute symptoms with 2-3 days , but can be more severe in vulnerable populations

Burden of Norovirus Infection



- ❑ #1 cause of acute gastroenteritis in U.S.
 - 21 million cases annually
 - 1 in 14 Americans become ill each year
 - 71,000 hospitalized annually in U.S.
 - 80 deaths annually among elderly in U.K.
 - 91,000 emergency room visits overall in the U.S.

- ❑ Occurs year round with peak activity during the winter months

- ❑ Cases occur in all settings, across the globe

Norovirus in Healthcare Facilities

- ❑ Norovirus is a recognized cause of gastroenteritis outbreaks in institutions.
- ❑ Healthcare facilities are the most commonly reported settings of norovirus gastroenteritis outbreaks in the US and other industrialized countries.
- ❑ Outbreaks of gastroenteritis in healthcare settings pose a risk to patients, healthcare personnel, and to the efficient provision of healthcare services.



Norovirus Activity in Healthcare

- Incidence of norovirus outbreaks in acute care facilities and community hospitals within the United States remains unclear.
- This is in contrast with the established high burden of acute care hospital outbreaks reported in many other industrialized countries.

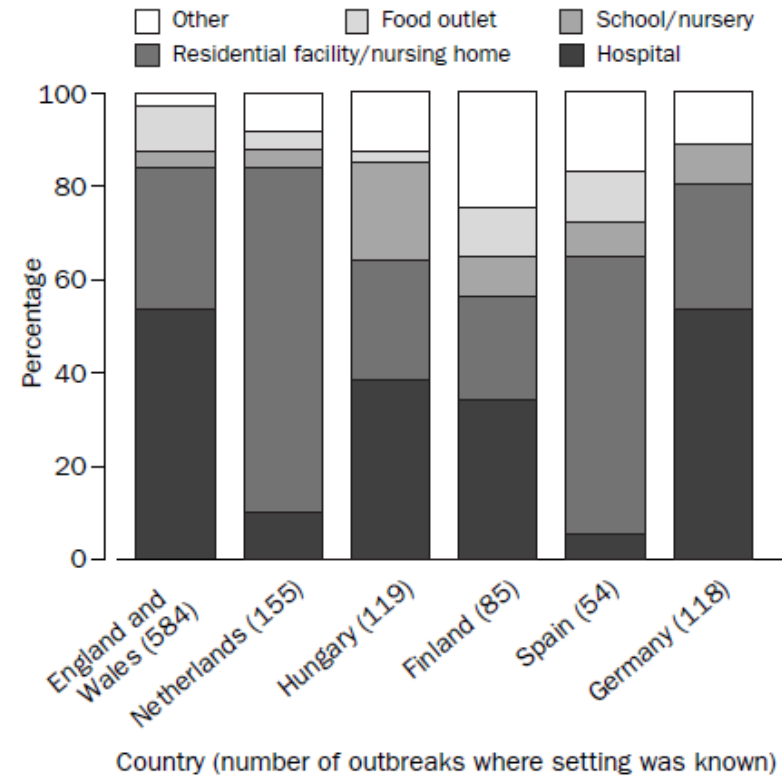
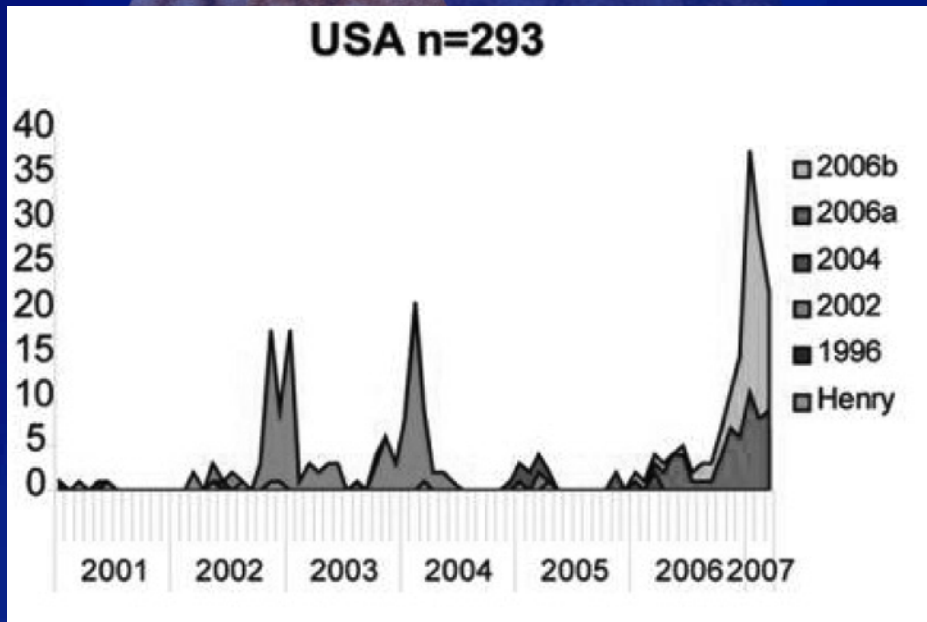


Figure 4: **Setting of norovirus outbreak in 2002 for six European regions**

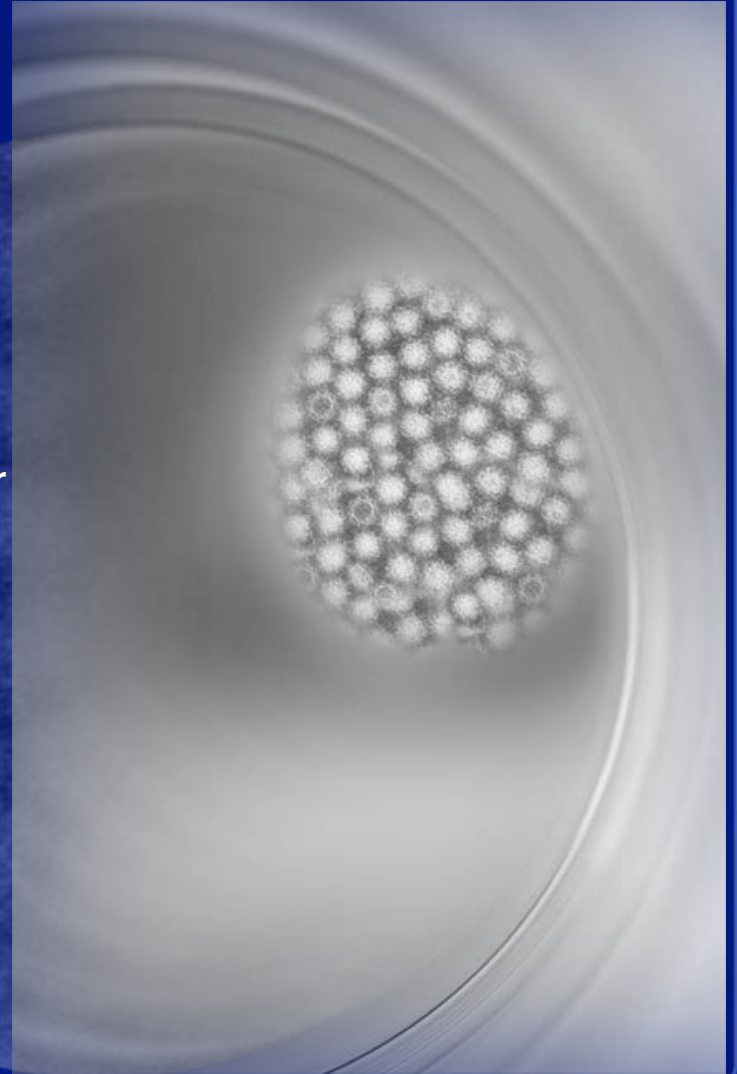
Dynamic Nature of Norovirus in the US



- Genogroup II type 4 (GII.4) noroviruses cause >75% of outbreaks worldwide
- New strains of GI.4 emerge every 3-5 years
- The periodic emergence of new strains is associated with heightened norovirus activity
- New strains in the 2002/03 and 2006/07 winters caused a surge in outbreaks

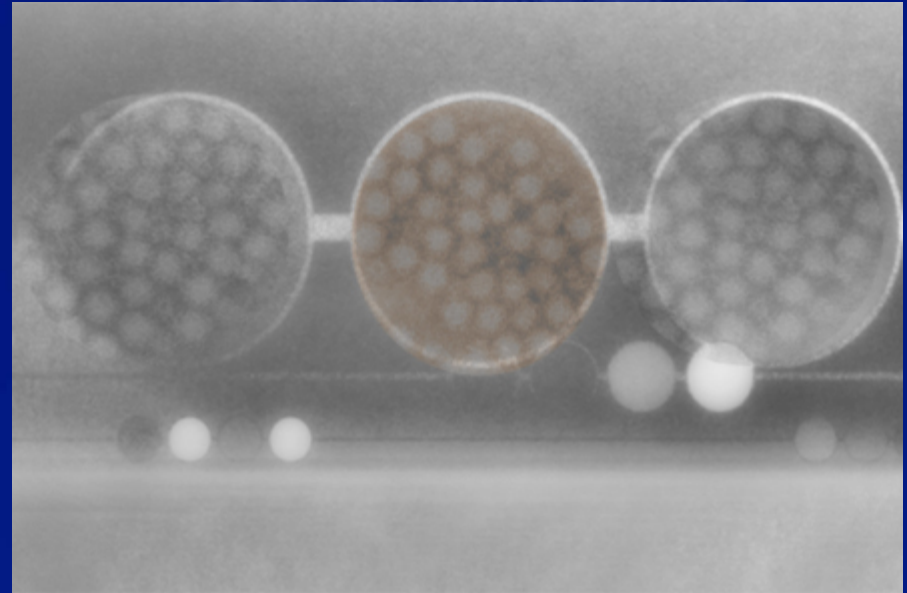
Clinical Disease

- ❑ Infectious dose: 18-1000 viral particles
- ❑ Incubation period: 12-48 hours
- ❑ Acute-onset vomiting and/or diarrhea
 - Watery, non-bloody stools
 - Abdominal cramps, nausea, low-grade fever
 - 30% infections asymptomatic
- ❑ Most recover after 12-72 hours
 - Up to 10% seek medical attention; some require hospitalization and fluid therapy
 - More severe illness and death possible in elderly and those with other illnesses



Viral Shedding

- ❑ Primarily in stool, but can also be present in vomitus
- ❑ Shedding peaks 4 days after exposure
- ❑ In some individuals, shedding may occur for at least 2-3 weeks
- ❑ $\sim 10^{12}$ viral copies/gram feces
- ❑ May occur after resolution of symptoms
- ❑ Infectivity of shed virus in environment unknown
- ❑ Shedding in asymptomatic individuals is common but their role in transmission is not known

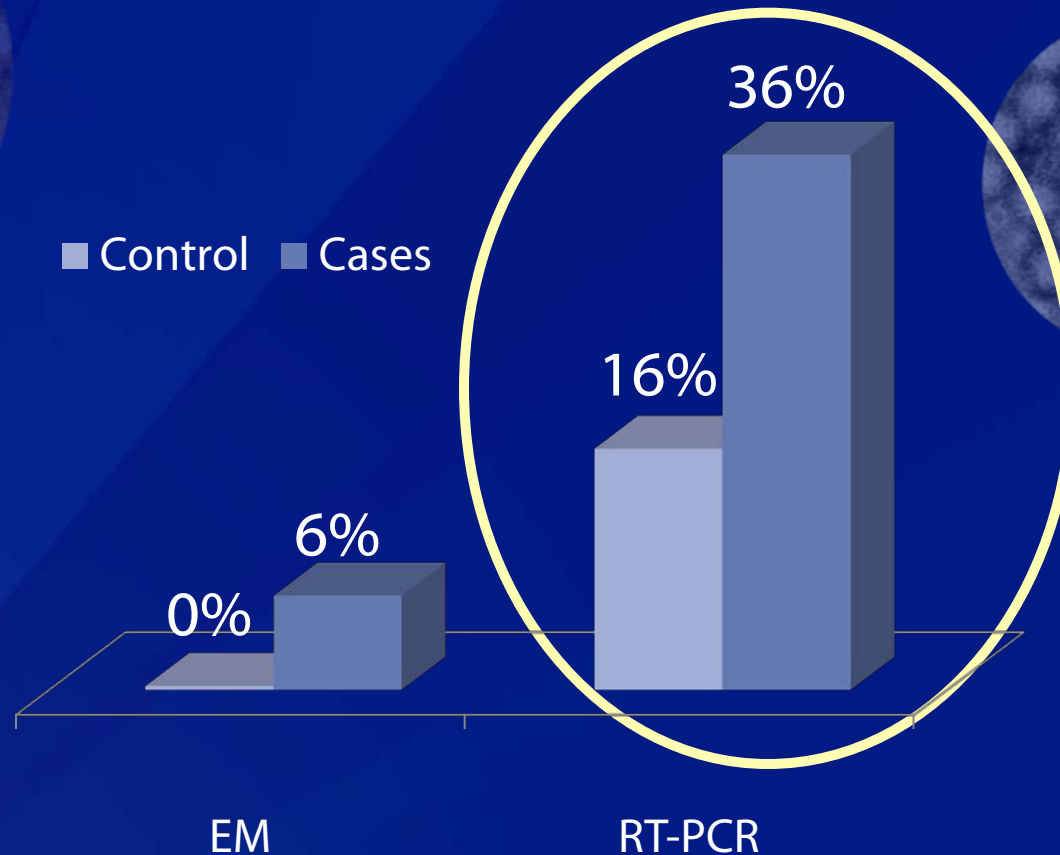


Immunity to Norovirus

- ❑ Short-term immunity after infection
- ❑ There is little cross protective immunity (against different genotypes)
- ❑ No long-term immunity
 - Protection believed to last less than one year, and in some studies, protection may only last a few months
- ❑ Genetic susceptibility
 - A portion of the population may be genetically resistant to norovirus infection
 - Currently no commercially available test to identify those who might carry genes conferring resistance to norovirus infection



Norovirus Prevalence in the Community



Using sensitive PCR diagnostics, norovirus is frequently detected in stools of both infected individuals (cases) and healthy asymptomatic individuals (controls)

Transmission of Disease

- ❑ Person to person
 - Direct fecal-oral
 - Ingestion of aerosolized vomitus
 - Indirect via fomites or contaminated environment

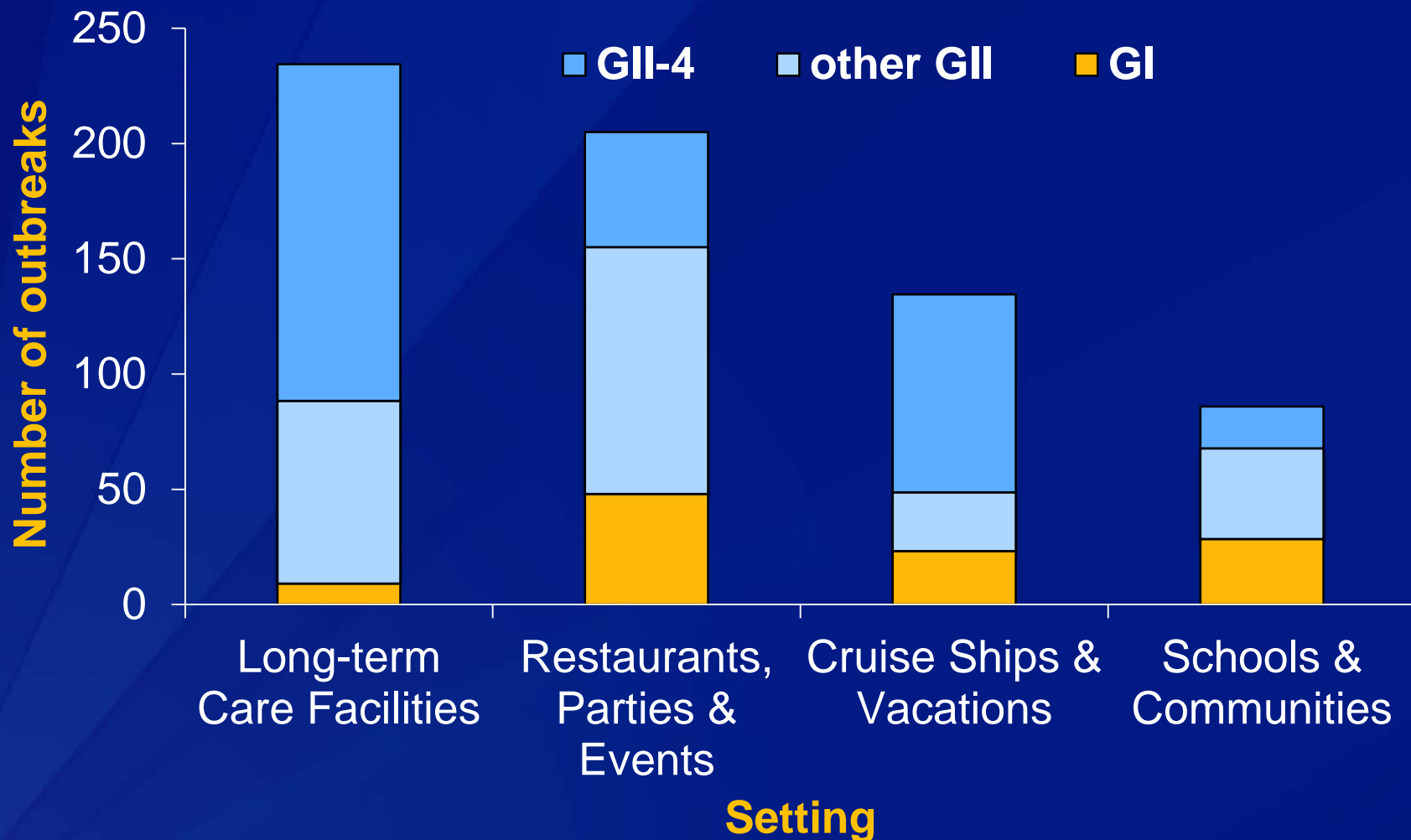
- ❑ Food
 - Contamination by infected food handlers
 - Point of service or source (e.g., raspberries, oysters)

- ❑ Recreational and Drinking Water
 - Well contamination from septic tank
 - Chlorination system breakdown

- ★ In healthcare, the most likely and common modes of transmission are through direct contact with infected persons or contaminated equipment

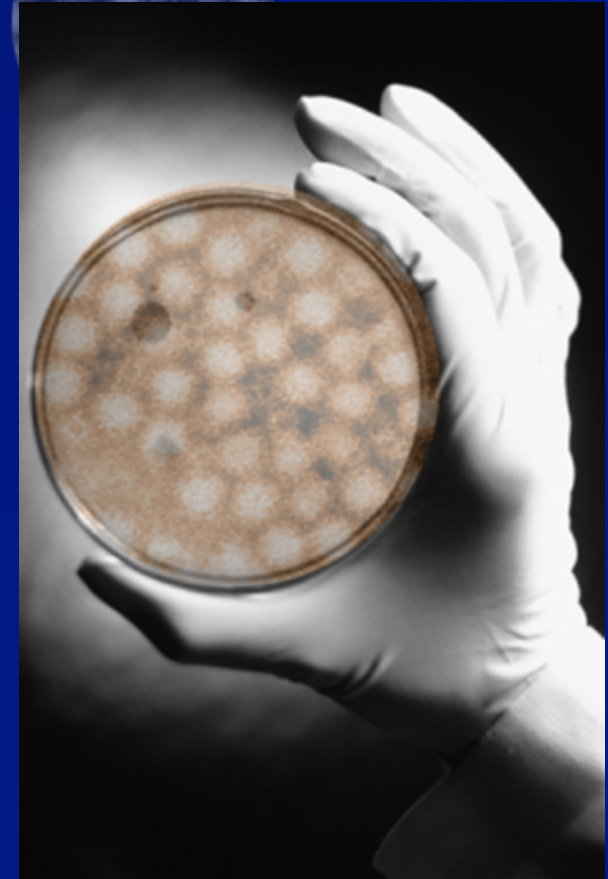


Setting of Norovirus Outbreaks Reported to CDC, United States 1994-2006



Laboratory Confirmation of Norovirus

- ❑ Where available, reverse transcription polymerase chain reaction (RT-PCR) confirmation is the preferred diagnostic for norovirus
- ❑ State public health laboratories may be able to provide RT-PCR diagnostics to confirm norovirus
- ❑ Typically, state laboratories require a minimum number of stool samples from a subset of symptomatic patients before initiating confirmatory testing



Submitting Clinical Samples for Norovirus Testing

- ❑ Consult with receiving clinical, local or state health labs prior to submitting samples for norovirus identification
 - Depending on laboratory policies, may need multiple suspect cases before specimen testing can be performed
- ❑ Stool specimens should be collected from individuals during acute phase of illness
 - Virus may be able to be detected in specimens taken later in the course of illness, but sensitivity is reduced
- ❑ Submit stool specimens as early as possible during a potential outbreak or cluster
- ❑ While not ideal, vomitus may be submitted for testing to some labs
- ❑ Both staff and patient cases can be tested

What should clinical staff do when they suspect norovirus?

- ❑ Key Infection Control Activities
 - Rapid identification and isolation of suspected cases of norovirus gastroenteritis
 - Communicating the presence of suspected cases to Infection Preventionists
 - Promoting increased adherence to hand hygiene, particularly the use of soap and water after contact with symptomatic patients
 - Enhanced environmental cleaning and disinfection

- ❑ Promptly initiate investigations
 - Collection of clinical and epidemiological information
 - Obtain clinical samples

Infection Control: Patient Isolation or Cohorting



- ❑ In healthcare settings where risk of transmission is high, use of isolation precautions is often the most effective means of interrupting transmission
- ❑ CONTACT PRECAUTIONS – single occupancy room with a dedicated bathroom, strict adherence to hand hygiene, wear gloves and gown upon room entry
 - Use Contact Precautions for a minimum of 48 hours after the resolution of symptoms
 - Symptomatic patients may be cohorted together
 - Exclude ill staff members and food handlers in healthcare facilities for a minimum of 48 hours following resolution of their symptoms
 - Exclude non-essential personnel and visitors

Infection Control: Hand Hygiene

- ❑ Wash with soap and water after contact with symptomatic patients
 - For all other indications, refer to the 2002 Guideline for Hand Hygiene*
- ❑ Alcohol-based hand sanitizers
 - Currently available products appear to be relatively ineffective against norovirus
 - Consider using FDA-compliant alcohol-based hand sanitizers for other indications (e.g., before contact with NV patient)*



*CDC HICPAC Guideline for Hand Hygiene in Health-Care Settings:
<http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf>

Infection Control: Environmental Cleaning and Disinfection

- ❑ The use of chemical cleaning and disinfecting agents are key in interrupting norovirus spread from contaminated environmental surfaces.
- ❑ Increase the frequency of cleaning and disinfection of patient care areas and frequently touched surfaces
e.g., increase ward/unit level cleaning to twice daily, with frequently touched surfaces cleaned and disinfected three times daily
- ❑ Use commercial cleaning and disinfection products registered with the U.S. Environmental Protection Agency (e.g., sodium hypochlorite (bleach) solution, hydrogen peroxide products, etc.)
http://www.epa.gov/pesticides/antimicrobials/list_g_norovirus.pdf
- ❑ It is critical to follow manufacturer instructions for methods of application, amount, dilution, and contact time

Infection Control: Other Considerations

- ❑ To reduce transmission, and depending on the magnitude of the outbreak, cohort staff to care for patients who are
 - asymptomatic unexposed
 - asymptomatic, potentially exposed
 - symptomatic
- ❑ Remove communal or shared food items for staff or patients for the duration of the outbreak
- ❑ Group activities for patients may need to be suspended; minimize patient movements within a patient care area to help control transmission



Surveillance for Norovirus Cases

- ❑ Units can use a “line list” to track symptomatic staff and patients
- ❑ During an outbreak, collect key information to assist with controlling the outbreak and to inform local/state health departments on outbreak details
- ❑ Suggested line list elements
 - Case (staff/patient) identifier
 - Case location
 - Symptoms
 - Outcome / Date of Resolution
 - Diagnostics submitted



Reporting Outbreaks

Internal Communication

- ❑ Report gastroenteritis outbreaks (e.g., 2 or more suspected or confirmed cases among staff or patients) to infection control units
- ❑ Outbreaks should also be reported to clinical management
- ❑ Important to include communications, laboratory, environmental services, admitting, occupational health departments



Reporting Outbreaks

External Reporting

- ❑ Report norovirus outbreaks to your local, county, or state health department
- ❑ In most states, all outbreaks of public health significance are reportable to the state health department
- ❑ Health departments enter norovirus outbreak data (among other pathogens) into National Outbreak Reporting System (NORS) → Centers for Disease Control and Prevention (CDC)



Summary: Management of Norovirus Outbreaks

- ❑ Create awareness of concurrent norovirus outbreaks in the community/ other local healthcare facilities
- ❑ Detect and confirm suspected norovirus cases rapidly
- ❑ During outbreaks, implement
 - Contact Precautions,
 - enhanced hand hygiene,
 - environmental infection control measures,
 - exclusion of ill staff from work for a minimum of 48 hrs after symptom resolution
 - surveillance for new and resolving cases,
- ❑ Develop a communication plan during outbreaks to include key departments and services
- ❑ Consult with and report outbreak to local/state health departments

Additional Resources

- ❑ **Norovirus in healthcare settings**

<http://www.cdc.gov/HAI/organisms/norovirus.html>

- ❑ **CDC HICPAC Guideline for the Prevention and Control of Norovirus Gastroenteritis Outbreaks in Healthcare Settings**

<http://www.cdc.gov/hicpac/pdf/norovirus/Norovirus-Guideline-2011.pdf>

- ❑ **Updated Norovirus Outbreak Management and Disease Prevention Guidelines**

http://www.cdc.gov/mmwr/preview/mmwrhtml/rr6003a1.htm?s_cid=rr6003a1_e

- ❑ **General information on norovirus**

<http://www.cdc.gov/ncidod/dvrd/revb/gastro/norovirus.htm>

For more information please contact Centers for Disease Control and Prevention

1600 Clifton Road NE, Atlanta, GA 30333

Telephone: 1-800-CDC-INFO (232-4636)/TTY: 1-888-232-6348

E-mail: cdcinfo@cdc.gov

Web: <http://www.cdc.gov>

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.



U.S. Department of Health and Human Services

Centers for Disease Control and Prevention

Sample Communication Framework: Suspected or Confirmed Norovirus Outbreaks

Department Notification	Role	Department Contact Position	Contact Name(s)	Contact email(s)/ phone #
Core groups to be notified : initial outbreak measures (2 or more cases, epidemiologically linked)				
EXAMPLE Infection prevention and control	Implementation of control measures and education, primary contact in facility outbreak control	e.g., Infection Preventionist	e.g., Malinda Smith	e.g., msmith@hospital.edu/ 515-555-1212
<input type="checkbox"/> Infection prevention and control	Implementation of control measures and education, primary contact in outbreak control			
<input type="checkbox"/> Unit / Ward leadership	Coordination of patient isolation requirements, patient/ staff case finding, modifications to staff assignments, staff absenteeism, visitor policy, etc			
<input type="checkbox"/> Clinical laboratory	Notify and coordinate testing incoming stool specimens for norovirus confirmation, estimate capacity for performing diagnostics, instructions on how to label and order specimens			
<input type="checkbox"/> Environmental services	Assess need for enhanced cleaning frequencies, changes to cleaning and disinfection products for outbreaks, coordinate needs for terminal cleaning of rooms/units, ward closure, ensure correct and complete adherence to cleaning protocols			
<input type="checkbox"/> Central supply/Distribution services	Assess need for increased personal protective equipment, etc.			
<input type="checkbox"/> Linen services	Anticipate increased need for linens (e.g., privacy curtains)			



Sample Communication Framework: Suspected or Confirmed Norovirus Outbreaks

Department Notification	Role	Department Contact Position	Contact Name(s)	Contact email(s)/phone #
<input type="checkbox"/> Occupational or Employee Health Services		Monitor and document staff reports of gastrointestinal illness from affected clinical areas; coordinate stool specimen collection and testing if required; monitor clinically adverse events		
<input type="checkbox"/> Relevant clinical care teams		Modifications to patient care plans if necessary (e.g., discharge planning)		
<input type="checkbox"/> Allied health services		Modifications to patient therapy for isolated patients (e.g., appointments postponed or rescheduled, location of care)		
<input type="checkbox"/> Patient placement/admitting services		Awareness/planning for potential increases in isolation needs, blocked beds, wing or unit closures		
<input type="checkbox"/> State or local health department		Preliminary and confirmatory reporting to outbreak coordination units, requests for assistance or follow-up if necessary, coordination with any media inquiries		
Core groups to be notified : uncontrolled transmission or requirements for expanded outbreak measures				
<input type="checkbox"/> Public relations		Preparations for press release, media and public inquiries, internal messaging		
<input type="checkbox"/> Risk management		Assist in response coordination, strategic planning		
<input type="checkbox"/> Healthcare facility management and administration		Assess impact of outbreak on operations, need for unit closure, notification, etc.		

Acute Gastroenteritis / Norovirus Case Report Worksheet

Reporting facility: _____ Contact Name/Phone Number: _____ Estimated number of exposed patients during outbreak

Street Address: _____ Outbreak Identification Number (Health Dept. assigned) _____ Estimated number of exposed staff during outbreak

Unit: _____

Patient/Staff Demographics					Case Location	Symptoms					Outcome		Diagnostics				
Name	Unique ID (optional)	Patient (P) Staff (S)	Age	Sex (M/F)	Patients only: Room/Bed	Symptom onset date (mm/dd/yy)	Vomiting (Y/N)	Diarrhea (Y/N)	Bloody stools (Y/N)	Fever (Y/N)	Abdominal cramps (Y/N)	First symptom-free date (mm/dd/yy)	Died (Y/N/Unk)	Specimen(s) collected for diagnostics (Y/N/Unk)	Date of specimen collection (mm/dd/yy)	Lab Results	Location of stool specimen testing (H=HCF lab, C=contracted lab, S=state lab, CD=CDC lab)
1.																	
2.																	
3.																	
4.																	
5.																	
6.																	
7.																	
8.																	
9.																	
10.																	
11.																	
12.																	
13.																	
14.																	
15.																	
16.																	
17.																	
18.																	
19.																	
20.																	

If required, REDACT Name column prior to faxing; FAX to local/state health department upon completion

NOROVIRUS

What healthcare providers should know

What is norovirus?

A virus that can cause severe and sudden gastroenteritis (i.e., inflammation of the lining of the stomach and intestines). Both healthy and compromised persons can be affected.

What are the symptoms?

Nausea, vomiting, diarrhea, and some stomach cramping

Is it contagious?

Norovirus is very easily transmitted through contaminated hands, equipment/surfaces, or food/water

What can I do to prevent norovirus?

Always perform appropriate hand hygiene, particularly after contact with fecal material or after contact with anyone suspected /confirmed with norovirus. Wear gloves when caring for symptomatic patients.

If you have symptoms consistent with norovirus infection, stay home for a *minimum* of 48 hrs after symptom resolution

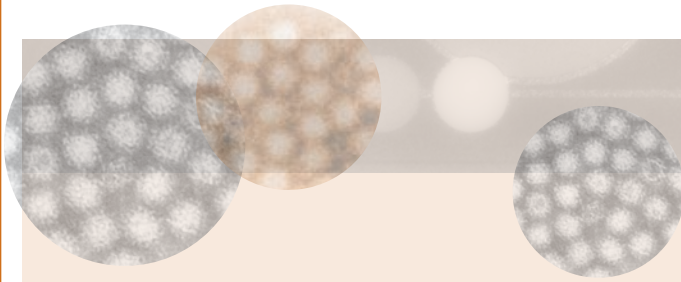
If an outbreak is suspected contact Infection Prevention and Control

► For more information, visit www.cdc.gov



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

Norovirus in Healthcare Facilities Fact Sheet



General Information

Virology

Noroviruses (genus *Norovirus*, family *Caliciviridae*) are a group of related, single-stranded RNA, non-enveloped viruses that cause acute gastroenteritis in humans. Norovirus is the official genus name for the group of viruses provisionally described as “Norwalk-like viruses”. Currently, human noroviruses belong to one of three norovirus genogroups (GI, GII, or GIV), which are further divided into >25 genetic clusters. Over 75% of confirmed human norovirus infections are associated with genotype GII.

Clinical manifestations

The average incubation period for norovirus-associated gastroenteritis is 12 to 48 hours, with a median period of approximately 33 hours. Illness is characterized by nausea, acute-onset vomiting, and watery, non-bloody diarrhea with abdominal cramps. In addition, myalgia, malaise, and headache are commonly reported. Low-grade fever is present in about half of cases. Dehydration is the most common complication and may require intravenous replacement fluids. Symptoms usually last 24 to 60 hours. Up to 30% of infections may be asymptomatic.

Epidemiology of transmission

Noroviruses are highly contagious, with as few as 18 virus particles thought to be sufficient to cause infection. This pathogen is estimated to be the causative agent in over 21 million gastroenteritis cases every year in the United States, representing approximately 60% of all acute gastroenteritis cases from known pathogens. Noroviruses are transmitted primarily through the fecal-oral route, either by direct person-to-person spread or fecally contaminated food or water. Noroviruses can also spread via a droplet route from vomitus. These viruses are relatively stable in the environment and can survive freezing and heating to 60°C (140°F). In healthcare facilities, transmission can also occur through

hand transfer of the virus to the oral mucosa via contact with materials, fomites, and environmental surfaces that have been contaminated with either feces or vomitus.

Norovirus infections are seen in all age groups, although severe outcomes and longer durations of illness are most likely to be reported among the elderly. Among hospitalized persons who are immunocompromised or have significant medical comorbidities, norovirus infection can directly result in prolonged hospital stays, additional medical complications, and, rarely, death. There is currently no vaccine available for norovirus and, generally, no specific medical treatment is offered for norovirus infection apart from oral or intravenous repletion of volume.

The ease of its transmission, a very low infectious dose, a short incubation period, environmental persistence, and lack of durable immunity following infection enables norovirus to spread rapidly through confined populations. Healthcare facilities and other institutional settings (e.g., daycare centers, schools, etc.) are particularly at-risk for outbreaks because of increased person-to-person contact. Healthcare facilities managing outbreaks of norovirus gastroenteritis may experience significant costs relating to isolation precautions and personal protective equipment, ward closures, supplemental environmental cleaning, staff cohorting or replacement, and sick time.

Diagnosis of norovirus infection

Diagnosis of norovirus infection relies on the detection of viral RNA in the stools of affected persons, by use of reverse transcription-polymerase chain reaction (RT-PCR) assays. This technology is available at CDC and most state public health laboratories and should be considered in the event of outbreaks of gastroenteritis in healthcare facilities. Enzyme immune-assays may also be used for identification of norovirus outbreak but are not recommended for diagnosis of individuals. Identification of the virus can be best made from stool specimens taken within 48 to 72 hours after onset of symptoms, although positive results can be obtained by using RT-PCR on samples taken as long as 7 days after symptom onset. Because of the limited availability of timely and routine laboratory diagnostic methods, a clinical diagnosis of norovirus infection is often used, especially when other agents of gastroenteritis have been ruled out.



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention



Measures to Limit Transmission*

Patient Cohorting and Isolation Precautions

Avoid exposure to vomitus or diarrhea. Place patients on Contact Precautions in a single occupancy room if they present with symptoms consistent with norovirus gastroenteritis.

Hand Hygiene

During outbreaks, use soap and water for hand hygiene after providing care or having contact with patients suspected or confirmed with norovirus gastroenteritis.

Patient Transfer and Ward Closure

Consider limiting transfers to those for which the receiving facility is able to maintain Contact Precautions; otherwise, it may be prudent to postpone transfers until patients no longer require Contact Precautions. During outbreaks, medically suitable individuals recovering from norovirus gastroenteritis can be discharged to their place of residence.

Diagnostics

In the absence of clinical laboratory diagnostics or in the case of delay in obtaining laboratory results, use Kaplan's clinical and epidemiologic criteria to identify a norovirus gastroenteritis outbreak.

Kaplan's Criteria

1. Vomiting in more than half of symptomatic cases and,
2. Mean (or median) incubation period of 24 to 48 hours and,
3. Mean (or median) duration of illness of 12 to 60 hours and,
4. No bacterial pathogen isolated in stool culture

Environmental Cleaning

Increase the frequency of cleaning and disinfection of patient care areas and frequently touched surfaces during outbreaks of norovirus gastroenteritis (e.g., increase ward/unit level cleaning to twice daily to maintain cleanliness, with frequently touched surfaces cleaned and disinfected three times daily using the US Environmental Protection Agency's list of approved products for healthcare settings (<http://www.epa.gov/oppad001/chemregindex.htm>).

Staff Leave and Policy

Develop and adhere to sick leave policies for healthcare personnel who have symptoms consistent with norovirus infection.

Exclude ill personnel from work for a minimum of 48 hours after the resolution of symptoms. Once personnel return to work, the importance of performing frequent hand hygiene should be reinforced, especially before and after each patient contact.

Establish protocols for staff cohorting in the event of an outbreak of norovirus gastroenteritis. Ensure staff care for one patient cohort on their ward and do not move between patient cohorts (e.g., patient cohorts may include symptomatic, asymptomatic exposed, or asymptomatic unexposed patient groups).

Communication and Notification

As with all outbreaks, notify appropriate local and state health departments, as required by state and local public health regulations, if an outbreak of norovirus gastroenteritis is suspected.

*Prevention and control recommendations taken from priority recommendations in the CDC HICPAC Guideline for the Prevention and Control of Norovirus Gastroenteritis Outbreaks in Healthcare Settings (<http://www.cdc.gov/hicpac/pdf/norovirus/Norovirus-Guideline-2011.pdf>)

Date last modified: September 6, 2011

Content source: Division of Healthcare Quality Promotion (DHQP), National Center for Preparedness, Detection, and Control of Infectious Diseases (NCEZID)

Contact Us: Centers for Disease Control and Prevention
1600 Clifton Road, Atlanta, GA 30333, USA

1-800-CDC-INFO (1-800-232-4636)

TTY:888-232-6348,

24 hours/everyday at cdcinfo@cdc.gov (TTY)



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

A Norovirus Outbreak Control Resource Toolkit for Healthcare Settings

Norovirus is the most common cause of sporadic gastroenteritis as well as gastroenteritis outbreaks. Because of high levels of contact and vulnerable patient populations, healthcare settings can be particularly susceptible to outbreaks of norovirus. To help address the challenges of managing and controlling norovirus gastroenteritis outbreaks in healthcare settings, the Centers for Disease Control and Prevention (CDC) is offering a toolkit for healthcare professionals including up-to-date information, recommended infection control measures, and tools for outbreak response coordination and reporting.

The toolkit serves as a complementary resource to the CDC HICPAC Guideline for the Prevention and Control of Norovirus Gastroenteritis Outbreaks in Healthcare Settings, 2011 (<http://www.cdc.gov/hicpac/norovirus/pubs.html>). These resources were jointly developed by CDC's Division of Healthcare Quality Promotion and Division of Viral Diseases and in consultation with infection preventionists around the country.

For healthcare professionals, the toolkit contains a variety of materials to support outbreak response as well as staff and patient education efforts including:

- ▶ A presentation on general norovirus epidemiology, infection control measures, and outbreak reporting guidance
- ▶ A norovirus fact sheet with general information and measures to limit transmission
- ▶ A poster for healthcare providers highlighting signs and symptoms of norovirus gastroenteritis and preventive infection control measures
- ▶ Key infection control recommendations based on the CDC HICPAC Guideline for the Prevention and Control of Norovirus Gastroenteritis Outbreaks in Healthcare Settings
- ▶ A sample line list for tracking and reporting norovirus cases among patients and healthcare personnel
- ▶ Sample worksheets to coordinate efforts to support
 - Laboratory confirmation of norovirus from stool (or vomitus) specimens
 - Internal and external communications for outbreak management

We encourage you to share these materials with your colleagues to help inform them about outbreaks of norovirus in healthcare settings and the recommended strategies for prevention and control.



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

Key Infection Control Recommendations

for the Control of Norovirus Outbreaks in Healthcare Settings

Patient Cohorting and Isolation Precautions

Place patients with norovirus gastroenteritis on Contact Precautions for a minimum of 48 hours after the resolution of symptoms

When symptomatic patients cannot be accommodated in single occupancy rooms, efforts should be made to separate them from asymptomatic patients. These efforts may include placing patients in multi-occupancy rooms, or designating patient care areas or contiguous sections within a facility for patient cohorts.

- ▶ Staff who have recovered from recent suspected norovirus infection associated with an outbreak may be best suited to care for symptomatic patients until the outbreak resolves.

Consider the following precautions:

- ▶ Minimize patient movements within a ward or unit during norovirus outbreaks
- ▶ Restrict symptomatic and recovering patients from leaving the patient-care area unless it is for essential care or treatment
- ▶ Suspend group activities (e.g., dining events) for the duration of a norovirus outbreak.

Hand Hygiene

- ▶ Actively promote adherence to hand hygiene among healthcare personnel, patients, and visitors in patient care areas affected by outbreaks of norovirus gastroenteritis
- ▶ During outbreaks, use soap and water for hand hygiene after providing care or having contact with patients suspected or confirmed with norovirus gastroenteritis.

*For all other hand hygiene indications refer to the 2002 HICPAC Guideline for Hand Hygiene in Health-Care Settings (<http://www.cdc.gov/mmwr/PDF/rr/rr51116.pdf>).



Personal Protective Equipment (PPE)

- ▶ If norovirus infection is suspected, adherence to PPE use according to Contact and Standard Precautions is recommended for individuals entering the patient care area (i.e., gowns and gloves upon entry).



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

Patient Transfer and Ward Closure

- ▶ Consider the closure of wards to new admissions or transfers as a measure to attenuate the magnitude of a norovirus outbreak.
- ▶ Consider limiting transfers to those for which the receiving facility is able to maintain Contact Precautions; otherwise, it may be prudent to postpone transfers until patients no longer require Contact Precautions. During outbreaks, medically suitable individuals recovering from norovirus gastroenteritis can be discharged to their place of residence.

Diagnostics

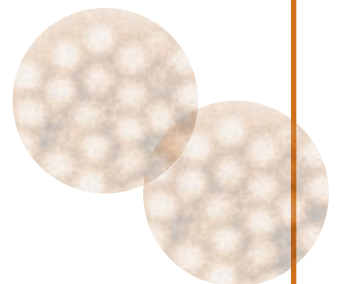
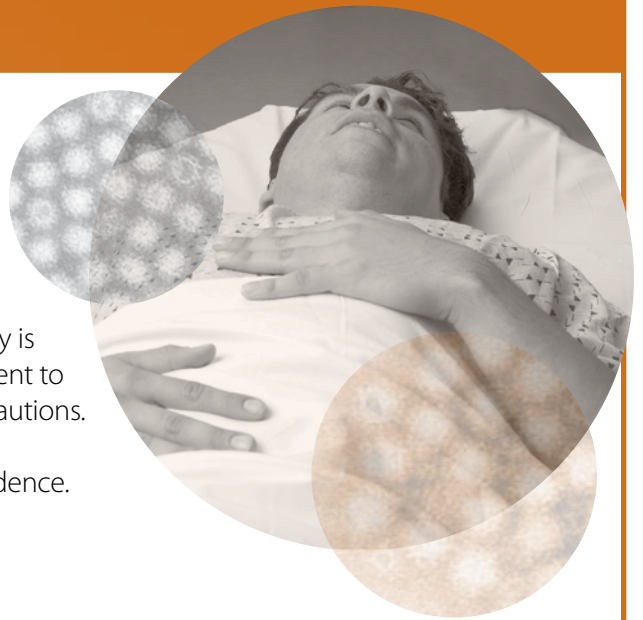
- ▶ In the absence of clinical laboratory diagnostics or in the case of delay in obtaining laboratory results, use Kaplan's clinical and epidemiologic criteria to identify a norovirus gastroenteritis outbreak.


Kaplan's Criteria:

1. Vomiting in more than half of symptomatic cases, and
 2. Mean (or median) incubation period of 24 to 48 hours, and
 3. Mean (or median) duration of illness of 12 to 60 hours, and
 4. No bacterial pathogen isolated from stool culture
- ▶ Consider submitting stool specimens as early as possible during a suspected norovirus gastroenteritis outbreak and ideally from individuals during the acute phase of illness (within 2-3 days of onset).
 - ▶ Specimens obtained from vomitus may be submitted for laboratory identification of norovirus when fecal specimens are unavailable (consult with your lab). Testing of vomitus as compared to fecal specimens may be less sensitive due to lower detectable viral concentrations.
 - ▶ Routine collecting and processing of environmental swabs during a norovirus outbreak is not required.

Environmental Cleaning

- ▶ Perform routine cleaning and disinfection of frequently touched environmental surfaces and equipment in isolation and cohorted areas, as well as high traffic clinical areas. Frequently touched surfaces include, but are not limited to, commodes, toilets, faucets, hand/bedrailing, telephones, door handles, computer equipment, and kitchen preparation surfaces.
- ▶ Increase the frequency of cleaning and disinfection of patient care areas and frequently touched surfaces during outbreaks of norovirus gastroenteritis (e.g., increase ward/unit level cleaning twice daily to maintain cleanliness, with frequently touched surfaces cleaned and disinfected three times daily using EPA-approved products for healthcare settings).



- 
- ▶ Clean and disinfect surfaces starting from the areas with a lower likelihood of norovirus contamination (e.g., tray tables, counter tops) to areas with highly contaminated surfaces (e.g., toilets, bathroom fixtures). Change mop heads when new solutions are prepared, or after cleaning large spills of emesis or fecal material.
 - ▶ No additional provisions for using disposable patient service items such as utensils or dishware are suggested for patients with symptoms of norovirus infection. Silverware and dishware may undergo normal processing and cleaning using standard procedures.
 - ▶ Use Standard Precautions for handling soiled patient-service items or linens, which includes the appropriate use of PPE.
 - ▶ Consider changing privacy curtains routinely and upon patient discharge or transfer.



Staff Leave and Policy

- ▶ Exclude ill personnel from work for a minimum of 48 hours after the resolution of symptoms. Once personnel return to work, the importance of performing frequent hand hygiene should be reinforced.
- ▶ Establish protocols for staff cohorting in the event of an outbreak of norovirus. Ensure staff care for one patient cohort on their ward and do not move between patient cohorts (e.g., patient cohorts may include symptomatic, asymptomatic exposed, or asymptomatic unexposed patient groups).
- ▶ Exclude non-essential staff, students, and volunteers from working in areas experiencing outbreaks of norovirus.

Communication and Notification

- ▶ Notify appropriate local and state health departments if an outbreak of norovirus gastroenteritis is suspected.



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention



Central Line-Associated Bloodstream Infections (CLABSI) in Non-Intensive Care Unit (non-ICU) Settings Toolkit

Activity C: ELC Prevention Collaboratives

Alex Kallen, MD, MPH and Priti Patel, MD, MPH

Division of Healthcare Quality Promotion

Centers for Disease Control and Prevention

Draft - 1/22111/09 --- Disclaimer: The findings and conclusions in this presentation are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.





Outline



- **Background**
 - Impact
 - HHS Prevention Targets
 - Pathogenesis
 - Epidemiology
- **Prevention Strategies**
 - Core
 - Supplemental
- **Measurement**
 - Process
 - Outcome
- **Tools for Implementation/Resources/References**



Background: Impact



- Bloodstream infections (BSIs) are a major cause of healthcare-associated morbidity and mortality
 - Up to 35% attributable mortality
 - BSI leads to excess hospital length of stay of 24 days
- Central Line (CL) use a major risk factor for BSI
- More than 250,000 central line-associated BSIs (CLABSIs) in US yearly
- Rates of CLABSI appear to vary by type of catheter

Pittet et al. JAMA 1994; 271 1598-1601.

Klevens et al. Public Health Reports 2007;122:160-6.



Background: HHS Prevention Targets



- Prevention of CLABSIs in Intensive Care Units (ICUs) and “other locations” have 2 associated goals in HHS HAI Prevention Plan:
 - Reduce CLABSIs by 50%
 - 100% adherence with CL insertion practices in non-emergent situations



Background: Impact Outside the ICU



- Most work aimed at reducing CLABSIs in the hospital has been done in ICUs
- Many CLs are found outside ICUs
 - In one study 55% of ICU patients had CL; 24% of non-ICU patients had CL
 - However, as more patients are located outside of the ICU, 70% of hospitalized patients with CLs were outside the ICU

Climo et al. ICHE 2003; 24:942-5.



Background: Impact CLABSI Rates



- CLABSI rates outside ICUs may be similar to rates of these infections in ICUs
- Although data are sparse, in one study CLABSI rates were:
 - 5.7 per 1,000 catheter-days in 4 inpatient wards
 - 5.2 per 1,000 catheter-days for medical ICU

Marschall et al. Infect Control Hospital Epidemiol 2007;28:905-9.



Background: Impact National Healthcare Safety Network (NHSN) CLABSI Rates



- From 2006 – 2008 NHSN report, pooled mean CLABSI rates were:
 - Medical-Surgical ICUs = 1.5 to 2.1 per 1,000 catheter-days
 - Medical-Surgical wards = 1.2 per 1,000 catheter-days

Edwards JR, et al. Am J Infect Control 2009;37:783-805.

<http://www.cdc.gov/nhsn/PDFs/dataStat/2009NHSNReport.PDF>

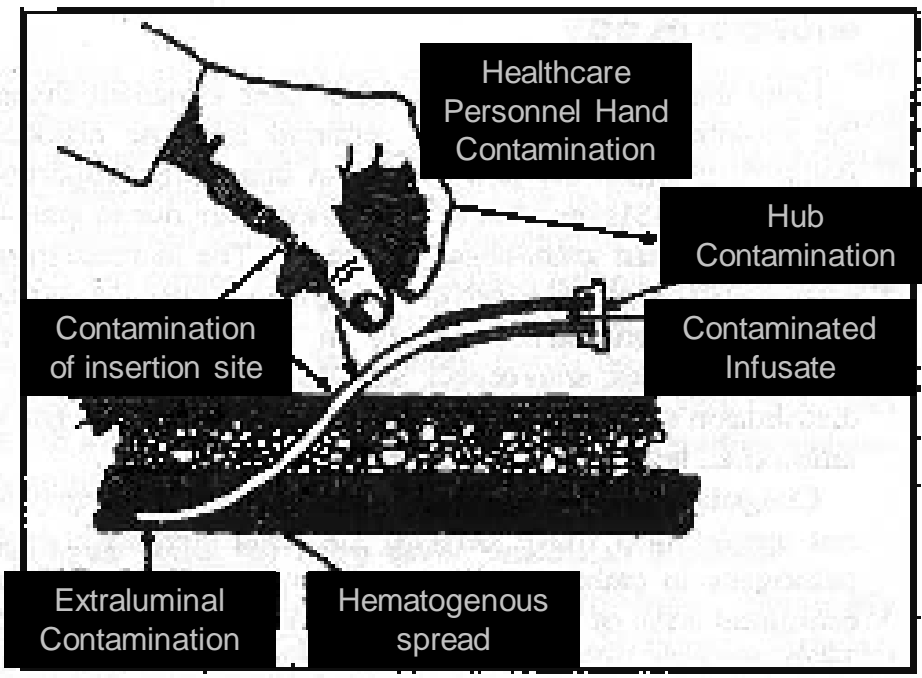


Background: Impact CLABSI in Outpatient Settings



- A number of patient groups may have long-term CLs as outpatients
 - Hemodialysis
 - Malignancy
 - Gastrointestinal tract disorders
 - Pulmonary hypertension
- Rates of CLABSI may be as high as those seen in ICUs
 - In hemodialysis - 1 to 4 per 1,000 catheter-days

Background: Pathogenesis CLABSI



HICPAC. Guideline for Prevention of Intravascular Device-Related Infections. 1996

More Common Mechanisms

1. Pathogen migration along external surface
 - more common early (< 7days)
2. Hub contamination with intraluminal colonization
 - more common >10 days

Less Common Mechanisms

1. Hematogenous seeding from another source
2. Contaminated infusates

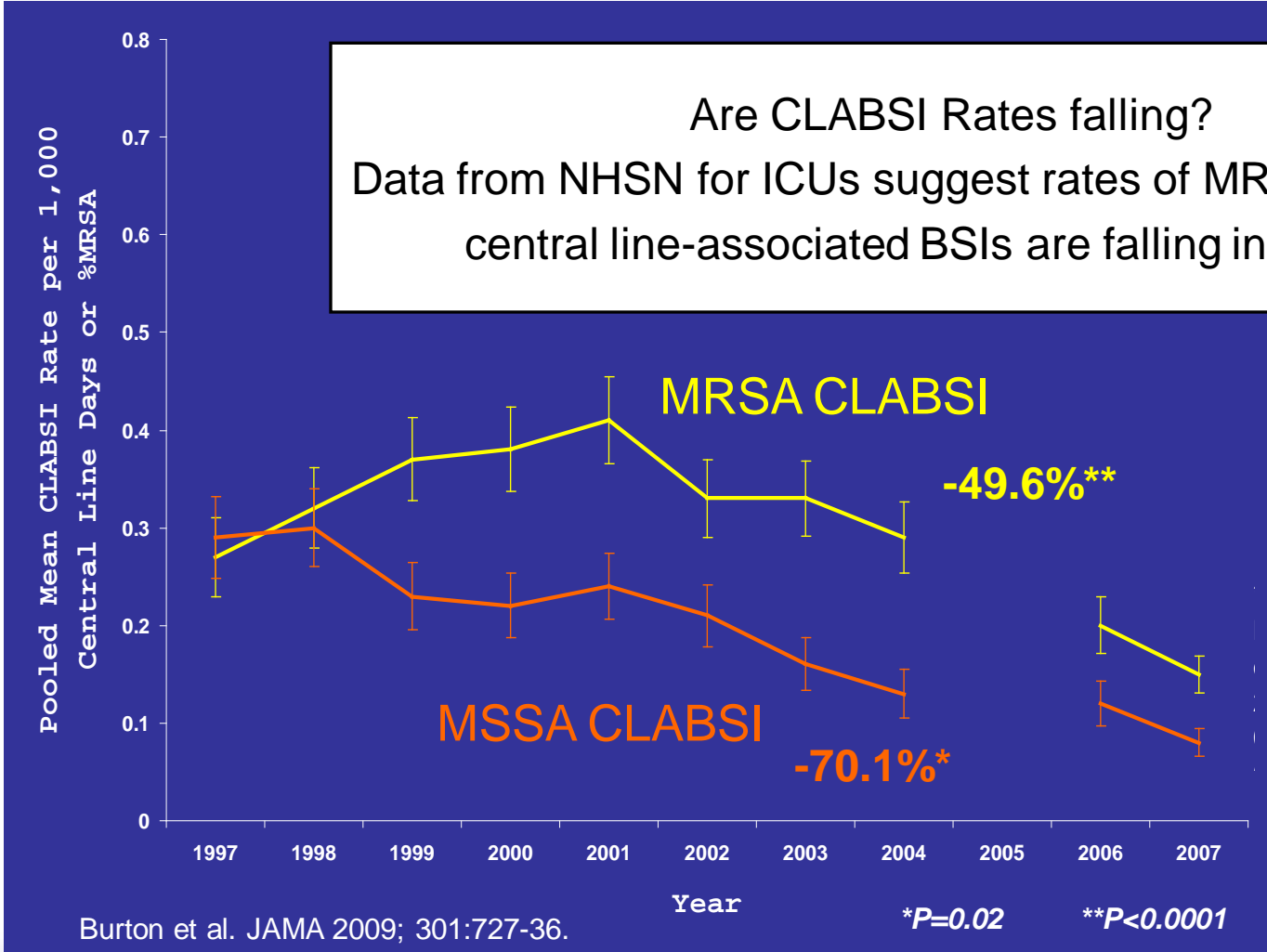


Background: Epidemiology

ALL ICU TYPES: Rates of Methicillin-Resistant and Methicillin-Susceptible *Staphylococcus aureus* CLABSIs—United States, 1997-2007



Are CLABSI Rates falling?
Data from NHSN for ICUs suggest rates of MRSA and MSSA central line-associated BSIs are falling in the U.S.



Burton et al. JAMA 2009; 301:727-36.



Background: Epidemiology Modifiable Risk Factors



Characteristic	Risk Factor Hierarchy
Insertion circumstances	Emergency > elective
Skill of inserter	General > specialized
Insertion site	Femoral > subclavian
Skin antisepsis	70% alcohol, 10% povidone-iodine > 2% chlorhexidine
Catheter lumens	Multilumen > single lumen
Duration of catheter use	Longer duration of use greater risk
Barrier precautions	Submaximal > maximal



Background: Prevention Strategies Interventions



- Pittsburgh Regional Health Initiative – Decrease in CLABSIs in 66 ICUs (68% decrease)
 - Interventions
 - Promotion of best practices
 - » Maximal barrier precautions
 - » Use of chlorhexidine for skin cleansing prior to insertion
 - » Avoidance of femoral site for CL
 - » Use of recommended insertion-site dressing practices
 - » Removal of CL when no longer needed
 - Educational module about BSI prevention
 - Engagement of leadership and clinicians
 - Standard tools for recording adherence to best practices
 - Standardizing catheter insertion kits
 - Measurement of CLABSI and reporting of rates back to facilities

CDC. MMWR 2005;54:1013-6.



Background: Prevention Strategies Interventions



- Michigan Keystone Project
- Decrease in CLABSI in 103 ICUs in Michigan (66% reduction)
- Basic interventions:
 - Hand hygiene
 - Full barrier precautions during CL insertion
 - Skin cleansing with chlorhexidine
 - Avoiding femoral site
 - Removing unnecessary catheters
 - Use of insertion checklist
 - Promotion of safety culture

Pronovost et al. NEJM 2006;355:2725-32.



Background: On the CUSP: Stop BSI project



- This national program is a collaboration between
 - Health Research and Educational Trust
 - Johns Hopkins University Quality and Safety Research Group
 - Michigan Health and Hospital Association Keystone Center for Patient Safety and Quality
- Builds on successes in Michigan Keystone project
 - CLABSI prevention bundle
 - Collaborative model
 - Promotion of safety culture
- Hospitals in all 50 states, the District of Columbia, and Puerto Rico are eligible to participate



Prevention Strategies

- **Core Strategies**

- High levels of scientific evidence
- Demonstrated feasibility

- **Supplemental Strategies**

- Some scientific evidence
- Variable levels of feasibility

The Collaborative should at a minimum include core prevention strategies. Supplemental prevention strategies also may be used. Most core and supplemental strategies are based on HICPAC guidelines. Strategies that are not included in HICPAC guidelines will be noted by an asterisk () after the strategy. HICPAC guidelines may be found at www.cdc.gov/hicpac



Prevention Strategies: Core



- Removing unnecessary CL
- Following proper insertion practices
- Facilitating proper insertion practices*
- Complying with hand hygiene recommendations
- Adequate skin antisepsis
- Choosing proper CL insertion sites
- Performing adequate hub/access port disinfection
- Providing education on CL maintenance and insertion

* Not part of 2002 HICPAC Guidelines for the Prevention of Intravascular Catheter-Related Infections





Prevention Strategies: Core Removing Unnecessary CL



- In one study, 9% of CLs outside of ICU deemed inappropriate
- Perform daily assessment of the need for the CL and promptly discontinue CLs that are no longer required
- Nursing staff should be encouraged to notify physicians of CLs that are unnecessary
- Use peripheral catheters instead
 - These generally have lower rates of BSIs than CL

Trick et al. Infect Control Hospital Epidemiol 2004;25:266-8.



Prevention Strategies: Core Proper Insertion Practices



- Ensure utilization of insertion bundle:
 - Chlorhexidine for skin antisepsis
 - Maximal sterile barrier precautions (e.g., mask, cap [i.e., similar to those worn in the O.R.], gown, sterile gloves, and large sterile drape)
 - Hand hygiene
- Many CLs in patients on non-ICU hospital wards are placed outside those wards (Emergency room, ICU, Operating room, or Pre-operative areas)
- In one study, 49% of CLs were present on admission to the ward. Rates of BSI in this study were higher in CLs placed in Emergency Room
- Define where placement occurs and review technique in those areas

Trick et al. Am J Infect Control 2006;34:636-41.



Prevention Strategies: Core

Facilitating Proper Insertion Practices*

- “Bundling” all needed supplies in one area (e.g., a cart or a kit) helps ensure items are available for use
- Use of a “checklist” to ensure all insertion practices are followed may be beneficial
- Empowering staff to stop a non-emergent CL insertion if proper procedures are not followed
- Promoting safety culture

* Not part of 2002 HICPAC Guidelines for the Prevention of Intravascular Catheter-Related Infections



Prevention Strategies: Core Hand Hygiene

- Hand hygiene should be a cornerstone of CLABSI prevention efforts
 - For both insertion and maintenance
- As part of a hand hygiene intervention, consider:
 - Ensuring easy access to soap and water and alcohol-based hand gels
 - Education for HCP and patients
 - Observation of practices - particularly around high-risk procedures (before and after contact with CL)
 - Feedback – “Just in time” feedback if failure to perform hand hygiene observed



Prevention Strategies: Core Chlorhexidine Skin Cleansing

- Chlorhexidine is the preferred agent for skin cleansing for both CL insertion and maintenance
 - Tincture of iodine, an iodophor, or 70% alcohol are alternatives
 - Recommended application methods and contact time should be followed for maximal effect
- Prior to use should ensure agent is compatible with catheter
 - Alcohol may interact with some polyurethane catheters
 - Some iodine-based compounds may interact with silicone catheters



Prevention Strategies: Core CL Site Choice



- For adult patients receiving non-tunneled CL, femoral site should be avoided due to an increased risk of infection and deep venous thrombosis
- Note:
 - In patients with renal failure, subclavian site should be avoided to minimize stenosis which may limit future vascular access options



Prevention Strategies: Core Hub/access port cleansing



- BSI “outbreaks” have been associated with failure to adequately decontaminate catheter hubs or failure to change them at appropriate intervals
- Cleanse hubs prior to use with an appropriate antiseptic (e.g., 70% alcohol)
- Manufacturer recommendations regarding cleansing and changing connectors should be followed



Prevention Strategies: Core

CL Maintenance and Insertion: Education

- Personnel responsible for insertion and maintenance of catheters should be trained and demonstrate competence
- Recurrent educational sessions for staff who care and/or insert CLs



Prevention Strategies: Supplemental



- Supplemental strategies include:
 - Chlorhexidine bathing*
 - Antimicrobial-impregnated catheters
 - Chlorhexidine-impregnated dressings*

* Not part of 2002 HICPAC Guidelines for the Prevention of Intravascular Catheter-Related Infections



Prevention Strategies: Supplemental Chlorhexidine Bathing*



- In an ICU at a single center, daily bathing with 2% chlorhexidine-impregnated cloths decreased the rate of BSIs compared to soap and water
- No data outside the ICU

Bleasdale, et al. Arch Intern Med 2007;167:2073-9.

* Not part of 2002 HICPAC Guidelines for the Prevention of Intravascular Catheter-Related Infections



Prevention Strategies: Supplemental Antimicrobial-Impregnated Catheters

- 2 types with most supporting evidence:
 - Minocycline-Rifampin
 - Chlorhexidine–Silver Sulfadiazine
- Platinum-Silver catheter available but less evidence to support use
- These may be appropriate for patients whose catheter is expected to be used for more than 5 days and when Core strategies have not decreased rates of CLABSI to established goals.



Prevention Strategies: Supplemental Chlorhexidine Dressings*



- Chlorhexidine-impregnated sponge dressings have been shown to decrease rates of CLABSIs in some studies and not in others.
- These dressings may be an option when Core interventions have not decreased rates of CLABSI to established goals

* Not part of 2002 HICPAC Guidelines for the Prevention of Intravascular Catheter-Related Infections



Summary of Prevention Strategies*

Core Measures

- Removing unnecessary CL
- Following proper insertion practices
- Facilitating proper insertion practices*
- Complying with hand hygiene recommendations
- Performing adequate skin cleaning
- Choosing proper CL insertion sites
- Performing adequate hub/access port cleaning
- Providing education on CL maintenance and insertion

Supplemental Measures

- Implementing chlorhexidine bathing*
- Using antimicrobial-impregnated catheters
- Applying chlorhexidine site dressings*

* Not part of 2002 HICPAC Guidelines for the Prevention of Intravascular Catheter-Related Infections



Measurement

- With CLABSI measurement it is important to
 - Have a definition that is consistent between sites
 - Collecting blood cultures in a similar fashion
 - For recommended indications
 - Via a peripheral venipuncture vs. via a CL



Measurement: Process Measures

- Process measures can help determine if interventions are being fully implemented
 - Ensuring interventions are being performed is itself a “core” intervention
- Potentially important process measures to consider are:
 - Hand hygiene adherence
 - Proportion of patients with CLs, and/or duration of CL use
 - Proportion of CL insertions in which maximal barrier precautions were used
- Consider using NHSN Central Line Insertion Practices (CLIP) option



Measurement: Outcome Calculating CLABSI Rates



$$\text{CLABSI Rate}^* = \frac{\text{\# CLABSIs identified}}{\text{\# central line-days}} \times 1000$$

* Stratify by:

- Type of ICU/Other Location
- For special care areas
 - Catheter type (temporary or permanent)
- For neonatal intensive care units
 - Birthweight category
 - Catheter type (umbilical or central)



Measurement: Outcome Device Utilization (DU) Ratio

$$\text{CL DU Ratio} = \frac{\# \text{ central line-days}}{\# \text{ patient-days}}$$

DU Ratio measures the proportion of total patient-days in which central lines were used.



Measurement: Process CLIP Adherence Rates



- **Using NHSN, adherence rates can be calculated for:**
 - Hand hygiene
 - Barrier precautions used including masks, sterile drape, gowns and sterile gloves
 - Skin preparation including type of agent and whether agent was allowed to dry
- **Other measures collected in the NHSN CLIP option that can be summarized include:**
 - CL type, location, and number of lumens
 - Antiseptic ointment applied to site



Measurement: Process

Calculating CLIP Adherence Rates

$$\text{Hand Hygiene Adherence Rate} = \frac{\text{\# hand hygiene performed for CL insertion}}{\text{\# CL insertions records completed}}$$

Adherence rates can also be measured for each of the barrier and prevention practices by using the number of CLIP records completed as the denominator.



Tools for Implementation

NHSN CLIP Option: Insertion Practices

Event Information [HELP](#)

Event Type*:

Location*:

Date of Insertion*:

Person recording insertion practice data >: Inserter Observer

Central Line Inserter ID:

Last Name: First Name:

Occupation of inserter >:

Insertion Details [HELP](#)

Reason for insertion >:

Inserter performed hand hygiene prior to central line insertion >:

Maximal sterile barrier precautions used >:

- Mask
- Sterile gown
- Large sterile drape
- Sterile gloves
- Cap

Skin Preparation (check all that apply) >: Chlorohexidine gluconate Povidone iodine Alcohol Other

Was skin preparation agent completely dry at the time of first skin puncture? >:

Insertion site >:

Antimicrobial coated catheter used:

Central line catheter type >:

Number of lumens >:

Central line exchanged over a guidewire >:

Antiseptic ointment applied to site >:



Evaluation Considerations

- **Assess baseline policies and procedures**
- **Areas to consider**
 - **Surveillance**
 - **Prevention strategies**
 - **Measurement**
- **Coordinator should track new policies/practices implemented during collaboration**



References

- Bleasdale SC, Trick WE, Gonzalez IM, et al. Effectiveness of chlorhexidine bathing to reduce catheter-associated bloodstream infections in medical intensive care unit patients. *Arch Intern Med* 2007; 67:2073-9.
- Burton DC, Edwards JR, Horan TC, et al. Methicillin-resistant *Staphylococcus aureus* central line-associated bloodstream infections in US intensive care units, 1997-2007. *JAMA* 2009;301:727-36.
- CDC. Reduction in central line-associated bloodstream infections among patients in intensive care units—Pennsylvania, April 2001-March 2005. *MMWR* 2005;54:1013-6.



References

- Climo M, Diekema D, Warren DK, et al. Prevalence of the use of central venous access devices within and outside of the intensive care unit: results of a survey among hospitals in the prevention epicenter program of the Centers for Disease Control and Prevention. *ICHE* 2003;24:942-5.
- Edwards, JR, Peterson KD, Mu Y, et al. National Healthcare Safety Network (NHSN) report: Data summary for 2006 through 2008, issued December 2009. *Am J Infect Control* 2009;37:783-805.



- Klevens RM, Edwards JR, Richards CI, et al. Estimating health care-associated infections and deaths in U.S. hospitals, 2002. *Public Health Reports* 2007;122:160-6.
- Pittet D, Tarara D, Wenzel RP. Nosocomial bloodstream infection in critically ill patients. Excess length of stay extra costs, and attributable mortality. *JAMA* 1994;271:1598-1601.



References

- Marschall J, Leone C, Jones M, et al. Catheter-associated bloodstream infections in general medical patients outside the intensive care unit : a surveillance study. *ICHE* 2007; 28:905-9.
- Pronovost P, Needham D, Berenholtz S, et al. An intervention to decrease catheter-related bloodstream infections in the ICU. *NEJM* 2006;355:2725-32.
- Trick WE, Vernon MO, Welbel SF, et al. Unnecessary use of central venous catheters: the need to look outside the intensive care unit. *Infect Control Hospital Epidemiol* 2004; 25:266-8.



References

- Trick WE, Miranda J, Evans AT, et al. Prospective cohort study of central venous catheters among internal medicine ward patients. *Am J Infect Control* 2006;34:636-41.



Clostridium difficile (CDI) Infections Toolkit

Activity C: ELC Prevention Collaboratives

Carolyn Gould, MD MSCR

Cliff McDonald, MD, FACP

Division of Healthcare Quality Promotion

Centers for Disease Control and Prevention

Draft - 12/23/09 --- Disclaimer: The findings and conclusions in this presentation are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.





Outline



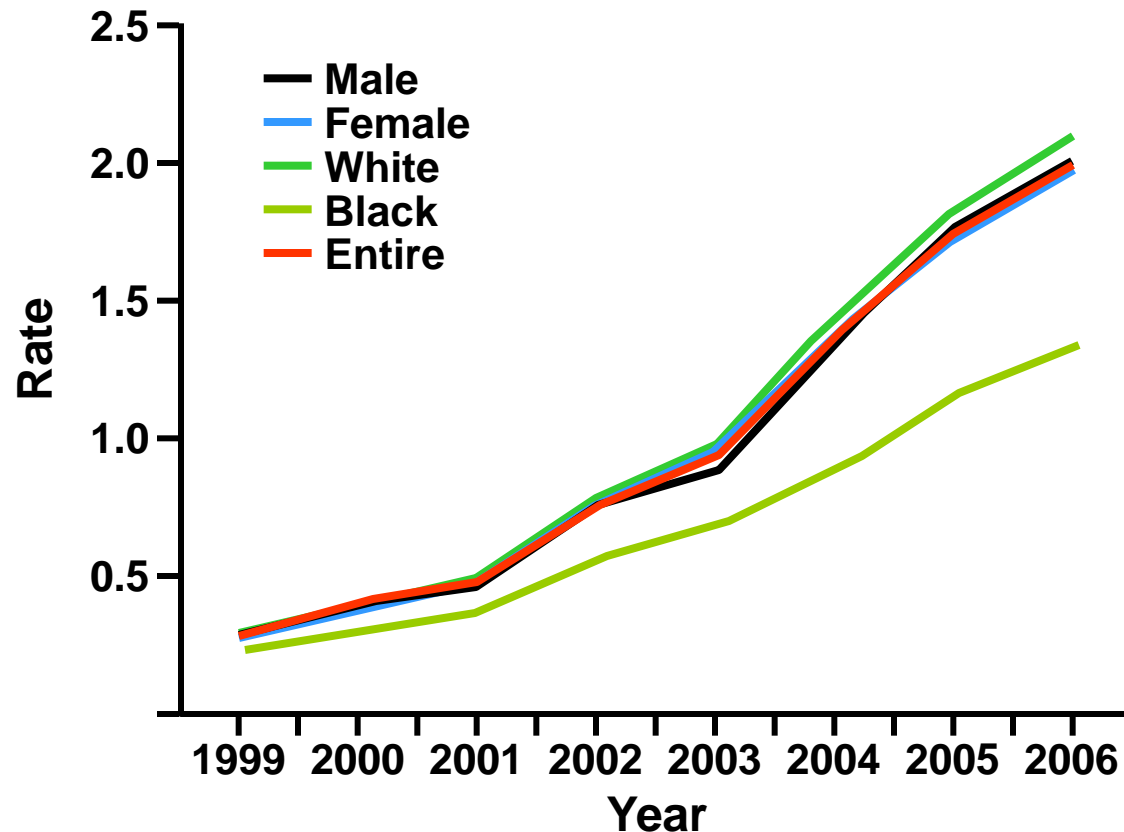
- **Background**
 - Impact
 - HHS Prevention Targets
 - Pathogenesis
 - Epidemiology
- **Prevention Strategies**
 - Core
 - Supplemental
- **Measurement**
 - Process
 - Outcome
- **Tools for Implementation/Resources/References**



Background: Impact



Age-Adjusted Death Rate* for Enterocolitis Due to *C. difficile*, 1999–2006



*Per 100,000 US standard population

Heron et al. Natl Vital Stat Rep 2009;57(14).

Available at http://www.cdc.gov/nchs/data/nvsr/nvsr57/nvsr57_14.pdf



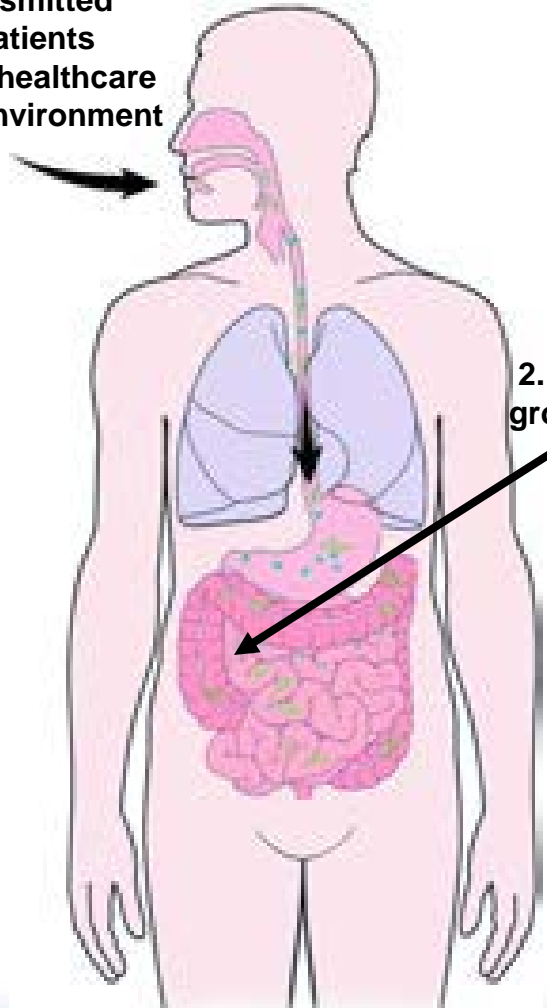
Background: HHS Prevention Targets

- **Case rate per 10,000 patient-days as measured in NHSN**
 - National 5-Year Prevention Target: 30% reduction
- **Because little baseline infection data exists, administrative data for ICD-9-CM coded *C. difficile* hospital discharges is also tracked**
 - National 5-Year Prevention Target: 30% reduction

<http://www.hhs.gov/ophs/initiatives/hai/prevtargets.html>

Background: Pathogenesis of CDI

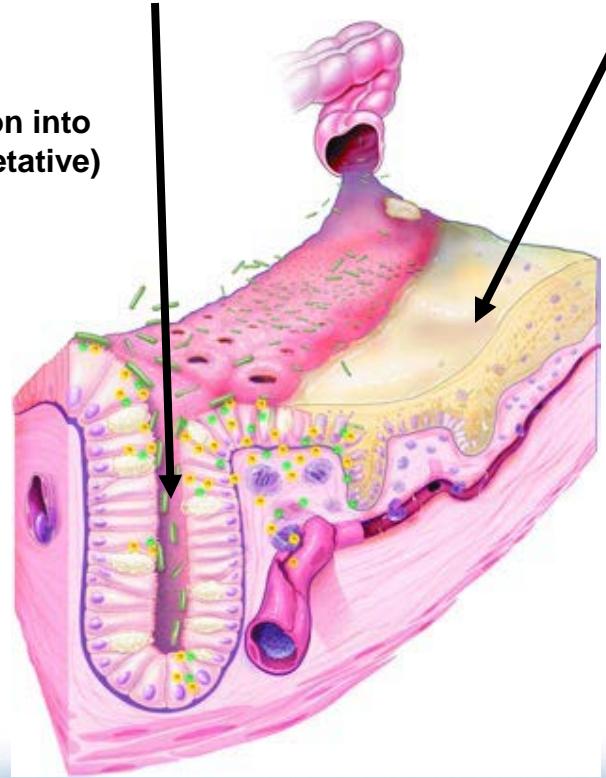
1. Ingestion of spores transmitted from other patients via the hands of healthcare personnel and environment



2. Germination into growing (vegetative) form

3. Altered lower intestine flora (due to antimicrobial use) allows proliferation of *C. difficile* in colon

4. Toxin A & B Production leads to colon damage +/- pseudomembrane



Sunenshine et al. Cleve Clin J Med. 2006;73:187-97.



Background: Epidemiology

Current epidemic strain of *C. difficile*

- BI/NAP1/027, toxinotype III
- Historically uncommon – epidemic since 2000
- More resistant to fluoroquinolones
 - Higher MICs compared to historic strains and current non-BI/NAP1 strains
- More virulent
 - Increased toxin A and B production
 - Polymorphisms in binding domain of toxin B
 - Increased sporulation

McDonald et al. N Engl J Med. 2005;353:2433-41.

Warny et al. Lancet. 2005;366:1079-84.

Stabler et al. J Med Micro. 2008;57:771-5.

Akerlund et al. J Clin Microbiol. 2008;46:1530-3.



Background: Epidemiology Risk Factors



- Antimicrobial exposure
- Acquisition of *C. difficile*
- Advanced age
- Underlying illness
- Immunosuppression
- Tube feeds
- ? Gastric acid suppression

Main modifiable risk factors



Prevention Strategies

- **Core Strategies**
 - High levels of scientific evidence
 - Demonstrated feasibility

- **Supplemental Strategies**
 - Some scientific evidence
 - Variable levels of feasibility

The Collaborative should at a minimum include core prevention strategies. Supplemental prevention strategies also may be used. Most core and supplemental strategies are based on HICPAC guidelines. Strategies that are not included in HICPAC guidelines will be noted by an asterisk () after the strategy. HICPAC guidelines may be found at www.cdc.gov/hicpac



Prevention Strategies: Core



- Implement an antimicrobial stewardship program
- Contact Precautions for duration of diarrhea
- Hand hygiene in compliance with CDC/WHO
- Cleaning and disinfection of equipment and environment
- Laboratory-based alert system for immediate notification of positive test results
- Educate about CDI: HCP, housekeeping, administration, patients, families

http://www.cdc.gov/ncidod/dhqp/id_CdiffFAQ_HCP.html

Dubberke et al. Infect Control Hosp Epidemiol 2008;29:S81-92.



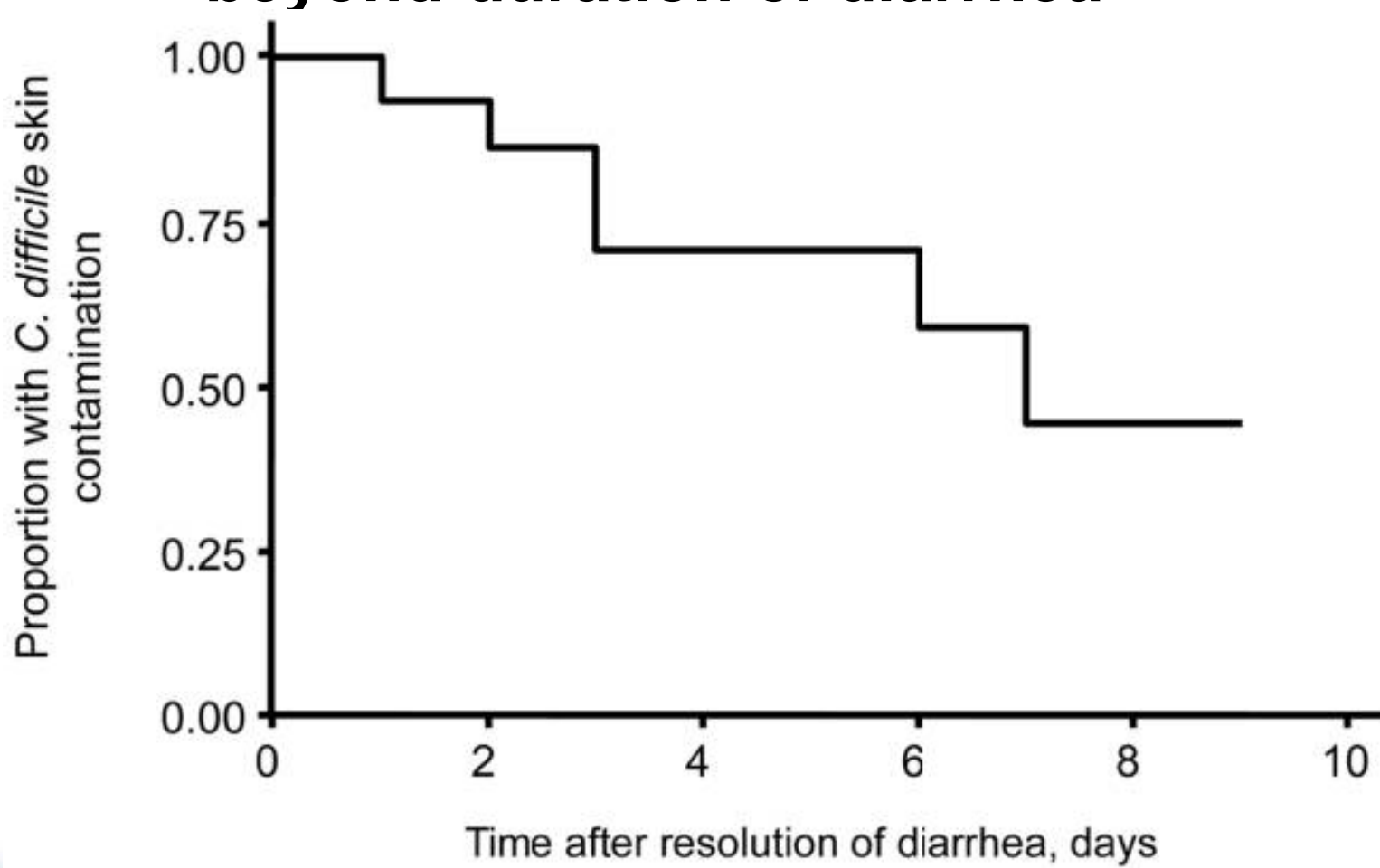
Prevention Strategies: Supplemental

- Extend use of Contact Precautions beyond duration of diarrhea (e.g., 48 hours)*
- Presumptive isolation for symptomatic patients pending confirmation of CDI
- Evaluate and optimize testing for CDI
- Implement soap and water for hand hygiene before exiting room of a patient with CDI
- Implement universal glove use on units with high CDI rates*
- Use sodium hypochlorite (bleach) – containing agents for environmental cleaning

* Not included in CDC/HICPAC 2007 Guideline for Isolation Precautions



Supplemental Prevention Strategies: Rationale for considering extending isolation beyond duration of diarrhea



Bobulsky et al. Clin Infect Dis 2008;46:447-50.



Supplemental Prevention Strategies:



Consider presumptive isolation for patients with ≥ 3 unformed stools within 24 hours

- Patients with CDI may contaminate environment and hands of healthcare personnel pending results of diagnostic testing
- CDI responsible for only ~30-40% of hospital-onset diarrhea
- However, CDI more likely among patients with ≥ 3 unformed (i.e. taking the shape of a container) stools within 24 hours
 - Send specimen for testing and presumptively isolate patient pending results
 - Positive predictive value of testing will also be optimized if focused on patients with ≥ 3 unformed stools within 24 hours
 - Exception: patient with possible recurrent CDI (isolate and test following first unformed stool)



Supplemental Prevention Strategies:

Evaluate and optimize test-ordering practices and diagnostic methods



- Most laboratories have relied on Toxin A/B enzyme immunoassays
 - Low sensitivities (70-80%) lead to low negative predictive value
- Despite high specificity, poor test ordering practices (i.e. testing formed stool or repeat testing in negative patients) may lead to many false positives
- Consider more sensitive diagnostic paradigms but apply these more judiciously across the patient population
 - Employ a highly sensitive screen with confirmatory test or a PCR-based molecular assay
 - Restrict testing to unformed stool only
 - Focus testing on patients with ≥ 3 unformed stools within 24 hours
 - Require expert consultation for repeat testing within 5 days

Peterson et al. Ann Intern Med 2009;15:176-9.



Supplemental Prevention Strategies: Hand Hygiene – Soap vs. Alcohol gel



- Alcohol not effective in eradicating *C. difficile* spores
- However, one hospital study found that from 2000-2003, despite increasing use of alcohol hand rub, there was no concomitant increase in CDI rates
- Discouraging alcohol gel use may undermine overall hand hygiene program with untoward consequences for HAIs in general

Boyce et al. Infect Control Hosp Epidemiol 2006;27:479-83.



Supplemental Prevention Strategies: Hand Washing: Product Comparison



Product	Log10 Reduction
Tap Water	0.76
4% CHG antimicrobial hand wash	0.77
Non-antimicrobial hand wash	0.78
Non-antimicrobial body wash	0.86
0.3% triclosan antimicrobial hand wash	0.99
Heavy duty hand cleaner used in manufacturing environments	1.21*

* Only value that was statistically better than others

Conclusion: Spores may be difficult to eradicate even with hand washing.

Edmonds, et al. Presented at: SHEA 2009; Abstract 43.



Supplemental Prevention Strategies: Hand Hygiene Methods



Since spores may be difficult to remove from hands even with hand washing, adherence to glove use, and Contact Precautions in general, should be emphasized for preventing *C. difficile* transmission via the hands of healthcare personnel

Johnson et al. Am J Med 1990;88:137-40.



Supplemental Prevention Strategies: **Glove Use**



Rationale for considering universal glove use (in addition to Contact Precautions for patients with known CDI) on units with high CDI rates

- Although the magnitude of their contribution is uncertain, asymptomatic carriers have a role in transmission
- Practical screening tests are not available
- There may be a role for universal glove use as a special approach to reducing transmission on units with longer lengths of stay and high endemic CDI rates
- Focus enhanced environmental cleaning strategies and avoid shared medical equipment on such units as well



Supplemental Prevention Strategies: Environmental Cleaning



- Bleach can kill spores, whereas other standard disinfectants cannot
- Limited data suggest cleaning with bleach (1:10 dilution prepared fresh daily) reduces *C. difficile* transmission
- Two before-after intervention studies demonstrated benefit of bleach cleaning in units with high endemic CDI rates
- Therefore, bleach may be most effective in reducing burden where CDI is highly endemic

Mayfield et al. Clin Infect Dis 2000;31:995-1000.

Wilcox et al. J Hosp Infect 2003;54:109-14.



Supplemental Prevention Strategies: Environmental Cleaning



Assess adequacy of cleaning before changing to new cleaning product such as bleach

- Ensure that environmental cleaning is adequate and high-touch surfaces are not being overlooked
- One study using a fluorescent environmental marker to assess cleaning showed:
 - only 47% of high-touch surfaces in 3 hospitals were cleaned
 - sustained improvement in cleaning of all objects, especially in previously poorly cleaned objects, following educational interventions with the environmental services staff
- The use of environmental markers is a promising method to improve cleaning in hospitals.

Carling et al. Clin Infect Dis 2006;42:385-8.



Summary of Prevention Measures

Core Measures

- Contact Precautions for duration of illness
- Hand hygiene in compliance with CDC/WHO
- Cleaning and disinfection of equipment and environment
- Laboratory-based alert system
- CDI surveillance
- Education

Supplemental Measures

- Prolonged duration of Contact Precautions*
- Presumptive isolation
- Evaluate and optimize testing
- Soap and water for HH upon exiting CDI room
- Universal glove use on units with high CDI rates*
- Bleach for environmental disinfection
- Antimicrobial stewardship program

* Not included in CDC/HICPAC 2007 Guideline for Isolation Precautions



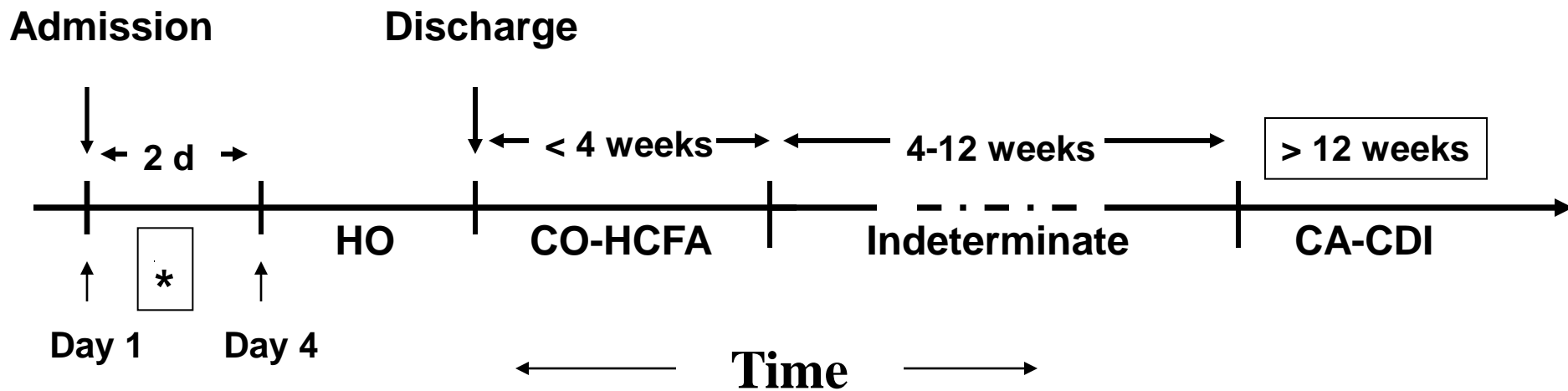
Measurement: Process Measures

- **Core Measures:**
 - Measure compliance with CDC/WHO recommendations for hand hygiene and Contact Precautions
 - Assess adherence to protocols and adequacy of environmental cleaning
- **Supplemental Measures:**
 - Intensify assessment of compliance with process measures
 - Track use of antibiotics associated with CDI in a facility



Measurement: Outcome

Categorize Cases by location and time of onset†



HO: Hospital (Healthcare)-Onset

CO-HCFA: Community-Onset , Healthcare Facility-Associated

CA: Community -Associated

* Depending upon whether patient was discharged within previous 4 weeks, CO-HCFA vs. CA

† Onset defined in NHSN LabID Event by specimen collection date

Modified from CDAD Surveillance Working Group. Infect Control Hosp Epidemiol 2007;28:140-5.



Measurement: Outcome Use NHSN CDAD Module



Laboratory-identified MDRO or CDAD Event

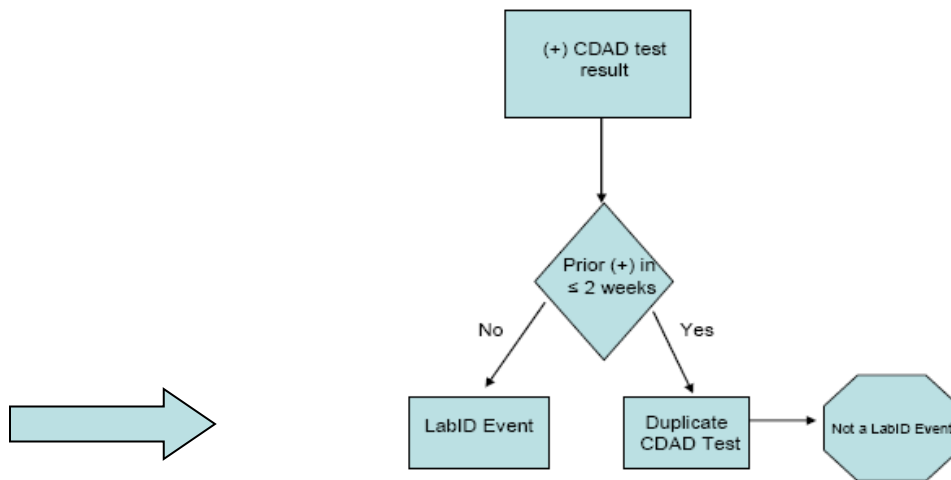
OMB No. 0920-0047
Exp. Date: 03-31-2017

*required for saving		
Facility ID:	Event #:	
*Patient ID:	Social Security #:	
Secondary ID:		
Patient Name, Last:	First:	Middle:
*Gender: M F	*Date of Birth:	
Ethnicity (Specify):	Race (Specify):	
Event Details		
*Event Type: LabID	*Date Specimen Collected:	
*Specific Organism Type: (Check one)		
<input type="checkbox"/> MRSA <input type="checkbox"/> MSSA <input type="checkbox"/> VRE <input type="checkbox"/> MDR- <i>Klebsiella</i> <input type="checkbox"/> MDR- <i>Acinetobacter</i> <input type="checkbox"/> C. <i>difficile</i>		
*Outpatient: Yes No	*Specimen Source:	
*Date Admitted	*Location:	*Date Admitted

Measurement: Outcome

Focus on Laboratory Identified (LabID) Events in NHSN

Figure 2. CDAD Test Result Algorithm for Laboratory-Identified (LabID) Events





Measurement: Outcome

NHSN Reporting: Definitions



Based on data submitted to NHSN, CDI LabID Events are categorized as:

- **Incident:** specimen obtained >8 weeks after the most recent LabID Event
- **Recurrent:** specimen obtained >2 weeks and ≤ 8 weeks after most recent LabID Event



Measurement: Outcome

NHSN Reporting: Definitions



Incident cases further characterized based on date of admission and date of specimen collection:

- **Healthcare Facility-Onset (HO):** LabID Event collected >3 days after admission to facility (i.e., on or after day 4)
- **Community-Onset (CO):** LabID Event collected as an outpatient or an inpatient ≤ 3 days after admission to the facility (i.e., days 1, 2, or 3 of admission)
- **Community-Onset Healthcare Facility-Associated (CO-HCFA):** CO LabID Event collected from a patient who was discharged from the facility ≤ 4 weeks prior to date stool specimen collected



Measurement: Outcome



Calculating CDI Incidence Rates*

- **Healthcare Facility-Onset Incidence Rate** =
Number of all Incident HO CDI LabID Events per
patient per month / Number of patient days for
the facility x 10,000
- **Combined Incidence Rate** = Number of all
Incident HO and CO-HCFA CDI LabID Events
per patient per month / Number of patient days
for the facility x 10,000

*For a given healthcare facility



Evaluation Considerations

- **Assess baseline policies and procedures**
- **Areas to consider**
 - **Surveillance**
 - **Prevention strategies**
 - **Measurement of effect of strategies**
- **Coordinator should track new policies/practices implemented during collaboration**



References

- Dubberke ER, Butler AM, Reske KA, et al. attributable outcomes of endemic *Clostridium difficile*-associated disease in nonsurgical patients. *Emerg Infect Dis* 2008;14:1031-8.
- Dubberke ER, Reske KA, Olssen MA, et al. Short- and long term attributable costs of *Clostridium difficile*-associated disease in nonsurgical inpatients. *Clin Infect Dis* 2008;46:497-504.
- Edmonds S, Kasper D, Zepka C, et al. *Clostridium difficile* and hand hygiene: spore removal effectiveness of handwash products. Presented at: SHEA 2009; Abstract 43.



References

- Elixhauser, A. (AHRQ), and Jhung, MA. (Centers for Disease Control and Prevention). *Clostridium Difficile-Associated Disease in U.S. Hospitals, 1993–2005*. HCUP Statistical Brief #50. April 2008. Agency for Healthcare Research and Quality, Rockville, MD. <http://www.hcup-us.ahrq.gov/reports/statbriefs/sb50.pdf>
- Fowler S, Webber A, Cooper BS, et al. Successful use of feedback to improve antibiotic prescribing and reduce *Clostridium difficile* infection: a controlled interrupted time series. *J Antimicrob Chemother* 2007;59:990-5.
- Heron MP, Hoyert D, Murphy SL, et al. *Natl Vital Stat Rep* 2009;57(14). US Dept of Health and Human Services, CDC; 2009. Available at http://www.cdc.gov/nchs/data/nvsr/nvsr57/nvsr57_14.pdf



References

- Johnson S, Gerding DN, Olson MM, et al. Prospective, controlled study of vinyl glove use to interrupt *Clostridium difficile* nosocomial transmission. *Am J Med* 1990;88:137-40.
- Mayfield JL, Leet T, Miller J, et al. Environmental control to reduce transmission of *Clostridium difficile*. *Clin Infect Dis* 2000;31:995–1000.
- McDonald LC, Killgore GE, Thompson A, et al. An epidemic, toxin gene–variant strain of *Clostridium difficile*. *N Engl J Med*. 2005;353:2433-41.



References

- McDonald LC, Coignard B, Dubberke E, et al. Ad Hoc CDAD Surveillance Working Group. Recommendations for surveillance of *Clostridium difficile*-associated disease. *Infect Control Hosp Epidemiol* 2007; 28:140-5.
- Oughton MT, Loo VG, Dendukuri N, et al. Hand hygiene with soap and water is superior to alcohol rum and antiseptic wipes for removal of *Clostridium difficile*. *Infect Control Hosp Epidemiol* 2009; 30:939-44.
- Peterson LR, Robicsek A. Does my patient have *Clostridium difficile* infection? *Ann Intern Med* 2009;15:176-9
- Riggs MM, Sethi AK, Zabarsky TF, et al. Asymptomatic carriers are a potential source for transmission of epidemic and nonepidemic *Clostridium difficile* strains among long-term care facility residents. *Clin Infect Dis* 2007; 45:992–8.



References

- SHEA/IDSA Compendium of Recommendations. Infect Control Hosp Epidemiol 2008;29:S81–S92.
<http://www.journals.uchicago.edu/doi/full/10.1086/591065>
- Stabler RA, Dawson LF, Phua LT, et al. Comparative analysis of BI/NAP1/027 hypervirulent strains reveals novel toxin B-encoding gene (tcdB) sequences. J Med Micro. 2008;57:771–5.
- Sunenshine RH, McDonald LC. *Clostridium difficile*-associated disease: new challenges from and established pathogen. Cleve Clin J Med. 2006;73:187-97.



References

- Warny M, Pepin J, Fang A, Killgore G, et al. Toxin production by an emerging strain of *Clostridium difficile* associated with outbreaks of severe disease in North America and Europe. Lancet. 2005;366:1079-84.
- Wilcox MF, Fawley WN, Wigglesworth N, et al. Comparison of the effect of detergent versus hypochlorite cleaning on environmental contamination and incidence of *Clostridium difficile* infection. *J Hosp Infect* 2003;54:109-14.



Additional resources



SHEA/IDSA Compendium of Recommendations

CDI Checklist Example

S81 INFECTION CONTROL AND HOSPITAL EPIDEMIOLOGY OCTOBER 2008, VOL. 29, SUPPLEMENT 1

SUPPLEMENT ARTICLE: SHEA/IDSA PRACTICE RECOMMENDATION

Strategies to Prevent *Clostridium difficile* Infections in Acute Care Hospitals

Erik R. Dubberke, MD; Dale N. Gerding, MD; David Classen, MD, MS; Kathleen M. Arias, MS, CIC;
 Kelly Podgorny, RN, MS, CPHQ; Deverick J. Anderson, MD, MPH; Helen Burstin, MD; David P. Calfee, MD, MS;
 Susan E. Coffin, MD, MPH; Victoria Fraser, MD; Frances A. Griffin, RRT, MPA; Peter Gross, MD; Keith S. Kaye, MD;
 Michael Klompas, MD; Evelyn Lo, MD; Jonas Marschall, MD; Leonard A. Mermel, DO, ScM; Lindsay Nicolle, MD;
 David A. Pegues, MD; Trish M. Perl, MD; Sanjay Saint, MD; Cassandra D. Salgado, MD, MS;
 Robert A. Weinstein, MD; Robert Wise, MD; Deborah S. Yokoe, MD, MPH

Clostridium difficile Infection (CDI) Checklist

Hospital interventions to decrease the incidence and mortality of healthcare-associated *C. difficile* infections

Prevention Checklist	Treatment Checklist
<ul style="list-style-type: none"> • When an MD, PA, NP, or RN suspects a patient has CDI: <ul style="list-style-type: none"> Physician, Physician Assistant, or Nurse Practitioner: <ul style="list-style-type: none"> <input type="checkbox"/> Initiate <i>Contact Precautions Plus</i> <input type="checkbox"/> Order stool <i>C. difficile</i> toxin testing <input type="checkbox"/> Discontinue non-essential antimicrobials <input type="checkbox"/> Discontinue all anti-peristaltic medications Registered Nurse: <ul style="list-style-type: none"> <input type="checkbox"/> Obtain stool sample for <i>C. difficile</i> toxin test <input type="checkbox"/> Place patient in single-patient room <input type="checkbox"/> Place <i>Contact Precautions Plus</i> sign on patient's door <input type="checkbox"/> Ensure that gloves and gowns are easily accessible from patient's room <input type="checkbox"/> Place dedicated stethoscope in patient's room <input type="checkbox"/> Remind staff to wash hands with soap and water following patient contact Microbiology Laboratory Staff Person: <ul style="list-style-type: none"> <input type="checkbox"/> Call relevant patient floor with positive <i>C. difficile</i> toxin test result <input type="checkbox"/> Provide daily list of positive test results for Infection Control Infection Control Practitioner: <ul style="list-style-type: none"> <input type="checkbox"/> Check microbiology results daily for positive <i>C. difficile</i> toxin results <input type="checkbox"/> Call relevant floor to confirm that patient with positive <i>C. difficile</i> toxin results is in a single-patient room and that the <i>Contact Precautions Plus</i> sign is on the patient's door <input type="checkbox"/> Flag the patient's <i>C. difficile</i> status in the hospital's clinical information system or in the patient's paper chart <input type="checkbox"/> Alert housekeeping that the patient is on <i>Contact Precautions Plus</i> Environmental Services Staff Person: <ul style="list-style-type: none"> <input type="checkbox"/> Prior to discharge cleaning, check for <i>Contact Precautions Plus</i> sign on the patient's door <input type="checkbox"/> If <i>Contact Precautions Plus</i> sign is on the door, clean the room with a bleach-based cleaning agent <input type="checkbox"/> Confirm for supervisor that bleach-based cleaning agent was used for discharge cleaning for every patient on <i>Contact Precautions Plus</i> 	<ul style="list-style-type: none"> • When an MD, PA, or NP diagnoses mild CDI: <i>All of the following criteria are present: diarrhea (>3 BM/day), no fever, WBC <15,000, no peritoneal signs, and no evidence of sepsis</i> Physician, Physician Assistant, or Nurse Practitioner: <ul style="list-style-type: none"> <input type="checkbox"/> Initiate oral metronidazole at dose 500mg every 8 hours <input type="checkbox"/> If no clinical improvement by 48-72 hours after diagnosis, treat patient as moderate CDI <input type="checkbox"/> Continue therapy for at least 14 days total and at least 10 days after symptoms have abated • When an MD, PA, or NP diagnoses moderate CDI: <i>At least one of the following criteria is present: diarrhea (≥12 BM/day), fever 37.5-38.5°C, WBC 15,000-25,000, or frankly visible stable lower gastrointestinal bleeding</i> Physician, Physician Assistant, or Nurse Practitioner: <ul style="list-style-type: none"> <input type="checkbox"/> Initiate oral vancomycin at dose 250mg every 6 hours <input type="checkbox"/> If no clinical improvement by 48 hours, add IV metronidazole at dose 500mg every 8 hours <input type="checkbox"/> Consider obtaining infectious disease consultation <input type="checkbox"/> Consider obtaining abdominal CT scan <input type="checkbox"/> Continue therapy for at least 14 days total and at least 10 days after symptoms have abated • When an MD, PA, or NP diagnoses severe CDI: <i>At least one of the following criteria is present: diarrhea (>12 BM/day), fever >38.5°C, WBC >25,000, hemodynamic instability, marked & continuous abdominal pain, ileus, absence of bowel sounds, evidence of sepsis, or intensive care unit level of care required</i> Physician, Physician Assistant, or Nurse Practitioner: <ul style="list-style-type: none"> <input type="checkbox"/> Obtain immediate infectious disease consultation <input type="checkbox"/> Obtain immediate general surgery consultation <input type="checkbox"/> Obtain abdominal CT scan <input type="checkbox"/> Initiate oral vancomycin at dose 250mg every 6 hours together with IV metronidazole at dose 500mg every 6 hours <input type="checkbox"/> Following consultation with general surgery regarding its use, consider rectal vancomycin <input type="checkbox"/> Ask general surgery service to assess the need for colectomy

Abbreviations: MD=medical doctor, PA=physician assistant, NP=nurse practitioner, RN=registered nurse, BM=bowel movement, WBC=white blood cell count, CT=computed tomography, IV=intravenous

FIGURE 1. *Clostridium difficile* infection checklist at Brigham and Women's Hospital.

Dubberke et al. Infect Control Hosp Epidemiol 2008;29:S81-92.
 Abbett SK et al. Infect Control Hosp Epidemiol 2009;30:1062-9.





Additional Reference Slides



- The following slides may be used for presentations regarding CDI.
- Explanations are available in the notes section of the slides.



Supplemental Prevention Strategies: Rationale for Soap and Water: Lack of efficacy of alcohol-based handrub against *C. difficile*

Interventions compared		Mean log reduction (95% CI), log ₁₀ CFU/mL
Intervention 1	Intervention 2	
Warm water and plain soap	No hand hygiene	2.14 (1.74–2.54)
Warm water and plain soap	Alcohol-based handrub	2.08 (1.69–2.47)
Cold water and plain soap	No hand hygiene	1.88 (1.48–2.28)
Cold water and plain soap	Alcohol-based handrub	1.82 (1.43–2.22)
Warm water and plain soap	Antiseptic hand wipe	1.57 (1.18–1.96)
Warm water and antibacterial soap	No hand hygiene	1.51 (1.12–1.91)
Warm water and antibacterial soap	Alcohol-based handrub	1.46 (1.06–1.85)
Cold water and plain soap	Antiseptic hand wipe	1.31 (0.92–1.71)
Warm water and antibacterial soap	Antiseptic hand wipe	0.94 (0.55–1.34)
Warm water and plain soap	Warm water and antibacterial soap	0.63 (0.23–1.02)
Antiseptic hand wipe	No hand hygiene	0.57 (0.17–0.96)
Antiseptic hand wipe	Alcohol-based handrub	0.51 (0.12–0.91)
Cold water and plain soap	Warm water and antibacterial soap	0.37 (–0.03 to 0.76)
Warm water and plain soap	Cold water and plain soap	0.26 (–0.14 to 0.66)
Alcohol-based handrub	No hand hygiene	0.06 (–0.34 to 0.45)

Oughton et al. Infect Control Hosp Epidemiol 2009;30:939-44.



Supplemental Prevention Strategies: Hand Hygiene – Alcohol Hand Rub Use 2000-2003

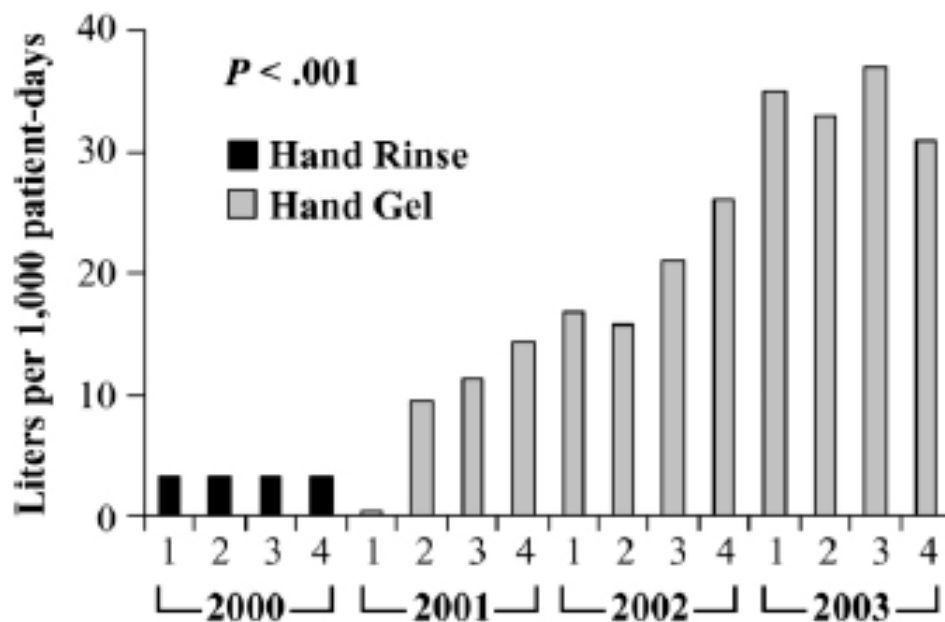


FIGURE 1. Use of alcohol hand rub by healthcare workers, in liters per 1,000 patient-days, per quarter, 2000-2003.

Boyce et al. Infect Control Hosp Epidemiol 2006; 27:479-83.



Supplemental Prevention Strategies: Hand Hygiene – CDI Rates 2000-2003

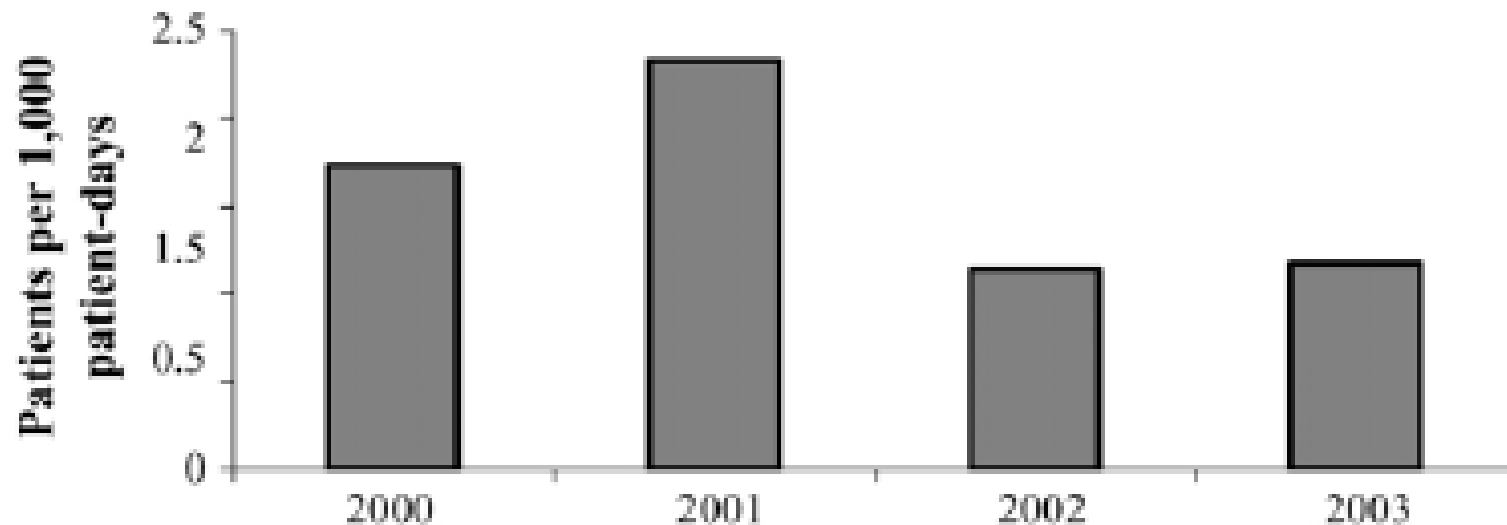


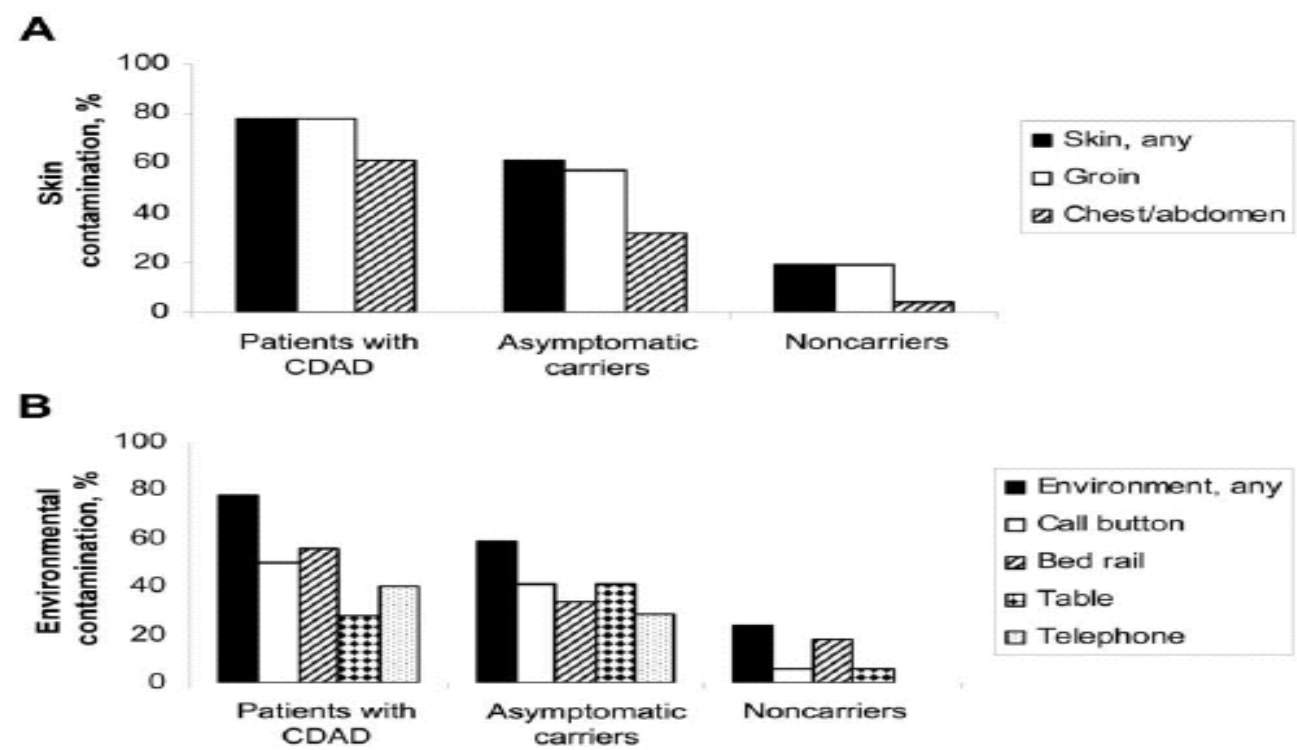
FIGURE 2. Number of patients with 1 or more tests positive for *Clostridium difficile* toxin per 1,000 patient-days, 2000-2003.

Boyce JM et al. Infect Control Hosp Epidemiol 2006; 27:479-83.

Supplemental Prevention Strategies: Universal Glove Use

Role of asymptomatic carriers?

Rationale for universal glove use on units with high CDI rates



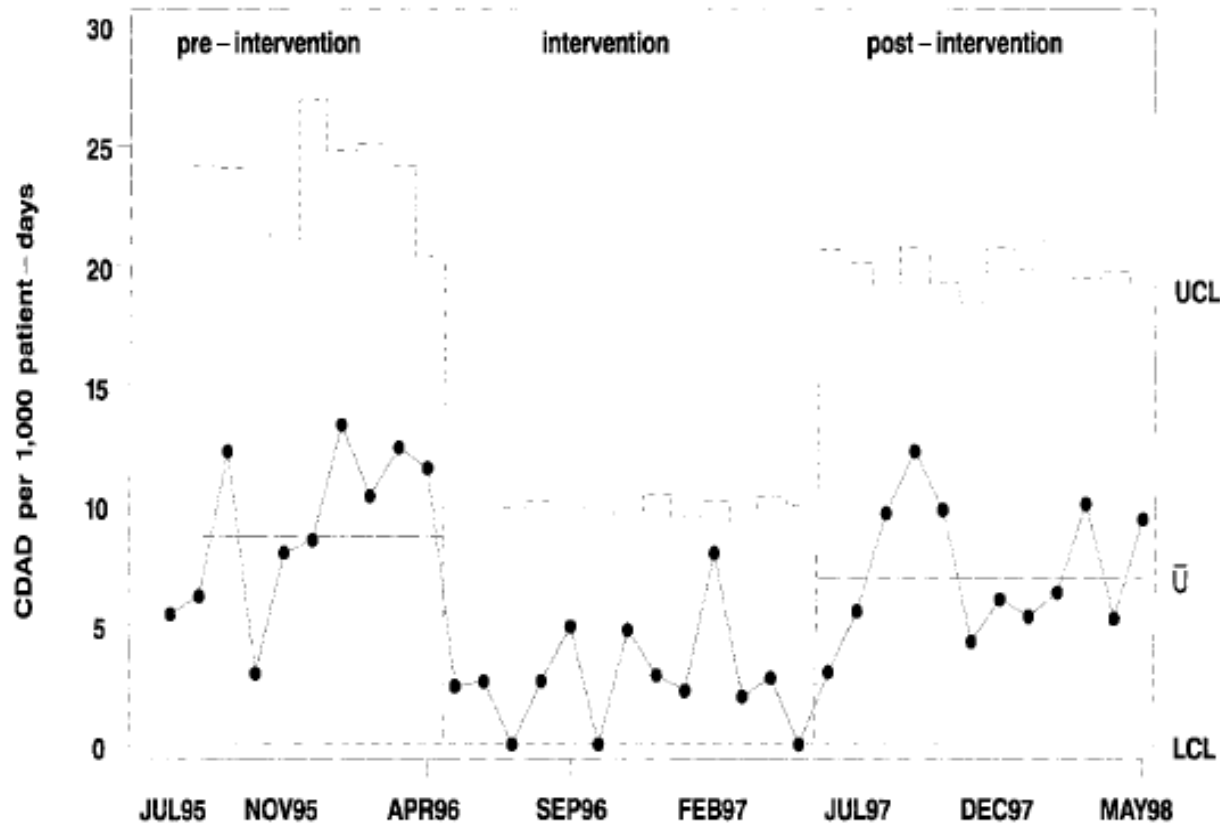
Riggs et al. Clin Infect Dis 2007;45:992–8.



Supplemental Prevention Strategies: Environmental Cleaning



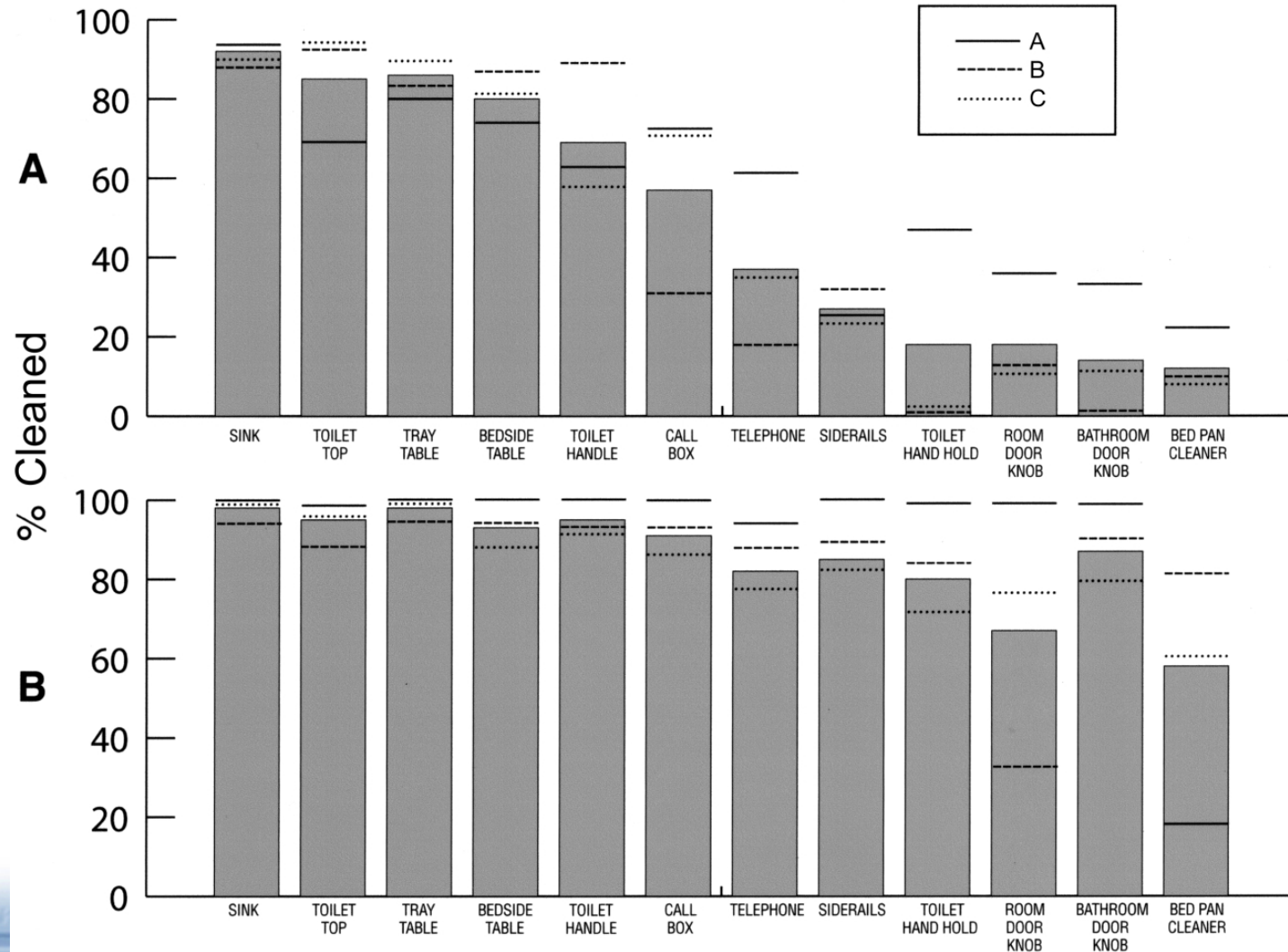
How Much Can be Achieved via Environmental Decontamination?



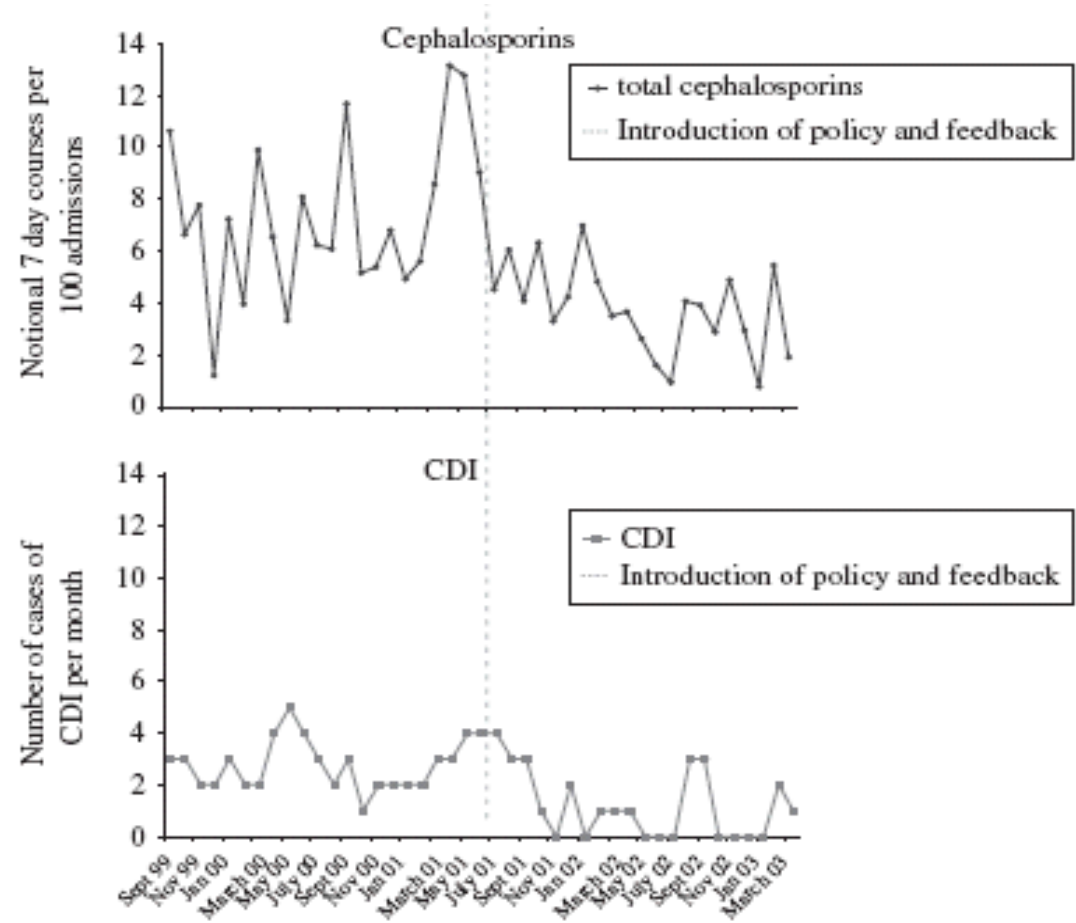
Mayfield et al. Clin Infect Dis 2000;31:995–1000.

Supplemental Prevention Strategies: Environmental Cleaning

Assess adequacy of cleaning before changing to new cleaning



Supplemental Prevention Strategies: Audit and feedback targeting broad-spectrum antibiotics



Fowler et al. J Antimicrob Chemother 2007;59:990-5.



Methicillin-Resistant *Staphylococcus aureus* (MRSA) Infections

Activity C: ELC Prevention Collaboratives

John Jernigan, MD, MS

Alex Kallen, MD, MPH

Division of Healthcare Quality Promotion

Centers for Disease Control and Prevention

Draft – 1/19/10 ---- Disclaimer: The findings and conclusions in this presentation are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.



Outline



- **Background**
 - Impact
 - HHS Prevention Targets
 - Pathogenesis
 - Epidemiology
- **Prevention Strategies**
 - Core
 - Supplemental
- **Measurement**
 - Process
 - Outcome
- **Tools for Implementation/Resources/References**



Background: Impact



Staphylococcus aureus is a common cause of healthcare-associated infections

- Second most common overall cause of healthcare-associated infections reported to the National Healthcare Safety Network (NHSN)
 - Coagulase-negative staphylococci (15%), *S. aureus* (14%)
 - Most common cause of surgical site infections(30%) and ventilator associated pneumonia (24%)
- Methicillin-resistance in *S. aureus* was first identified in the 1960s primarily among hospitalized patients
- Since that time, methicillin-resistant *S. aureus* (MRSA) has become a predominant cause of *S. aureus* infections in both healthcare and community settings
 - Primarily due to transmission of relatively few ancestral clones rather than the de novo development of methicillin-resistance among susceptible strains

Hidron et al. Infect Control Hosp Epidemiol 2008;29:996-1011



Background: Impact



- Current estimates suggest that 49-65% of healthcare-associated *S. aureus* infections reported to NHSN are caused by methicillin-resistant strains
- National population-based estimates of invasive MRSA infections
 - 94,360 invasive MRSA infections annually in the US
 - Associated 18,650 deaths each year
 - 86% of all invasive MRSA infections are healthcare-associated

Hidron et al. Infect Control Hosp Epidemiol 2008;29:996-1011

Klevens et al. JAMA 2007;298:1763-71



Background: Impact



Why the Emergence of MRSA is a Healthcare Pathogen is Important (1)

- MRSA has emerged as one of the predominant pathogens in healthcare-associated infections
- Treatment options for MRSA are limited and less effective than options available for susceptible *S. aureus* infections and result in higher morbidity and mortality
- High prevalence influences unfavorable antibiotic prescribing, which contributes to further spread of resistance
 - prevalent MRSA → more vancomycin use → more vancomycin resistance (VRE and VRSA)
more linezolid/daptomycin use → more resistance



Background: Impact



Why the Emergence of MRSA is a Healthcare Pathogen is Important (2)

- MRSA adds to overall *S. aureus* infection burden
 - Preventing MRSA infections reduces overall burden of *S. aureus* infections
- MRSA is a marker for ability to contain transmission of important pathogens in the healthcare setting
 - Programs that successfully prevent MRSA transmission are likely to have benefit when applied to other epidemiologically important healthcare pathogens that spread by patient-to-patient transmission



Background: HHS Prevention Targets



- Population-based surveillance
 - 50% reduction in incidence rate of all healthcare-associated invasive MRSA infections
- National Healthcare Safety Network
 - 50% reduction in incidence rate of hospital-onset MRSA bacteremia

HHS Action Plan to Prevent HAI

(<http://www.hhs.gov/ophs/initiatives/hai/infection.html>)



Background: Pathogenesis



- For MRSA, colonization generally precedes infection
- In addition, colonization can be long-lasting -- months or years in some subpopulations
- In general, MRSA is transmitted person to person; the “*de novo*” generation of resistance in *S. aureus* is very rare
- Transmission of MRSA from the environment to people, although it can occur, is less common than transmission from person to person



Background: Epidemiology



- Once acquired, MRSA colonization can be long-lasting -- months or years in some subpopulations
 - A patient acquiring MRSA colonization during a hospital stay has increased risk for MRSA infections following discharge, or during subsequent acute and long-term care admissions
- MRSA carriers also serve as reservoirs for further transmission as they move through and across healthcare facilities
- Healthcare facilities that share patients are interdependent upon one another with regard to their MRSA experience
 - The quality of MRSA control in one facility may influence the MRSA experience in others
 - There may be advantages to coordinated multicenter control programs involving facilities that share patients with one another



Background: Epidemiology



- Successful MRSA prevention is possible
 - Single and multi-center studies have demonstrated that MRSA prevention programs can be effective
 - Reductions in incidence of MRSA disease by up to 70% have been documented in acute-care facilities
 - Significant intervention-associated reductions in the proportion of *S. aureus* infections caused by MRSA have also been documented in these studies

Ellingson K et al. Presented at SHEA 2009, Abstract 512.

Huang et al. Clin Infect Dis 2006; 43:971-88.

Robicsek et al. Ann Intern Med 2008; 148:409-18.



Epidemiology

- Successful MRSA prevention is possible
 - According to NSHN data, rates of central line-associated BSI (CLABSI) caused by MRSA have declined by nearly 50% in the past decade
 - This observation may be primarily attributable to successful CLABSI prevention efforts
 - The proportion of all *S. aureus* CLABSI caused by MRSA has continued to increase during the same time period
- Population-based estimates suggest the incidence of invasive healthcare-associated MRSA disease decreased by 11-17% in the US between 2005-2007

Burton et al. JAMA 2009; 301:727-36

Kallen AJ, et al. Presented at SHEA 2009 Abstract 49



Prevention Strategies

- **Core Strategies**
 - High levels of scientific evidence
 - Demonstrated feasibility

- **Supplemental Strategies**
 - Some scientific evidence
 - Variable levels of feasibility

*The Collaborative should at a minimum include core prevention strategies. Supplemental prevention strategies also may be used. Hospitals should not be excluded from participation if they already have ongoing interventions using supplemental prevention strategies. Project coordinators should carefully track which prevention strategies are being used by participating facilities.



Prevention Strategies: Basic Rationale



- Because MRSA colonization generally precedes infection with this organism, MRSA interventions primarily have targeted two broad areas:
 - Preventing transmission from colonized to uncolonized persons – a focus of most of the interventions in this toolkit
 - Preventing infection in colonized individuals:
 - Not MRSA-specific: Strategies aimed at preventing device and procedure-associated infections (e.g., ventilator associated pneumonias, central line associated bloodstream infections, etc), not necessarily MRSA-specific
 - MRSA-specific: MRSA decolonization strategies



Core Prevention Strategies



- Assessing hand hygiene practices
- Implementing Contact Precautions
- Recognizing previously colonized patients
- Rapidly reporting MRSA lab results
- Providing MRSA education for healthcare providers



Core Prevention Strategies: Hand Hygiene

- Hand hygiene should be a cornerstone of prevention efforts
 - Prevents transmission of pathogens via hands of healthcare personnel
- As part of a hand hygiene intervention, consider:
 - Ensuring easy access to soap and water/alcohol-based hand gels
 - Education for healthcare personnel and patients
 - Observation of practices - particularly around high-risk procedures (before and after contact with colonized or infected patients)
 - Feedback – “Just in time” feedback if failure to perform hand hygiene observed



Core Prevention Strategies: Contact Precautions



- Involves use of gown and gloves for patient care
 - Don equipment prior to room entry
 - Remove prior to room exit
- Single room (preferred) or cohorting for MRSA colonized/infected patients
- Use of dedicated non-essential items may help decrease transmission due to contact with these fomites
 - Blood pressure cuffs
 - Stethoscopes
 - IV poles and pumps



Core Prevention Strategies: Recognizing Previously Colonized

- Patients can be colonized with MRSA for months
- There is no single 'best' strategy for discontinuation of isolation precautions for MRSA patients
- Being able to recognize previously colonized or infected patients who have not met criteria for discontinuing isolation allows them to be subject to interventions in a timely fashion



Core Prevention Strategies: Laboratory Reporting



- Facilities should have a mechanism for rapidly communicating positive MRSA results from laboratory to clinical area
- Allows for rapid institution of interventions on newly identified MRSA patients



Core Prevention Strategies: Education



- To improve adherence to hand hygiene
- To improve adherence to interventions (e.g., Contact Precautions)
- Encourage behavioral change through a better understanding of the problem



Core Prevention Strategies: Device and Procedure-Associated Prevention Measures

- In addition to measures designed to prevent MRSA transmission, healthcare facilities should routinely implement strategies for preventing device- and procedure-associated infections
 - Central line-associated bloodstream infections
 - Surgical site infections
 - Catheter-associated urinary tract infections
 - Ventilator-associated pneumonia



Supplemental Prevention Strategies

- Active surveillance testing – screening of patients to detect colonization even if no evidence of infection
 - Widely used and even recommended as a core prevention strategy by some, but precise role remains controversial
- Other novel strategies
 - Decolonization
 - Chlorhexidine bathing



Supplemental Prevention Strategies: Active Surveillance Testing (AST)



- When clinical culture results alone are used to identify MRSA carriers, more than half of all MRSA-colonized patients remain unrecognized*
 - The rationale for active surveillance testing is to identify all colonized patients so that additional precautions can be applied (e.g. Contact Precautions)
- To date, results of studies evaluating AST have had mixed results
 - Huang et al. Clin Infect Dis 2006; 43:971-978
 - Observational study
 - Found largest decrease in MRSA bacteremia associated with institution of active surveillance
 - Robicsek et al. Ann Intern Med 2008; 148:409-418
 - Observational study
 - Found significant decrease in MRSA disease with universal institution of AST combined with decolonization regimens
 - Harbarth et al. JAMA 2008; 299:1149-1157
 - Observational study
 - No significant decrease in MRSA disease with institution of rapid AST
- Several successful MRSA prevention collaboratives have used AST as one of their interventions

*Salgado CD, Farr BM. Infect Control Hosp Epidemiol 2006; 27:116-121.



Supplemental Prevention Strategies: Active Surveillance Testing (2)



Testing methods:

- Culture
 - Pros
 - Generally less costly
 - A common practice most labs are used to
 - Cons
 - May take 72 hours to identify MRSA colonized patients. If pre-emptive isolation not employed, may allow for transmission prior to recognizing patient as positive
- Polymerase chain reaction
 - Pros
 - Rapid results
 - Cons
 - Expensive
 - Technically more challenging



Supplemental Prevention Strategies: Active Surveillance Testing (3)



Unknowns:

- **Which body sites should be tested?**
 - Nares most common
 - Other potential sites include wounds, axillae, groin
 - Adding more sites increases yield of testing; contribution to goal of decreasing transmission unclear
- **Frequency of testing?**
 - Generally done at time of admission, sometimes repeated weekly
 - Including discharge AST allows for identification of transmission events that occurred during hospitalization
- **Who should be tested?**
 - One commonly employed strategy: focusing on patients in high-risk areas (e.g., ICUs)
 - Some employ facility-wide AST



Supplemental Prevention Strategies: Decolonization Therapy for MRSA Carriers

- Decolonization is use of topical and/or systemic agents to suppress or eliminate colonization
- May reduce risk of subsequent infections in MRSA carriers
- May help decrease MRSA spread by reducing reservoir of transmission
- No data yet to definitively support its routine use in general patient care settings
 - Robicsek and Harbarth studies used decolonization in addition to AST with mixed results
 - Growing evidence suggests that pre-operative *S. aureus* decolonization regimens decrease risk of subsequent *S. aureus* infection in some surgical populations



Supplemental Prevention Strategies: Decolonization Therapy for MRSA Carriers (2)

Unknowns:

- Which body sites should be targeted?
 - just nares or whole body
- Which decolonization regimen?
 - Intranasal mupirocin, chlorhexidine baths
 - May be advantageous to use combination of mupirocin and chlorhexidine
 - Other agents (oral agents)
- Will emergence of mupirocin resistance be a limiting factor?
 - Also potential cross-resistance to other therapeutic agents



Supplemental Prevention Strategies: Universal use of Chlorhexidine Bathing in High-Risk Patient Populations

- Use of daily chlorhexidine baths in ICU populations may decrease overall rates of bloodstream infections and MRSA acquisition, but effect on MRSA infections less clear
- Does not require AST since applied to all patients in the target population

Climo MW, et al. Crit Care Med 2009; 37:1858-65



Summary of Prevention Strategies



Core Measures

- Assessing hand hygiene practices
- Implementing Contact Precautions
- Recognizing previously colonized patients
- Rapidly reporting MRSA lab results
- Providing MRSA education for healthcare providers

Supplemental Measures

- Active surveillance testing
- Decolonization
- Chlorhexidine bathing



Measurement: Outcome Using NHSN to support MRSA Prevention Collaboratives

- NHSN provides a module designed to facilitate prevention of healthcare-associated MRSA and other multidrug-resistant organisms
 - Provides methods and reporting mechanisms for both outcome and process measures

<http://www.cdc.gov/nhsn>



Measurement: Outcome MRSA Outcome Measures



- MRSA Infection Incidence Rate
 - Numerator = Number of MRSA infections*
 - Denominator = Number of patient-days (stratified by time and location)

**per current NHSN definitions for healthcare associated infection*

<http://www.cdc.gov/nhsn>



Measurement: Outcome NHSN



- **Laboratory Identified MRSA Events**
 - Proxy Measure for MDRO Healthcare Acquisition
 - Numerator = Number of 1st MRSA isolates per patient (infection or colonization) identified from a clinical culture (i.e. not from AST) among those with no documented prior evidence of infection or colonization
 - Denominator = number of patient days for the location or facility
 - Proxy Measure for MDRO Bloodstream Infection
 - Numerator = Total number of patients with MRSA blood isolate and no prior positive blood culture in ≤ 2 weeks
 - Denominator = Number of patient-days for same period

Note : isolates of MRSA are generally attributed to the location or facility under surveillance if they come from cultures collected more than 3 calendar days after admission (if day of admission is day 1)

<http://www.cdc.gov/nhsn>



Measurement: Outcome

Other Potential Measures Available in NHSN



- **Measures Based on Active Surveillance Testing**
 - Admission prevalence rate
 - Incidence of MRSA colonization
- **Other Laboratory Identified MRSA Events**
 - Admission prevalence rate (community-onset MRSA)
 - Overall prevalence rate (community-onset plus healthcare facility-onset)
 - MRSA bloodstream infection admission prevalence rate
 - Proportion of *S. aureus* resistant to methicillin

<http://www.cdc.gov/nhsn>



Measurement: Process

MRSA Process Measures



- As part of the MDRO module, NHSN allows facilities to track adherence to:
 - Active surveillance testing
 - Contact Precautions
 - Hand hygiene

<http://www.cdc.gov/nhsn>



Evaluation Considerations

- **Assess baseline policies and procedures**
- **Areas to consider**
 - Surveillance
 - Prevention strategies
 - Measurement
- **Coordinator should track new policies/practices implemented during collaboration**



References

- Burton DC, Edwards JR, Horan TC, et al. Methicillin-resistant *Staphylococcus aureus* central line-associated bloodstream infections in US intensive care units. JAMA 2009;301:727-36.
- Calfee D, Salgado CD, Classen D, et al. SHEA Compendium: Strategies to Prevent MRSA Transmission in Acute Care Hospitals Infect Control Hosp Epidemiol 2008; 29:S62-S80.



References

- Climo MW, Sepkowitz KA, Zuccotti G, et al. The effectiveness of daily bathing with chlorhexidine on the acquisition of methicillin-resistant *Staphylococcus aureus*, vancomycin-resistant *Enterococcus*, and healthcare-associated bloodstream infections: results of a quasi-experimental multicenter trial. *Crit Care Med* 2009;37:1858-65.
- Cohen AI, Calfee D, Fridkin SK, et al. Recommendations for metrics for multidrug-resistant organisms in healthcare settings: SHEA/HICPAC Position Paper. *Infect Control Hosp Epidemiol* 2008; 29:901-13.



References

- Ellingson K, Iversen N, Zuckerman JM, et al. A successful multi-center intervention to prevent transmission of methicillin-resistant *Staphylococcus aureus*. Presented at SHEA 2009, Abstract 512.
- HICPAC – Management of Multidrug Resistant Organisms in Healthcare Settings, 2006
<http://www.cdc.gov/ncidod/dhqp/pdf/ar/MDROGuideline2006.pdf>
- Hidron AL, Edwards JR, Patel J, et al. NHSN annual update: antimicrobial-resistant pathogens associated with healthcare-associated infections: annual summary of data reported to the National Healthcare Safety Network at the Centers for Disease Control and Prevention, 2006-2007. *Infect Control Hosp Epidemiol*; 2008;29:966-1011.



References

- Huang SS, Yokoe, DS, Hinrichsen VL, et al. Impact of routine intensive care unit surveillance cultures and resultant barrier precautions on hospital-wide methicillin-resistant *Staphylococcus aureus* bacteremia. *CLin Infect Dis* 2006;43:971-8.
- Kallen AJ, Yi Mu, Bulens SN, et al. Changes in the incidence of healthcare-associated invasive MRSA infections and concurrent MRSA control practices in the US, 2005 to 2007 Presented at SHEA 2009. Abstract 49.
- Klevens, RM, Morrison MA, Nadle J, et al. Invasive methicillin-resistant *Staphylococcus aureus* infections in the United States. *JAMA*;2007:298:1763-71.



References

- Robicsek A, Beaumont JL, Paule SM, et al. Universal surveillance for methicillin-resistant *Staphylococcus aureus* in 3 affiliated hospitals. *Ann Intern Med* 2008;148:409-18.
- Harbarth S, Fankhauser C, Srenzel J, et al. Universal screening for methicillin-resistant *Staphylococcus aureus* at hospital admission and nosocomial infection in surgical patients. *JAMA* 2008;229:1149-57.



Additional Resources

- HHS Action Plan to Prevent Healthcare Associated Infections. June 2009 <http://www.hhs.gov/ophs/initiatives/hai/infection.html>
- Overview of Methicillin-Resistant *Staphylococcus aureus* Surveillance through the National Healthcare Safety Network
http://www.cdc.gov/nhsn/PDFs/Overview_MRSA_Surveillance_Final12_08.pdf
- Multidrug-Resistant Organism & *Clostridium difficile*-Associated Disease (MDRO/CDAD) Module
http://www.cdc.gov/nhsn/PDFs/pscManual/12pscMDRO_CDA_Dcurrent.pdf
- NHSN Web site – www.cdc.gov/nhsn



Additional Reference Slides



- The following slides may be used for presentations on MRSA
- Explanations are available in the notes sections of the slides



Distribution and Rank Order of 9 Most Common Pathogens Reported for 28,502 HAIs, NHSN 2006-2007



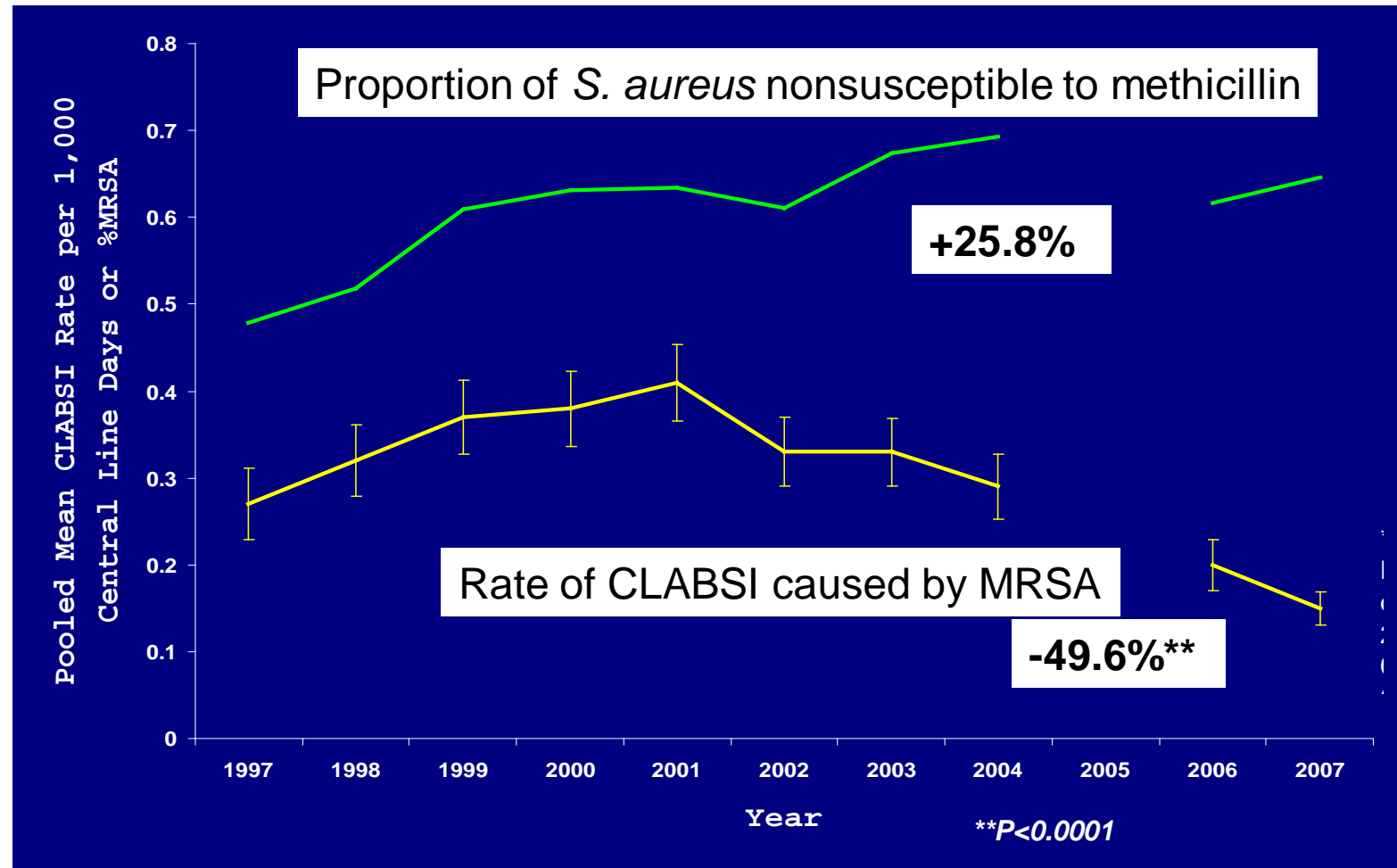
Pathogen	Column %				Total*
	CLABSI 11,428	CAUTI 9,377	VAP 5,960	SSI 7,025	
CoNS	34	3	1	14	15
<i>S. aureus</i>	10	2	24	30	14
<i>Enterococcus</i> spp.	15	15	1	11	12
<i>Candida</i> spp.	12	21	<1	2	11
<i>E. coli</i>	3	22	5	10	10
<i>P. aeruginosa</i>	3	10	16	5	8
<i>K. pneumoniae</i>	5	8	7	3	6
<i>Enterobacter</i> spp.	4	4	8	4	5
<i>A. baumannii</i>	2	1	8	1	3

15.6% of healthcare-associated infections had >1 pathogen (polymicrobial)

Hidron et al. Infect Control Hosp Epidemiol 2008;29:996-1011



Trends in % MRSA and Rates of MRSA Central Line-Associated Bloodstream Infections (CLABSI) — United States, 1997-2007



Burton et al. JAMA 2009; 301:727-36





Modeled Incidence and Percent Change for All Invasive Hospital-Onset and Healthcare-Associated, Community-Onset MRSA infections, 2005-2007



<i>Year</i>	<i>Modeled incidence per 100,000 population</i>	<i>Modeled percent change from previous year</i>	<i>Total modeled percent change</i>	<i>P-value</i>
<i>Hospital-onset</i>				
2005	9.95			
2006	8.96	-9.97%		
2007	8.24	-8.08%	-17.2%	0.01
<i>Healthcare-associated, community-onset</i>				
2005	22.13			
2006	21.11	-4.59%		
2007	19.70	-6.71%	-11.0%	0.04

Kallen AJ, et al, SHEA 2009, Abstract 49





Surgical Site Infection (SSI) Toolkit

Activity C: ELC Prevention Collaboratives

S.I. Berríos-Torres, MD

Division of Healthcare Quality Promotion
Centers for Disease Control and Prevention

Draft - 12/21/09 --- Disclaimer: The findings and conclusions in this presentation are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention.





Outline



- **Background**
 - Impact
 - HHS Prevention Targets
 - Pathogenesis
 - Epidemiology
- **Prevention Strategies**
 - Core
 - Supplemental
- **Measurement**
 - Process
 - Outcome
- **Tools for Implementation/Resources/References**



Background: Impact

Burden-US

- ~300,000 SSIs/yr (17% of all HAI; second to UTI)
- 2%-5% of patients undergoing inpatient surgery

Mortality

- 3 % mortality
- 2-11 times higher risk of death
- 75% of deaths among patients with SSI are directly attributable to SSI

Morbidity

- long-term disabilities

Anderson DJ, et al. Strategies to prevent surgical site infections in acute care hospitals. Infect Control Hosp Epidemiol 2008;29:S51-S61 for individual references





Background: Impact



Length of Hospital Stay

- ~7-10 additional postoperative hospital days

Cost

- \$3000-\$29,000/SSI depending on procedure & pathogen
- Up to \$10 billion annually
- Most estimates are based on inpatient costs at time of index operation and do not account for the additional costs of rehospitalization, post-discharge outpatient expenses, and long term disabilities

Anderson DJ, et al. Strategies to prevent surgical site infections in acute care hospitals. Infect Control Hosp Epidemiol 2008;29:S51-S61 for individual references



Background: HHS Prevention Targets

- **Reduce the admission and readmission SSI Standardized Incidence Ratio (SIR) by at least 25% from baseline**
 - Outcome – SSI SIR

- **95% adherence rates to each SCIP/NQF infection process measure**
 - Process - Adherence to SCIP/NQF infection process measures

<http://www.hhs.gov/ophs/initiatives/hai/prevtargets.html>
Appendix G



Background: Pathogenesis

Pathogen Sources



Endogenous

- Patient flora
 - skin
 - mucous membranes
 - GI tract
- Seeding from a distant focus of infection



Background: Pathogenesis

Pathogen Sources



Exogenous

- Surgical Personnel (surgeon and team)
 - Soiled attire
 - Breaks in aseptic technique
 - Inadequate hand hygiene
- OR physical environment and ventilation
- Tools, equipment, materials brought to the operative field



Background: Pathogenesis Organisms Causing SSI January 2006-October 2007

<i>Staphylococcus aureus</i>	30.0%
Coagulase-negative staphylococci	13.7%
Enterococcus spp.	11.2%
<i>Escherichia coli</i>	9.6%
<i>Pseudomonas aeruginosa</i>	5.6%
Enterobacter spp	4.2%
<i>Klebsiella pneumoniae</i>	3.0%
Candida spp.	2.0%
<i>Klebsiella oxytoca</i>	0.7%
<i>Acinetobacter baumannii</i>	0.6%

N=7,025

Hidron AI, et.al., Infect Control Hosp Epidemiol 2008;29:996-1011
Hidron AI et.al., Infect Control Hosp Epidemiol 2009;30:107–107(ERRATUM)



Background: Epidemiology Emerging Challenges

Challenges in detecting SSIs

- Lack of standardized methods for post-discharge/outpatient surveillance
 - Increased number of outpatient surgeries
 - Shorter postoperative inpatient stays

Antimicrobial Prophylaxis

- Increasing trend toward resistant organisms may undermine the effectiveness of existing recommendations for antimicrobial prophylaxis



Background: Epidemiology

Important Modifiable Risk Factors

- Antimicrobial prophylaxis
 - Inappropriate choice (procedure specific)
 - Improper timing (pre-incision dose)
 - Inadequate dose based on body mass index, procedures >3h, or increased blood loss
- Skin or site preparation ineffective
 - Removal of hair with razors
- Colorectal procedures
 - Inadequate bowel prep/antibiotics
 - Improper intraoperative temperature regulation



Background: Epidemiology

Additional Modifiable Risk Factors

- Excessive OR traffic
- Inadequate wound dressing protocol
- Improper glucose control
- Colonization with preexisting microorganisms
- Inadequate intraoperative oxygen levels



Prevention Strategies

- **Core Strategies**

- High levels of scientific evidence
- Demonstrated feasibility

- **Supplemental Strategies**

- Some scientific evidence
- Variable levels of feasibility

The Collaborative should at a minimum include core prevention strategies. Supplemental prevention strategies also may be used. Most core and supplemental strategies are based on HICPAC guidelines. Strategies that are not included in HICPAC guidelines will be noted by an asterisk () after the strategy. HICPAC guidelines may be found at www.cdc.gov/hicpac



Prevention Strategies: Core Preoperative Measures

Administer antimicrobial prophylaxis in accordance with evidence based standards and guidelines

- Administer within 1 hour prior to incision*
 - 2hr for vancomycin and fluoroquinolones
- Select appropriate agents on basis of
 - Surgical procedure
 - Most common SSI pathogens for the procedure
 - Published recommendations

*Fry DE. Surgical Site Infections and the Surgical Care Improvement Project (SCIP): Evolution of National Quality Measures. Surg Infect 2008;9(6):579-84.



Prevention Strategies: Core Preoperative Measures

- **Remote infections-whenever possible:**
 - Identify and treat before elective operation
 - Postpone operation until infection has resolved
- **Do not remove hair at the operative site unless it will interfere with the operation; do not use razors**
 - If necessary, remove by clipping or by use of a depilatory agent



Prevention Strategies: Core



Preoperative Measures (continued)

- **Skin Prep**
 - Use appropriate antiseptic agent and technique for skin preparation
- **Maintain immediate postoperative normothermia***
- **Colorectal surgery patients**
 - Mechanically prepare the colon (Enemas, cathartic agents)
 - Administer non-absorbable oral antimicrobial agents in divided doses on the day before the operation

*Fry DE. Surgical Site Infections and the Surgical Care Improvement Project (SCIP): Evolution of National Quality Measures. Surg Infect 2008;9(6):579-84.



Prevention Strategies: Core Intraoperative Measures



- **Operating Room (OR) Traffic**
 - Keep OR doors closed during surgery except as needed for passage of equipment, personnel, and the patient



Prevention Strategies: Core Postoperative Measures



- **Surgical Wound Dressing**
 - Protect primary closure incisions with sterile dressing for 24-48 hrs post-op
- **Control blood glucose level during the immediate post-operative period (cardiac)***
 - Measure blood glucose level at 6AM on POD#1 and #2 with procedure day = POD#0
 - Maintain post-op blood glucose level at <200mg/dL
- **Discontinue antibiotics within 24hrs after surgery end time (48hrs for cardiac)***

*Fry DE. Surgical Site Infections and the Surgical Care Improvement Project (SCIP): Evolution of National Quality Measures. Surg Infect 2008;9(6):579-84.





Prevention Strategies: Supplemental Preoperative



- Nasal screen and decolonize only *Staphylococcus aureus* carriers undergoing elective cardiac and other procedures (i.e., orthopaedic, neurosurgery procedures with implants) with preoperative mupirocin therapy*
Bode LGM, et al. Preventing SSI in nasal carriers of Staph aureus. NEJM 2010;362:9-17
- Screen preoperative blood glucose levels and maintain tight glucose control POD#1 and POD#2 in patients undergoing select elective procedures (e.g., arthroplasties, spinal fusions)*

NOTE: These supplemental strategies are not part of the 1999 HICPAC Guideline for Prevention of Surgical Site Infections



Prevention Strategies: Supplemental Perioperative



- Redose antibiotic at the 3 hr interval in procedures with duration >3hrs (* See exceptions to this recommendation in*Engelman R, et al. The Society of Thoracic Surgeons Practice Guideline Series:Antibiotic Prophylaxis in Cardiac Surgery, Part II:Antibiotic Choice. Ann Thor Surg 2007;83:1569-76
- Adjust antimicrobial prophylaxis dose for obese patients (body mass index >30)* Anderson DJ, Kaye KS, Classen D, et al. Strategies to prevent surgical site infections in acute care hospitals. Infect Control Hosp Epidemiol 2008;29 (Suppl 1):S51-S61
- Use at least 50% fraction of inspired oxygen intraoperatively and immediately postoperatively in select procedure(s)* Maragakis LL, Cosgrove SE, Martinez EA, et al. Intraoperative fraction of inspired oxygen is a modifiable risk factor for surgical site infection after spinal surgery. Anesthesiology 2009;110:556-562. and Meyhoff CS, Wetterslev J, Jorgensen LN, et al. Effect of high perioperative oxygen fraction on surgical site infection and pulmonary complications after abdominal surgery: The PROXI randomized clinical trial. JAMA 2009;302:1543-1550.

NOTE: These supplemental strategies are not part of the 1999 HICPAC Guideline for Prevention of Surgical Site Infections





Prevention Strategies: Supplemental Postoperative



- Feedback of surgeon specific infection rates.



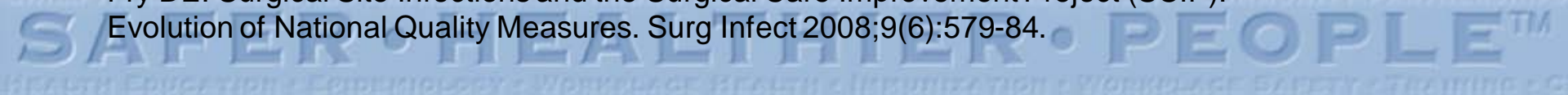
Measurement: Surgical Care Improvement Project (SCIP)



Process Measures

Quality Indicator	Numerator	Denominator
Appropriate antibiotic choice	Number of patients who received the appropriate prophylactic antibiotic	All patients for whom prophylactic antibiotics are indicated
Appropriate timing of prophylactic antibiotics	Number of patients who received the prophylactic antibiotic within 1hr prior to incision (2hr: Vancomycin or Fluoroquinolones)	All patients for whom prophylactic antibiotics are indicated
Appropriate discontinuation of antibiotics	Number of patients who received prophylactic antibiotics and had them discontinued in 24 h (48h cardiac)	All patients who received prophylactic antibiotics

Fry DE. Surgical Site Infections and the Surgical Care Improvement Project (SCIP): Evolution of National Quality Measures. Surg Infect 2008;9(6):579-84.





Measurement: Surgical Care Improvement Project (SCIP)



Process Measures (continued)

Quality Indicator	Numerator	Denominator
Appropriate hair removal	Number of patients who did not have hair removed or who had hair removed with clippers	All surgical patients
Normothermia	Number of patients with postoperative temperature $\geq 36.0^{\circ}\text{C}$	All surgical patients
Glucose control	Number of cardiac surgery patients with glucose control at 6AM POD1 and POD2 (operation = POD0)	Patients undergoing cardiac surgery

Fry DE. Surgical Site Infections and the Surgical Care Improvement Project (SCIP): Evolution of National Quality Measures. Surg Infect 2008;9(6):579-84.





Measurement: Outcome Measures

SSI Rate



Patients with SSI after selected operations X100
Total # of selected operations performed

- Crude, unadjusted rate
- Can lead to erroneous conclusions regarding SSI risk by institution and/or surgeon
- NOT for reporting or inter-hospital comparisons



Measurement: Outcome Measures Risk Adjustment (1) NNIS Risk Index

Score to predict risk of acquiring SSI

- Widely used-targeted at surveillance
- Operation-specific
- Allows monitoring of trends
- Facilitates comparison
 - facility vs. national

Culver DH, Horan TC, Gaines RP. Surgical infection rates by wound class, operative procedure, patient risk index. Am J Med;1991:152S-157S.



Measurement: Outcome Measures

Risk Adjustment (2)

NNIS Risk Index



- Focus on high volume operations
- Employs Risk Stratification
 - American Society of Anesthesiologists (ASA) score (3, 4, or 5)
 - Wound Classification (contaminated or dirty)
 - Duration of Procedure (over T [proc specific] hours)
- Does not include many patient & perioperative related SSI risk factors
- Increased NNIS Risk index = Increased risk of SSI

Culver DH, Horan TC, Gaines RP. Surgical infection rates by wound class, operative procedure, patient risk index. Am J Med;1991:152S-157S.



Measurement: Outcome Measures

Risk Adjustment (2)

Standardized Incidence Ratio - SIR

$$\text{SIR} = \frac{\text{Observed \# SSI}}{\text{Expected \# SSI}}$$

Expected # SSI =
operations* in each proc risk category X NNIS rate
100

- Value >1.0 = more SSIs than expected
- Helps better identify outliers
- Will be used for comparison within NHSN in 2010

*Performed by a surgeon, a surgical subspecialty service or a hospital
Detailed explanation and examples in: Edwards JR, Horan TC. Risk-adjusted Comparisons.
In: Carrico R, ed. APIC Text of Infection Control and Epidemiology, 3rd ed. Washington DC
APIC 2009. Chapter 7, p.1-7.



Evaluation Considerations

- **Assess baseline policies and procedures**
- **Areas to consider**
 - **Surveillance**
 - **Prevention strategies**
 - **Measurement**
- **Coordinator should track new policies/practices implemented during collaboration**



References

- Casey AL, Elliott TSJ. Progress in the prevention of surgical site infection. *Curr Opin Infect Dis* 2009;22:370-375
- Chong T, Sawyer R. Update on the epidemiology and prevention of surgical site infections. *Curr Infect Dis Rep* 2002;4:484-490)
- Department of Health and Human Services. Action Plan to Prevent Healthcare-Associated Infections. <http://www.hhs.gov/ophs/initiatives/hai/infection.html> Accessed 17 February 2010
- Fry DE. A systems approach to the prevention of surgical infections. *Surg Clin N Am* 2009;89:521-537.
- Haynes AB, Weiser TG, Berry WR, et al,. A surgical safety checklist to reduce morbidity and mortality in a global population. *N Eng J Med* 2009;360(5):491-499.



References

- Horan TC, Andrus M, Dudeck MA. CDC/NHSN surveillance definition of health care-associated infection and criteria for specific types of infections in the acute care setting. *Am J Infect Control* 2008;36:309-32
- Kirby JP, Mazuski JE. Prevention of surgical site infection. *Surg Clin N Am* 2009;89:365-389.
- Mangram AJ, Horan TC, Pearson ML, et al. Guideline for the prevention of surgical site infection, 1999. Hospital Infection Control Practices Advisory Committee. *Infect Control Hosp Epidemiol* 1999; 20:250-278.
- McKibben L, Horan T, Tokars JI, et al. Guidance on Public Reporting of Healthcare-Associated Infections: Recommendations of the Healthcare Infection Control Practices Advisory Committee. *Am J Infect Control* 2005;33:217-26.



References

- Nichols RL. Preventing surgical site infections. Clin Med Res 2004;2(2):115-118.
- Travis J, Carr JB, Saylor D, et.al., Coronary Artery Bypass Graft Surgery: Surgical Site Infection Prevention. J Healthcare Quality 2009;31:16-23
- Trussell J, Impact of a patient care pathway protocol on surgical site infection rates in cardiothoracic surgery patients. Am J Surg 2008;196:883-889.
- World Alliance for Patient Safety. WHO guidelines for safe surgery. Geneva: World Health Organization, 2008.
- Yokoe DS, Mermel LA, Anderson DJ, et.al. A compendium of strategies to prevent healthcare-associated infections in acute care hospital. Infect Control Hosp Epidemiol 2008;29:S12-S21.



References

SSI Bundles



- Canadian Getting Started Kit:
<http://www.saferhealthcarenow.ca/EN/Interventions/SSI/Pages/ask.aspx> (Select SSI Getting Started Kit)
- IHI:
<http://www.ihl.org/IHI/Programs/Campaign/SSI.htm>
(Select “Power Point Presentation with Facilitator Notes)”) <http://www.100liveswashington.org/resources/SSI-summary.pdf>



References

SSI Bundles



- Australian:
http://www.health.vic.gov.au/sss1/downloads/prev_surgical.pdf
- Scottish:
<http://www.hps.scot.nhs.uk/haiic/ic/SSIPreventionBundle.aspx>



Resources for Implementation

WHO Surgical Safety Checklist



Surgical Safety Checklist



World Health Organization

Patient Safety
A World Alliance for Safer Health Care

Before induction of anaesthesia

(with at least nurse and anaesthetist)

Has the patient confirmed his/her identity, site, procedure, and consent?

Yes

Is the site marked?

Yes

Not applicable

Is the anaesthesia machine and medication check complete?

Yes

Is the pulse oximeter on the patient and functioning?

Yes

Does the patient have a:

Known allergy?

No

Yes

Difficult airway or aspiration risk?

No

Yes, and equipment/assistance available

Risk of >500ml blood loss (7ml/kg in children)?

No

Yes, and two IVs/central access and fluids planned

Before skin incision

(with nurse, anaesthetist and surgeon)

Confirm all team members have introduced themselves by name and role.

Confirm the patient's name, procedure, and where the incision will be made.

Has antibiotic prophylaxis been given within the last 60 minutes?

Yes

Not applicable

Anticipated Critical Events

To Surgeon:

What are the critical or non-routine steps?

How long will the case take?

What is the anticipated blood loss?

To Anaesthetist:

Are there any patient-specific concerns?

To Nursing Team:

Has sterility (including indicator results) been confirmed?

Are there equipment issues or any concerns?

Is essential imaging displayed?

Yes

Not applicable

Before patient leaves operating room

(with nurse, anaesthetist and surgeon)

Nurse Verbally Confirms:

The name of the procedure

Completion of instrument, sponge and needle counts

Specimen labelling (read specimen labels aloud, including patient name)

Whether there are any equipment problems to be addressed

To Surgeon, Anaesthetist and Nurse:

What are the key concerns for recovery and management of this patient?

This checklist is not intended to be comprehensive. Additions and modifications to fit local practice are encouraged.

Revised 1 / 2009

© WHO, 2009

Infection Prevention and Control Assessment Tool for Outpatient Settings

This tool is intended to assist in the assessment of infection control programs and practices in outpatient settings. In order to complete the assessment, direct observation of infection control practices will be necessary. To facilitate the assessment, health departments are encouraged to share this tool with facilities in advance of their visit.

Overview

Section 1: Facility Demographics

Section 2: Infection Control Program and Infrastructure

Section 3: Direct Observation of Facility Practices

Section 4: Infection Control Guidelines and Other Resources

Infection Control Domains for Gap Assessment

- I. Infection Control Program and Infrastructure
- II. Infection Control Training and Competency
- III. Healthcare Personnel Safety
- IV. Surveillance and Disease Reporting
- V.a/b. Hand Hygiene
- VI.a/b. Personal Protective Equipment (PPE)
- VII.a/b. Injection Safety
- VIII.a/b. Respiratory Hygiene/Cough Etiquette
- IX.a/b. Point-of-Care Testing (if applicable)
- X.a/b. Environmental Cleaning
- XI.a/b. Device Reprocessing (if applicable)
- XII. Sterilization of Reusable Devices (if applicable)
- XIII. High-level Disinfection of Reusable Devices (if applicable)



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

Section 1: Facility Demographics			
Facility Name (for health department use only)			
NHSN Facility Organization ID (for health department use only)			
State-assigned Unique ID			
Date of Assessment			
Type of Assessment	<input type="checkbox"/> On-site <input type="checkbox"/> Other (specify):		
Rationale for Assessment (Select all that apply)	<input type="checkbox"/> Outbreak <input type="checkbox"/> Input from accrediting organization or state survey agency <input type="checkbox"/> Other (specify):		
Is the facility licensed by the state?	<input type="checkbox"/> Yes <input type="checkbox"/> No		
Is the facility certified by the Centers for Medicare & Medicaid Services (CMS)?	<input type="checkbox"/> Yes <input type="checkbox"/> No		
Is the facility accredited?	<input type="checkbox"/> Yes <input type="checkbox"/> No If yes, list the accreditation organization: <ul style="list-style-type: none"> <input type="checkbox"/> Accreditation Association for Ambulatory Health Care (AAAHC) <input type="checkbox"/> American Association for Accreditation of Ambulatory Surgery Facilities (AAAASF) <input type="checkbox"/> American Osteopathic Association (AOA) <input type="checkbox"/> The Joint Commission (TJC) <input type="checkbox"/> Other (specify): 		
Is the facility affiliated with a hospital?	<input type="checkbox"/> Yes (specify – for health department use only): <input type="checkbox"/> No		
Which procedures are performed by the facility? Select all that apply.	<input type="checkbox"/> Chemotherapy	<input type="checkbox"/> Endoscopy	<input type="checkbox"/> Ear/Nose/Throat
	<input type="checkbox"/> Imaging (MRI/CT)	<input type="checkbox"/> Immunizations	<input type="checkbox"/> OB/Gyn
	<input type="checkbox"/> Ophthalmologic	<input type="checkbox"/> Orthopedic	<input type="checkbox"/> Pain remediation
	<input type="checkbox"/> Plastic/reconstructive	<input type="checkbox"/> Podiatry	<input type="checkbox"/> Other (specify):
What is the primary procedure-type performed by the facility? Select only one.	<input type="checkbox"/> Chemotherapy	<input type="checkbox"/> Endoscopy	<input type="checkbox"/> Ear/Nose/Throat
	<input type="checkbox"/> Imaging (MRI/CT)	<input type="checkbox"/> Immunizations	<input type="checkbox"/> OB/Gyn
	<input type="checkbox"/> Ophthalmologic	<input type="checkbox"/> Orthopedic	<input type="checkbox"/> Pain remediation
	<input type="checkbox"/> Plastic/reconstructive	<input type="checkbox"/> Podiatry	<input type="checkbox"/> Other (specify):
How many physicians work at the facility?			
What is the average number of patients seen per week?			

Section 2: Infection Control Program and Infrastructure

I. Infection Control Program and Infrastructure		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Written infection prevention policies and procedures are available, current, and based on evidence-based guidelines (e.g., CDC/HICPAC), regulations, or standards.</p> <p><i>Note: Policies and procedures should be appropriate for the services provided by the facility and should extend beyond OSHA bloodborne pathogen training</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>B. Infection prevention policies and procedures are re-assessed at least annually or according to state or federal requirements, and updated if appropriate.</p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>C. At least one individual trained in infection prevention is employed by or regularly available (e.g., by contract) to manage the facility's infection control program.</p> <p><i>Note: Examples of training may include: Successful completion of initial and/or recertification exams developed by the Certification Board for Infection Control & Epidemiology; participation in infection control courses organized by the state or recognized professional societies (e.g., APIC, SHEA).</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>D. Facility has system for early detection and management of potentially infectious persons at initial points of patient encounter.</p> <p><i>Note: System may include taking a travel and occupational history, as appropriate, and elements described under respiratory hygiene/cough etiquette.</i></p>	<input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training and Competency		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Facility has a competency-based training program that provides job-specific training on infection prevention policies and procedures to healthcare personnel.</p> <p><i>Note: This includes those employed by outside agencies and available by contract or on a volunteer basis to the facility.</i></p> <p><i>See sections below for more specific assessment of training related to: hand hygiene, personal protective equipment (PPE), injection safety, environmental cleaning, point-of-care testing, and device reprocessing</i></p>	<input type="radio"/> Yes <input type="radio"/> No	

III. Healthcare Personnel Safety		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Facility has an exposure control plan that is tailored to the specific requirements of the facility (e.g., addresses potential hazards posed by specific services provided by the facility).</p> <p><i>Note: A model template, which includes a guide for creating an exposure control plan that meets the requirements of the OSHA Bloodborne Pathogens Standard is available at: https://www.osha.gov/Publications/osha3186.pdf</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>B. HCP for whom contact with blood or other potentially infectious material is anticipated are trained on the OSHA bloodborne pathogen standard upon hire and at least annually.</p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>C. Following an exposure event, post-exposure evaluation and follow-up, including prophylaxis as appropriate, are available at no cost to employee and are supervised by a licensed healthcare professional.</p> <p><i>Note: An exposure incident refers to a specific eye, mouth, other mucous membrane, non-intact skin, or parenteral contact with blood or other potentially infectious materials that results from the performance of an individual's duties.</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>D. Facility tracks HCP exposure events and evaluates event data and develops/implements corrective action plans to reduce incidence of such events.</p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>E. Facility follows recommendations of the Advisory Committee on Immunization Practices (ACIP) for immunization of HCP, including offering Hepatitis B and influenza vaccination.</p> <p><i>Note: Immunization of Health-Care Personnel: Recommendations of the ACIP available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/rr6007a1.htm</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>F. All HCP receive baseline tuberculosis (TB) screening prior to placement, and those with potential for ongoing exposure to TB receive periodic screening (if negative) at least annually.</p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>G. If respirators are used, the facility has a respiratory protection program that details required worksite-specific procedures and elements for required respirator use, including provision of medical clearance, training, and fit testing as appropriate.</p>	<input type="radio"/> Yes <input type="radio"/> No Not Applicable <input type="radio"/>	
<p>H. Facility has well-defined policies concerning contact of personnel with patients when personnel have potentially transmissible conditions. These policies include:</p> <ul style="list-style-type: none"> i. Work-exclusion policies that encourage reporting of illnesses and do not penalize with loss of wages, benefits, or job status. ii. Education of personnel on prompt reporting of illness to supervisor. 	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	

IV. Surveillance and Disease Reporting		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. An updated list of diseases reportable to the public health authority is readily available to all personnel.	<input type="radio"/> Yes <input type="radio"/> No	
B. Facility can demonstrate knowledge of and compliance with mandatory reporting requirements for notifiable diseases, healthcare associated infections (as appropriate), and for potential outbreaks.	<input type="radio"/> Yes <input type="radio"/> No	
C. Patients who have undergone procedures at the facility are educated regarding signs and symptoms of infection that may be associated with the procedure and instructed to notify the facility if such signs or symptoms occur.	<input type="radio"/> Yes <input type="radio"/> No	

V.a. Hand Hygiene		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. All HCP are educated regarding appropriate indications for hand hygiene: i. Upon hire, prior to provision of care ii. Annually	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	
B. HCP are required to demonstrate competency with hand hygiene following each training	<input type="radio"/> Yes <input type="radio"/> No	
C. Facility regularly audits (monitors and documents) adherence to hand hygiene.	<input type="radio"/> Yes <input type="radio"/> No	
D. Facility provides feedback from audits to personnel regarding their hand hygiene performance.	<input type="radio"/> Yes <input type="radio"/> No	
E. Hand hygiene policies promote preferential use of alcohol-based hand rub over soap and water in all clinical situations except when hands are visibly soiled (e.g., blood, body fluids) or after caring for a patient with known or suspected <i>C. difficile</i> or norovirus.	<input type="radio"/> Yes <input type="radio"/> No	

VI.a. Personal Protective Equipment (PPE)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. HCP who use PPE receive training on proper selection and use of PPE: i. Upon hire, prior to provision of care ii. Annually iii. When new equipment or protocols are introduced	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	
B. HCP are required to demonstrate competency with selection and use of PPE following each training.	<input type="radio"/> Yes <input type="radio"/> No	
C. Facility regularly audits (monitors and documents) adherence to proper PPE selection and use.	<input type="radio"/> Yes <input type="radio"/> No	
D. Facility provides feedback from audits to personnel regarding their performance with selection and use of PPE.	<input type="radio"/> Yes <input type="radio"/> No	

VII.a. Injection Safety (This element does not include assessment of pharmacy/compounding practices)

Elements to be assessed	Assessment	Notes/Areas for Improvement
A. HCP who prepare and/or administer parenteral medications receive training on safe injection practices: <ul style="list-style-type: none"> i. Upon hire, prior to being allowed to prepare and/or administer parenteral medications ii. Annually iii. When new equipment or protocols are introduced 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Yes <input type="radio"/> No</p>	
B. HCP are required to demonstrate competency with safe injection practices following each training.	<input type="radio"/> Yes <input type="radio"/> No	
C. Facility regularly audits (monitors and documents) adherence to safe injection practices.	<input type="radio"/> Yes <input type="radio"/> No	
D. Facility provides feedback from audits to personnel regarding their adherence to safe injection practices.	<input type="radio"/> Yes <input type="radio"/> No	
E. Facility has policies and procedures to track HCP access to controlled substances to prevent narcotics theft/diversion.	<input type="radio"/> Yes <input type="radio"/> No	
<p><i>Note: Policies and procedures should address: how data are reviewed, how facility would respond to unusual access patterns, how facility would assess risk to patients if tampering (alteration or substitution) is suspected or identified, and who the facility would contact if diversion is suspected or identified.</i></p>		

VIII.a. Respiratory Hygiene/Cough Etiquette

Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Facility has policies and procedures to contain respiratory secretions in persons who have signs and symptoms of a respiratory infection, beginning at point of entry to the facility and continuing through the duration of the visit. Policies include: <ul style="list-style-type: none"> i. Offering facemasks to coughing patients and other symptomatic persons upon entry to the facility, at a minimum, during periods of increased respiratory infection activity in the community. ii. Providing space in waiting rooms and encouraging persons with symptoms of respiratory infections to sit as far away from others as possible. <p><i>Note: If available, facilities may wish to place patients with symptoms of a respiratory infection in a separate area while waiting for care.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Yes <input type="radio"/> No</p>	
B. Facility educates HCP on the importance of infection prevention measures to contain respiratory secretions to prevent the spread of respiratory pathogens.	<input type="radio"/> Yes <input type="radio"/> No	

IX.a. Point-of-Care Testing (e.g., blood glucose meters, INR monitor)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. HCP who perform point-of-care testing receive training on recommended practices: i. Upon hire, prior to being allowed to perform point-of-care testing ii. Annually iii. When new equipment or protocols are introduced	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
B. HCP are required to demonstrate competency with recommended practices for point-of-care testing following each training.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
C. Facility regularly audits (monitors and documents) adherence to recommended practices during point-of-care testing.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
D. Facility provides feedback from audits to personnel regarding their adherence to recommended practices.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

X.a. Environmental Cleaning		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Facility has written policies and procedures for routine cleaning and disinfection of environmental surfaces, including identification of responsible personnel.	<input type="radio"/> Yes <input type="radio"/> No	
B. Personnel who clean and disinfect patient care areas (e.g., environmental services, technicians, nurses) receive training on cleaning procedures i. Upon hire, prior to being allowed to perform environmental cleaning ii. Annually iii. When new equipment or protocols are introduced <i>Note: If environmental cleaning is performed by contract personnel, facility should verify this is provided by contracting company.</i>	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	
C. HCP are required to demonstrate competency with environmental cleaning procedures following each training.	<input type="radio"/> Yes <input type="radio"/> No	
D. Facility regularly audits (monitors and documents) adherence to cleaning and disinfection procedures, including using products in accordance with manufacturer's instructions (e.g., dilution, storage, shelf-life, contact time).	<input type="radio"/> Yes <input type="radio"/> No	
E. Facility provides feedback from audits to personnel regarding their adherence to cleaning and disinfection procedures.	<input type="radio"/> Yes <input type="radio"/> No	
F. Facility has a policy/procedure for decontamination of spills of blood or other body fluids.	<input type="radio"/> Yes <input type="radio"/> No	

X.a. Environmental Cleaning, continued

Operating Room

Elements to be assessed	Assessment	Notes/Areas for Improvement
G. Operating rooms are terminally cleaned after last procedure of the day.	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Not applicable	
H. Facility regularly audits (monitors and documents) adherence to recommended infection control practices for surgical infection prevention including: <ul style="list-style-type: none"> i. Adherence to preoperative surgical scrub and hand hygiene ii. Appropriate use of surgical attire and drapes iii. Adherence to aseptic technique and sterile field iv. Proper ventilation requirements in surgical suites v. Minimization of traffic in the operating room vi. Adherence to cleaning and disinfection of environmental surfaces 	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Not applicable	
I. Facility provides feedback from audits to personnel regarding their adherence to surgical infection prevention practices.	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Not applicable	

XI.a. Device Reprocessing

The following basic information allows for a general assessment of policies and procedures related to reprocessing of reusable medical devices. Outpatient facilities that are performing on-site sterilization or high-level disinfection of reusable medical devices should refer to the more detailed checklists in separate sections of this document devoted to those issues.

Categories of Medical Devices:

- **Critical items** (e.g., surgical instruments) are objects that enter sterile tissue or the vascular system and must be sterile prior to use (see Sterilization Section).
- **Semi-critical items** (e.g., endoscopes for upper endoscopy and colonoscopy, vaginal probes) are objects that contact mucous membranes or non-intact skin and require, at a minimum, high-level disinfection prior to reuse (see High-level Disinfection Section).
- **Non-critical items** (e.g., blood pressure cuffs) are objects that may come in contact with intact skin but not mucous membranes and should undergo cleaning and low- or intermediate-level disinfection depending on the nature and degree of contamination.

Single-use devices (SUDs) are labeled by the manufacturer for a single use and do not have reprocessing instructions. They may *not* be reprocessed for reuse except by entities which have complied with FDA regulatory requirements and have received FDA clearance to reprocess specific SUDs.

Note: Cleaning must always be performed prior to sterilization and disinfection

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Facility has policies and procedures to ensure that reusable medical devices are cleaned and reprocessed appropriately prior to use on another patient.</p> <p><i>Note: This includes clear delineation of responsibility among HCP for cleaning and disinfection of equipment including, non-critical equipment, mobile devices, and other electronics (e.g., point-of-care devices) that might not be reprocessed in a centralized reprocessing area.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>B. The individual(s) in charge of infection prevention at the facility is consulted whenever new devices or products will be purchased or introduced to ensure implementation of appropriate reprocessing policies and procedures.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>C. HCP responsible for reprocessing reusable medical devices receive hands-on training on proper selection and use of PPE and recommended steps for reprocessing assigned devices:</p> <ul style="list-style-type: none"> i. Upon hire, prior to being allowed to reprocess devices ii. Annually iii. When new devices are introduced or policies/procedures change. <p><i>Note: If device reprocessing is performed by contract personnel, facility should verify this is provided by contracting company.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>D. HCP are required to demonstrate competency with reprocessing procedures (i.e., correct technique is observed by trainer) following each training.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	

XI.a. Device Reprocessing, continued

Elements to be assessed	Assessment	Notes/Areas for Improvement
E. Facility regularly audits (monitors and documents) adherence to reprocessing procedures.	<input type="radio"/> Yes <input type="radio"/> No	
F. Facility provides feedback from audits to personnel regarding their adherence to reprocessing procedures.	<input type="radio"/> Yes <input type="radio"/> No	
G. Facility has protocols to ensure that HCP can readily identify devices that have been properly reprocessed and are ready for patient use (e.g., tagging system, storage in designated area).	<input type="radio"/> Yes <input type="radio"/> No	
H. Facility has policies and procedures outlining facility response (i.e., risk assessment and recall of device) in the event of a reprocessing error or failure.	<input type="radio"/> Yes <input type="radio"/> No	
I. Routine maintenance for reprocessing equipment (e.g., automated endoscope reprocessors, steam autoclave) is performed by qualified personnel in accordance with manufacturer instructions; confirm maintenance records are available.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

Section 3: Direct Observation of Facility Practices

Certain infection control lapses (e.g., reuse of syringes on more than one patient or to access a medication container that is used for subsequent patients; reuse of lancets) have resulted in bloodborne pathogen transmission and should be halted immediately. Identification of such lapses warrants appropriate notification and testing of potentially affected patients.

If an element is unable to be observed during an assessment (e.g., no patients received point-of-care testing during the visit), assess the element by interviewing appropriate personnel about facility practices. Notation should also be made in the notes section that the element was not able to be directly observed.

V.b. Hand hygiene		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Supplies necessary for adherence to hand hygiene (e.g., soap, water, paper towels, alcohol-based hand rub) are readily accessible to HCP in patient care areas.	<input type="radio"/> Yes <input type="radio"/> No	
Hand hygiene is performed correctly:		
B. Before contact with the patient	<input type="radio"/> Yes <input type="radio"/> No	
C. Before performing an aseptic task (e.g., insertion of IV or preparing an injection)	<input type="radio"/> Yes <input type="radio"/> No	
D. After contact with the patient	<input type="radio"/> Yes <input type="radio"/> No	
E. After contact with objects in the immediate vicinity of the patient	<input type="radio"/> Yes <input type="radio"/> No	
F. After contact with blood, body fluids or contaminated surfaces	<input type="radio"/> Yes <input type="radio"/> No	
G. After removing gloves	<input type="radio"/> Yes <input type="radio"/> No	
H. When moving from a contaminated-body site to a clean-body site during patient care	<input type="radio"/> Yes <input type="radio"/> No	

VI.b. Personal Protective Equipment (PPE)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Sufficient and appropriate PPE is available and readily accessible to HCP.	<input type="radio"/> Yes <input type="radio"/> No	
PPE is used correctly:		
B. PPE, other than respirator, is removed and discarded prior to leaving the patient's room or care area. If a respirator is used, it is removed and discarded (or reprocessed if reusable) <u>after</u> leaving the patient room or care area and closing the door.	<input type="radio"/> Yes <input type="radio"/> No	
C. Hand hygiene is performed immediately after removal of PPE.	<input type="radio"/> Yes <input type="radio"/> No	

VI.b. Personal Protective Equipment (PPE), continued		
Elements to be assessed	Assessment	Notes/Areas for Improvement
D. Gloves		
i. HCP wear gloves for potential contact with blood, body fluids, mucous membranes, non-intact skin, or contaminated equipment.	<input type="radio"/> Yes <input type="radio"/> No	
ii. HCP <u>do not</u> wear the same pair of gloves for the care of more than one patient.	<input type="radio"/> Yes <input type="radio"/> No	
iii. HCP <u>do not</u> wash gloves for the purpose of reuse.	<input type="radio"/> Yes <input type="radio"/> No	
E. Gowns		
i. HCP wear gowns to protect skin and clothing during procedures or activities where contact with blood or body fluids is anticipated.	<input type="radio"/> Yes <input type="radio"/> No Not Applicable <input type="radio"/>	
ii. HCP <u>do not</u> wear the same gown for the care of more than one patient.	<input type="radio"/> Yes <input type="radio"/> No Not Applicable <input type="radio"/>	
F. Facial protection		
i. HCP wear mouth, nose, and eye protection during procedures that are likely to generate splashes or sprays of blood or other body fluids.	<input type="radio"/> Yes <input type="radio"/> No Not Applicable <input type="radio"/>	

VII.b. Injection safety (This element does not include assessment of pharmacy/compounding practices)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Injections are prepared using aseptic technique in a clean area free from contamination or contact with blood, body fluids or contaminated equipment.	<input type="radio"/> Yes <input type="radio"/> No	
B. Needles and syringes are used for only one patient (this includes manufactured prefilled syringes and cartridge devices such as insulin pens).	<input type="radio"/> Yes <input type="radio"/> No	
C. The rubber septum on a medication vial is disinfected with alcohol prior to piercing.	<input type="radio"/> Yes <input type="radio"/> No	
D. Medication containers are entered with a new needle and a new syringe, even when obtaining additional doses for the same patient.	<input type="radio"/> Yes <input type="radio"/> No	
E. Single dose (single-use) medication vials, ampules, and bags or bottles of intravenous solution are used for only one patient.	<input type="radio"/> Yes <input type="radio"/> No	
F. Medication administration tubing and connectors are used for only one patient.	<input type="radio"/> Yes <input type="radio"/> No	
G. Multi-dose vials are dated by HCP when they are first opened and discarded within 28 days unless the manufacturer specifies a different (shorter or longer) date for that opened vial. <i>Note: This is different from the expiration date printed on the vial.</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/> (Facility does not use multi-dose vials or discards them after single patient use)	

VII.b. Injection safety (This element does not include assessment of pharmacy/compounding practices), continued

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>H. Multi-dose vials to be used for more than one patient are kept in a centralized medication area and <u>do not</u> enter the immediate patient treatment area (e.g., operating room, patient room/cubicle).</p> <p><i>Note: If multi-dose vials enter the immediate patient treatment area they should be dedicated for single-patient use and discarded immediately after use.</i></p>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/> (Facility does not use multi-dose vials or discards them after single patient use)	
I. All sharps are disposed of in a puncture-resistant sharps container.	<input type="radio"/> Yes <input type="radio"/> No	
J. Filled sharps containers are disposed of in accordance with state regulated medical waste rules.	<input type="radio"/> Yes <input type="radio"/> No	
K. All controlled substances (e.g., Schedule II, III, IV, V drugs) are kept locked within a secure area.	<input type="radio"/> Yes <input type="radio"/> No	
L. HCP wear a facemask (e.g., surgical mask) when placing a catheter or injecting material into the epidural or subdural space (e.g., during myelogram, epidural or spinal anesthesia).	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/> (Facility does not perform spinal injection procedures)	

VIII.b. Respiratory Hygiene/Cough Etiquette

Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Facility:		
i. Posts signs at entrances with instructions to patients with symptoms of respiratory infection to: <ul style="list-style-type: none"> a. Inform HCP of symptoms of a respiratory infection when they first register for care, and b. Practice Respiratory Hygiene/Cough Etiquette (cover their mouths/noses when coughing or sneezing, use and dispose of tissues, and perform hand hygiene after hands have been covered with respiratory secretions). 	<input type="radio"/> Yes <input type="radio"/> No	
ii. Provides tissues and no-touch receptacles for disposal of tissues.	<input type="radio"/> Yes <input type="radio"/> No	
iii. Provides resources for performing hand hygiene in or near waiting areas.	<input type="radio"/> Yes <input type="radio"/> No	

IX.b. Point-of-Care Testing (e.g., blood glucose meters, INR monitor)

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. New single-use, auto-disabling lancing device is used for each patient.</p> <p><i>Note: Lancet holder devices are not suitable for multi-patient use.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/></p>	
<p>B. If used for more than one patient, the point-of-care testing meter is cleaned and disinfected after every use according to manufacturer’s instructions.</p> <p><i>Note: If the manufacturer does not provide instructions for cleaning and disinfection, then the testing meter should not be used for >1 patient.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/></p>	

X.b. Environmental Cleaning

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Supplies necessary for appropriate cleaning and disinfection procedures (e.g., EPA-registered disinfectants) are available.</p> <p><i>Note: If environmental services are performed by contract personnel, facility should verify that appropriate EPA-registered products are provided by contracting company</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>B. High-touch surfaces in rooms where surgical or other invasive procedures (e.g., endoscopy, spinal injections) are performed are cleaned and then disinfected with an EPA-registered disinfectant after each procedure.</p>	<p><input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/></p>	
<p>C. Cleaners and disinfectants are used in accordance with manufacturer’s instructions (e.g., dilution, storage, shelf-life, contact time).</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>D. HCP engaged in environmental cleaning wear appropriate PPE to prevent exposure to infectious agents or chemicals (PPE can include gloves, gowns, masks, and eye protection).</p> <p><i>Note: The exact type of correct PPE depends on infectious or chemical agent and anticipated type of exposure.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	

XI.b. Device Reprocessing		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Policies, procedures, and manufacturer reprocessing instructions for reusable medical devices used in the facility are available in the reprocessing area(s).	<input type="radio"/> Yes <input type="radio"/> No	
B. Reusable medical devices are cleaned, reprocessed (disinfection or sterilization) and maintained according to the manufacturer instructions. <i>Note: If the manufacturer does not provide such instructions, the device may not be suitable for multi-patient use.</i>	<input type="radio"/> Yes <input type="radio"/> No	
C. Single-use devices are discarded after use and not used for more than one patient. <i>Note: If the facility elects to reuse single-use devices, these devices must be reprocessed prior to reuse by a third-party reprocessor that it is registered with the FDA as a third-party reprocessor and cleared by the FDA to reprocess the specific device in question. The facility should have documentation from the third party reprocessor confirming this is the case.</i>	<input type="radio"/> Yes <input type="radio"/> No	
D. Reprocessing area: i. Adequate space is allotted for reprocessing activities. ii. A workflow pattern is followed such that devices clearly flow from high contamination areas to clean/sterile areas (i.e., there is clear separation between soiled and clean workspaces).	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	
E. Adequate time for reprocessing is allowed to ensure adherence to all steps recommended by the device manufacturer, including drying and proper storage. <i>Note: Facilities should have an adequate supply of instruments for the volume of procedures performed and should schedule procedures to allow sufficient time for all reprocessing steps.</i>	<input type="radio"/> Yes <input type="radio"/> No	
F. HCP engaged in device reprocessing wear appropriate PPE to prevent exposure to infectious agents or chemicals (PPE can include gloves, gowns, masks, and eye protection). <i>Note: The exact type of correct PPE depends on infectious or chemical agent and anticipated type of exposure.</i>	<input type="radio"/> Yes <input type="radio"/> No	
G. Medical devices are stored in a manner to protect from damage and contamination.	<input type="radio"/> Yes <input type="radio"/> No	

XII. Sterilization of Reusable Devices

Note: If all device sterilization is performed off-site, skip to items M-O below.

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Devices are thoroughly cleaned according to manufacturer instructions and visually inspected for residual soil prior to sterilization.</p> <p><i>Note: Cleaning may be manual (i.e., using friction) and/or mechanical (e.g., with ultrasonic cleaners, washer-disinfector, washer-sterilizers).</i></p> <p><i>Ensure appropriately sized cleaning brushes are selected for cleaning device channels and lumens.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>B. Cleaning is performed as soon as practical after use (e.g., at the point of use) to prevent soiled materials from becoming dried onto devices.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>C. Enzymatic cleaner or detergent is used for cleaning and discarded according to manufacturer's instructions (typically after each use)</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>D. Cleaning brushes are disposable or, if reusable, cleaned and high-level disinfected or sterilized (per manufacturer's instructions) after use.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>E. After cleaning, instruments are appropriately wrapped/packaged for sterilization (e.g., package system selected is compatible with the sterilization process being performed, items are placed correctly into the basket, shelf or cart of the sterilizer so as not to impede the penetration of the sterilant, hinged instruments are open, instruments are disassembled if indicated by the manufacturer).</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>F. A chemical indicator (process indicator) is placed correctly in the instrument packs in every load.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>G. A biological indicator, intended specifically for the type and cycle parameters of the sterilizer, is used at least weekly for each sterilizer and with every load containing implantable items.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>H. For dynamic air removal-type sterilizers (e.g., prevacuum steam sterilizer), an air removal test (Bowie-Dick test) is performed in an empty dynamic-air removal sterilizer each day the sterilizer is used to verify efficacy of air removal.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>I. Sterile packs are labeled with a load number that indicates the sterilizer used, the cycle or load number, the date of sterilization, and, if applicable, the expiration date.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>J. Sterilization logs are current and include results from each load.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>K. Immediate-use steam sterilization, if performed, is only done in circumstances in which routine sterilization procedures cannot be performed.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	

XII. Sterilization of Reusable Devices, continued		
Note: If all device sterilization is performed off-site, skip to items M-O below.		
Elements to be assessed	Assessment	Notes/Areas for Improvement
L. Instruments that undergo immediate-use steam sterilization are used immediately and not stored.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
M. After sterilization, medical devices are stored so that sterility is not compromised.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
N. Sterile packages are inspected for integrity and compromised packages are reprocessed prior to use.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
O. The facility has a process to perform initial cleaning of devices (to prevent soiled materials from becoming dried onto devices) prior to transport to the off-site reprocessing facility.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

XIII. High-Level Disinfection of Reusable Devices		
Note: If all high-level disinfection is performed off-site, skip to items L-N below.		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Flexible endoscopes are inspected for damage and leak tested as part of each reprocessing cycle. Any device that fails the leak test is removed from clinical use and repaired.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
B. Devices are thoroughly cleaned according to manufacturer instructions and visually inspected for residual soil prior to high-level disinfection. <i>Note: Cleaning may be manual (i.e., using friction) and/or mechanical (e.g., with ultrasonic cleaners, washer-disinfector, washer-sterilizers).</i> <i>Ensure appropriately sized cleaning brushes are selected for cleaning device channels and lumens.</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
C. Cleaning is performed as soon as practical after use (e.g., at the point of use) to prevent soiled materials from becoming dried onto instruments.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
D. Enzymatic cleaner or detergent is used and discarded according to manufacturer instructions (typically after each use).	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
E. Cleaning brushes are disposable or, if reusable, cleaned and high-level disinfected or sterilized (per manufacturer instructions) after use.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
F. For chemicals used in high-level disinfection, manufacturer instructions are followed for: <ul style="list-style-type: none"> i. Preparation ii. Testing for appropriate concentration iii. Replacement (i.e., upon expiration or loss of efficacy) 	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

XIII. High-Level Disinfection of Reusable Devices, continued

Note: If all high-level disinfection is performed off-site, skip to items L-N below.

Elements to be assessed	Assessment	Notes/Areas for Improvement
G. If automated reprocessing equipment is used, proper connectors are used to assure that channels and lumens are appropriately disinfected.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
H. Devices are disinfected for the appropriate length of time as specified by manufacturer instructions.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
I. Devices are disinfected at the appropriate temperature as specified by manufacturer instructions.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
J. After high-level disinfection, devices are rinsed with sterile water, filtered water, or tap water followed by a rinse with 70% - 90% ethyl or isopropyl alcohol. <i>Note: There is no recommendation to use sterile or filtered water rather than tap water for rinsing semi-critical equipment that contact the mucous membranes of the rectum or vagina</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
K. Devices are dried thoroughly prior to reuse. <i>Note: For lumened instruments (e.g., endoscopes) this includes flushing all channels with alcohol and forcing air through channels.</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
L. After high-level disinfection, devices are stored in a manner to protect from damage or contamination. <i>Note: Endoscopes should be hung in a vertical position.</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
M. Facility maintains a log for each endoscopy procedure which includes: patient's name and medical record number (if available), procedure, date, endoscopist, system used to reprocess the endoscope (if more than one system could be used in the reprocessing area), and serial number or other identifier of the endoscope used.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
N. The facility has a process to perform initial cleaning of devices (to prevent soiled materials from becoming dried onto devices) prior to transport to the off-site reprocessing facility.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

Section 4: Infection Control Guidelines and Other Resources

- **General Infection Prevention**

- CDC/HICPAC Guidelines and recommendations: http://www.cdc.gov/HAI/prevent/prevent_pubs.html

- **Healthcare Personnel Safety**

- Guideline for Infection Control in Healthcare Personnel: <http://www.cdc.gov/hicpac/pdf/InfectControl98.pdf>
- Immunization of HealthCare Personnel: <http://www.cdc.gov/vaccines/spec-grps/hcw.htm>
- Occupational Safety & Health Administration (OSHA) Bloodborne Pathogens and Needlestick Prevention Standard: <http://www.osha.gov/SLTC/bloodbornepathogens/index.html>
- OSHA Respiratory Protection Standard: [https://www.osha.gov/pls/oshaweb/owadisp.show_document?p_id=12716&p_table=STANDARD S](https://www.osha.gov/pls/oshaweb/owadisp.show_document?p_id=12716&p_table=STANDARD_S)
- OSHA Respirator Fit Testing: https://www.osha.gov/video/respiratory_protection/fittesting_transcript.html

- **Hand Hygiene**

- Guideline for Hand Hygiene in Healthcare Settings: <http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf>
- Hand Hygiene in Healthcare Settings: <http://www.cdc.gov/handhygiene/>

Examples of tools that can be used to conduct a formal audit of hand hygiene practices:

- http://www.jointcommission.org/assets/1/18/hh_monograph.pdf
- <http://comepepi.cs.uiowa.edu/index.php/Research/IScrub>

- **Personal Protective Equipment**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
- Guidance for the Selection and Use of Personal Protective Equipment in Healthcare Settings: <http://www.cdc.gov/HAI/prevent/ppe.html>

- **Injection Safety**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
- CDC Injection Safety Web Materials: <http://www.cdc.gov/injectionsafety/>

- CDC training video and related Safe Injection Practices Campaign materials: <http://www.oneandonlycampaign.org/>

- **Respiratory Hygiene/Cough Etiquette**
 - 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
 - Recommendations for preventing the spread of influenza: <http://www.cdc.gov/flu/professionals/infectioncontrol/>

- **Environmental Cleaning**
 - Guidelines for Environmental Infection Control in Healthcare Facilities: http://www.cdc.gov/hicpac/pdf/guidelines/eic_in_HCF_03.pdf
 - Options for Evaluating Environmental Infection Control: <http://www.cdc.gov/HAI/toolkits/Evaluating-Environmental-Cleaning.html>

- **Equipment Reprocessing**
 - Guideline for Disinfection and Sterilization in Healthcare Facilities: http://www.cdc.gov/hicpac/pdf/guidelines/Disinfection_Nov_2008.pdf
 - FDA regulations on reprocessing of single-use devices: <http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm071434>

- **Point-of-Care Testing**
 - Infection Prevention during Blood Glucose Monitoring and Insulin Administration: <http://www.cdc.gov/injectionsafety/blood-glucose-monitoring.html>
 - Frequently Asked Questions (FAQs) regarding Assisted Blood Glucose Monitoring and Insulin Administration: http://www.cdc.gov/injectionsafety/providers/blood-glucose-monitoring_faqs.html

- **Resources to assist with evaluation and response to breaches in infection control**
 - Patel PR, Srinivasan A, Perz JF. Developing a broader approach to management of infection control breaches in health care settings. Am J Infect Control. 2008 Dec;36(10):685-90
 - Steps for Evaluating an Infection Control Breach: http://www.cdc.gov/hai/outbreaks/steps_for_eval_IC_breach.html
 - Patient Notification Toolkit: <http://www.cdc.gov/injectionsafety/pntoolkit/index.html>

Infection Control Assessment Tool for Acute Care Hospitals

This tool is intended to assist in the assessment of infection control programs and practices in acute care hospitals. If feasible, direct observations of infection control practices are encouraged. To facilitate the assessment, health departments are encouraged to share this tool with hospitals in advance of their visit.

Overview

Section 1: Facility Demographics

Section 2: Infection Control Program and Infrastructure

Section 3: Direct Observation of Facility Practices (optional)

Section 4: Infection Control Guidelines and Other Resources

Infection Control Domains for Gap Assessment

- I. Infection Control Program and Infrastructure
- II. Infection Control Training, Competency, and Implementation of Policies and Practices
 - A. Hand Hygiene
 - B. Personal Protective Equipment (PPE)
 - C. Prevention of Catheter-associated Urinary Tract Infection (CAUTI)
 - D. Prevention of Central Line-associated Bloodstream Infection (CLABSI)
 - E. Prevention of Ventilator-associated Event (VAE)
 - F. Injection Safety
 - G. Prevention of Surgical Site Infection
 - H. Prevention of *Clostridium difficile* Infection (CDI)
 - I. Environmental Cleaning
 - J. Device Reprocessing
- III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs)



Section 1. Facility Demographics	
Facility Name (for health department use only)	
NHSN Facility Organization ID (for health department use only)	
State-assigned Unique ID	
Date of Assessment	
Type of Assessment	<input type="checkbox"/> On-site <input type="checkbox"/> Other (specify):
Rationale for Assessment (Select all that apply)	<input type="checkbox"/> Outbreak <input type="checkbox"/> Input from accrediting organization or state survey agency <input type="checkbox"/> NHSN data If YES, specify: <input type="checkbox"/> CAUTI <input type="checkbox"/> CLABSI <input type="checkbox"/> SSI <input type="checkbox"/> CDI <input type="checkbox"/> Other (specify:) <input type="checkbox"/> Collaborative (specify partner[s]):) <input type="checkbox"/> Other (specify):
Facility type	<input type="checkbox"/> Acute Care Hospital <input type="checkbox"/> Critical Access Hospital <input type="checkbox"/> Long-term Acute Care Hospital (LTACH) <input type="checkbox"/> Other (specify):
Number of Licensed Beds	
Number of Infection Preventionist Full-Time Equivalents	

Section 2: Infection Control Program and Infrastructure

I. Infection Control Program and Infrastructure		
Elements to be assessed	Assessment	Notes/Areas for Improvement
1. Hospital provides fiscal and human resource support for maintaining the infection prevention and control program.	<input type="radio"/> Yes <input type="radio"/> No	
2. The person(s) charged with directing the infection prevention and control program at the hospital is/are qualified and trained in infection control. Verify qualifications, which should include: (Check all that apply) <input type="checkbox"/> Successful completion of initial and recertification exams developed by the Certification Board for Infection Control & Epidemiology (CIC) AND/OR <input type="checkbox"/> Participation in infection control courses organized by recognized professional societies (e.g., APIC, SHEA)	<input type="radio"/> Yes <input type="radio"/> No	
3. Infection prevention and control program performs an annual facility infection risk assessment that evaluates and prioritizes potential risks for infections, contamination, and exposures and the program's preparedness to eliminate or mitigate such risks. <i>Note: Example of Facility Infection Risk Assessment Report and Plan is available in Section 4.</i>	<input type="radio"/> Yes <input type="radio"/> No	
4. Written infection control policies and procedures are available, current, and based on evidence-based guidelines (e.g., CDC/HICPAC), regulations, or standards. Verify the following: a. Respondent can describe the process for reviewing and updating policies (e.g., policies are dated and reviewed annually and when new guidelines are issued)	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No	
5. Infection prevention and control program provides infection prevention education to patients, family members, and other caregivers. Verify the following: a. Respondent can describe how this education is provided (e.g., information included in the admission or discharge packet, videos, signage, in-person training)	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Hand Hygiene		
<p>1. Hospital has a competency-based training program for hand hygiene.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all healthcare personnel, including all ancillary personnel not directly involved in patient care but potentially exposed to infectious agents (e.g., food tray handlers, housekeeping, volunteer personnel). b. Training is provided upon hire, prior to provision of care at this hospital. c. Training is provided at least annually. d. Personnel are required to demonstrate competency with hand hygiene following each training. e. Hospital maintains current documentation of hand hygiene competency for all personnel. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No 	
<p>2. Hospital regularly audits (monitors and documents) adherence to hand hygiene.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>3. Hospital provides feedback from audits to personnel regarding their hand hygiene performance.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	
<p>4. Supplies necessary for adherence to hand hygiene (e.g., soap, water, paper towels, alcohol-based hand rub) are readily accessible in patient care areas.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>5. Hand hygiene policies promote preferential use of alcohol-based hand rub over soap and water except when hands are visibly soiled (e.g., blood, body fluids) or after caring for a patient with known or suspected <i>C. difficile</i> or norovirus.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
B. Personal Protective Equipment (PPE)		
<p>1. Hospital has a competency-based training program for use of personal protective equipment (PPE).</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who use PPE. b. Training is provided upon hire, prior to provision of care at this hospital. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Training includes 1) appropriate indications for specific PPE components, 2) proper donning, doffing, adjustment, and wear of PPE, and 3) proper care, maintenance, useful life, and disposal of PPE. f. Personnel are required to demonstrate competency with selection and use of PPE (i.e., correct technique is observed by trainer) following each training. g. Hospital maintains current documentation of PPE competency for all personnel who use PPE. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No g. <input type="radio"/> Yes <input type="radio"/> No 	
<p>2. Hospital regularly audits (monitors and documents) adherence to proper PPE selection and use, including donning and doffing.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>3. Hospital provides feedback to personnel regarding their performance with selection and use of PPE.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	
<p>4. Supplies necessary for adherence to personal protective equipment recommendations specified under Standard and Transmission-based Precautions (e.g., gloves, gowns, mouth, eye, nose, and face protection) are available and located near point of use.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>5. The hospital's respiratory protection program provides annual respiratory fit testing for all personnel who are anticipated to require respiratory protection.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Hospital maintains supplies of respiratory protection devices (e.g., Powered air purifying respirator) to be used by personnel who cannot be fitted. b. Healthcare personnel are educated about factors that may compromise proper fit and function of respiratory protection devices (e.g., weight gain/loss, facial hair). 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
C. Prevention of Catheter-associated Urinary Tract Infection (CAUTI)		
1. Hospital has physician and/or nurse champions for CAUTI prevention activities.	<input type="radio"/> Yes <input type="radio"/> No	
2. Hospital has a competency-based training program for insertion of urinary catheters. Verify the following: <ol style="list-style-type: none"> a. Training is provided to all personnel who are given responsibility for insertion of urinary catheters. <i>Personnel</i> may include, but are not limited to, nurses, nursing assistants, medical assistants, technicians, and physicians. b. Training is provided upon hire, prior to being allowed to perform urinary catheter insertion. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with insertion (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with urinary catheter insertion for all personnel who insert urinary catheters. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No	
3. Hospital regularly audits (monitors and documents) adherence to recommended practices for insertion of urinary catheters. Verify the following: <ol style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No	
4. Hospital provides feedback from audits to personnel regarding their performance for insertion of urinary catheters. Verify the following: <ol style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
C. Prevention of Catheter-associated Urinary Tract Infection (CAUTI), continued		
<p>5. Hospital has a competency-based training program for <u>maintenance</u> of urinary catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who are given responsibility for urinary catheter maintenance (e.g., perineal care, emptying the drainage bag aseptically, maintaining the closed drainage system, maintaining unobstructed urine flow). Personnel may include, but are not limited to, nurses, nursing assistants, medical assistants, technicians, and transport personnel. b. Training is provided upon hire, prior to being allowed to perform urinary catheter maintenance. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with catheter maintenance (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with urinary catheter maintenance for all personnel who maintain urinary catheters. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p> <p>d. <input type="radio"/> Yes <input type="radio"/> No</p> <p>e. <input type="radio"/> Yes <input type="radio"/> No</p> <p>f. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>6. Hospital regularly audits (monitors and documents) adherence to recommended practices for <u>maintenance</u> of urinary catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>7. Hospital provides feedback from audits to personnel regarding their performance for <u>maintenance</u> of urinary catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>8. Patients with urinary catheters are assessed, at least daily, for continued need for the catheter.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods used to trigger the daily assessments (e.g., patient safety checklist, daily rounds, nurse directed protocol, reminders or stop orders). b. Hospital routinely audits adherence to daily assessment of urinary catheter need. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
C. Prevention of Catheter-associated Urinary Tract Infection (CAUTI), continued		
<p>9. Hospital monitors CAUTI data and uses it to direct prevention activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent is familiar with National Healthcare Safety Network (NHSN) CAUTI data. b. Respondent can describe how CAUTI data are used to direct prevention activities. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>10. Hospital provides feedback of CAUTI data to frontline personnel.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
D. Prevention of Central line-associated Bloodstream Infection (CLABSI)		
1. Hospital has physician and/or nurse champions for CLABSI prevention activities.	<input type="radio"/> Yes <input type="radio"/> No	
2. Hospital has a competency-based training program for insertion of central venous catheters. Verify the following: <ul style="list-style-type: none"> a. Training is provided to all personnel who are given responsibility for insertion of central venous catheters. Personnel may include, but are not limited to, physicians, physician assistants, and members of line insertion teams. b. Training is provided upon hire, prior to being allowed to perform central venous catheter insertion. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with insertion (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with central venous catheter insertion for all personnel who insert central venous catheters. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No	
3. Hospital regularly audits (monitors and documents) adherence to recommended practices for insertion of central venous catheters. Verify the following: <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No	
4. Hospital provides feedback from audits to personnel regarding their performance for insertion of central venous catheters. Verify the following: <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
D. Prevention of Central line-associated Bloodstream Infection (CLABSI), continued		
<p>5. Hospital has a competency-based training program for maintenance of central venous catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who maintain central venous catheters (e.g., scrub the hub, accessing the catheter, dressing changes). Personnel may include, but are not limited to, nurses, nursing assistants, physicians, and physician assistants. b. Training is provided upon hire, prior to being allowed to perform central venous catheter maintenance. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with maintenance (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with central venous catheter maintenance for all personnel who maintain central venous catheters. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p> <p>d. <input type="radio"/> Yes <input type="radio"/> No</p> <p>e. <input type="radio"/> Yes <input type="radio"/> No</p> <p>f. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>6. Hospital regularly audits (monitors and documents) adherence to recommended practices for maintenance of central venous catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>7. Hospital provides feedback from audits to personnel regarding their performance for maintenance of central venous catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>8. Patients with central venous catheters are assessed, at least daily, for continued need for the catheter.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods used to trigger the daily assessments (e.g., patient safety checklist, daily rounds, reminders). b. Hospital routinely audits adherence to daily assessment of central venous catheter need. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
D. Prevention of Central line-associated Bloodstream Infection (CLABSI), continued		
<p>9. Hospital monitors CLABSI data and uses it to direct prevention activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent is familiar with National Healthcare Safety network (NHSN) CLABSI data. b. Respondent can describe how CLABSI data are used to direct prevention activities. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>10. Hospital provides feedback of CLABSI data to frontline personnel.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
E. Prevention of Ventilator-associated Event (VAE)		
1. Hospital has physician and/or nurse champions for VAE prevention activities.	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Check if facility does not provide care to ventilated patients and move to item F. Injection Safety.	
2. Hospital has a competency-based training program addressing prevention of VAEs. Verify the following: <ul style="list-style-type: none"> a. Training is provided to all personnel who provide respiratory therapy for ventilated patients (e.g., suctioning, administration of aerosolized medications). Personnel may include, but are not limited to, respiratory therapists and nurses. b. Training is provided upon hire, prior to being allowed to provide respiratory therapy for ventilated patients. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with respiratory therapy practices (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with respiratory practices for all personnel who provide respiratory therapy for ventilated patients. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No	
3. Hospital regularly audits (monitors and documents) adherence to recommended practices for management of ventilated patients (e.g., suctioning, administration of aerosolized medications). Verify the following: <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No	
4. Hospital provides feedback from audits to personnel regarding their performance for management of ventilated patients. Verify the following: <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
E. Prevention of Ventilator-associated Event (VAE), continued		
<p>5. Patients requiring invasive ventilation are assessed, at least daily, for continued need for the ventilator.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods used to trigger the daily assessments (e.g., patient safety checklist, daily rounds, reminders) b. Hospital routinely audits adherence to daily assessment of ventilator need. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>6. Hospital has a program that includes daily spontaneous breathing trials and lightening of sedation in eligible patient.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>7. Hospital has an oral-hygiene program.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>8. Hospital monitors VAE data and uses it to direct prevention activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how VAE data are used to direct prevention activities. <p>If the hospital reports VAE data to NHSN, verify the following:</p> <ul style="list-style-type: none"> b. Respondent is familiar with NHSN VAE data. <p>If the hospital does not report VAE data to NHSN, verify the following:</p> <ul style="list-style-type: none"> c. Respondent can describe how VAE data are collected. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>Not Applicable <input type="radio"/></p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p> <p>Not Applicable <input type="radio"/></p>	
<p>9. Hospital provides feedback of VAE data to frontline personnel.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
F. Injection Safety (This element does not include assessment of pharmacy practices)		
<p>1. Hospital has a competency-based training program for preparation and administration of parenteral medications (e.g., SQ, IM, IV) outside of the pharmacy.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who prepare and/or administer injections and parenteral infusions. b. Training is provided upon hire, prior to being allowed to prepare and/or administer injections and parenteral infusions. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with preparation and/or administration of injections and parenteral infusions following each training. f. Hospital maintains current documentation of competency with preparation and/or administration procedures for all personnel who prepare and/or administer injections and parenteral infusions. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No 	
<p>2. Hospital regularly audits (monitors and documents) adherence to safe injection practices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>3. Hospital provides feedback from audits to personnel regarding their adherence to safe injection practices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	
<p>4. Hospital has a drug diversion prevention program that includes consultation with the IP program when drug tampering (involving alteration or substitution) is suspected or identified to assess patient safety risks.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how the hospital would assess risk to patients if tampering is suspected or identified. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
G. Prevention of Surgical Site Infection (SSI)		
<p>1. Hospital has a surgical care improvement program.</p> <p>Verify the following: The surgical care improvement program addresses appropriate prophylactic antibiotic use including:</p> <ul style="list-style-type: none"> a. Preoperative timing of prophylactic antibiotic administration (within 1 hour prior to incision or 2 hours for vancomycin or fluoroquinolones). b. Appropriate prophylactic antibiotic selection based on procedure type. c. Discontinuation of prophylactic antibiotics within 24 hours (48 hours for CABG or other cardiac surgery) after surgical end time. d. The surgical care improvement program addresses prompt removal of urinary catheter on post-op day 1 or 2, unless there is a documented appropriate reason for continued use. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Check if facility does not perform surgeries and move to item H. <i>Clostridium difficile</i> Infection.</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No 	
<p>2. Hospital regularly audits (monitors and documents) adherence to elements of surgical care improvement program.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>3. Hospital provides feedback from audits to personnel regarding their adherence to elements of the surgical care improvement program.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
G. Prevention of Surgical Site Infection (SSI) , continued		
<p>4. Hospital regularly audits (monitors and documents) adherence to recommended infection control practices for SSI prevention.</p> <p>Verify the following:</p> <p>Auditing includes:</p> <ul style="list-style-type: none"> a. Adherence to preoperative surgical scrub and hand hygiene b. Appropriate use of surgical attire and drapes c. Adherence to aseptic technique and sterile field d. Proper ventilation requirements in surgical suites e. Minimization of traffic in the operating room f. Adherence to cleaning and disinfection of environmental surfaces g. Respondent can describe process used for audits. h. Respondent can describe frequency of audits. i. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p> <p>d. <input type="radio"/> Yes <input type="radio"/> No</p> <p>e. <input type="radio"/> Yes <input type="radio"/> No</p> <p>f. <input type="radio"/> Yes <input type="radio"/> No</p> <p>g. <input type="radio"/> Yes <input type="radio"/> No</p> <p>h. <input type="radio"/> Yes <input type="radio"/> No</p> <p>i. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>5. Hospital provides feedback from audits to personnel regarding their adherence to surgical infection control practices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>6. Hospital monitors SSI data and uses it to direct prevention activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent is familiar with NHSN SSI data. b. Respondent can describe how SSI data are used to direct prevention activities. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>7. Hospital provides feedback of SSI data to surgeons and other surgical personnel.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
H. Prevention of <i>Clostridium difficile</i> Infection (CDI)		
1. Hospital has physician and/or nurse champions for CDI prevention activities.	<input type="radio"/> Yes <input type="radio"/> No	
2. Hospital regularly audits (monitors and documents) adherence to recommended infection control practices for CDI prevention. Verify the following: Auditing includes: <ol style="list-style-type: none"> a. Adherence to hand hygiene b. Appropriate use of PPE c. Compliance with Contact Precautions, including use of dedicated or disposable equipment d. Adherence to cleaning and disinfection procedures, including use of sporicidal disinfectants if part of hospital policy e. Respondent can describe process used for audits. f. Respondent can describe frequency of audits. g. Respondent can describe process for improvement when non-adherence is observed. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No g. <input type="radio"/> Yes <input type="radio"/> No	
3. Hospital provides feedback from audits to personnel regarding their adherence to recommended infection control practices for CDI prevention. Verify the following: <ol style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	
4. Hospital has specific antibiotic stewardship strategies in place to reduce CDI. <i>Note: Please see section III.8 for full assessment of antibiotic stewardship program.</i> Verify the following: <ol style="list-style-type: none"> a. Hospital has strategies to reduce unnecessary use of antibiotics that are high-risk for CDI (e.g., fluoroquinolones, 3rd/4th generation cephalosporins). b. Hospital reviews appropriateness of antibiotics prescribed for treatment of other conditions (e.g., urinary tract infection) for patients with new or recent CDI diagnosis. c. Hospital educates providers about the risk of CDI with antibiotics. d. Hospital educates patients and family members about the risk of CDI with antibiotics. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No	
5. Hospital monitors CDI data and uses it to direct prevention activities. Verify the following: <ol style="list-style-type: none"> a. Respondent is familiar with NHSN CDI data. b. Respondent can describe how CDI data are used to direct prevention activities. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	
6. Hospital provides feedback of CDI data to frontline personnel. Verify the following: <ol style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
I. Environmental Cleaning		
<p>1. Hospital has a competency-based training program for environmental cleaning.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who clean and disinfect patient care areas. Personnel may include, but are not limited to, environmental services staff, nurses, nursing assistants, and technicians. b. Training is provided upon hire, prior to being allowed to perform environmental cleaning. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with environmental cleaning (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with environmental cleaning procedures for all personnel who clean and disinfect patient care areas. g. If the hospital contracts environmental services, the contractor has a comparable training program. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No g. <input type="radio"/> Yes <input type="radio"/> No <p>Not Applicable <input type="radio"/></p>	
<p>2. Hospital has policies that clearly define responsibilities for cleaning and disinfection of non-critical equipment, mobile devices, and other electronics (e.g., ICU monitors, ventilator surfaces, bar code scanners, point-of-care devices, mobile work stations, code carts, airway boxes).</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>3. Hospital has protocols to ensure that healthcare personnel can readily identify equipment that has been properly cleaned and disinfected and is ready for patient use (e.g., tagging system, placement in dedicated clean area).</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>4. Hospital regularly audits (monitors and documents) adherence to cleaning and disinfection procedures, including use of products in accordance with manufacturers' instructions (e.g., dilution, storage, shelf-life, contact time).</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits (e.g., monitoring technology, direct observation). b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>5. Hospital provides feedback from audits to personnel regarding their adherence to cleaning and disinfection procedures.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>J. Device Reprocessing</p> <p>This section refers to all medical devices that may be reused in the hospital. Device categories include:</p> <ul style="list-style-type: none"> • Critical items (e.g., surgical instruments) are objects that enter sterile tissue or the vascular system and must be sterile prior to use. • Semi-critical items (e.g., endoscopes for upper endoscopy and colonoscopy, laryngoscope blades) are objects that contact mucous membranes or non-intact skin and require, at a minimum, high-level disinfection prior to reuse. • Non-critical items (e.g., blood pressure cuffs, point-of-care devices) are objects that may come in contact with intact skin but not mucous membranes and should undergo cleaning and low- or intermediate-level disinfection depending on the nature and degree of contamination (See Environmental Cleaning Section I. above). <p>Single-use devices (SUDs) are labeled by the manufacturer for a single use and do not have reprocessing instructions. They may not be reused unless they have been reprocessed for reuse by entities which have complied with FDA regulatory requirements and have received FDA clearance to reprocess specific SUDs.</p>		
<p>1. Hospital has a competency-based training program for reprocessing of critical devices.</p> <p>Verify the following:</p> <ol style="list-style-type: none"> Training is provided to all personnel who reprocess critical devices. Training is provided upon hire, prior to being allowed to reprocess critical devices. Training is provided at least annually. Training is provided when new devices or protocols are introduced. Personnel are required to demonstrate competency with device reprocessing (i.e., correct technique is observed by trainer) following each training. Hospital maintains current documentation of competency with reprocessing procedures for all personnel who reprocess critical devices. If the hospital contracts reprocessing of critical devices, the contractor has a comparable training program which includes the specific devices used by the hospital. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ol style="list-style-type: none"> <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <p>Not Applicable <input type="radio"/></p>	
<p>2. Hospital regularly audits (monitors and documents) adherence to reprocessing procedures for critical devices.</p> <p>Verify the following:</p> <ol style="list-style-type: none"> Respondent can describe process used for audits. Respondent can describe frequency of audits. Audits occur in all locations where critical devices are reprocessed (e.g., central sterile reprocessing, operating suites), including locations where initial cleaning steps are performed (e.g., point of use). Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ol style="list-style-type: none"> <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
J. Device Reprocessing, continued		
<p>3. Hospital provides feedback from audits to personnel regarding their adherence to reprocessing procedures for critical devices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	
<p>4. Hospital has a competency-based training program for reprocessing of semi-critical devices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who reprocess semi-critical devices. b. Training is provided upon hire, prior to being allowed to reprocess semi-critical devices. c. Training is provided at least annually. d. Training is provided when new devices or protocols are introduced. e. Personnel are required to demonstrate competency with device reprocessing (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with reprocessing procedures for all personnel who reprocess semi-critical devices. g. If the hospital contracts reprocessing of semi-critical devices, the contractor has a comparable training program which includes the specific devices used by the hospital. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No g. <input type="radio"/> Yes <input type="radio"/> No <p>Not Applicable <input type="radio"/></p>	
<p>5. Hospital regularly audits (monitors and documents) adherence to reprocessing procedures for semi-critical devices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Audits occur in all locations where semi-critical devices are reprocessed (e.g., central sterile reprocessing, endoscopy suites), including locations where initial cleaning steps are performed (e.g., point of use). d. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No 	
<p>6. Hospital provides feedback from audits to personnel regarding their adherence to reprocessing procedures for semi-critical devices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
J. Device Reprocessing, continued		
<p>7. If hospital reuses single-use devices, the devices are reprocessed by an FDA-approved entity.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not Applicable <input type="radio"/> (hospital does not reuse single-use devices)</p>	
<p>8. Hospital maintains documentation of reprocessing activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Hospital maintains logs for each sterilizer cycle that include the results from each load. b. Hospital has documentation that the chemicals used for high-level disinfection are routinely tested for appropriate concentration and replaced appropriately. c. Hospital maintains documentation of reprocessing activities. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>9. Hospital allows adequate time for reprocessing to ensure adherence to all steps recommended by the device manufacturer, including drying and proper storage.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Hospital has an adequate supply of instruments for the volume of procedures performed to allow sufficient time for all reprocessing steps. b. Scheduling of procedures allows sufficient time for all reprocessing steps. c. Hospital does not routinely use immediate-use steam sterilization (IUSS). 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>10. IP program is consulted whenever new devices or products will be purchased or introduced to ensure implementation of appropriate reprocessing policies and procedures.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>11. Hospital has policies and procedures outlining hospital response (i.e., risk assessment and recall of device) in the event of a reprocessing error or failure.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. The IP can describe how the risk assessment would be performed including how the hospital would identify which patients may have been exposed to an improperly reprocessed device. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No 	

III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>1. Hospital has system in place for early detection and management of potentially infectious persons at initial points of entry to the hospital, including rapid isolation as appropriate.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Travel and occupational history is included as part of admission and triage protocols. b. Hospital has system to identify (flag) patients with targeted MDROs upon readmission so appropriate precautions can be applied. <p>The hospital has a respiratory/hygiene cough etiquette program that includes:</p> <ul style="list-style-type: none"> c. Posting signs at entrances d. Providing tissues and no-touch receptacles for disposal of tissues e. Providing hand hygiene supplies in or near waiting areas f. Offering facemasks to coughing patients and other symptomatic individuals upon entry to the facility g. Providing space in patient waiting areas (e.g., ED waiting room) and encouraging individuals with symptoms of respiratory infections to sit as far away from others as possible 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p> <p>d. <input type="radio"/> Yes <input type="radio"/> No</p> <p>e. <input type="radio"/> Yes <input type="radio"/> No</p> <p>f. <input type="radio"/> Yes <input type="radio"/> No</p> <p>g. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>2. Hospital has systems in place for early detection and isolation of infectious patients identified during the hospital stay, including rapid isolation of patients as appropriate.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. There is a mechanism for prompt notification of the IP by the clinical microbiology laboratory when novel resistance patterns and/or targeted antimicrobial-resistant pathogens are detected. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>3. Hospital has system in place for INTER-facility communication of infectious status and isolation needs of patients prior to transfer to other facilities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods employed to ensure infectious status and isolation needs are communicated with receiving facilities. b. The hospital has system to notify receiving facilities of microbiological tests (e.g., cultures) that are pending at the time of transfer. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs), continued		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>4. Hospital has system in place for INTER-facility communication to identify infectious status and isolation needs of patients prior to accepting patients from other facilities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods employed to ensure infectious status and isolation needs are obtained from transferring facilities. b. The hospital has system to follow-up on microbiological results (e.g., cultures) that are pending at the time of transfer. c. If the hospital identifies an infection that may be related to care provided at another facility (e.g., hospital, nursing home, clinic), the facility is notified. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>5. Hospital has system in place for INTRA-facility communication to identify infectious status and isolation needs of patients prior to transfer to other units or shared spaces (e.g., radiology, physical therapy, emergency department) within the hospital.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods employed to ensure infectious status and isolation needs are communicated with receiving units. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>6. Hospital has a surveillance program to monitor incidence of epidemiologically-important organisms (e.g., CRE) and targeted healthcare-associated infections.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how the hospital determines which organisms and HAIs to track. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>7. Hospital uses surveillance data to implement corrective actions rapidly when transmission of epidemiologically-important organisms (e.g., CRE) or increased rates or persistently elevated rates of healthcare-associated infections are detected.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Data collection method allows for timely response to identified problems. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p>	

III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs), continued

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>8. Hospital has an antibiotic stewardship program that meets the 7 CDC core elements listed below (a – g).</p> <p><i>Note: The antibiotic stewardship program should be assessed in consultation with personnel knowledgeable about antibiotic stewardship activities (e.g., physician or pharmacist stewardship lead). Responses can be obtained from or cross-checked with the NHSN Annual Hospital Survey Antibiotic Stewardship Practice questions (Q 23 – 34) if available.</i></p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Hospital leadership commitment <ul style="list-style-type: none"> o Hospital has a written statement of support from leadership that supports efforts to improve antibiotic use (antibiotic stewardship) <u>AND/OR</u> o Hospital provides salary support for dedicated time for antibiotic stewardship activities. b. Program leadership (accountability) <ul style="list-style-type: none"> o There is a leader responsible for outcomes of stewardship activities at the hospital. c. Drug expertise <ul style="list-style-type: none"> o There is at least one pharmacist responsible for improving antibiotic use at the hospital. d. Act (at least one prescribing improvement action below) <ul style="list-style-type: none"> o Hospital has a policy that requires prescribers to document an indication for all antibiotics in the medical record or during order entry. o Hospital has hospital-specific treatment recommendations, based on national guidelines and local susceptibility, to assist with antibiotic selection for common clinical conditions. o There is a formal procedure for all clinicians to review the appropriateness of all antibiotics at or after 48 hours from the initial orders (e.g., antibiotic time out). o Hospital has specified antibiotic agents that need to be approved by a physician or pharmacist prior to dispensing at the hospital. o Physician or pharmacist reviews courses of therapy for specified antibiotic agents and communicates results with prescribers. e. Track <ul style="list-style-type: none"> o Hospital monitors antibiotic use (consumption). f. Report <ul style="list-style-type: none"> o Prescribers receive feedback by the stewardship program about how they can improve their antibiotic prescribing. g. Educate <ul style="list-style-type: none"> o Stewardship program provides education to clinicians and other relevant staff on improving antibiotic use. 	<p>○ Yes ○ No</p> <p>a. ○ Yes ○ No</p> <p>b. ○ Yes ○ No</p> <p>c. ○ Yes ○ No</p> <p>d. ○ Yes ○ No</p> <p>e. ○ Yes ○ No</p> <p>f. ○ Yes ○ No</p> <p>g. ○ Yes ○ No</p>	

III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs), continued		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>9. Hospital has occupational health program that, in addition to complying with state and federal requirements (e.g., OSHA), has policies regarding contact of personnel with patients when personnel have potentially transmissible conditions.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. The program has work-exclusion policies that encourage reporting of illnesses and do not penalize with loss of wages, benefits or job status. b. Personnel are educated regarding prompt reporting of illness to their supervisor and the occupational health programs. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>10. Hospital follows recommendations of the Advisory Committee on Immunization Practices (ACIP) for immunization of healthcare personnel, including offering Hepatitis B and influenza vaccination.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>11. Hospital is compliant with mandatory reporting requirements for notifiable diseases, healthcare-associated infections (as appropriate), and potential outbreaks.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Hospital can identify point(s) of contact at the local or state health department for HAI concerns. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>12. Hospital implements infection control measures relevant to construction, renovation, demolition, and repairs including performance of an infection control risk assessment (ICRA) before a project gets underway.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. IP program is consulted anytime construction, renovation, demolition, or repairs will be performed. b. ICRA elements are included in all contracts related to construction, renovation, demolition, and repairs. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

Section 3: Direct Observation of Facility Practices (optional)

Certain infection control lapses (e.g., reuse of syringes on more than one patient or to access a medication container that is used for subsequent patients; reuse of lancets) can result in bloodborne pathogen transmission and should be halted immediately. Identification of such lapses warrants appropriate notification and testing of potentially affected patients.

Examples of Auditing Tools for Direct Observations:

- **General Infection Control**

Centers for Medicare & Medicaid Services Hospital Infection Control

Worksheet: <http://www.cms.gov/Medicare/Provider-Enrollment-and-Certification/SurveyCertificationGenInfo/Downloads/Survey-and-Cert-Letter-15-12-Attachment-1.pdf>

Auditing checklists available for observations of:

- Hand hygiene
- Personal protective equipment use
- Indwelling urinary catheter insertion and maintenance
- Central venous catheter insertion and maintenance
- Injection safety
- Environmental services
- Equipment reprocessing (non-critical, semi-critical, critical reusable and single-use devices)
- Ventilator/respiratory therapy
- Spinal injection procedures
- Point of care devices
- Transmission-based precautions (Contact, Droplet, Airborne)
- Surgical procedures

- **Hand Hygiene Auditing Tools**

- Measuring Hand Hygiene Adherence: Overcoming the Challenges: http://www.jointcommission.org/assets/1/18/hh_monograph.pdf
- iScrub: <http://compepi.cs.uiowa.edu/index.php/Research/iScrub>

- **Personal Protective Equipment (PPE) Donning and Doffing**

- CDC Sequence for Donning and Removing Personal Protective Equipment <http://www.cdc.gov/hai/pdfs/ppe/PPE-Sequence.pdf>

- **Urinary Catheter Appropriate Use, Insertion, and Maintenance**

- American Nurses Association CAUTI Prevention Tool: <http://nursingworld.org/CAUTI-Tool>
- CDC TAP CAUTI Toolkit Implementation Guide: <http://www.cdc.gov/hai/prevent/tap/resources.html>

- **Central Venous Catheter Appropriate Use, Insertion, and Maintenance**

- CDC Checklist for Prevention of Central Line-Associated Blood Stream Infections: <http://www.cdc.gov/HAI/pdfs/bsi/checklist-for-CLABSI.pdf>
- AHRQ Tools for Reducing CLABSI: <http://www.ahrq.gov/professionals/education/curriculum-tools/clabsitools/index.html>

- **Safe Injection Practices**

- Injection Safety
Checklist: <http://www.oneandonlycampaign.org/sites/default/files/upload/pdf/Injection%20Safety%20Checklist-508.pdf>

- **Environmental Infection Control**

- CDC Environmental Checklist for Monitoring Terminal Cleaning: <http://www.cdc.gov/HAI/toolkits/Environmental-Cleaning-Checklist-10-6-2010.pdf>
- CDC Environmental Cleaning Evaluation Worksheet: <http://www.cdc.gov/HAI/toolkits/Evaluating-Environmental-Cleaning.html>
- Infection Control Risk Assessment (ICRA) Matrix of Precautions for Construction & Renovation: http://www.ashe.org/advocacy/organizations/CDC/pdfs/assessment_icra.pdf

Section 4: Infection Control Guidelines and Other Resources

- **General Infection Prevention**

- CDC/HICPAC Guidelines and recommendations: http://www.cdc.gov/HAI/prevent/prevent_pubs.html

- **Facility Infection Risk Assessment**

- Infection Prevention Annual Report and Plan: <http://apicchapter26.org/Data%20files/Minutes%202011/IC%20Risk%20Assessment%20guide.pdf>

- **Hand Hygiene**

- Guideline for Hand Hygiene in Healthcare Settings: <http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf>
- Hand Hygiene in Healthcare Settings: <http://www.cdc.gov/handhygiene>

- **Personal Protective Equipment**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation2007.pdf>
- Guidance for the Selection and Use of Personal Protective Equipment in Healthcare Settings: <http://www.cdc.gov/HAI/prevent/ppe.html>

- **Catheter-associated Urinary Tract Infection (CAUTI)**

- Guideline for Prevention of Catheter-associated Urinary Tract Infections, 2009: <http://www.cdc.gov/hicpac/pdf/CAUTI/CAUTIGuideline2009final.pdf>

- **Central line-associated Bloodstream Infection (CLABSI)**

- Guideline for Prevention of Intravascular Catheter-related Infections, 2011: <http://www.cdc.gov/hicpac/pdf/guidelines/bsi-guidelines-2011.pdf>

- **Ventilator-associated Event (VAE)**

- Guidelines for Preventing Healthcare-associated Pneumonia, 2003: http://www.cdc.gov/hicpac/pdf/guidelines/CDCpneumo_guidelines.pdf

- **Surgical Site Infection (SSI)**

- Guidelines for the Prevention of Surgical Site Infection, 1999: http://www.cdc.gov/hicpac/pdf/guidelines/SSI_1999.pdf

- **Safe Injection Practices**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
- CDC Injection Safety Web Materials: <http://www.cdc.gov/injectionsafety>
- CDC training video and related Safe Injection Practices Campaign materials: <http://oneandonlycampaign.org>

- ***Clostridium difficile* Infection (CDI) and Multidrug-Resistant Organisms (MDRO), including antimicrobial stewardship**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
- Management of Multi-Drug Resistant Organisms in Healthcare Settings, 2006: <http://www.cdc.gov/hicpac/pdf/guidelines/MDROGuideline2006.pdf>
- SHEA-IDSA Strategies to Prevention *Clostridium difficile* Infections in Acute Care Hospitals: 2014 Update: <http://www.jstor.org/stable/10.1086/676023>
- SHEA-IDSA Guideline: <http://www.cdc.gov/HAI/pdfs/cdiff/Cohen-IDSA-SHEA-CDI-guidelines-2010.pdf>
- CDC's Core Elements of Hospital Antibiotic Stewardship Program: <http://www.cdc.gov/getsmart/healthcare/implementation/core-elements.html>
- CDC Implementation Resources for Antibiotic Stewardship: <http://www.cdc.gov/getsmart/healthcare/implementation.html>
- EPA Listing of disinfectant products with sporicidal activity against *C. difficile*: http://www.epa.gov/oppad001/list_k_clostridium.pdf

- **Environmental Infection Control, including Infection Control Risk Assessment (ICRA)**

- Guidelines for Environmental Infection Control in Healthcare Facilities: http://www.cdc.gov/hicpac/pdf/guidelines/eic_in_HCF_03.pdf
- 2014 Facility Guidelines Institute (FGI) Guidelines for Hospitals and Outpatient Facilities: http://www.fgiguideines.org/guidelines2014_HOP.php

- **Equipment Reprocessing**

- Guideline for Disinfection and Sterilization in Healthcare Facilities: http://www.cdc.gov/hicpac/pdf/guidelines/Disinfection_Nov_2008.pdf
- FDA regulations on reprocessing of single-use devices: <http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm071434>

- **Point-of-Care Testing**

- Infection Prevention during Blood Glucose Monitoring and Insulin Administration: <http://www.cdc.gov/injectionsafety/blood-glucose-monitoring.html>
- Frequently Asked Questions (FAQs) regarding Assisted Blood Glucose Monitoring and Insulin Administration: http://www.cdc.gov/injectionsafety/providers/blood-glucose-monitoring_faqs.html

- **Respiratory Hygiene/Cough Etiquette**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
- Recommendations for Preventing the Spread of Influenza: <http://www.cdc.gov/flu/professionals/infectioncontrol/>

- **Healthcare Personnel Safety**

- Guideline for Infection Control in Healthcare Personnel: <http://www.cdc.gov/hicpac/pdf/InfectControl98.pdf>
- Immunization of Healthcare Personnel: <http://www.cdc.gov/vaccines/adults/rec-vac/hcw.html>
- Occupational Safety & Health Administration (OSHA) Bloodborne Pathogen and Needlestick Prevention Standard: <https://www.osha.gov/SLTC/bloodbornepathogens/index.html>
- Hospital Respiratory Protection Program Toolkit: <http://www.cdc.gov/niosh/docs/2015-117/pdfs/2015-117.pdf>

- **Resources to assist with evaluation and response to breaches in infection control**

- Patel PR, Srinivasan A, Perz JF. Developing a broader approach to management of infection control breaches in health care settings. Am J Infect Control 2008; 36(10):685-90. [http://www.ajicjournal.org/article/S0196-6553\(08\)00683-4/abstract](http://www.ajicjournal.org/article/S0196-6553(08)00683-4/abstract)
- Steps for Evaluating an Infection Control Breach: http://www.cdc.gov/hai/outbreaks/steps_for_eval_IC_breach.html
- Patient Notification Toolkit: <http://www.cdc.gov/injectionsafety/pntoolkit/index.html>

Infection Control Assessment Tool for Acute Care Hospitals

This tool is intended to assist in the assessment of infection control programs and practices in acute care hospitals. If feasible, direct observations of infection control practices are encouraged. To facilitate the assessment, health departments are encouraged to share this tool with hospitals in advance of their visit.

Overview

Section 1: Facility Demographics

Section 2: Infection Control Program and Infrastructure

Section 3: Direct Observation of Facility Practices (optional)

Section 4: Infection Control Guidelines and Other Resources

Infection Control Domains for Gap Assessment

- I. Infection Control Program and Infrastructure
- II. Infection Control Training, Competency, and Implementation of Policies and Practices
 - A. Hand Hygiene
 - B. Personal Protective Equipment (PPE)
 - C. Prevention of Catheter-associated Urinary Tract Infection (CAUTI)
 - D. Prevention of Central Line-associated Bloodstream Infection (CLABSI)
 - E. Prevention of Ventilator-associated Event (VAE)
 - F. Injection Safety
 - G. Prevention of Surgical Site Infection
 - H. Prevention of *Clostridium difficile* Infection (CDI)
 - I. Environmental Cleaning
 - J. Device Reprocessing
- III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs)



Section 1. Facility Demographics	
Facility Name (for health department use only)	
NHSN Facility Organization ID (for health department use only)	
State-assigned Unique ID	
Date of Assessment	
Type of Assessment	<input type="checkbox"/> On-site <input type="checkbox"/> Other (specify):
Rationale for Assessment (Select all that apply)	<input type="checkbox"/> Outbreak <input type="checkbox"/> Input from accrediting organization or state survey agency <input type="checkbox"/> NHSN data If YES, specify: <input type="checkbox"/> CAUTI <input type="checkbox"/> CLABSI <input type="checkbox"/> SSI <input type="checkbox"/> CDI <input type="checkbox"/> Other (specify:) <input type="checkbox"/> Collaborative (specify partner[s]):) <input type="checkbox"/> Other (specify):
Facility type	<input type="checkbox"/> Acute Care Hospital <input type="checkbox"/> Critical Access Hospital <input type="checkbox"/> Long-term Acute Care Hospital (LTACH) <input type="checkbox"/> Other (specify):
Number of Licensed Beds	
Number of Infection Preventionist Full-Time Equivalents	

Section 2: Infection Control Program and Infrastructure

I. Infection Control Program and Infrastructure		
Elements to be assessed	Assessment	Notes/Areas for Improvement
1. Hospital provides fiscal and human resource support for maintaining the infection prevention and control program.	<input type="radio"/> Yes <input type="radio"/> No	
2. The person(s) charged with directing the infection prevention and control program at the hospital is/are qualified and trained in infection control. Verify qualifications, which should include: (Check all that apply) <input type="checkbox"/> Successful completion of initial and recertification exams developed by the Certification Board for Infection Control & Epidemiology (CIC) AND/OR <input type="checkbox"/> Participation in infection control courses organized by recognized professional societies (e.g., APIC, SHEA)	<input type="radio"/> Yes <input type="radio"/> No	
3. Infection prevention and control program performs an annual facility infection risk assessment that evaluates and prioritizes potential risks for infections, contamination, and exposures and the program's preparedness to eliminate or mitigate such risks. <i>Note: Example of Facility Infection Risk Assessment Report and Plan is available in Section 4.</i>	<input type="radio"/> Yes <input type="radio"/> No	
4. Written infection control policies and procedures are available, current, and based on evidence-based guidelines (e.g., CDC/HICPAC), regulations, or standards. Verify the following: a. Respondent can describe the process for reviewing and updating policies (e.g., policies are dated and reviewed annually and when new guidelines are issued)	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No	
5. Infection prevention and control program provides infection prevention education to patients, family members, and other caregivers. Verify the following: a. Respondent can describe how this education is provided (e.g., information included in the admission or discharge packet, videos, signage, in-person training)	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Hand Hygiene		
<p>1. Hospital has a competency-based training program for hand hygiene.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all healthcare personnel, including all ancillary personnel not directly involved in patient care but potentially exposed to infectious agents (e.g., food tray handlers, housekeeping, volunteer personnel). b. Training is provided upon hire, prior to provision of care at this hospital. c. Training is provided at least annually. d. Personnel are required to demonstrate competency with hand hygiene following each training. e. Hospital maintains current documentation of hand hygiene competency for all personnel. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No 	
<p>2. Hospital regularly audits (monitors and documents) adherence to hand hygiene.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>3. Hospital provides feedback from audits to personnel regarding their hand hygiene performance.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	
<p>4. Supplies necessary for adherence to hand hygiene (e.g., soap, water, paper towels, alcohol-based hand rub) are readily accessible in patient care areas.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>5. Hand hygiene policies promote preferential use of alcohol-based hand rub over soap and water except when hands are visibly soiled (e.g., blood, body fluids) or after caring for a patient with known or suspected <i>C. difficile</i> or norovirus.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
B. Personal Protective Equipment (PPE)		
<p>1. Hospital has a competency-based training program for use of personal protective equipment (PPE). Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who use PPE. b. Training is provided upon hire, prior to provision of care at this hospital. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Training includes 1) appropriate indications for specific PPE components, 2) proper donning, doffing, adjustment, and wear of PPE, and 3) proper care, maintenance, useful life, and disposal of PPE. f. Personnel are required to demonstrate competency with selection and use of PPE (i.e., correct technique is observed by trainer) following each training. g. Hospital maintains current documentation of PPE competency for all personnel who use PPE. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No g. <input type="radio"/> Yes <input type="radio"/> No 	
<p>2. Hospital regularly audits (monitors and documents) adherence to proper PPE selection and use, including donning and doffing. Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>3. Hospital provides feedback to personnel regarding their performance with selection and use of PPE. Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	
<p>4. Supplies necessary for adherence to personal protective equipment recommendations specified under Standard and Transmission-based Precautions (e.g., gloves, gowns, mouth, eye, nose, and face protection) are available and located near point of use.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>5. The hospital's respiratory protection program provides annual respiratory fit testing for all personnel who are anticipated to require respiratory protection. Verify the following:</p> <ul style="list-style-type: none"> a. Hospital maintains supplies of respiratory protection devices (e.g., Powered air purifying respirator) to be used by personnel who cannot be fitted. b. Healthcare personnel are educated about factors that may compromise proper fit and function of respiratory protection devices (e.g., weight gain/loss, facial hair). 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
C. Prevention of Catheter-associated Urinary Tract Infection (CAUTI)		
1. Hospital has physician and/or nurse champions for CAUTI prevention activities.	<input type="radio"/> Yes <input type="radio"/> No	
2. Hospital has a competency-based training program for insertion of urinary catheters. Verify the following: <ol style="list-style-type: none"> a. Training is provided to all personnel who are given responsibility for insertion of urinary catheters. <i>Personnel</i> may include, but are not limited to, nurses, nursing assistants, medical assistants, technicians, and physicians. b. Training is provided upon hire, prior to being allowed to perform urinary catheter insertion. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with insertion (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with urinary catheter insertion for all personnel who insert urinary catheters. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No	
3. Hospital regularly audits (monitors and documents) adherence to recommended practices for insertion of urinary catheters. Verify the following: <ol style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No	
4. Hospital provides feedback from audits to personnel regarding their performance for insertion of urinary catheters. Verify the following: <ol style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
C. Prevention of Catheter-associated Urinary Tract Infection (CAUTI), continued		
<p>5. Hospital has a competency-based training program for <u>maintenance</u> of urinary catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who are given responsibility for urinary catheter maintenance (e.g., perineal care, emptying the drainage bag aseptically, maintaining the closed drainage system, maintaining unobstructed urine flow). Personnel may include, but are not limited to, nurses, nursing assistants, medical assistants, technicians, and transport personnel. b. Training is provided upon hire, prior to being allowed to perform urinary catheter maintenance. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with catheter maintenance (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with urinary catheter maintenance for all personnel who maintain urinary catheters. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p> <p>d. <input type="radio"/> Yes <input type="radio"/> No</p> <p>e. <input type="radio"/> Yes <input type="radio"/> No</p> <p>f. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>6. Hospital regularly audits (monitors and documents) adherence to recommended practices for <u>maintenance</u> of urinary catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>7. Hospital provides feedback from audits to personnel regarding their performance for <u>maintenance</u> of urinary catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>8. Patients with urinary catheters are assessed, at least daily, for continued need for the catheter.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods used to trigger the daily assessments (e.g., patient safety checklist, daily rounds, nurse directed protocol, reminders or stop orders). b. Hospital routinely audits adherence to daily assessment of urinary catheter need. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
C. Prevention of Catheter-associated Urinary Tract Infection (CAUTI), continued		
<p>9. Hospital monitors CAUTI data and uses it to direct prevention activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent is familiar with National Healthcare Safety Network (NHSN) CAUTI data. b. Respondent can describe how CAUTI data are used to direct prevention activities. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>10. Hospital provides feedback of CAUTI data to frontline personnel.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
D. Prevention of Central line-associated Bloodstream Infection (CLABSI)		
1. Hospital has physician and/or nurse champions for CLABSI prevention activities.	<input type="radio"/> Yes <input type="radio"/> No	
2. Hospital has a competency-based training program for insertion of central venous catheters. Verify the following: <ul style="list-style-type: none"> a. Training is provided to all personnel who are given responsibility for insertion of central venous catheters. Personnel may include, but are not limited to, physicians, physician assistants, and members of line insertion teams. b. Training is provided upon hire, prior to being allowed to perform central venous catheter insertion. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with insertion (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with central venous catheter insertion for all personnel who insert central venous catheters. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No	
3. Hospital regularly audits (monitors and documents) adherence to recommended practices for insertion of central venous catheters. Verify the following: <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No	
4. Hospital provides feedback from audits to personnel regarding their performance for insertion of central venous catheters. Verify the following: <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
D. Prevention of Central line-associated Bloodstream Infection (CLABSI), continued		
<p>5. Hospital has a competency-based training program for maintenance of central venous catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who maintain central venous catheters (e.g., scrub the hub, accessing the catheter, dressing changes). Personnel may include, but are not limited to, nurses, nursing assistants, physicians, and physician assistants. b. Training is provided upon hire, prior to being allowed to perform central venous catheter maintenance. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with maintenance (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with central venous catheter maintenance for all personnel who maintain central venous catheters. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p> <p>d. <input type="radio"/> Yes <input type="radio"/> No</p> <p>e. <input type="radio"/> Yes <input type="radio"/> No</p> <p>f. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>6. Hospital regularly audits (monitors and documents) adherence to recommended practices for maintenance of central venous catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>7. Hospital provides feedback from audits to personnel regarding their performance for maintenance of central venous catheters.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>8. Patients with central venous catheters are assessed, at least daily, for continued need for the catheter.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods used to trigger the daily assessments (e.g., patient safety checklist, daily rounds, reminders). b. Hospital routinely audits adherence to daily assessment of central venous catheter need. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
D. Prevention of Central line-associated Bloodstream Infection (CLABSI), continued		
<p>9. Hospital monitors CLABSI data and uses it to direct prevention activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent is familiar with National Healthcare Safety network (NHSN) CLABSI data. b. Respondent can describe how CLABSI data are used to direct prevention activities. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>10. Hospital provides feedback of CLABSI data to frontline personnel.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
E. Prevention of Ventilator-associated Event (VAE)		
1. Hospital has physician and/or nurse champions for VAE prevention activities.	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Check if facility does not provide care to ventilated patients and move to item F. Injection Safety.	
2. Hospital has a competency-based training program addressing prevention of VAEs. Verify the following: <ul style="list-style-type: none"> a. Training is provided to all personnel who provide respiratory therapy for ventilated patients (e.g., suctioning, administration of aerosolized medications). Personnel may include, but are not limited to, respiratory therapists and nurses. b. Training is provided upon hire, prior to being allowed to provide respiratory therapy for ventilated patients. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with respiratory therapy practices (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with respiratory practices for all personnel who provide respiratory therapy for ventilated patients. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No	
3. Hospital regularly audits (monitors and documents) adherence to recommended practices for management of ventilated patients (e.g., suctioning, administration of aerosolized medications). Verify the following: <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No	
4. Hospital provides feedback from audits to personnel regarding their performance for management of ventilated patients. Verify the following: <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
E. Prevention of Ventilator-associated Event (VAE), continued		
<p>5. Patients requiring invasive ventilation are assessed, at least daily, for continued need for the ventilator.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods used to trigger the daily assessments (e.g., patient safety checklist, daily rounds, reminders) b. Hospital routinely audits adherence to daily assessment of ventilator need. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>6. Hospital has a program that includes daily spontaneous breathing trials and lightening of sedation in eligible patient.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>7. Hospital has an oral-hygiene program.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>8. Hospital monitors VAE data and uses it to direct prevention activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how VAE data are used to direct prevention activities. <p>If the hospital reports VAE data to NHSN, verify the following:</p> <ul style="list-style-type: none"> b. Respondent is familiar with NHSN VAE data. <p>If the hospital does not report VAE data to NHSN, verify the following:</p> <ul style="list-style-type: none"> c. Respondent can describe how VAE data are collected. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>Not Applicable <input type="radio"/></p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p> <p>Not Applicable <input type="radio"/></p>	
<p>9. Hospital provides feedback of VAE data to frontline personnel.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
F. Injection Safety (This element does not include assessment of pharmacy practices)		
<p>1. Hospital has a competency-based training program for preparation and administration of parenteral medications (e.g., SQ, IM, IV) outside of the pharmacy.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who prepare and/or administer injections and parenteral infusions. b. Training is provided upon hire, prior to being allowed to prepare and/or administer injections and parenteral infusions. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with preparation and/or administration of injections and parenteral infusions following each training. f. Hospital maintains current documentation of competency with preparation and/or administration procedures for all personnel who prepare and/or administer injections and parenteral infusions. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No 	
<p>2. Hospital regularly audits (monitors and documents) adherence to safe injection practices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>3. Hospital provides feedback from audits to personnel regarding their adherence to safe injection practices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	
<p>4. Hospital has a drug diversion prevention program that includes consultation with the IP program when drug tampering (involving alteration or substitution) is suspected or identified to assess patient safety risks.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how the hospital would assess risk to patients if tampering is suspected or identified. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
G. Prevention of Surgical Site Infection (SSI)		
<p>1. Hospital has a surgical care improvement program.</p> <p>Verify the following: The surgical care improvement program addresses appropriate prophylactic antibiotic use including:</p> <ul style="list-style-type: none"> a. Preoperative timing of prophylactic antibiotic administration (within 1 hour prior to incision or 2 hours for vancomycin or fluoroquinolones). b. Appropriate prophylactic antibiotic selection based on procedure type. c. Discontinuation of prophylactic antibiotics within 24 hours (48 hours for CABG or other cardiac surgery) after surgical end time. d. The surgical care improvement program addresses prompt removal of urinary catheter on post-op day 1 or 2, unless there is a documented appropriate reason for continued use. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Check if facility does not perform surgeries and move to item H. <i>Clostridium difficile</i> Infection.</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p> <p>d. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>2. Hospital regularly audits (monitors and documents) adherence to elements of surgical care improvement program.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>3. Hospital provides feedback from audits to personnel regarding their adherence to elements of the surgical care improvement program.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
G. Prevention of Surgical Site Infection (SSI) , continued		
<p>4. Hospital regularly audits (monitors and documents) adherence to recommended infection control practices for SSI prevention.</p> <p>Verify the following:</p> <p>Auditing includes:</p> <ul style="list-style-type: none"> a. Adherence to preoperative surgical scrub and hand hygiene b. Appropriate use of surgical attire and drapes c. Adherence to aseptic technique and sterile field d. Proper ventilation requirements in surgical suites e. Minimization of traffic in the operating room f. Adherence to cleaning and disinfection of environmental surfaces g. Respondent can describe process used for audits. h. Respondent can describe frequency of audits. i. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No g. <input type="radio"/> Yes <input type="radio"/> No h. <input type="radio"/> Yes <input type="radio"/> No i. <input type="radio"/> Yes <input type="radio"/> No 	
<p>5. Hospital provides feedback from audits to personnel regarding their adherence to surgical infection control practices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	
<p>6. Hospital monitors SSI data and uses it to direct prevention activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent is familiar with NHSN SSI data. b. Respondent can describe how SSI data are used to direct prevention activities. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	
<p>7. Hospital provides feedback of SSI data to surgeons and other surgical personnel.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
H. Prevention of <i>Clostridium difficile</i> Infection (CDI)		
1. Hospital has physician and/or nurse champions for CDI prevention activities.	<input type="radio"/> Yes <input type="radio"/> No	
2. Hospital regularly audits (monitors and documents) adherence to recommended infection control practices for CDI prevention. Verify the following: Auditing includes: <ol style="list-style-type: none"> a. Adherence to hand hygiene b. Appropriate use of PPE c. Compliance with Contact Precautions, including use of dedicated or disposable equipment d. Adherence to cleaning and disinfection procedures, including use of sporicidal disinfectants if part of hospital policy e. Respondent can describe process used for audits. f. Respondent can describe frequency of audits. g. Respondent can describe process for improvement when non-adherence is observed. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No g. <input type="radio"/> Yes <input type="radio"/> No	
3. Hospital provides feedback from audits to personnel regarding their adherence to recommended infection control practices for CDI prevention. Verify the following: <ol style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	
4. Hospital has specific antibiotic stewardship strategies in place to reduce CDI. <i>Note: Please see section III.8 for full assessment of antibiotic stewardship program.</i> Verify the following: <ol style="list-style-type: none"> a. Hospital has strategies to reduce unnecessary use of antibiotics that are high-risk for CDI (e.g., fluoroquinolones, 3rd/4th generation cephalosporins). b. Hospital reviews appropriateness of antibiotics prescribed for treatment of other conditions (e.g., urinary tract infection) for patients with new or recent CDI diagnosis. c. Hospital educates providers about the risk of CDI with antibiotics. d. Hospital educates patients and family members about the risk of CDI with antibiotics. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No	
5. Hospital monitors CDI data and uses it to direct prevention activities. Verify the following: <ol style="list-style-type: none"> a. Respondent is familiar with NHSN CDI data. b. Respondent can describe how CDI data are used to direct prevention activities. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	
6. Hospital provides feedback of CDI data to frontline personnel. Verify the following: <ol style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<input type="radio"/> Yes <input type="radio"/> No a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
I. Environmental Cleaning		
<p>1. Hospital has a competency-based training program for environmental cleaning.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who clean and disinfect patient care areas. Personnel may include, but are not limited to, environmental services staff, nurses, nursing assistants, and technicians. b. Training is provided upon hire, prior to being allowed to perform environmental cleaning. c. Training is provided at least annually. d. Training is provided when new equipment or protocols are introduced. e. Personnel are required to demonstrate competency with environmental cleaning (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with environmental cleaning procedures for all personnel who clean and disinfect patient care areas. g. If the hospital contracts environmental services, the contractor has a comparable training program. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No g. <input type="radio"/> Yes <input type="radio"/> No <p>Not Applicable <input type="radio"/></p>	
<p>2. Hospital has policies that clearly define responsibilities for cleaning and disinfection of non-critical equipment, mobile devices, and other electronics (e.g., ICU monitors, ventilator surfaces, bar code scanners, point-of-care devices, mobile work stations, code carts, airway boxes).</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>3. Hospital has protocols to ensure that healthcare personnel can readily identify equipment that has been properly cleaned and disinfected and is ready for patient use (e.g., tagging system, placement in dedicated clean area).</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>4. Hospital regularly audits (monitors and documents) adherence to cleaning and disinfection procedures, including use of products in accordance with manufacturers' instructions (e.g., dilution, storage, shelf-life, contact time).</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits (e.g., monitoring technology, direct observation). b. Respondent can describe frequency of audits. c. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>5. Hospital provides feedback from audits to personnel regarding their adherence to cleaning and disinfection procedures.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>J. Device Reprocessing</p> <p>This section refers to all medical devices that may be reused in the hospital. Device categories include:</p> <ul style="list-style-type: none"> • Critical items (e.g., surgical instruments) are objects that enter sterile tissue or the vascular system and must be sterile prior to use. • Semi-critical items (e.g., endoscopes for upper endoscopy and colonoscopy, laryngoscope blades) are objects that contact mucous membranes or non-intact skin and require, at a minimum, high-level disinfection prior to reuse. • Non-critical items (e.g., blood pressure cuffs, point-of-care devices) are objects that may come in contact with intact skin but not mucous membranes and should undergo cleaning and low- or intermediate-level disinfection depending on the nature and degree of contamination (See Environmental Cleaning Section I. above). <p>Single-use devices (SUDs) are labeled by the manufacturer for a single use and do not have reprocessing instructions. They may not be reused unless they have been reprocessed for reuse by entities which have complied with FDA regulatory requirements and have received FDA clearance to reprocess specific SUDs.</p>		
<p>1. Hospital has a competency-based training program for reprocessing of critical devices.</p> <p>Verify the following:</p> <ol style="list-style-type: none"> Training is provided to all personnel who reprocess critical devices. Training is provided upon hire, prior to being allowed to reprocess critical devices. Training is provided at least annually. Training is provided when new devices or protocols are introduced. Personnel are required to demonstrate competency with device reprocessing (i.e., correct technique is observed by trainer) following each training. Hospital maintains current documentation of competency with reprocessing procedures for all personnel who reprocess critical devices. If the hospital contracts reprocessing of critical devices, the contractor has a comparable training program which includes the specific devices used by the hospital. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ol style="list-style-type: none"> <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <p>Not Applicable <input type="radio"/></p>	
<p>2. Hospital regularly audits (monitors and documents) adherence to reprocessing procedures for critical devices.</p> <p>Verify the following:</p> <ol style="list-style-type: none"> Respondent can describe process used for audits. Respondent can describe frequency of audits. Audits occur in all locations where critical devices are reprocessed (e.g., central sterile reprocessing, operating suites), including locations where initial cleaning steps are performed (e.g., point of use). Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ol style="list-style-type: none"> <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
J. Device Reprocessing, continued		
<p>3. Hospital provides feedback from audits to personnel regarding their adherence to reprocessing procedures for critical devices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	
<p>4. Hospital has a competency-based training program for reprocessing of semi-critical devices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Training is provided to all personnel who reprocess semi-critical devices. b. Training is provided upon hire, prior to being allowed to reprocess semi-critical devices. c. Training is provided at least annually. d. Training is provided when new devices or protocols are introduced. e. Personnel are required to demonstrate competency with device reprocessing (i.e., correct technique is observed by trainer) following each training. f. Hospital maintains current documentation of competency with reprocessing procedures for all personnel who reprocess semi-critical devices. g. If the hospital contracts reprocessing of semi-critical devices, the contractor has a comparable training program which includes the specific devices used by the hospital. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No g. <input type="radio"/> Yes <input type="radio"/> No Not Applicable <input type="radio"/> 	
<p>5. Hospital regularly audits (monitors and documents) adherence to reprocessing procedures for semi-critical devices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe process used for audits. b. Respondent can describe frequency of audits. c. Audits occur in all locations where semi-critical devices are reprocessed (e.g., central sterile reprocessing, endoscopy suites), including locations where initial cleaning steps are performed (e.g., point of use). d. Respondent can describe process for improvement when non-adherence is observed. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No 	
<p>6. Hospital provides feedback from audits to personnel regarding their adherence to reprocessing procedures for semi-critical devices.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how feedback is provided. b. Respondent can describe frequency of feedback. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	

II. Infection Control Training, Competency, and Implementation of Policies and Procedures

Elements to be assessed	Assessment	Notes/Areas for Improvement
J. Device Reprocessing, continued		
<p>7. If hospital reuses single-use devices, the devices are reprocessed by an FDA-approved entity.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not Applicable <input type="radio"/> (hospital does not reuse single-use devices)</p>	
<p>8. Hospital maintains documentation of reprocessing activities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Hospital maintains logs for each sterilizer cycle that include the results from each load. b. Hospital has documentation that the chemicals used for high-level disinfection are routinely tested for appropriate concentration and replaced appropriately. c. Hospital maintains documentation of reprocessing activities. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>9. Hospital allows adequate time for reprocessing to ensure adherence to all steps recommended by the device manufacturer, including drying and proper storage.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Hospital has an adequate supply of instruments for the volume of procedures performed to allow sufficient time for all reprocessing steps. b. Scheduling of procedures allows sufficient time for all reprocessing steps. c. Hospital does not routinely use immediate-use steam sterilization (IUSS). 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No 	
<p>10. IP program is consulted whenever new devices or products will be purchased or introduced to ensure implementation of appropriate reprocessing policies and procedures.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>11. Hospital has policies and procedures outlining hospital response (i.e., risk assessment and recall of device) in the event of a reprocessing error or failure.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. The IP can describe how the risk assessment would be performed including how the hospital would identify which patients may have been exposed to an improperly reprocessed device. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No 	

III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>1. Hospital has system in place for early detection and management of potentially infectious persons at initial points of entry to the hospital, including rapid isolation as appropriate.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Travel and occupational history is included as part of admission and triage protocols. b. Hospital has system to identify (flag) patients with targeted MDROs upon readmission so appropriate precautions can be applied. <p>The hospital has a respiratory/hygiene cough etiquette program that includes:</p> <ul style="list-style-type: none"> c. Posting signs at entrances d. Providing tissues and no-touch receptacles for disposal of tissues e. Providing hand hygiene supplies in or near waiting areas f. Offering facemasks to coughing patients and other symptomatic individuals upon entry to the facility g. Providing space in patient waiting areas (e.g., ED waiting room) and encouraging individuals with symptoms of respiratory infections to sit as far away from others as possible 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No c. <input type="radio"/> Yes <input type="radio"/> No d. <input type="radio"/> Yes <input type="radio"/> No e. <input type="radio"/> Yes <input type="radio"/> No f. <input type="radio"/> Yes <input type="radio"/> No g. <input type="radio"/> Yes <input type="radio"/> No 	
<p>2. Hospital has systems in place for early detection and isolation of infectious patients identified during the hospital stay, including rapid isolation of patients as appropriate.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. There is a mechanism for prompt notification of the IP by the clinical microbiology laboratory when novel resistance patterns and/or targeted antimicrobial-resistant pathogens are detected. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No 	
<p>3. Hospital has system in place for INTER-facility communication of infectious status and isolation needs of patients prior to transfer to other facilities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods employed to ensure infectious status and isolation needs are communicated with receiving facilities. b. The hospital has system to notify receiving facilities of microbiological tests (e.g., cultures) that are pending at the time of transfer. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <ul style="list-style-type: none"> a. <input type="radio"/> Yes <input type="radio"/> No b. <input type="radio"/> Yes <input type="radio"/> No 	

III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs), continued		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>4. Hospital has system in place for INTER-facility communication to identify infectious status and isolation needs of patients prior to accepting patients from other facilities.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods employed to ensure infectious status and isolation needs are obtained from transferring facilities. b. The hospital has system to follow-up on microbiological results (e.g., cultures) that are pending at the time of transfer. c. If the hospital identifies an infection that may be related to care provided at another facility (e.g., hospital, nursing home, clinic), the facility is notified. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p> <p>c. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>5. Hospital has system in place for INTRA-facility communication to identify infectious status and isolation needs of patients prior to transfer to other units or shared spaces (e.g., radiology, physical therapy, emergency department) within the hospital.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe methods employed to ensure infectious status and isolation needs are communicated with receiving units. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>6. Hospital has a surveillance program to monitor incidence of epidemiologically-important organisms (e.g., CRE) and targeted healthcare-associated infections.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Respondent can describe how the hospital determines which organisms and HAIs to track. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>7. Hospital uses surveillance data to implement corrective actions rapidly when transmission of epidemiologically-important organisms (e.g., CRE) or increased rates or persistently elevated rates of healthcare-associated infections are detected.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Data collection method allows for timely response to identified problems. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p>	

III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs), continued

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>8. Hospital has an antibiotic stewardship program that meets the 7 CDC core elements listed below (a – g).</p> <p><i>Note: The antibiotic stewardship program should be assessed in consultation with personnel knowledgeable about antibiotic stewardship activities (e.g., physician or pharmacist stewardship lead). Responses can be obtained from or cross-checked with the NHSN Annual Hospital Survey Antibiotic Stewardship Practice questions (Q 23 – 34) if available.</i></p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Hospital leadership commitment <ul style="list-style-type: none"> o Hospital has a written statement of support from leadership that supports efforts to improve antibiotic use (antibiotic stewardship) <u>AND/OR</u> o Hospital provides salary support for dedicated time for antibiotic stewardship activities. b. Program leadership (accountability) <ul style="list-style-type: none"> o There is a leader responsible for outcomes of stewardship activities at the hospital. c. Drug expertise <ul style="list-style-type: none"> o There is at least one pharmacist responsible for improving antibiotic use at the hospital. d. Act (at least one prescribing improvement action below) <ul style="list-style-type: none"> o Hospital has a policy that requires prescribers to document an indication for all antibiotics in the medical record or during order entry. o Hospital has hospital-specific treatment recommendations, based on national guidelines and local susceptibility, to assist with antibiotic selection for common clinical conditions. o There is a formal procedure for all clinicians to review the appropriateness of all antibiotics at or after 48 hours from the initial orders (e.g., antibiotic time out). o Hospital has specified antibiotic agents that need to be approved by a physician or pharmacist prior to dispensing at the hospital. o Physician or pharmacist reviews courses of therapy for specified antibiotic agents and communicates results with prescribers. e. Track <ul style="list-style-type: none"> o Hospital monitors antibiotic use (consumption). f. Report <ul style="list-style-type: none"> o Prescribers receive feedback by the stewardship program about how they can improve their antibiotic prescribing. g. Educate <ul style="list-style-type: none"> o Stewardship program provides education to clinicians and other relevant staff on improving antibiotic use. 	<p>○ Yes ○ No</p> <p>a. ○ Yes ○ No</p> <p>b. ○ Yes ○ No</p> <p>c. ○ Yes ○ No</p> <p>d. ○ Yes ○ No</p> <p>e. ○ Yes ○ No</p> <p>f. ○ Yes ○ No</p> <p>g. ○ Yes ○ No</p>	

III. Systems to Detect, Prevent, and Respond to Healthcare-Associated Infections and Multidrug-Resistant Organisms (MDROs), continued		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>9. Hospital has occupational health program that, in addition to complying with state and federal requirements (e.g., OSHA), has policies regarding contact of personnel with patients when personnel have potentially transmissible conditions.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. The program has work-exclusion policies that encourage reporting of illnesses and do not penalize with loss of wages, benefits or job status. b. Personnel are educated regarding prompt reporting of illness to their supervisor and the occupational health programs. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>10. Hospital follows recommendations of the Advisory Committee on Immunization Practices (ACIP) for immunization of healthcare personnel, including offering Hepatitis B and influenza vaccination.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>11. Hospital is compliant with mandatory reporting requirements for notifiable diseases, healthcare-associated infections (as appropriate), and potential outbreaks.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. Hospital can identify point(s) of contact at the local or state health department for HAI concerns. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>12. Hospital implements infection control measures relevant to construction, renovation, demolition, and repairs including performance of an infection control risk assessment (ICRA) before a project gets underway.</p> <p>Verify the following:</p> <ul style="list-style-type: none"> a. IP program is consulted anytime construction, renovation, demolition, or repairs will be performed. b. ICRA elements are included in all contracts related to construction, renovation, demolition, and repairs. 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>a. <input type="radio"/> Yes <input type="radio"/> No</p> <p>b. <input type="radio"/> Yes <input type="radio"/> No</p>	

Section 3: Direct Observation of Facility Practices (optional)

Certain infection control lapses (e.g., reuse of syringes on more than one patient or to access a medication container that is used for subsequent patients; reuse of lancets) can result in bloodborne pathogen transmission and should be halted immediately. Identification of such lapses warrants appropriate notification and testing of potentially affected patients.

Examples of Auditing Tools for Direct Observations:

- **General Infection Control**

Centers for Medicare & Medicaid Services Hospital Infection Control

Worksheet: <http://www.cms.gov/Medicare/Provider-Enrollment-and-Certification/SurveyCertificationGenInfo/Downloads/Survey-and-Cert-Letter-15-12-Attachment-1.pdf>

Auditing checklists available for observations of:

- Hand hygiene
- Personal protective equipment use
- Indwelling urinary catheter insertion and maintenance
- Central venous catheter insertion and maintenance
- Injection safety
- Environmental services
- Equipment reprocessing (non-critical, semi-critical, critical reusable and single-use devices)
- Ventilator/respiratory therapy
- Spinal injection procedures
- Point of care devices
- Transmission-based precautions (Contact, Droplet, Airborne)
- Surgical procedures

- **Hand Hygiene Auditing Tools**

- Measuring Hand Hygiene Adherence: Overcoming the Challenges: http://www.jointcommission.org/assets/1/18/hh_monograph.pdf
- iScrub: <http://compepi.cs.uiowa.edu/index.php/Research/iScrub>

- **Personal Protective Equipment (PPE) Donning and Doffing**

- CDC Sequence for Donning and Removing Personal Protective Equipment <http://www.cdc.gov/hai/pdfs/ppe/PPE-Sequence.pdf>

- **Urinary Catheter Appropriate Use, Insertion, and Maintenance**

- American Nurses Association CAUTI Prevention Tool: <http://nursingworld.org/CAUTI-Tool>
- CDC TAP CAUTI Toolkit Implementation Guide: <http://www.cdc.gov/hai/prevent/tap/resources.html>

- **Central Venous Catheter Appropriate Use, Insertion, and Maintenance**

- CDC Checklist for Prevention of Central Line-Associated Blood Stream Infections: <http://www.cdc.gov/HAI/pdfs/bsi/checklist-for-CLABSI.pdf>

- AHRQ Tools for Reducing CLABSI: <http://www.ahrq.gov/professionals/education/curriculum-tools/clabsitools/index.html>

- **Safe Injection Practices**

- Injection Safety
Checklist: <http://www.oneandonlycampaign.org/sites/default/files/upload/pdf/Injection%20Safety%20Checklist-508.pdf>

- **Environmental Infection Control**

- CDC Environmental Checklist for Monitoring Terminal Cleaning: <http://www.cdc.gov/HAI/toolkits/Environmental-Cleaning-Checklist-10-6-2010.pdf>

- CDC Environmental Cleaning Evaluation Worksheet: <http://www.cdc.gov/HAI/toolkits/Evaluating-Environmental-Cleaning.html>

- Infection Control Risk Assessment (ICRA) Matrix of Precautions for Construction & Renovation: http://www.ashe.org/advocacy/organizations/CDC/pdfs/assessment_icra.pdf

Section 4: Infection Control Guidelines and Other Resources

- **General Infection Prevention**

- CDC/HICPAC Guidelines and recommendations: http://www.cdc.gov/HAI/prevent/prevent_pubs.html

- **Facility Infection Risk Assessment**

- Infection Prevention Annual Report and Plan: <http://apicchapter26.org/Data%20files/Minutes%202011/IC%20Risk%20Assessment%20guide.pdf>

- **Hand Hygiene**

- Guideline for Hand Hygiene in Healthcare Settings: <http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf>
- Hand Hygiene in Healthcare Settings: <http://www.cdc.gov/handhygiene>

- **Personal Protective Equipment**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation2007.pdf>
- Guidance for the Selection and Use of Personal Protective Equipment in Healthcare Settings: <http://www.cdc.gov/HAI/prevent/ppe.html>

- **Catheter-associated Urinary Tract Infection (CAUTI)**

- Guideline for Prevention of Catheter-associated Urinary Tract Infections, 2009: <http://www.cdc.gov/hicpac/pdf/CAUTI/CAUTIGuideline2009final.pdf>

- **Central line-associated Bloodstream Infection (CLABSI)**

- Guideline for Prevention of Intravascular Catheter-related Infections, 2011: <http://www.cdc.gov/hicpac/pdf/guidelines/bsi-guidelines-2011.pdf>

- **Ventilator-associated Event (VAE)**

- Guidelines for Preventing Healthcare-associated Pneumonia, 2003: http://www.cdc.gov/hicpac/pdf/guidelines/CDCpneumo_guidelines.pdf

- **Surgical Site Infection (SSI)**

- Guidelines for the Prevention of Surgical Site Infection, 1999: http://www.cdc.gov/hicpac/pdf/guidelines/SSI_1999.pdf

- **Safe Injection Practices**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
- CDC Injection Safety Web Materials: <http://www.cdc.gov/injectionsafety>
- CDC training video and related Safe Injection Practices Campaign materials: <http://oneandonlycampaign.org>

- ***Clostridium difficile* Infection (CDI) and Multidrug-Resistant Organisms (MDRO), including antimicrobial stewardship**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
- Management of Multi-Drug Resistant Organisms in Healthcare Settings, 2006: <http://www.cdc.gov/hicpac/pdf/guidelines/MDROGuideline2006.pdf>
- SHEA-IDSA Strategies to Prevention *Clostridium difficile* Infections in Acute Care Hospitals: 2014 Update: <http://www.jstor.org/stable/10.1086/676023>
- SHEA-IDSA Guideline: <http://www.cdc.gov/HAI/pdfs/cdiff/Cohen-IDSA-SHEA-CDI-guidelines-2010.pdf>
- CDC's Core Elements of Hospital Antibiotic Stewardship Program: <http://www.cdc.gov/getsmart/healthcare/implementation/core-elements.html>
- CDC Implementation Resources for Antibiotic Stewardship: <http://www.cdc.gov/getsmart/healthcare/implementation.html>
- EPA Listing of disinfectant products with sporicidal activity against *C. difficile*: http://www.epa.gov/oppad001/list_k_clostridium.pdf

- **Environmental Infection Control, including Infection Control Risk Assessment (ICRA)**

- Guidelines for Environmental Infection Control in Healthcare Facilities: http://www.cdc.gov/hicpac/pdf/guidelines/eic_in_HCF_03.pdf
- 2014 Facility Guidelines Institute (FGI) Guidelines for Hospitals and Outpatient Facilities: http://www.fgiguideines.org/guidelines2014_HOP.php

- **Equipment Reprocessing**

- Guideline for Disinfection and Sterilization in Healthcare Facilities: http://www.cdc.gov/hicpac/pdf/guidelines/Disinfection_Nov_2008.pdf
- FDA regulations on reprocessing of single-use devices: <http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm071434>

- **Point-of-Care Testing**

- Infection Prevention during Blood Glucose Monitoring and Insulin Administration: <http://www.cdc.gov/injectionsafety/blood-glucose-monitoring.html>
- Frequently Asked Questions (FAQs) regarding Assisted Blood Glucose Monitoring and Insulin Administration: http://www.cdc.gov/injectionsafety/providers/blood-glucose-monitoring_faqs.html

- **Respiratory Hygiene/Cough Etiquette**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
- Recommendations for Preventing the Spread of Influenza: <http://www.cdc.gov/flu/professionals/infectioncontrol/>

- **Healthcare Personnel Safety**

- Guideline for Infection Control in Healthcare Personnel: <http://www.cdc.gov/hicpac/pdf/InfectControl98.pdf>
- Immunization of Healthcare Personnel: <http://www.cdc.gov/vaccines/adults/rec-vac/hcw.html>
- Occupational Safety & Health Administration (OSHA) Bloodborne Pathogen and Needlestick Prevention Standard: <https://www.osha.gov/SLTC/bloodbornepathogens/index.html>
- Hospital Respiratory Protection Program Toolkit: <http://www.cdc.gov/niosh/docs/2015-117/pdfs/2015-117.pdf>

- **Resources to assist with evaluation and response to breaches in infection control**

- Patel PR, Srinivasan A, Perz JF. Developing a broader approach to management of infection control breaches in health care settings. Am J Infect Control 2008; 36(10):685-90. [http://www.ajicjournal.org/article/S0196-6553\(08\)00683-4/abstract](http://www.ajicjournal.org/article/S0196-6553(08)00683-4/abstract)
- Steps for Evaluating an Infection Control Breach: http://www.cdc.gov/hai/outbreaks/steps_for_eval_IC_breach.html
- Patient Notification Toolkit: <http://www.cdc.gov/injectionsafety/pntoolkit/index.html>

Infection Prevention and Control Assessment Tool for Outpatient Settings

This tool is intended to assist in the assessment of infection control programs and practices in outpatient settings. In order to complete the assessment, direct observation of infection control practices will be necessary. To facilitate the assessment, health departments are encouraged to share this tool with facilities in advance of their visit.

Overview

Section 1: Facility Demographics

Section 2: Infection Control Program and Infrastructure

Section 3: Direct Observation of Facility Practices

Section 4: Infection Control Guidelines and Other Resources

Infection Control Domains for Gap Assessment

- I. Infection Control Program and Infrastructure
- II. Infection Control Training and Competency
- III. Healthcare Personnel Safety
- IV. Surveillance and Disease Reporting
- V.a/b. Hand Hygiene
- VI.a/b. Personal Protective Equipment (PPE)
- VII.a/b. Injection Safety
- VIII.a/b. Respiratory Hygiene/Cough Etiquette
- IX.a/b. Point-of-Care Testing (if applicable)
- X.a/b. Environmental Cleaning
- XI.a/b. Device Reprocessing (if applicable)
- XII. Sterilization of Reusable Devices (if applicable)
- XIII. High-level Disinfection of Reusable Devices (if applicable)



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

Section 1: Facility Demographics			
Facility Name (for health department use only)			
NHSN Facility Organization ID (for health department use only)			
State-assigned Unique ID			
Date of Assessment			
Type of Assessment	<input type="checkbox"/> On-site <input type="checkbox"/> Other (specify):		
Rationale for Assessment (Select all that apply)	<input type="checkbox"/> Outbreak <input type="checkbox"/> Input from accrediting organization or state survey agency <input type="checkbox"/> Other (specify):		
Is the facility licensed by the state?	<input type="checkbox"/> Yes <input type="checkbox"/> No		
Is the facility certified by the Centers for Medicare & Medicaid Services (CMS)?	<input type="checkbox"/> Yes <input type="checkbox"/> No		
Is the facility accredited?	<input type="checkbox"/> Yes <input type="checkbox"/> No If yes, list the accreditation organization: <ul style="list-style-type: none"> <input type="checkbox"/> Accreditation Association for Ambulatory Health Care (AAAHC) <input type="checkbox"/> American Association for Accreditation of Ambulatory Surgery Facilities (AAAASF) <input type="checkbox"/> American Osteopathic Association (AOA) <input type="checkbox"/> The Joint Commission (TJC) <input type="checkbox"/> Other (specify): 		
Is the facility affiliated with a hospital?	<input type="checkbox"/> Yes (specify – for health department use only): <input type="checkbox"/> No		
Which procedures are performed by the facility? Select all that apply.	<input type="checkbox"/> Chemotherapy	<input type="checkbox"/> Endoscopy	<input type="checkbox"/> Ear/Nose/Throat
	<input type="checkbox"/> Imaging (MRI/CT)	<input type="checkbox"/> Immunizations	<input type="checkbox"/> OB/Gyn
	<input type="checkbox"/> Ophthalmologic	<input type="checkbox"/> Orthopedic	<input type="checkbox"/> Pain remediation
	<input type="checkbox"/> Plastic/reconstructive	<input type="checkbox"/> Podiatry	<input type="checkbox"/> Other (specify):
What is the primary procedure-type performed by the facility? Select only one.	<input type="checkbox"/> Chemotherapy	<input type="checkbox"/> Endoscopy	<input type="checkbox"/> Ear/Nose/Throat
	<input type="checkbox"/> Imaging (MRI/CT)	<input type="checkbox"/> Immunizations	<input type="checkbox"/> OB/Gyn
	<input type="checkbox"/> Ophthalmologic	<input type="checkbox"/> Orthopedic	<input type="checkbox"/> Pain remediation
	<input type="checkbox"/> Plastic/reconstructive	<input type="checkbox"/> Podiatry	<input type="checkbox"/> Other (specify):
How many physicians work at the facility?			
What is the average number of patients seen per week?			

Section 2: Infection Control Program and Infrastructure

I. Infection Control Program and Infrastructure		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Written infection prevention policies and procedures are available, current, and based on evidence-based guidelines (e.g., CDC/HICPAC), regulations, or standards.</p> <p><i>Note: Policies and procedures should be appropriate for the services provided by the facility and should extend beyond OSHA bloodborne pathogen training</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>B. Infection prevention policies and procedures are re-assessed at least annually or according to state or federal requirements, and updated if appropriate.</p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>C. At least one individual trained in infection prevention is employed by or regularly available (e.g., by contract) to manage the facility's infection control program.</p> <p><i>Note: Examples of training may include: Successful completion of initial and/or recertification exams developed by the Certification Board for Infection Control & Epidemiology; participation in infection control courses organized by the state or recognized professional societies (e.g., APIC, SHEA).</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>D. Facility has system for early detection and management of potentially infectious persons at initial points of patient encounter.</p> <p><i>Note: System may include taking a travel and occupational history, as appropriate, and elements described under respiratory hygiene/cough etiquette.</i></p>	<input type="radio"/> Yes <input type="radio"/> No	

II. Infection Control Training and Competency		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Facility has a competency-based training program that provides job-specific training on infection prevention policies and procedures to healthcare personnel.</p> <p><i>Note: This includes those employed by outside agencies and available by contract or on a volunteer basis to the facility.</i></p> <p><i>See sections below for more specific assessment of training related to: hand hygiene, personal protective equipment (PPE), injection safety, environmental cleaning, point-of-care testing, and device reprocessing</i></p>	<input type="radio"/> Yes <input type="radio"/> No	

III. Healthcare Personnel Safety		
Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Facility has an exposure control plan that is tailored to the specific requirements of the facility (e.g., addresses potential hazards posed by specific services provided by the facility).</p> <p><i>Note: A model template, which includes a guide for creating an exposure control plan that meets the requirements of the OSHA Bloodborne Pathogens Standard is available at: https://www.osha.gov/Publications/osha3186.pdf</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>B. HCP for whom contact with blood or other potentially infectious material is anticipated are trained on the OSHA bloodborne pathogen standard upon hire and at least annually.</p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>C. Following an exposure event, post-exposure evaluation and follow-up, including prophylaxis as appropriate, are available at no cost to employee and are supervised by a licensed healthcare professional.</p> <p><i>Note: An exposure incident refers to a specific eye, mouth, other mucous membrane, non-intact skin, or parenteral contact with blood or other potentially infectious materials that results from the performance of an individual's duties.</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>D. Facility tracks HCP exposure events and evaluates event data and develops/implements corrective action plans to reduce incidence of such events.</p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>E. Facility follows recommendations of the Advisory Committee on Immunization Practices (ACIP) for immunization of HCP, including offering Hepatitis B and influenza vaccination.</p> <p><i>Note: Immunization of Health-Care Personnel: Recommendations of the ACIP available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/rr6007a1.htm</i></p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>F. All HCP receive baseline tuberculosis (TB) screening prior to placement, and those with potential for ongoing exposure to TB receive periodic screening (if negative) at least annually.</p>	<input type="radio"/> Yes <input type="radio"/> No	
<p>G. If respirators are used, the facility has a respiratory protection program that details required worksite-specific procedures and elements for required respirator use, including provision of medical clearance, training, and fit testing as appropriate.</p>	<input type="radio"/> Yes <input type="radio"/> No Not Applicable <input type="radio"/>	
<p>H. Facility has well-defined policies concerning contact of personnel with patients when personnel have potentially transmissible conditions. These policies include:</p> <ul style="list-style-type: none"> i. Work-exclusion policies that encourage reporting of illnesses and do not penalize with loss of wages, benefits, or job status. ii. Education of personnel on prompt reporting of illness to supervisor. 	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	

IV. Surveillance and Disease Reporting		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. An updated list of diseases reportable to the public health authority is readily available to all personnel.	<input type="radio"/> Yes <input type="radio"/> No	
B. Facility can demonstrate knowledge of and compliance with mandatory reporting requirements for notifiable diseases, healthcare associated infections (as appropriate), and for potential outbreaks.	<input type="radio"/> Yes <input type="radio"/> No	
C. Patients who have undergone procedures at the facility are educated regarding signs and symptoms of infection that may be associated with the procedure and instructed to notify the facility if such signs or symptoms occur.	<input type="radio"/> Yes <input type="radio"/> No	

V.a. Hand Hygiene		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. All HCP are educated regarding appropriate indications for hand hygiene: i. Upon hire, prior to provision of care ii. Annually	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	
B. HCP are required to demonstrate competency with hand hygiene following each training	<input type="radio"/> Yes <input type="radio"/> No	
C. Facility regularly audits (monitors and documents) adherence to hand hygiene.	<input type="radio"/> Yes <input type="radio"/> No	
D. Facility provides feedback from audits to personnel regarding their hand hygiene performance.	<input type="radio"/> Yes <input type="radio"/> No	
E. Hand hygiene policies promote preferential use of alcohol-based hand rub over soap and water in all clinical situations except when hands are visibly soiled (e.g., blood, body fluids) or after caring for a patient with known or suspected <i>C. difficile</i> or norovirus.	<input type="radio"/> Yes <input type="radio"/> No	

VI.a. Personal Protective Equipment (PPE)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. HCP who use PPE receive training on proper selection and use of PPE: i. Upon hire, prior to provision of care ii. Annually iii. When new equipment or protocols are introduced	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	
B. HCP are required to demonstrate competency with selection and use of PPE following each training.	<input type="radio"/> Yes <input type="radio"/> No	
C. Facility regularly audits (monitors and documents) adherence to proper PPE selection and use.	<input type="radio"/> Yes <input type="radio"/> No	
D. Facility provides feedback from audits to personnel regarding their performance with selection and use of PPE.	<input type="radio"/> Yes <input type="radio"/> No	

VII.a. Injection Safety (This element does not include assessment of pharmacy/compounding practices)

Elements to be assessed	Assessment	Notes/Areas for Improvement
A. HCP who prepare and/or administer parenteral medications receive training on safe injection practices: <ul style="list-style-type: none"> i. Upon hire, prior to being allowed to prepare and/or administer parenteral medications ii. Annually iii. When new equipment or protocols are introduced 	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Yes <input type="radio"/> No</p>	
B. HCP are required to demonstrate competency with safe injection practices following each training.	<input type="radio"/> Yes <input type="radio"/> No	
C. Facility regularly audits (monitors and documents) adherence to safe injection practices.	<input type="radio"/> Yes <input type="radio"/> No	
D. Facility provides feedback from audits to personnel regarding their adherence to safe injection practices.	<input type="radio"/> Yes <input type="radio"/> No	
E. Facility has policies and procedures to track HCP access to controlled substances to prevent narcotics theft/diversion.	<input type="radio"/> Yes <input type="radio"/> No	
<p><i>Note: Policies and procedures should address: how data are reviewed, how facility would respond to unusual access patterns, how facility would assess risk to patients if tampering (alteration or substitution) is suspected or identified, and who the facility would contact if diversion is suspected or identified.</i></p>		

VIII.a. Respiratory Hygiene/Cough Etiquette

Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Facility has policies and procedures to contain respiratory secretions in persons who have signs and symptoms of a respiratory infection, beginning at point of entry to the facility and continuing through the duration of the visit. Policies include: <ul style="list-style-type: none"> i. Offering facemasks to coughing patients and other symptomatic persons upon entry to the facility, at a minimum, during periods of increased respiratory infection activity in the community. ii. Providing space in waiting rooms and encouraging persons with symptoms of respiratory infections to sit as far away from others as possible. <p><i>Note: If available, facilities may wish to place patients with symptoms of a respiratory infection in a separate area while waiting for care.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Yes <input type="radio"/> No</p> <p><input type="radio"/> Yes <input type="radio"/> No</p>	
B. Facility educates HCP on the importance of infection prevention measures to contain respiratory secretions to prevent the spread of respiratory pathogens.	<input type="radio"/> Yes <input type="radio"/> No	

IX.a. Point-of-Care Testing (e.g., blood glucose meters, INR monitor)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. HCP who perform point-of-care testing receive training on recommended practices: i. Upon hire, prior to being allowed to perform point-of-care testing ii. Annually iii. When new equipment or protocols are introduced	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
B. HCP are required to demonstrate competency with recommended practices for point-of-care testing following each training.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
C. Facility regularly audits (monitors and documents) adherence to recommended practices during point-of-care testing.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
D. Facility provides feedback from audits to personnel regarding their adherence to recommended practices.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

X.a. Environmental Cleaning		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Facility has written policies and procedures for routine cleaning and disinfection of environmental surfaces, including identification of responsible personnel.	<input type="radio"/> Yes <input type="radio"/> No	
B. Personnel who clean and disinfect patient care areas (e.g., environmental services, technicians, nurses) receive training on cleaning procedures i. Upon hire, prior to being allowed to perform environmental cleaning ii. Annually iii. When new equipment or protocols are introduced <i>Note: If environmental cleaning is performed by contract personnel, facility should verify this is provided by contracting company.</i>	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	
C. HCP are required to demonstrate competency with environmental cleaning procedures following each training.	<input type="radio"/> Yes <input type="radio"/> No	
D. Facility regularly audits (monitors and documents) adherence to cleaning and disinfection procedures, including using products in accordance with manufacturer's instructions (e.g., dilution, storage, shelf-life, contact time).	<input type="radio"/> Yes <input type="radio"/> No	
E. Facility provides feedback from audits to personnel regarding their adherence to cleaning and disinfection procedures.	<input type="radio"/> Yes <input type="radio"/> No	
F. Facility has a policy/procedure for decontamination of spills of blood or other body fluids.	<input type="radio"/> Yes <input type="radio"/> No	

X.a. Environmental Cleaning, continued

Operating Room

Elements to be assessed	Assessment	Notes/Areas for Improvement
G. Operating rooms are terminally cleaned after last procedure of the day.	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Not applicable	
H. Facility regularly audits (monitors and documents) adherence to recommended infection control practices for surgical infection prevention including: <ul style="list-style-type: none"> i. Adherence to preoperative surgical scrub and hand hygiene ii. Appropriate use of surgical attire and drapes iii. Adherence to aseptic technique and sterile field iv. Proper ventilation requirements in surgical suites v. Minimization of traffic in the operating room vi. Adherence to cleaning and disinfection of environmental surfaces 	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Not applicable	
I. Facility provides feedback from audits to personnel regarding their adherence to surgical infection prevention practices.	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Not applicable	

XI.a. Device Reprocessing

The following basic information allows for a general assessment of policies and procedures related to reprocessing of reusable medical devices. Outpatient facilities that are performing on-site sterilization or high-level disinfection of reusable medical devices should refer to the more detailed checklists in separate sections of this document devoted to those issues.

Categories of Medical Devices:

- **Critical items** (e.g., surgical instruments) are objects that enter sterile tissue or the vascular system and must be sterile prior to use (see Sterilization Section).
- **Semi-critical items** (e.g., endoscopes for upper endoscopy and colonoscopy, vaginal probes) are objects that contact mucous membranes or non-intact skin and require, at a minimum, high-level disinfection prior to reuse (see High-level Disinfection Section).
- **Non-critical items** (e.g., blood pressure cuffs) are objects that may come in contact with intact skin but not mucous membranes and should undergo cleaning and low- or intermediate-level disinfection depending on the nature and degree of contamination.

Single-use devices (SUDs) are labeled by the manufacturer for a single use and do not have reprocessing instructions. They may *not* be reprocessed for reuse except by entities which have complied with FDA regulatory requirements and have received FDA clearance to reprocess specific SUDs.

Note: Cleaning must always be performed prior to sterilization and disinfection

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Facility has policies and procedures to ensure that reusable medical devices are cleaned and reprocessed appropriately prior to use on another patient.</p> <p><i>Note: This includes clear delineation of responsibility among HCP for cleaning and disinfection of equipment including, non-critical equipment, mobile devices, and other electronics (e.g., point-of-care devices) that might not be reprocessed in a centralized reprocessing area.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>B. The individual(s) in charge of infection prevention at the facility is consulted whenever new devices or products will be purchased or introduced to ensure implementation of appropriate reprocessing policies and procedures.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>C. HCP responsible for reprocessing reusable medical devices receive hands-on training on proper selection and use of PPE and recommended steps for reprocessing assigned devices:</p> <ul style="list-style-type: none"> i. Upon hire, prior to being allowed to reprocess devices ii. Annually iii. When new devices are introduced or policies/procedures change. <p><i>Note: If device reprocessing is performed by contract personnel, facility should verify this is provided by contracting company.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No</p>	
<p>D. HCP are required to demonstrate competency with reprocessing procedures (i.e., correct technique is observed by trainer) following each training.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	

XI.a. Device Reprocessing, continued

Elements to be assessed	Assessment	Notes/Areas for Improvement
E. Facility regularly audits (monitors and documents) adherence to reprocessing procedures.	<input type="radio"/> Yes <input type="radio"/> No	
F. Facility provides feedback from audits to personnel regarding their adherence to reprocessing procedures.	<input type="radio"/> Yes <input type="radio"/> No	
G. Facility has protocols to ensure that HCP can readily identify devices that have been properly reprocessed and are ready for patient use (e.g., tagging system, storage in designated area).	<input type="radio"/> Yes <input type="radio"/> No	
H. Facility has policies and procedures outlining facility response (i.e., risk assessment and recall of device) in the event of a reprocessing error or failure.	<input type="radio"/> Yes <input type="radio"/> No	
I. Routine maintenance for reprocessing equipment (e.g., automated endoscope reprocessors, steam autoclave) is performed by qualified personnel in accordance with manufacturer instructions; confirm maintenance records are available.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

Section 3: Direct Observation of Facility Practices

Certain infection control lapses (e.g., reuse of syringes on more than one patient or to access a medication container that is used for subsequent patients; reuse of lancets) have resulted in bloodborne pathogen transmission and should be halted immediately. Identification of such lapses warrants appropriate notification and testing of potentially affected patients.

If an element is unable to be observed during an assessment (e.g., no patients received point-of-care testing during the visit), assess the element by interviewing appropriate personnel about facility practices. Notation should also be made in the notes section that the element was not able to be directly observed.

V.b. Hand hygiene		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Supplies necessary for adherence to hand hygiene (e.g., soap, water, paper towels, alcohol-based hand rub) are readily accessible to HCP in patient care areas.	<input type="radio"/> Yes <input type="radio"/> No	
Hand hygiene is performed correctly:		
B. Before contact with the patient	<input type="radio"/> Yes <input type="radio"/> No	
C. Before performing an aseptic task (e.g., insertion of IV or preparing an injection)	<input type="radio"/> Yes <input type="radio"/> No	
D. After contact with the patient	<input type="radio"/> Yes <input type="radio"/> No	
E. After contact with objects in the immediate vicinity of the patient	<input type="radio"/> Yes <input type="radio"/> No	
F. After contact with blood, body fluids or contaminated surfaces	<input type="radio"/> Yes <input type="radio"/> No	
G. After removing gloves	<input type="radio"/> Yes <input type="radio"/> No	
H. When moving from a contaminated-body site to a clean-body site during patient care	<input type="radio"/> Yes <input type="radio"/> No	

VI.b. Personal Protective Equipment (PPE)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Sufficient and appropriate PPE is available and readily accessible to HCP.	<input type="radio"/> Yes <input type="radio"/> No	
PPE is used correctly:		
B. PPE, other than respirator, is removed and discarded prior to leaving the patient's room or care area. If a respirator is used, it is removed and discarded (or reprocessed if reusable) <u>after</u> leaving the patient room or care area and closing the door.	<input type="radio"/> Yes <input type="radio"/> No	
C. Hand hygiene is performed immediately after removal of PPE.	<input type="radio"/> Yes <input type="radio"/> No	

VI.b. Personal Protective Equipment (PPE), continued		
Elements to be assessed	Assessment	Notes/Areas for Improvement
D. Gloves		
i. HCP wear gloves for potential contact with blood, body fluids, mucous membranes, non-intact skin, or contaminated equipment.	<input type="radio"/> Yes <input type="radio"/> No	
ii. HCP <u>do not</u> wear the same pair of gloves for the care of more than one patient.	<input type="radio"/> Yes <input type="radio"/> No	
iii. HCP <u>do not</u> wash gloves for the purpose of reuse.	<input type="radio"/> Yes <input type="radio"/> No	
E. Gowns		
i. HCP wear gowns to protect skin and clothing during procedures or activities where contact with blood or body fluids is anticipated.	<input type="radio"/> Yes <input type="radio"/> No Not Applicable <input type="radio"/>	
ii. HCP <u>do not</u> wear the same gown for the care of more than one patient.	<input type="radio"/> Yes <input type="radio"/> No Not Applicable <input type="radio"/>	
F. Facial protection		
i. HCP wear mouth, nose, and eye protection during procedures that are likely to generate splashes or sprays of blood or other body fluids.	<input type="radio"/> Yes <input type="radio"/> No Not Applicable <input type="radio"/>	

VII.b. Injection safety (This element does not include assessment of pharmacy/compounding practices)		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Injections are prepared using aseptic technique in a clean area free from contamination or contact with blood, body fluids or contaminated equipment.	<input type="radio"/> Yes <input type="radio"/> No	
B. Needles and syringes are used for only one patient (this includes manufactured prefilled syringes and cartridge devices such as insulin pens).	<input type="radio"/> Yes <input type="radio"/> No	
C. The rubber septum on a medication vial is disinfected with alcohol prior to piercing.	<input type="radio"/> Yes <input type="radio"/> No	
D. Medication containers are entered with a new needle and a new syringe, even when obtaining additional doses for the same patient.	<input type="radio"/> Yes <input type="radio"/> No	
E. Single dose (single-use) medication vials, ampules, and bags or bottles of intravenous solution are used for only one patient.	<input type="radio"/> Yes <input type="radio"/> No	
F. Medication administration tubing and connectors are used for only one patient.	<input type="radio"/> Yes <input type="radio"/> No	
G. Multi-dose vials are dated by HCP when they are first opened and discarded within 28 days unless the manufacturer specifies a different (shorter or longer) date for that opened vial. <i>Note: This is different from the expiration date printed on the vial.</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/> (Facility does not use multi-dose vials or discards them after single patient use)	

VII.b. Injection safety (This element does not include assessment of pharmacy/compounding practices), continued

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>H. Multi-dose vials to be used for more than one patient are kept in a centralized medication area and <u>do not</u> enter the immediate patient treatment area (e.g., operating room, patient room/cubicle).</p> <p><i>Note: If multi-dose vials enter the immediate patient treatment area they should be dedicated for single-patient use and discarded immediately after use.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/> (Facility does not use multi-dose vials or discards them after single patient use)</p>	
I. All sharps are disposed of in a puncture-resistant sharps container.	<input type="radio"/> Yes <input type="radio"/> No	
J. Filled sharps containers are disposed of in accordance with state regulated medical waste rules.	<input type="radio"/> Yes <input type="radio"/> No	
K. All controlled substances (e.g., Schedule II, III, IV, V drugs) are kept locked within a secure area.	<input type="radio"/> Yes <input type="radio"/> No	
L. HCP wear a facemask (e.g., surgical mask) when placing a catheter or injecting material into the epidural or subdural space (e.g., during myelogram, epidural or spinal anesthesia).	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/> (Facility does not perform spinal injection procedures)</p>	

VIII.b. Respiratory Hygiene/Cough Etiquette

Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Facility:		
<p>i. Posts signs at entrances with instructions to patients with symptoms of respiratory infection to:</p> <p>a. Inform HCP of symptoms of a respiratory infection when they first register for care, and</p> <p>b. Practice Respiratory Hygiene/Cough Etiquette (cover their mouths/noses when coughing or sneezing, use and dispose of tissues, and perform hand hygiene after hands have been covered with respiratory secretions).</p>	<input type="radio"/> Yes <input type="radio"/> No	
ii. Provides tissues and no-touch receptacles for disposal of tissues.	<input type="radio"/> Yes <input type="radio"/> No	
iii. Provides resources for performing hand hygiene in or near waiting areas.	<input type="radio"/> Yes <input type="radio"/> No	

IX.b. Point-of-Care Testing (e.g., blood glucose meters, INR monitor)

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. New single-use, auto-disabling lancing device is used for each patient.</p> <p><i>Note: Lancet holder devices are not suitable for multi-patient use.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/></p>	
<p>B. If used for more than one patient, the point-of-care testing meter is cleaned and disinfected after every use according to manufacturer’s instructions.</p> <p><i>Note: If the manufacturer does not provide instructions for cleaning and disinfection, then the testing meter should not be used for >1 patient.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/></p>	

X.b. Environmental Cleaning

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Supplies necessary for appropriate cleaning and disinfection procedures (e.g., EPA-registered disinfectants) are available.</p> <p><i>Note: If environmental services are performed by contract personnel, facility should verify that appropriate EPA-registered products are provided by contracting company</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>B. High-touch surfaces in rooms where surgical or other invasive procedures (e.g., endoscopy, spinal injections) are performed are cleaned and then disinfected with an EPA-registered disinfectant after each procedure.</p>	<p><input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/></p>	
<p>C. Cleaners and disinfectants are used in accordance with manufacturer’s instructions (e.g., dilution, storage, shelf-life, contact time).</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	
<p>D. HCP engaged in environmental cleaning wear appropriate PPE to prevent exposure to infectious agents or chemicals (PPE can include gloves, gowns, masks, and eye protection).</p> <p><i>Note: The exact type of correct PPE depends on infectious or chemical agent and anticipated type of exposure.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p>	

XI.b. Device Reprocessing		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Policies, procedures, and manufacturer reprocessing instructions for reusable medical devices used in the facility are available in the reprocessing area(s).	<input type="radio"/> Yes <input type="radio"/> No	
B. Reusable medical devices are cleaned, reprocessed (disinfection or sterilization) and maintained according to the manufacturer instructions. <i>Note: If the manufacturer does not provide such instructions, the device may not be suitable for multi-patient use.</i>	<input type="radio"/> Yes <input type="radio"/> No	
C. Single-use devices are discarded after use and not used for more than one patient. <i>Note: If the facility elects to reuse single-use devices, these devices must be reprocessed prior to reuse by a third-party reprocessor that it is registered with the FDA as a third-party reprocessor and cleared by the FDA to reprocess the specific device in question. The facility should have documentation from the third party reprocessor confirming this is the case.</i>	<input type="radio"/> Yes <input type="radio"/> No	
D. Reprocessing area: i. Adequate space is allotted for reprocessing activities. ii. A workflow pattern is followed such that devices clearly flow from high contamination areas to clean/sterile areas (i.e., there is clear separation between soiled and clean workspaces).	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No	
E. Adequate time for reprocessing is allowed to ensure adherence to all steps recommended by the device manufacturer, including drying and proper storage. <i>Note: Facilities should have an adequate supply of instruments for the volume of procedures performed and should schedule procedures to allow sufficient time for all reprocessing steps.</i>	<input type="radio"/> Yes <input type="radio"/> No	
F. HCP engaged in device reprocessing wear appropriate PPE to prevent exposure to infectious agents or chemicals (PPE can include gloves, gowns, masks, and eye protection). <i>Note: The exact type of correct PPE depends on infectious or chemical agent and anticipated type of exposure.</i>	<input type="radio"/> Yes <input type="radio"/> No	
G. Medical devices are stored in a manner to protect from damage and contamination.	<input type="radio"/> Yes <input type="radio"/> No	

XII. Sterilization of Reusable Devices

Note: If all device sterilization is performed off-site, skip to items M-O below.

Elements to be assessed	Assessment	Notes/Areas for Improvement
<p>A. Devices are thoroughly cleaned according to manufacturer instructions and visually inspected for residual soil prior to sterilization.</p> <p><i>Note: Cleaning may be manual (i.e., using friction) and/or mechanical (e.g., with ultrasonic cleaners, washer-disinfector, washer-sterilizers).</i></p> <p><i>Ensure appropriately sized cleaning brushes are selected for cleaning device channels and lumens.</i></p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>B. Cleaning is performed as soon as practical after use (e.g., at the point of use) to prevent soiled materials from becoming dried onto devices.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>C. Enzymatic cleaner or detergent is used for cleaning and discarded according to manufacturer's instructions (typically after each use)</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>D. Cleaning brushes are disposable or, if reusable, cleaned and high-level disinfected or sterilized (per manufacturer's instructions) after use.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>E. After cleaning, instruments are appropriately wrapped/packaged for sterilization (e.g., package system selected is compatible with the sterilization process being performed, items are placed correctly into the basket, shelf or cart of the sterilizer so as not to impede the penetration of the sterilant, hinged instruments are open, instruments are disassembled if indicated by the manufacturer).</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>F. A chemical indicator (process indicator) is placed correctly in the instrument packs in every load.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>G. A biological indicator, intended specifically for the type and cycle parameters of the sterilizer, is used at least weekly for each sterilizer and with every load containing implantable items.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>H. For dynamic air removal-type sterilizers (e.g., prevacuum steam sterilizer), an air removal test (Bowie-Dick test) is performed in an empty dynamic-air removal sterilizer each day the sterilizer is used to verify efficacy of air removal.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>I. Sterile packs are labeled with a load number that indicates the sterilizer used, the cycle or load number, the date of sterilization, and, if applicable, the expiration date.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>J. Sterilization logs are current and include results from each load.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	
<p>K. Immediate-use steam sterilization, if performed, is only done in circumstances in which routine sterilization procedures cannot be performed.</p>	<p><input type="radio"/> Yes <input type="radio"/> No</p> <p>Not applicable <input type="radio"/></p>	

XII. Sterilization of Reusable Devices, continued		
Note: If all device sterilization is performed off-site, skip to items M-O below.		
Elements to be assessed	Assessment	Notes/Areas for Improvement
L. Instruments that undergo immediate-use steam sterilization are used immediately and not stored.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
M. After sterilization, medical devices are stored so that sterility is not compromised.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
N. Sterile packages are inspected for integrity and compromised packages are reprocessed prior to use.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
O. The facility has a process to perform initial cleaning of devices (to prevent soiled materials from becoming dried onto devices) prior to transport to the off-site reprocessing facility.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

XIII. High-Level Disinfection of Reusable Devices		
Note: If all high-level disinfection is performed off-site, skip to items L-N below.		
Elements to be assessed	Assessment	Notes/Areas for Improvement
A. Flexible endoscopes are inspected for damage and leak tested as part of each reprocessing cycle. Any device that fails the leak test is removed from clinical use and repaired.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
B. Devices are thoroughly cleaned according to manufacturer instructions and visually inspected for residual soil prior to high-level disinfection. <i>Note: Cleaning may be manual (i.e., using friction) and/or mechanical (e.g., with ultrasonic cleaners, washer-disinfector, washer-sterilizers).</i> <i>Ensure appropriately sized cleaning brushes are selected for cleaning device channels and lumens.</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
C. Cleaning is performed as soon as practical after use (e.g., at the point of use) to prevent soiled materials from becoming dried onto instruments.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
D. Enzymatic cleaner or detergent is used and discarded according to manufacturer instructions (typically after each use).	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
E. Cleaning brushes are disposable or, if reusable, cleaned and high-level disinfected or sterilized (per manufacturer instructions) after use.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
F. For chemicals used in high-level disinfection, manufacturer instructions are followed for: <ul style="list-style-type: none"> i. Preparation ii. Testing for appropriate concentration iii. Replacement (i.e., upon expiration or loss of efficacy) 	<input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

XIII. High-Level Disinfection of Reusable Devices, continued

Note: If all high-level disinfection is performed off-site, skip to items L-N below.

Elements to be assessed	Assessment	Notes/Areas for Improvement
G. If automated reprocessing equipment is used, proper connectors are used to assure that channels and lumens are appropriately disinfected.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
H. Devices are disinfected for the appropriate length of time as specified by manufacturer instructions.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
I. Devices are disinfected at the appropriate temperature as specified by manufacturer instructions.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
J. After high-level disinfection, devices are rinsed with sterile water, filtered water, or tap water followed by a rinse with 70% - 90% ethyl or isopropyl alcohol. <i>Note: There is no recommendation to use sterile or filtered water rather than tap water for rinsing semi-critical equipment that contact the mucous membranes of the rectum or vagina</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
K. Devices are dried thoroughly prior to reuse. <i>Note: For lumened instruments (e.g., endoscopes) this includes flushing all channels with alcohol and forcing air through channels.</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
L. After high-level disinfection, devices are stored in a manner to protect from damage or contamination. <i>Note: Endoscopes should be hung in a vertical position.</i>	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
M. Facility maintains a log for each endoscopy procedure which includes: patient's name and medical record number (if available), procedure, date, endoscopist, system used to reprocess the endoscope (if more than one system could be used in the reprocessing area), and serial number or other identifier of the endoscope used.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	
N. The facility has a process to perform initial cleaning of devices (to prevent soiled materials from becoming dried onto devices) prior to transport to the off-site reprocessing facility.	<input type="radio"/> Yes <input type="radio"/> No Not applicable <input type="radio"/>	

Section 4: Infection Control Guidelines and Other Resources

- **General Infection Prevention**

- CDC/HICPAC Guidelines and recommendations: http://www.cdc.gov/HAI/prevent/prevent_pubs.html

- **Healthcare Personnel Safety**

- Guideline for Infection Control in Healthcare Personnel: <http://www.cdc.gov/hicpac/pdf/InfectControl98.pdf>
- Immunization of HealthCare Personnel: <http://www.cdc.gov/vaccines/spec-grps/hcw.htm>
- Occupational Safety & Health Administration (OSHA) Bloodborne Pathogens and Needlestick Prevention Standard: <http://www.osha.gov/SLTC/bloodbornepathogens/index.html>
- OSHA Respiratory Protection Standard: [https://www.osha.gov/pls/oshaweb/owadisp.show_document?p_id=12716&p_table=STANDARD S](https://www.osha.gov/pls/oshaweb/owadisp.show_document?p_id=12716&p_table=STANDARD_S)
- OSHA Respirator Fit Testing: https://www.osha.gov/video/respiratory_protection/fittesting_transcript.html

- **Hand Hygiene**

- Guideline for Hand Hygiene in Healthcare Settings: <http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf>
- Hand Hygiene in Healthcare Settings: <http://www.cdc.gov/handhygiene/>

Examples of tools that can be used to conduct a formal audit of hand hygiene practices:

- http://www.jointcommission.org/assets/1/18/hh_monograph.pdf
- <http://comepepi.cs.uiowa.edu/index.php/Research/IScrub>

- **Personal Protective Equipment**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
- Guidance for the Selection and Use of Personal Protective Equipment in Healthcare Settings: <http://www.cdc.gov/HAI/prevent/ppe.html>

- **Injection Safety**

- 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>
- CDC Injection Safety Web Materials: <http://www.cdc.gov/injectionsafety/>

- CDC training video and related Safe Injection Practices Campaign materials: <http://www.oneandonlycampaign.org/>

- **Respiratory Hygiene/Cough Etiquette**
 - 2007 Guidelines for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings: <http://www.cdc.gov/hicpac/pdf/isolation/Isolation2007.pdf>

 - Recommendations for preventing the spread of influenza: <http://www.cdc.gov/flu/professionals/infectioncontrol/>

- **Environmental Cleaning**
 - Guidelines for Environmental Infection Control in Healthcare Facilities: http://www.cdc.gov/hicpac/pdf/guidelines/eic_in_HCF_03.pdf

 - Options for Evaluating Environmental Infection Control: <http://www.cdc.gov/HAI/toolkits/Evaluating-Environmental-Cleaning.html>

- **Equipment Reprocessing**
 - Guideline for Disinfection and Sterilization in Healthcare Facilities: http://www.cdc.gov/hicpac/pdf/guidelines/Disinfection_Nov_2008.pdf

 - FDA regulations on reprocessing of single-use devices: <http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm071434>

- **Point-of-Care Testing**
 - Infection Prevention during Blood Glucose Monitoring and Insulin Administration: <http://www.cdc.gov/injectionsafety/blood-glucose-monitoring.html>

 - Frequently Asked Questions (FAQs) regarding Assisted Blood Glucose Monitoring and Insulin Administration: http://www.cdc.gov/injectionsafety/providers/blood-glucose-monitoring_faqs.html

- **Resources to assist with evaluation and response to breaches in infection control**
 - Patel PR, Srinivasan A, Perz JF. Developing a broader approach to management of infection control breaches in health care settings. Am J Infect Control. 2008 Dec;36(10):685-90

 - Steps for Evaluating an Infection Control Breach: http://www.cdc.gov/hai/outbreaks/steps_for_eval_IC_breach.html

 - Patient Notification Toolkit: <http://www.cdc.gov/injectionsafety/pntoolkit/index.html>



Clostridium difficile (CDI) Infections Toolkit

Activity C: ELC Prevention Collaboratives

Carolyn Gould, MD MSCR

Cliff McDonald, MD, FACP

Division of Healthcare Quality Promotion

Centers for Disease Control and Prevention

Draft - 12/23/09 --- Disclaimer: The findings and conclusions in this presentation are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.





Outline



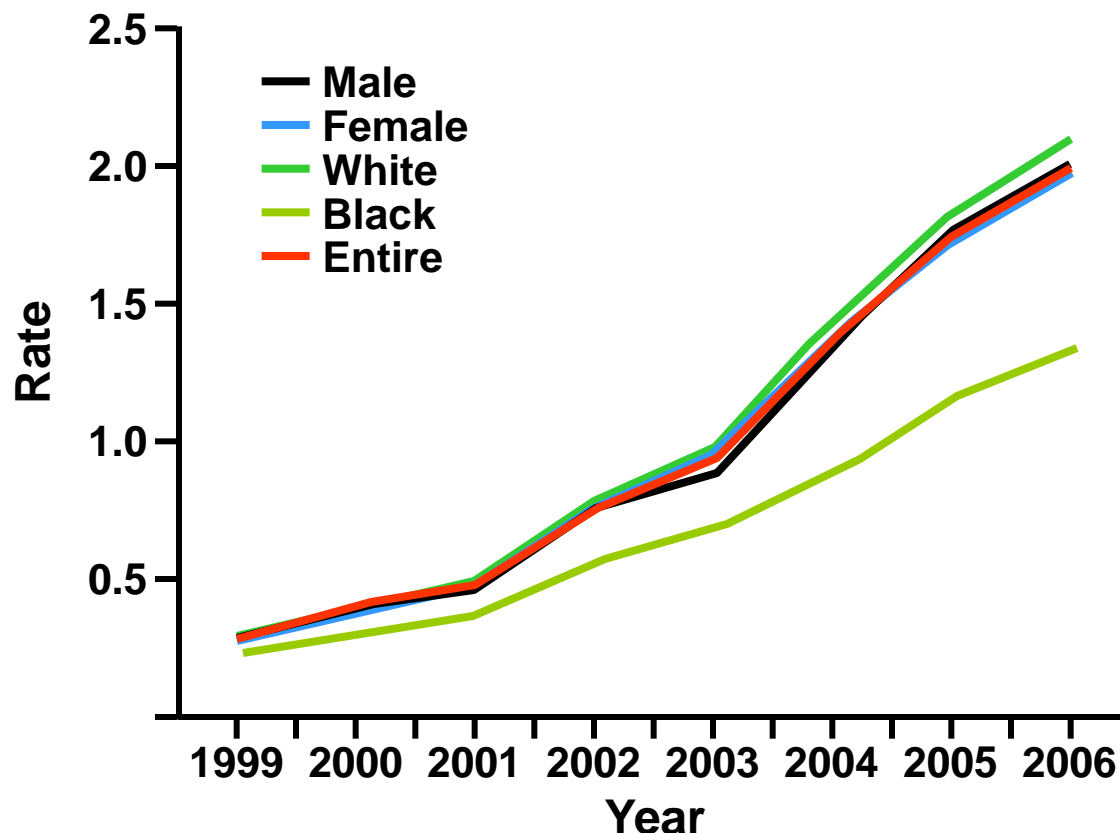
- **Background**
 - Impact
 - HHS Prevention Targets
 - Pathogenesis
 - Epidemiology
- **Prevention Strategies**
 - Core
 - Supplemental
- **Measurement**
 - Process
 - Outcome
- **Tools for Implementation/Resources/References**



Background: Impact



Age-Adjusted Death Rate* for Enterocolitis Due to *C. difficile*, 1999–2006



*Per 100,000 US standard population

Heron et al. Natl Vital Stat Rep 2009;57(14).

Available at http://www.cdc.gov/nchs/data/nvsr/nvsr57/nvsr57_14.pdf



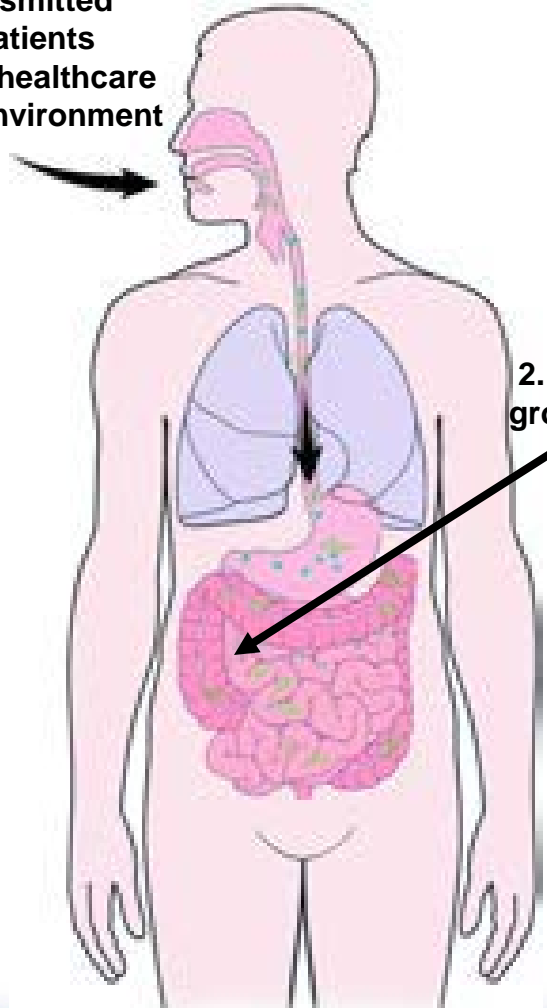
Background: HHS Prevention Targets

- **Case rate per 10,000 patient-days as measured in NHSN**
 - National 5-Year Prevention Target: 30% reduction
- **Because little baseline infection data exists, administrative data for ICD-9-CM coded *C. difficile* hospital discharges is also tracked**
 - National 5-Year Prevention Target: 30% reduction

<http://www.hhs.gov/ophs/initiatives/hai/prevtargets.html>

Background: Pathogenesis of CDI

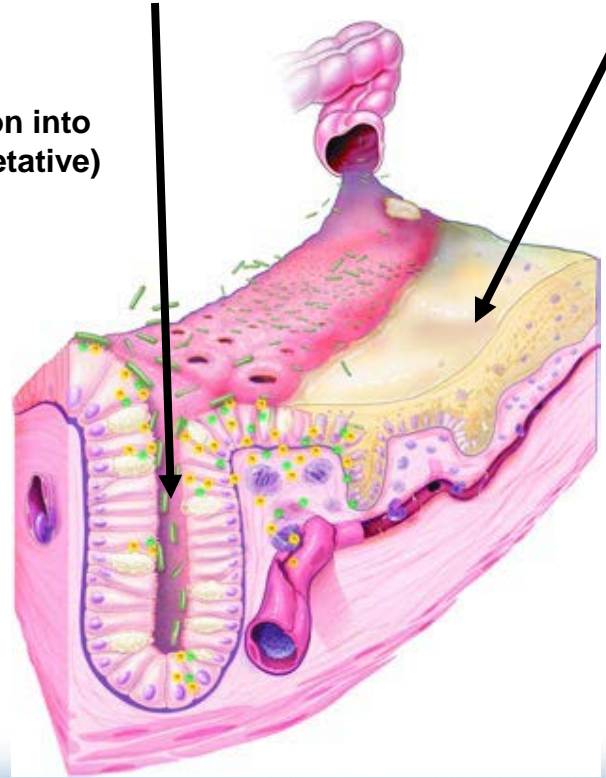
1. Ingestion of spores transmitted from other patients via the hands of healthcare personnel and environment



2. Germination into growing (vegetative) form

3. Altered lower intestine flora (due to antimicrobial use) allows proliferation of *C. difficile* in colon

4. Toxin A & B Production leads to colon damage +/- pseudomembrane



Sunenshine et al. Cleve Clin J Med. 2006;73:187-97.



Background: Epidemiology

Current epidemic strain of *C. difficile*

- BI/NAP1/027, toxinotype III
- Historically uncommon – epidemic since 2000
- More resistant to fluoroquinolones
 - Higher MICs compared to historic strains and current non-BI/NAP1 strains
- More virulent
 - Increased toxin A and B production
 - Polymorphisms in binding domain of toxin B
 - Increased sporulation

McDonald et al. N Engl J Med. 2005;353:2433-41.

Warny et al. Lancet. 2005;366:1079-84.

Stabler et al. J Med Micro. 2008;57:771–5.

Akerlund et al. J Clin Microbiol. 2008;46:1530–3.



Background: Epidemiology Risk Factors



- Antimicrobial exposure
- Acquisition of *C. difficile*
- Advanced age
- Underlying illness
- Immunosuppression
- Tube feeds
- ? Gastric acid suppression

Main modifiable risk factors



Prevention Strategies

- **Core Strategies**

- High levels of scientific evidence
- Demonstrated feasibility

- **Supplemental Strategies**

- Some scientific evidence
- Variable levels of feasibility

The Collaborative should at a minimum include core prevention strategies. Supplemental prevention strategies also may be used. Most core and supplemental strategies are based on HICPAC guidelines. Strategies that are not included in HICPAC guidelines will be noted by an asterisk () after the strategy. HICPAC guidelines may be found at www.cdc.gov/hicpac



Prevention Strategies: Core



- Implement an antimicrobial stewardship program
- Contact Precautions for duration of diarrhea
- Hand hygiene in compliance with CDC/WHO
- Cleaning and disinfection of equipment and environment
- Laboratory-based alert system for immediate notification of positive test results
- Educate about CDI: HCP, housekeeping, administration, patients, families

http://www.cdc.gov/ncidod/dhqp/id_CdiffFAQ_HCP.html

Dubberke et al. Infect Control Hosp Epidemiol 2008;29:S81-92.



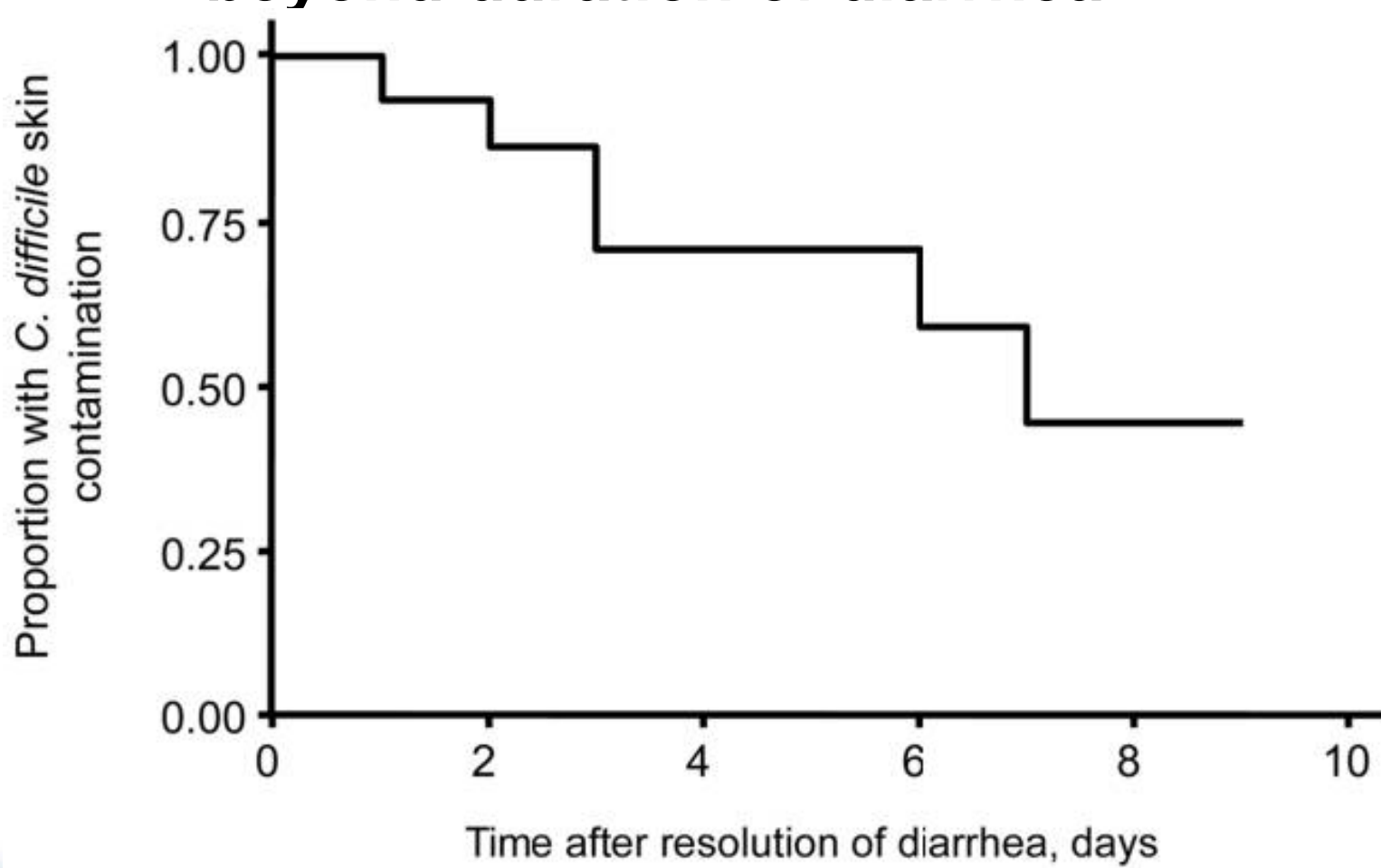
Prevention Strategies: Supplemental

- Extend use of Contact Precautions beyond duration of diarrhea (e.g., 48 hours)*
- Presumptive isolation for symptomatic patients pending confirmation of CDI
- Evaluate and optimize testing for CDI
- Implement soap and water for hand hygiene before exiting room of a patient with CDI
- Implement universal glove use on units with high CDI rates*
- Use sodium hypochlorite (bleach) – containing agents for environmental cleaning

* Not included in CDC/HICPAC 2007 Guideline for Isolation Precautions



Supplemental Prevention Strategies: Rationale for considering extending isolation beyond duration of diarrhea



Bobulsky et al. Clin Infect Dis 2008;46:447-50.



Supplemental Prevention Strategies:



Consider presumptive isolation for patients with ≥ 3 unformed stools within 24 hours

- Patients with CDI may contaminate environment and hands of healthcare personnel pending results of diagnostic testing
- CDI responsible for only ~30-40% of hospital-onset diarrhea
- However, CDI more likely among patients with ≥ 3 unformed (i.e. taking the shape of a container) stools within 24 hours
 - Send specimen for testing and presumptively isolate patient pending results
 - Positive predictive value of testing will also be optimized if focused on patients with ≥ 3 unformed stools within 24 hours
 - Exception: patient with possible recurrent CDI (isolate and test following first unformed stool)



Supplemental Prevention Strategies:

Evaluate and optimize test-ordering practices and diagnostic methods



- Most laboratories have relied on Toxin A/B enzyme immunoassays
 - Low sensitivities (70-80%) lead to low negative predictive value
- Despite high specificity, poor test ordering practices (i.e. testing formed stool or repeat testing in negative patients) may lead to many false positives
- Consider more sensitive diagnostic paradigms but apply these more judiciously across the patient population
 - Employ a highly sensitive screen with confirmatory test or a PCR-based molecular assay
 - Restrict testing to unformed stool only
 - Focus testing on patients with ≥ 3 unformed stools within 24 hours
 - Require expert consultation for repeat testing within 5 days

Peterson et al. Ann Intern Med 2009;15:176-9.



Supplemental Prevention Strategies: Hand Hygiene – Soap vs. Alcohol gel



- Alcohol not effective in eradicating *C. difficile* spores
- However, one hospital study found that from 2000-2003, despite increasing use of alcohol hand rub, there was no concomitant increase in CDI rates
- Discouraging alcohol gel use may undermine overall hand hygiene program with untoward consequences for HAIs in general

Boyce et al. Infect Control Hosp Epidemiol 2006;27:479-83.



Supplemental Prevention Strategies: Hand Washing: Product Comparison



Product	Log10 Reduction
Tap Water	0.76
4% CHG antimicrobial hand wash	0.77
Non-antimicrobial hand wash	0.78
Non-antimicrobial body wash	0.86
0.3% triclosan antimicrobial hand wash	0.99
Heavy duty hand cleaner used in manufacturing environments	1.21*

* Only value that was statistically better than others

Conclusion: Spores may be difficult to eradicate even with hand washing.

Edmonds, et al. Presented at: SHEA 2009; Abstract 43.



Supplemental Prevention Strategies: Hand Hygiene Methods



Since spores may be difficult to remove from hands even with hand washing, adherence to glove use, and Contact Precautions in general, should be emphasized for preventing *C. difficile* transmission via the hands of healthcare personnel

Johnson et al. Am J Med 1990;88:137-40.



Supplemental Prevention Strategies: **Glove Use**



Rationale for considering universal glove use (in addition to Contact Precautions for patients with known CDI) on units with high CDI rates

- Although the magnitude of their contribution is uncertain, asymptomatic carriers have a role in transmission
- Practical screening tests are not available
- There may be a role for universal glove use as a special approach to reducing transmission on units with longer lengths of stay and high endemic CDI rates
- Focus enhanced environmental cleaning strategies and avoid shared medical equipment on such units as well



Supplemental Prevention Strategies: Environmental Cleaning



- Bleach can kill spores, whereas other standard disinfectants cannot
- Limited data suggest cleaning with bleach (1:10 dilution prepared fresh daily) reduces *C. difficile* transmission
- Two before-after intervention studies demonstrated benefit of bleach cleaning in units with high endemic CDI rates
- Therefore, bleach may be most effective in reducing burden where CDI is highly endemic

Mayfield et al. Clin Infect Dis 2000;31:995-1000.

Wilcox et al. J Hosp Infect 2003;54:109-14.



Supplemental Prevention Strategies: Environmental Cleaning



Assess adequacy of cleaning before changing to new cleaning product such as bleach

- Ensure that environmental cleaning is adequate and high-touch surfaces are not being overlooked
- One study using a fluorescent environmental marker to assess cleaning showed:
 - only 47% of high-touch surfaces in 3 hospitals were cleaned
 - sustained improvement in cleaning of all objects, especially in previously poorly cleaned objects, following educational interventions with the environmental services staff
- The use of environmental markers is a promising method to improve cleaning in hospitals.

Carling et al. Clin Infect Dis 2006;42:385-8.



Summary of Prevention Measures

Core Measures

- Contact Precautions for duration of illness
- Hand hygiene in compliance with CDC/WHO
- Cleaning and disinfection of equipment and environment
- Laboratory-based alert system
- CDI surveillance
- Education

Supplemental Measures

- Prolonged duration of Contact Precautions*
- Presumptive isolation
- Evaluate and optimize testing
- Soap and water for HH upon exiting CDI room
- Universal glove use on units with high CDI rates*
- Bleach for environmental disinfection
- Antimicrobial stewardship program

* Not included in CDC/HICPAC 2007 Guideline for Isolation Precautions



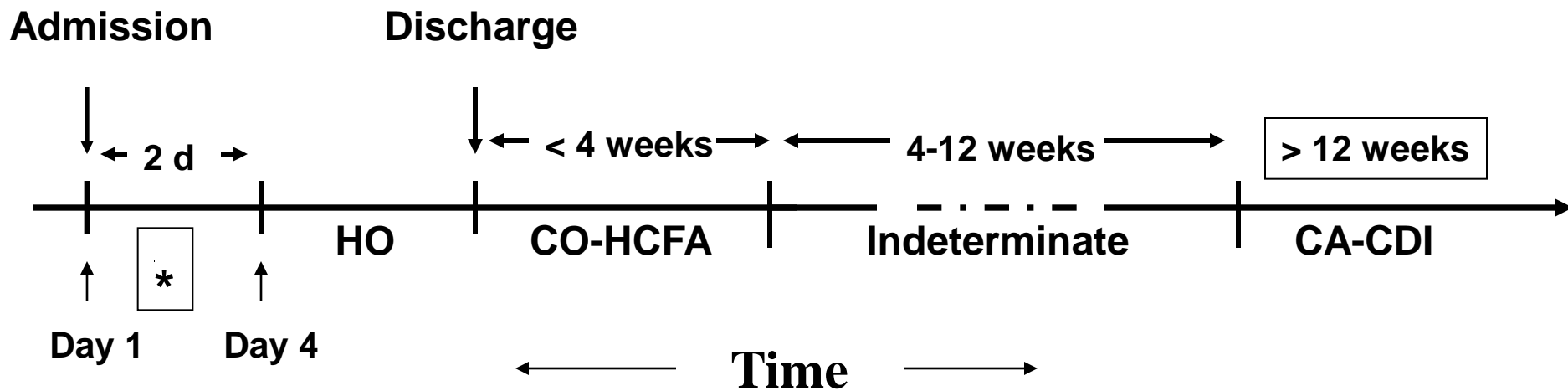
Measurement: Process Measures

- **Core Measures:**
 - Measure compliance with CDC/WHO recommendations for hand hygiene and Contact Precautions
 - Assess adherence to protocols and adequacy of environmental cleaning
- **Supplemental Measures:**
 - Intensify assessment of compliance with process measures
 - Track use of antibiotics associated with CDI in a facility



Measurement: Outcome

Categorize Cases by location and time of onset†



HO: Hospital (Healthcare)-Onset

CO-HCFA: Community-Onset, Healthcare Facility-Associated

CA: Community-Associated

* Depending upon whether patient was discharged within previous 4 weeks, CO-HCFA vs. CA

† Onset defined in NHSN LabID Event by specimen collection date

Modified from CDAD Surveillance Working Group. Infect Control Hosp Epidemiol 2007;28:140-5.



Measurement: Outcome

Use NHSN CDAD Module



Laboratory-identified MDRO or CDAD Event

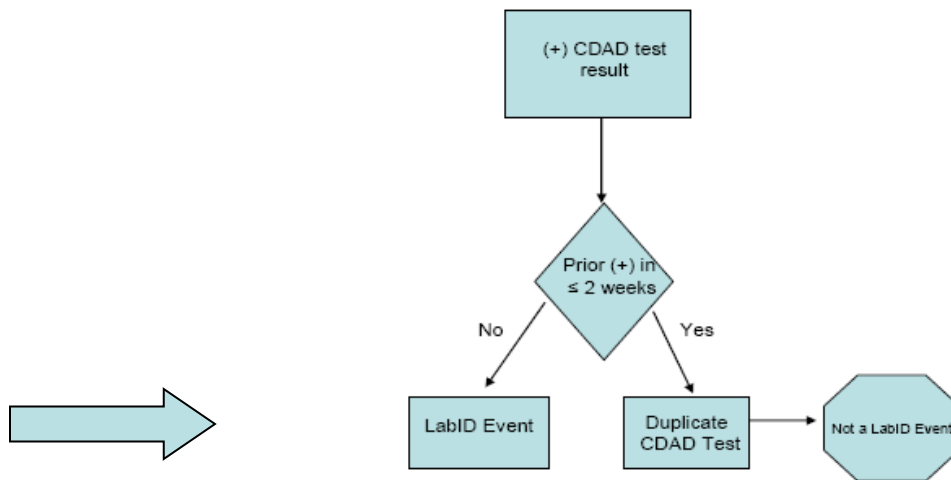
OMB No. 0920-0047
Exp. Date: 03-31-2017

*required for saving	
Facility ID:	Event #:
*Patient ID:	Social Security #:
Secondary ID:	
Patient Name, Last:	First: Middle:
*Gender: M F	*Date of Birth:
Ethnicity (Specify):	Race (Specify):
Event Details	
*Event Type: LabID	*Date Specimen Collected:
*Specific Organism Type: (Check one)	
<input type="checkbox"/> MRSA <input type="checkbox"/> MSSA <input type="checkbox"/> VRE <input type="checkbox"/> MDR- <i>Klebsiella</i> <input type="checkbox"/> MDR- <i>Acinetobacter</i> <input type="checkbox"/> C. <i>difficile</i>	
*Outpatient: Yes No	*Specimen Source:
*Date Admitted	*Location: *Date Admitted

Measurement: Outcome

Focus on Laboratory Identified (LabID) Events in NHSN

Figure 2. CDAD Test Result Algorithm for Laboratory-Identified (LabID) Events





Measurement: Outcome

NHSN Reporting: Definitions



Based on data submitted to NHSN, CDI LabID Events are categorized as:

- **Incident:** specimen obtained >8 weeks after the most recent LabID Event
- **Recurrent:** specimen obtained >2 weeks and ≤ 8 weeks after most recent LabID Event



Measurement: Outcome

NHSN Reporting: Definitions



Incident cases further characterized based on date of admission and date of specimen collection:

- **Healthcare Facility-Onset (HO):** LabID Event collected >3 days after admission to facility (i.e., on or after day 4)
- **Community-Onset (CO):** LabID Event collected as an outpatient or an inpatient ≤ 3 days after admission to the facility (i.e., days 1, 2, or 3 of admission)
- **Community-Onset Healthcare Facility-Associated (CO-HCFA):** CO LabID Event collected from a patient who was discharged from the facility ≤ 4 weeks prior to date stool specimen collected



Measurement: Outcome



Calculating CDI Incidence Rates*

- **Healthcare Facility-Onset Incidence Rate** =
Number of all Incident HO CDI LabID Events per
patient per month / Number of patient days for
the facility x 10,000
- **Combined Incidence Rate** = Number of all
Incident HO and CO-HCFA CDI LabID Events
per patient per month / Number of patient days
for the facility x 10,000

*For a given healthcare facility



Evaluation Considerations

- **Assess baseline policies and procedures**
- **Areas to consider**
 - **Surveillance**
 - **Prevention strategies**
 - **Measurement of effect of strategies**
- **Coordinator should track new policies/practices implemented during collaboration**



References

- Dubberke ER, Butler AM, Reske KA, et al. attributable outcomes of endemic *Clostridium difficile*-associated disease in nonsurgical patients. *Emerg Infect Dis* 2008;14:1031-8.
- Dubberke ER, Reske KA, Olssen MA, et al. Short- and long term attributable costs of *Clostridium difficile*-associated disease in nonsurgical inpatients. *Clin Infect Dis* 2008;46:497-504.
- Edmonds S, Kasper D, Zepka C, et al. *Clostridium difficile* and hand hygiene: spore removal effectiveness of handwash products. Presented at: SHEA 2009; Abstract 43.



References

- Elixhauser, A. (AHRQ), and Jhung, MA. (Centers for Disease Control and Prevention). *Clostridium Difficile-Associated Disease in U.S. Hospitals, 1993–2005*. HCUP Statistical Brief #50. April 2008. Agency for Healthcare Research and Quality, Rockville, MD. <http://www.hcup-us.ahrq.gov/reports/statbriefs/sb50.pdf>
- Fowler S, Webber A, Cooper BS, et al. Successful use of feedback to improve antibiotic prescribing and reduce *Clostridium difficile* infection: a controlled interrupted time series. *J Antimicrob Chemother* 2007;59:990-5.
- Heron MP, Hoyert D, Murphy SL, et al. *Natl Vital Stat Rep* 2009;57(14). US Dept of Health and Human Services, CDC; 2009. Available at http://www.cdc.gov/nchs/data/nvsr/nvsr57/nvsr57_14.pdf



References

- Johnson S, Gerding DN, Olson MM, et al. Prospective, controlled study of vinyl glove use to interrupt *Clostridium difficile* nosocomial transmission. *Am J Med* 1990;88:137-40.
- Mayfield JL, Leet T, Miller J, et al. Environmental control to reduce transmission of *Clostridium difficile*. *Clin Infect Dis* 2000;31:995–1000.
- McDonald LC, Killgore GE, Thompson A, et al. An epidemic, toxin gene–variant strain of *Clostridium difficile*. *N Engl J Med*. 2005;353:2433-41.



References

- McDonald LC, Coignard B, Dubberke E, et al. Ad Hoc CDAD Surveillance Working Group. Recommendations for surveillance of *Clostridium difficile*-associated disease. *Infect Control Hosp Epidemiol* 2007; 28:140-5.
- Oughton MT, Loo VG, Dendukuri N, et al. Hand hygiene with soap and water is superior to alcohol rum and antiseptic wipes for removal of *Clostridium difficile*. *Infect Control Hosp Epidemiol* 2009; 30:939-44.
- Peterson LR, Robicsek A. Does my patient have *Clostridium difficile* infection? *Ann Intern Med* 2009;15:176-9
- Riggs MM, Sethi AK, Zabarsky TF, et al. Asymptomatic carriers are a potential source for transmission of epidemic and nonepidemic *Clostridium difficile* strains among long-term care facility residents. *Clin Infect Dis* 2007; 45:992–8.



References

- SHEA/IDSA Compendium of Recommendations. Infect Control Hosp Epidemiol 2008;29:S81–S92.
<http://www.journals.uchicago.edu/doi/full/10.1086/591065>
- Stabler RA, Dawson LF, Phua LT, et al. Comparative analysis of BI/NAP1/027 hypervirulent strains reveals novel toxin B-encoding gene (tcdB) sequences. J Med Micro. 2008;57:771–5.
- Sunenshine RH, McDonald LC. *Clostridium difficile*-associated disease: new challenges from and established pathogen. Cleve Clin J Med. 2006;73:187-97.



References

- Warny M, Pepin J, Fang A, Killgore G, et al. Toxin production by and emerging strain of *Clostridium difficile* associated with outbreaks of severe disease in North America and Europe. Lancet. 2005;366:1079-84.
- Wilcox MF, Fawley WN, Wigglesworth N, et al. Comparison of the effect of detergent versus hypochlorite cleaning on environmental contamination and incidence of *Clostridium difficile* infection. *J Hosp Infect* 2003;54:109-14.



Additional resources



SHEA/IDSA Compendium of Recommendations

CDI Checklist Example

S81 INFECTION CONTROL AND HOSPITAL EPIDEMIOLOGY OCTOBER 2008, VOL. 29, SUPPLEMENT 1

SUPPLEMENT ARTICLE: SHEA/IDSA PRACTICE RECOMMENDATION

Strategies to Prevent *Clostridium difficile* Infections in Acute Care Hospitals

Erik R. Dubberke, MD; Dale N. Gerding, MD; David Classen, MD, MS; Kathleen M. Arias, MS, CIC;
 Kelly Podgorny, RN, MS, CPHQ; Deverick J. Anderson, MD, MPH; Helen Burstin, MD; David P. Calfee, MD, MS;
 Susan E. Coffin, MD, MPH; Victoria Fraser, MD; Frances A. Griffin, RRT, MPA; Peter Gross, MD; Keith S. Kaye, MD;
 Michael Klompas, MD; Evelyn Lo, MD; Jonas Marschall, MD; Leonard A. Mermel, DO, ScM; Lindsay Nicolle, MD;
 David A. Pegues, MD; Trish M. Perl, MD; Sanjay Saint, MD; Cassandra D. Salgado, MD, MS;
 Robert A. Weinstein, MD; Robert Wise, MD; Deborah S. Yokoe, MD, MPH

Clostridium difficile Infection (CDI) Checklist

Hospital interventions to decrease the incidence and mortality of healthcare-associated *C. difficile* infections

Prevention Checklist	Treatment Checklist
<ul style="list-style-type: none"> • When an MD, PA, NP, or RN suspects a patient has CDI: <ul style="list-style-type: none"> Physician, Physician Assistant, or Nurse Practitioner: <ul style="list-style-type: none"> <input type="checkbox"/> Initiate <i>Contact Precautions Plus</i> <input type="checkbox"/> Order stool <i>C. difficile</i> toxin testing <input type="checkbox"/> Discontinue non-essential antimicrobials <input type="checkbox"/> Discontinue all anti-peristaltic medications Registered Nurse: <ul style="list-style-type: none"> <input type="checkbox"/> Obtain stool sample for <i>C. difficile</i> toxin test <input type="checkbox"/> Place patient in single-patient room <input type="checkbox"/> Place <i>Contact Precautions Plus</i> sign on patient's door <input type="checkbox"/> Ensure that gloves and gowns are easily accessible from patient's room <input type="checkbox"/> Place dedicated stethoscope in patient's room <input type="checkbox"/> Remind staff to wash hands with soap and water following patient contact Microbiology Laboratory Staff Person: <ul style="list-style-type: none"> <input type="checkbox"/> Call relevant patient floor with positive <i>C. difficile</i> toxin test result <input type="checkbox"/> Provide daily list of positive test results for Infection Control Infection Control Practitioner: <ul style="list-style-type: none"> <input type="checkbox"/> Check microbiology results daily for positive <i>C. difficile</i> toxin results <input type="checkbox"/> Call relevant floor to confirm that patient with positive <i>C. difficile</i> toxin results is in a single-patient room and that the <i>Contact Precautions Plus</i> sign is on the patient's door <input type="checkbox"/> Flag the patient's <i>C. difficile</i> status in the hospital's clinical information system or in the patient's paper chart <input type="checkbox"/> Alert housekeeping that the patient is on <i>Contact Precautions Plus</i> Environmental Services Staff Person: <ul style="list-style-type: none"> <input type="checkbox"/> Prior to discharge cleaning, check for <i>Contact Precautions Plus</i> sign on the patient's door <input type="checkbox"/> If <i>Contact Precautions Plus</i> sign is on the door, clean the room with a bleach-based cleaning agent <input type="checkbox"/> Confirm for supervisor that bleach-based cleaning agent was used for discharge cleaning for every patient on <i>Contact Precautions Plus</i> 	<ul style="list-style-type: none"> • When an MD, PA, or NP diagnoses mild CDI: <i>All of the following criteria are present: diarrhea (>3 BM/day), no fever, WBC<15,000, no peritoneal signs, and no evidence of sepsis</i> Physician, Physician Assistant, or Nurse Practitioner: <ul style="list-style-type: none"> <input type="checkbox"/> Initiate oral metronidazole at dose 500mg every 8 hours <input type="checkbox"/> If no clinical improvement by 48-72 hours after diagnosis, treat patient as moderate CDI <input type="checkbox"/> Continue therapy for at least 14 days total and at least 10 days after symptoms have abated • When an MD, PA, or NP diagnoses moderate CDI: <i>At least one of the following criteria is present: diarrhea (6-12 BM/day), fever 37.5-38.5°C, WBC 15,000-25,000, or frankly visible stable lower gastrointestinal bleeding</i> Physician, Physician Assistant, or Nurse Practitioner: <ul style="list-style-type: none"> <input type="checkbox"/> Initiate oral vancomycin at dose 250mg every 6 hours <input type="checkbox"/> If no clinical improvement by 48 hours, add IV metronidazole at dose 500mg every 8 hours <input type="checkbox"/> Consider obtaining infectious disease consultation <input type="checkbox"/> Consider obtaining abdominal CT scan <input type="checkbox"/> Continue therapy for at least 14 days total and at least 10 days after symptoms have abated • When an MD, PA, or NP diagnoses severe CDI: <i>At least one of the following criteria is present: diarrhea (>12 BM/day), fever >38.5°C, WBC >25,000, hemodynamic instability, marked & continuous abdominal pain, ileus, absence of bowel sounds, evidence of sepsis, or intensive care unit level of care required</i> Physician, Physician Assistant, or Nurse Practitioner: <ul style="list-style-type: none"> <input type="checkbox"/> Obtain immediate infectious disease consultation <input type="checkbox"/> Obtain immediate general surgery consultation <input type="checkbox"/> Obtain abdominal CT scan <input type="checkbox"/> Initiate oral vancomycin at dose 250mg every 6 hours together with IV metronidazole at dose 500mg every 6 hours <input type="checkbox"/> Following consultation with general surgery regarding its use, consider rectal vancomycin <input type="checkbox"/> Ask general surgery service to assess the need for colectomy

Abbreviations: MD=medical doctor, PA=physician assistant, NP=nurse practitioner, RN=registered nurse, BM=bowel movement, WBC=white blood cell count, CT=computed tomography, IV=intravenous

FIGURE 1. *Clostridium difficile* infection checklist at Brigham and Women's Hospital.

Dubberke et al. Infect Control Hosp Epidemiol 2008;29:S81-92.
 Abbett SK et al. Infect Control Hosp Epidemiol 2009;30:1062-9.





Additional Reference Slides



- The following slides may be used for presentations regarding CDI.
- Explanations are available in the notes section of the slides.



Supplemental Prevention Strategies: Rationale for Soap and Water: Lack of efficacy of alcohol-based handrub against *C. difficile*

Interventions compared		Mean log reduction (95% CI), log ₁₀ CFU/mL
Intervention 1	Intervention 2	
Warm water and plain soap	No hand hygiene	2.14 (1.74–2.54)
Warm water and plain soap	Alcohol-based handrub	2.08 (1.69–2.47)
Cold water and plain soap	No hand hygiene	1.88 (1.48–2.28)
Cold water and plain soap	Alcohol-based handrub	1.82 (1.43–2.22)
Warm water and plain soap	Antiseptic hand wipe	1.57 (1.18–1.96)
Warm water and antibacterial soap	No hand hygiene	1.51 (1.12–1.91)
Warm water and antibacterial soap	Alcohol-based handrub	1.46 (1.06–1.85)
Cold water and plain soap	Antiseptic hand wipe	1.31 (0.92–1.71)
Warm water and antibacterial soap	Antiseptic hand wipe	0.94 (0.55–1.34)
Warm water and plain soap	Warm water and antibacterial soap	0.63 (0.23–1.02)
Antiseptic hand wipe	No hand hygiene	0.57 (0.17–0.96)
Antiseptic hand wipe	Alcohol-based handrub	0.51 (0.12–0.91)
Cold water and plain soap	Warm water and antibacterial soap	0.37 (–0.03 to 0.76)
Warm water and plain soap	Cold water and plain soap	0.26 (–0.14 to 0.66)
Alcohol-based handrub	No hand hygiene	0.06 (–0.34 to 0.45)

Oughton et al. Infect Control Hosp Epidemiol 2009;30:939-44.



Supplemental Prevention Strategies: Hand Hygiene – Alcohol Hand Rub Use 2000-2003

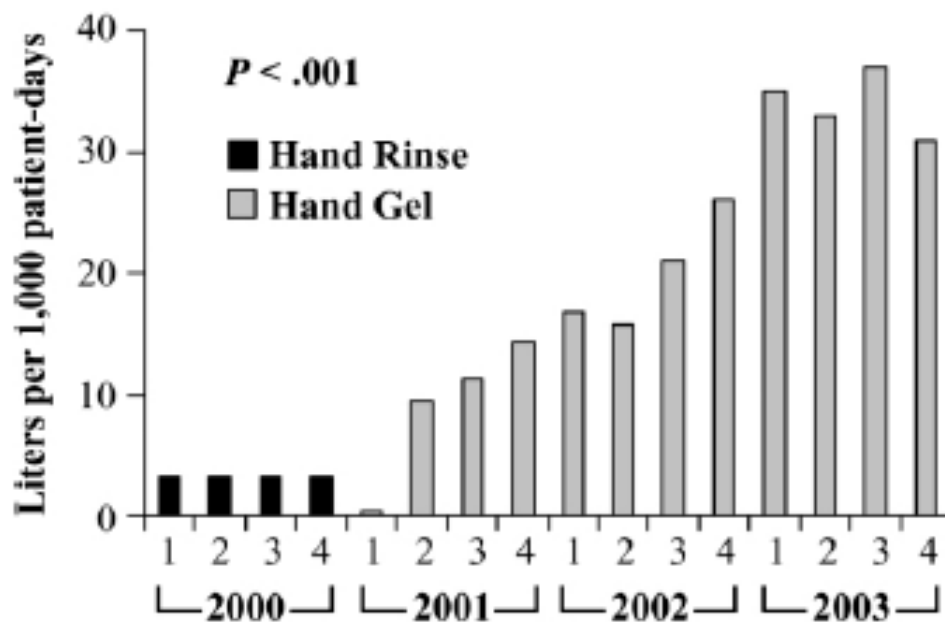


FIGURE 1. Use of alcohol hand rub by healthcare workers, in liters per 1,000 patient-days, per quarter, 2000-2003.

Boyce et al. Infect Control Hosp Epidemiol 2006; 27:479-83.



Supplemental Prevention Strategies: Hand Hygiene – CDI Rates 2000-2003

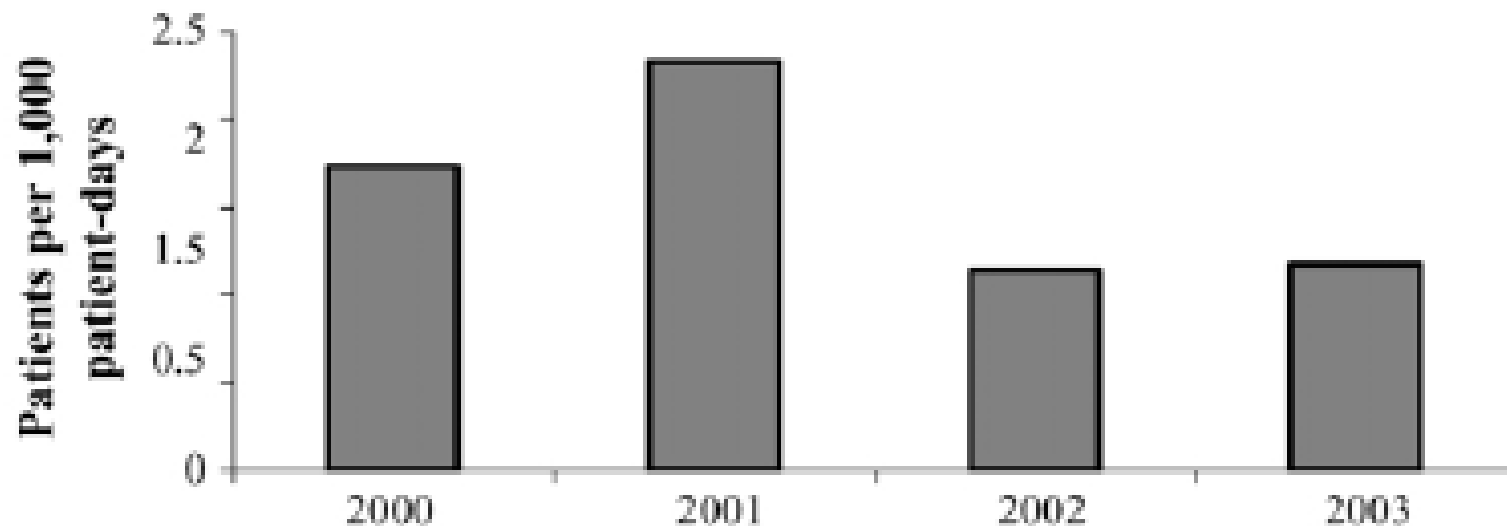


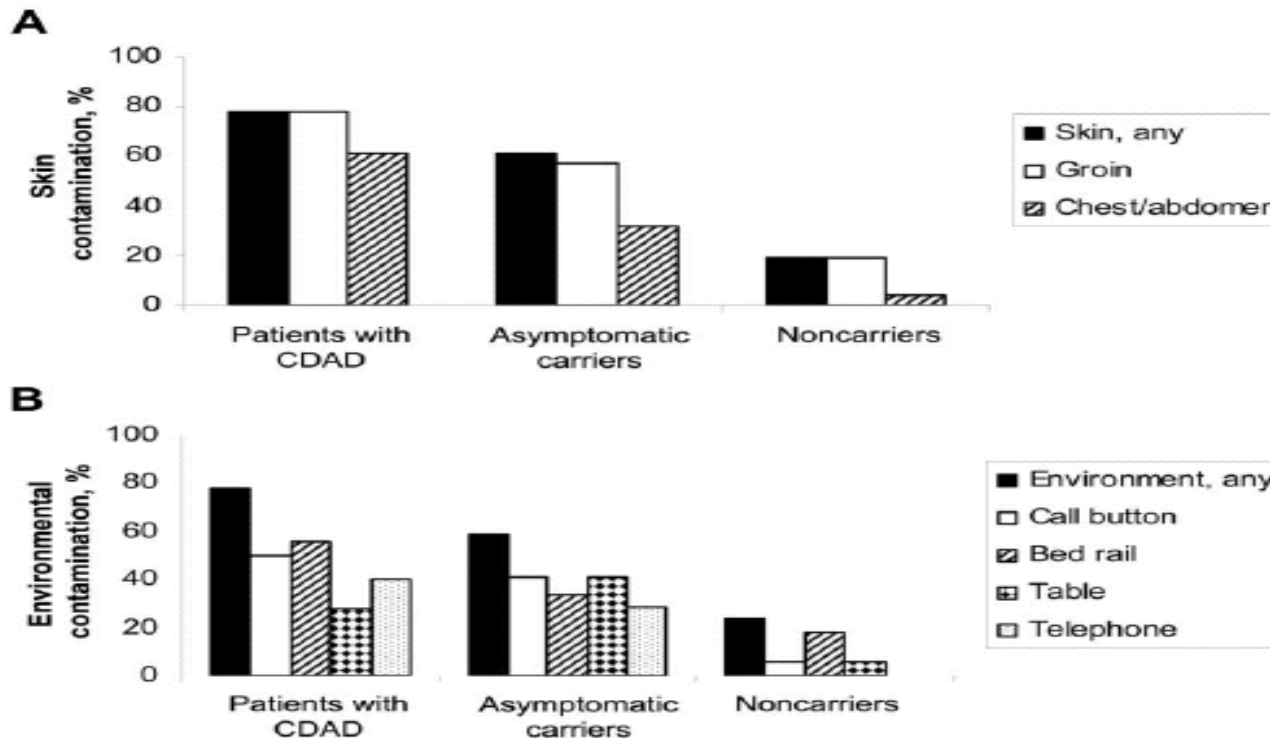
FIGURE 2. Number of patients with 1 or more tests positive for *Clostridium difficile* toxin per 1,000 patient-days, 2000-2003.

Boyce JM et al. Infect Control Hosp Epidemiol 2006; 27:479-83.

Supplemental Prevention Strategies: Universal Glove Use

Role of asymptomatic carriers?

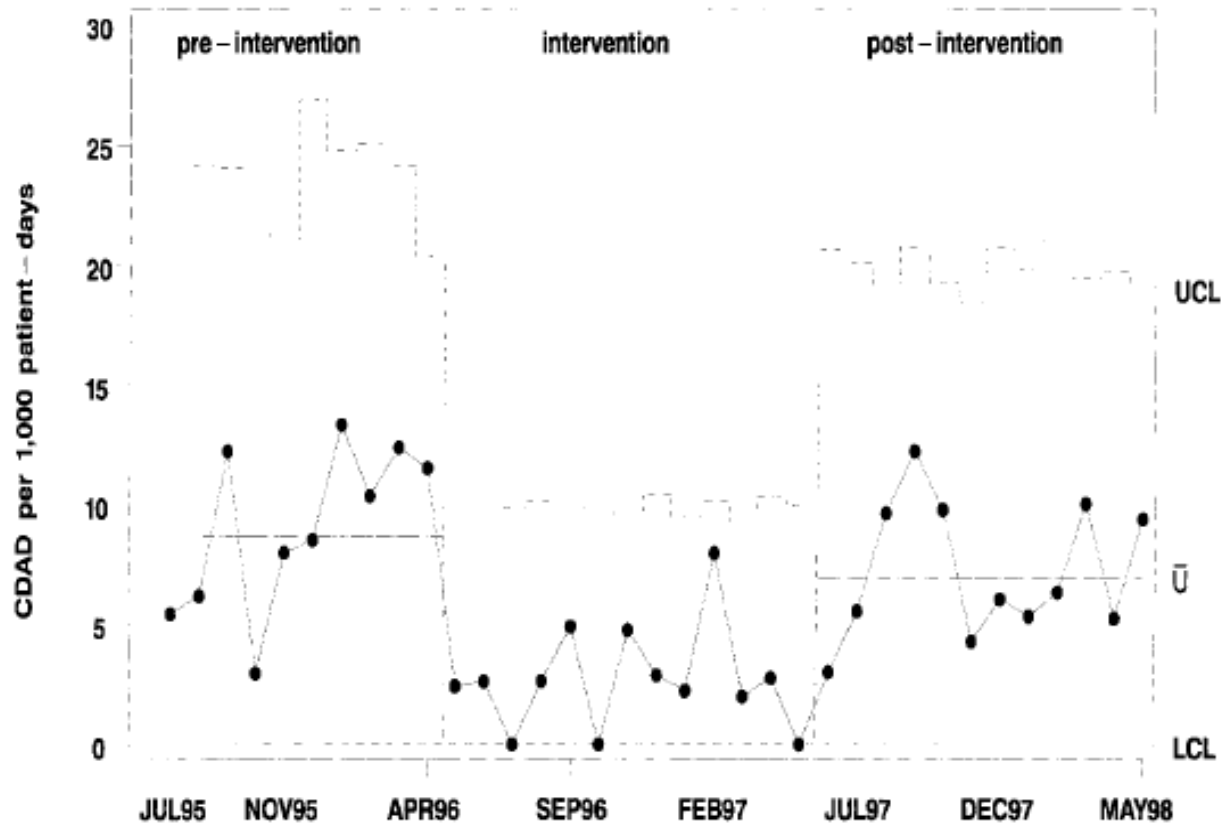
Rationale for universal glove use on units with high CDI rates



Riggs et al. Clin Infect Dis 2007;45:992–8.

Supplemental Prevention Strategies: Environmental Cleaning

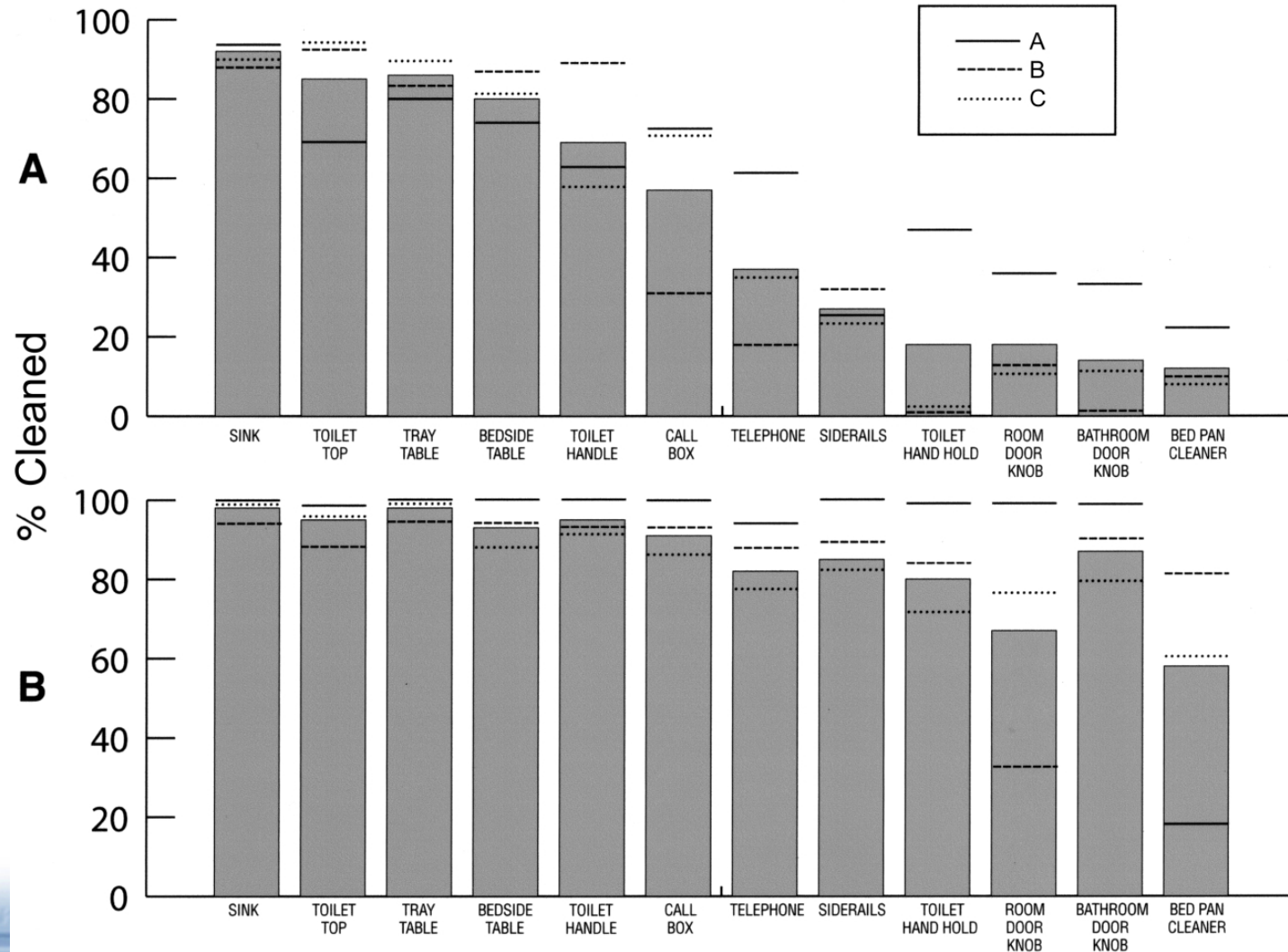
How Much Can be Achieved via Environmental Decontamination?



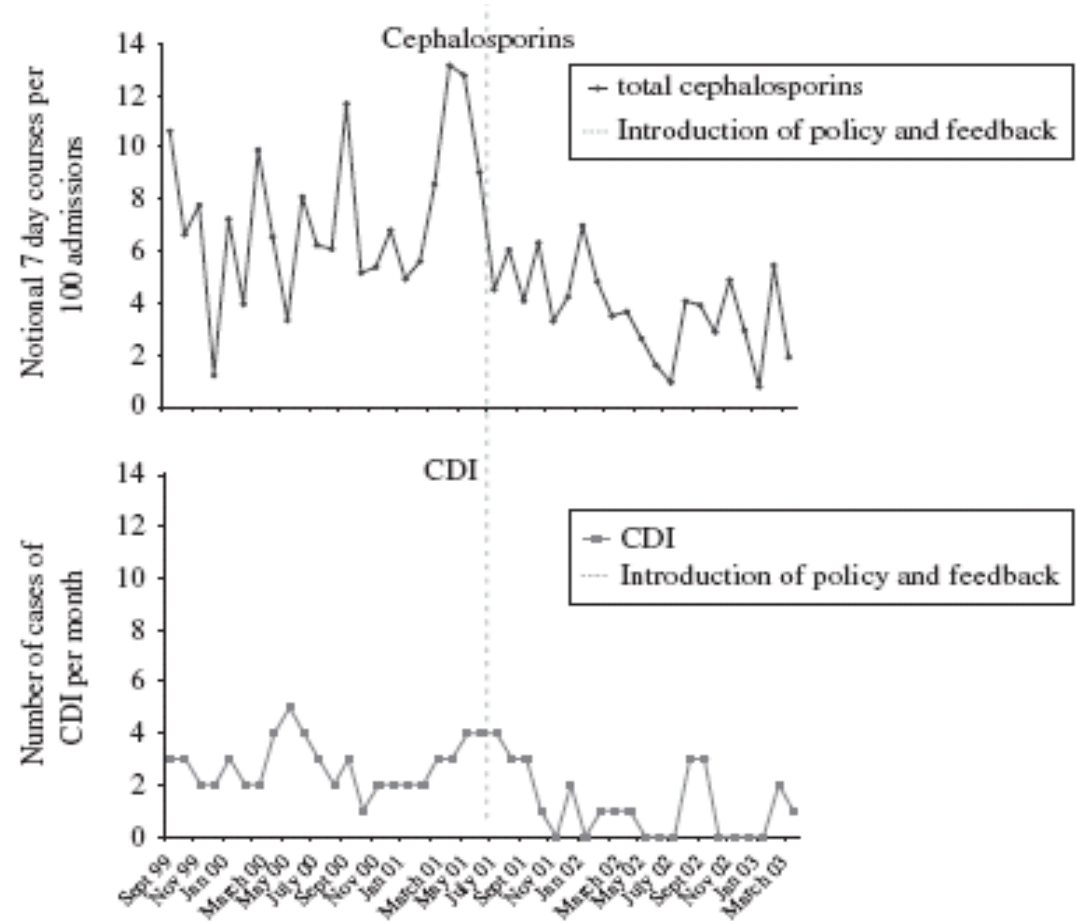
Mayfield et al. Clin Infect Dis 2000;31:995–1000.

Supplemental Prevention Strategies: Environmental Cleaning

Assess adequacy of cleaning before changing to new cleaning



Supplemental Prevention Strategies: Audit and feedback targeting broad-spectrum antibiotics



Fowler et al. J Antimicrob Chemother 2007;59:990-5.



Central Line-Associated Bloodstream Infections (CLABSI) in Non-Intensive Care Unit (non-ICU) Settings Toolkit

Activity C: ELC Prevention Collaboratives

Alex Kallen, MD, MPH and Priti Patel, MD, MPH

Division of Healthcare Quality Promotion

Centers for Disease Control and Prevention

Draft - 1/22111/09 --- Disclaimer: The findings and conclusions in this presentation are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.





Outline



- **Background**
 - Impact
 - HHS Prevention Targets
 - Pathogenesis
 - Epidemiology
- **Prevention Strategies**
 - Core
 - Supplemental
- **Measurement**
 - Process
 - Outcome
- **Tools for Implementation/Resources/References**



Background: Impact



- Bloodstream infections (BSIs) are a major cause of healthcare-associated morbidity and mortality
 - Up to 35% attributable mortality
 - BSI leads to excess hospital length of stay of 24 days
- Central Line (CL) use a major risk factor for BSI
- More than 250,000 central line-associated BSIs (CLABSIs) in US yearly
- Rates of CLABSI appear to vary by type of catheter

Pittet et al. JAMA 1994; 271 1598-1601.

Klevens et al. Public Health Reports 2007;122:160-6.



Background: HHS Prevention Targets



- Prevention of CLABSIs in Intensive Care Units (ICUs) and “other locations” have 2 associated goals in HHS HAI Prevention Plan:
 - Reduce CLABSIs by 50%
 - 100% adherence with CL insertion practices in non-emergent situations



Background: Impact Outside the ICU



- Most work aimed at reducing CLABSIs in the hospital has been done in ICUs
- Many CLs are found outside ICUs
 - In one study 55% of ICU patients had CL; 24% of non-ICU patients had CL
 - However, as more patients are located outside of the ICU, 70% of hospitalized patients with CLs were outside the ICU

Climo et al. ICHE 2003; 24:942-5.



Background: Impact CLABSI Rates



- CLABSI rates outside ICUs may be similar to rates of these infections in ICUs
- Although data are sparse, in one study CLABSI rates were:
 - 5.7 per 1,000 catheter-days in 4 inpatient wards
 - 5.2 per 1,000 catheter-days for medical ICU

Marschall et al. Infect Control Hospital Epidemiol 2007;28:905-9.



Background: Impact National Healthcare Safety Network (NHSN) CLABSI Rates



- From 2006 – 2008 NHSN report, pooled mean CLABSI rates were:
 - Medical-Surgical ICUs = 1.5 to 2.1 per 1,000 catheter-days
 - Medical-Surgical wards = 1.2 per 1,000 catheter-days

Edwards JR, et al. Am J Infect Control 2009;37:783-805.

<http://www.cdc.gov/nhsn/PDFs/dataStat/2009NHSNReport.PDF>

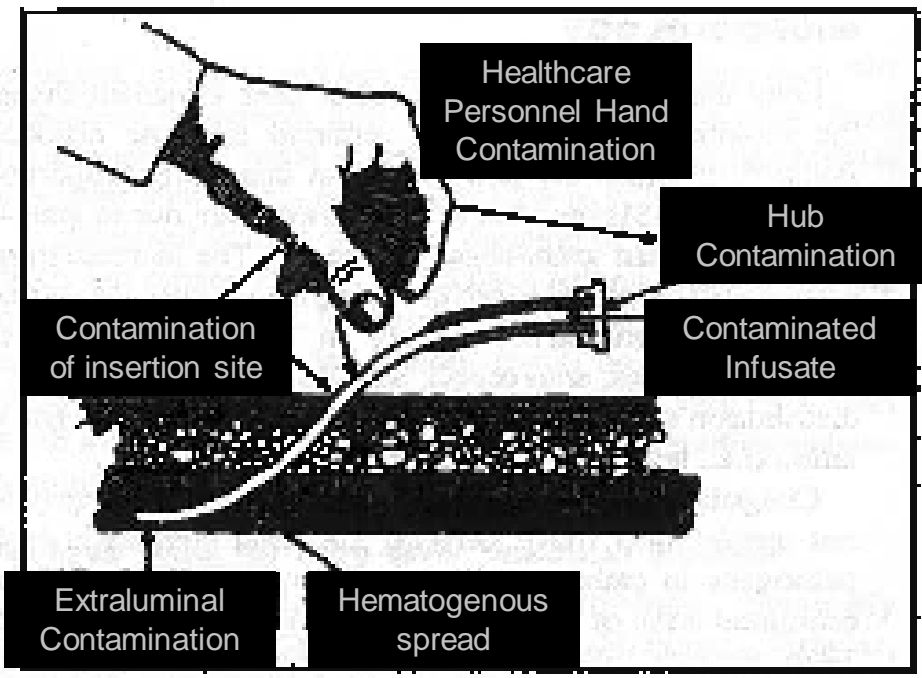


Background: Impact CLABSI in Outpatient Settings



- A number of patient groups may have long-term CLs as outpatients
 - Hemodialysis
 - Malignancy
 - Gastrointestinal tract disorders
 - Pulmonary hypertension
- Rates of CLABSI may be as high as those seen in ICUs
 - In hemodialysis - 1 to 4 per 1,000 catheter-days

Background: Pathogenesis CLABSI



HICPAC. Guideline for Prevention of Intravascular Device-Related Infections. 1996

More Common Mechanisms

1. Pathogen migration along external surface
 - more common early (< 7days)
2. Hub contamination with intraluminal colonization
 - more common >10 days

Less Common Mechanisms

1. Hematogenous seeding from another source
2. Contaminated infusates

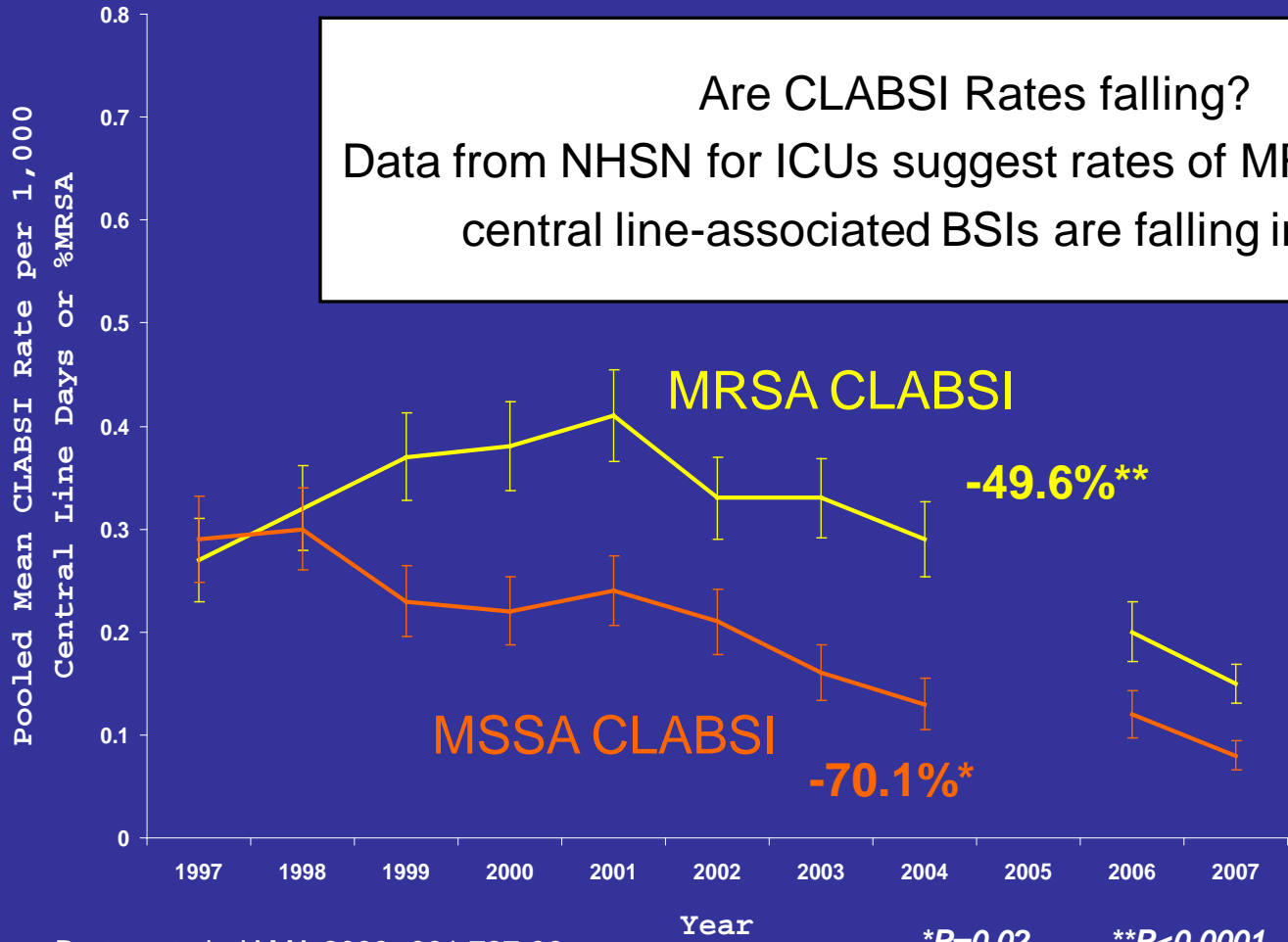


Background: Epidemiology

ALL ICU TYPES: Rates of Methicillin-Resistant and Methicillin-Susceptible *Staphylococcus aureus* CLABSIs—United States, 1997-2007



Are CLABSI Rates falling?
Data from NHSN for ICUs suggest rates of MRSA and MSSA central line-associated BSIs are falling in the U.S.



Burton et al. JAMA 2009; 301:727-36.

*P=0.02

**P<0.0001



Background: Epidemiology Modifiable Risk Factors



Characteristic	Risk Factor Hierarchy
Insertion circumstances	Emergency > elective
Skill of inserter	General > specialized
Insertion site	Femoral > subclavian
Skin antisepsis	70% alcohol, 10% povidone-iodine > 2% chlorhexidine
Catheter lumens	Multilumen > single lumen
Duration of catheter use	Longer duration of use greater risk
Barrier precautions	Submaximal > maximal



Background: Prevention Strategies Interventions



- Pittsburgh Regional Health Initiative – Decrease in CLABSIs in 66 ICUs (68% decrease)
 - Interventions
 - Promotion of best practices
 - » Maximal barrier precautions
 - » Use of chlorhexidine for skin cleansing prior to insertion
 - » Avoidance of femoral site for CL
 - » Use of recommended insertion-site dressing practices
 - » Removal of CL when no longer needed
 - Educational module about BSI prevention
 - Engagement of leadership and clinicians
 - Standard tools for recording adherence to best practices
 - Standardizing catheter insertion kits
 - Measurement of CLABSI and reporting of rates back to facilities

CDC. MMWR 2005;54:1013-6.



Background: Prevention Strategies Interventions



- Michigan Keystone Project
- Decrease in CLABSI in 103 ICUs in Michigan (66% reduction)
- Basic interventions:
 - Hand hygiene
 - Full barrier precautions during CL insertion
 - Skin cleansing with chlorhexidine
 - Avoiding femoral site
 - Removing unnecessary catheters
 - Use of insertion checklist
 - Promotion of safety culture

Pronovost et al. NEJM 2006;355:2725-32.



Background: On the CUSP: Stop BSI project



- This national program is a collaboration between
 - Health Research and Educational Trust
 - Johns Hopkins University Quality and Safety Research Group
 - Michigan Health and Hospital Association Keystone Center for Patient Safety and Quality
- Builds on successes in Michigan Keystone project
 - CLABSI prevention bundle
 - Collaborative model
 - Promotion of safety culture
- Hospitals in all 50 states, the District of Columbia, and Puerto Rico are eligible to participate



Prevention Strategies

- **Core Strategies**

- High levels of scientific evidence
- Demonstrated feasibility

- **Supplemental Strategies**

- Some scientific evidence
- Variable levels of feasibility

The Collaborative should at a minimum include core prevention strategies. Supplemental prevention strategies also may be used. Most core and supplemental strategies are based on HICPAC guidelines. Strategies that are not included in HICPAC guidelines will be noted by an asterisk () after the strategy. HICPAC guidelines may be found at www.cdc.gov/hicpac



Prevention Strategies: Core



- Removing unnecessary CL
- Following proper insertion practices
- Facilitating proper insertion practices*
- Complying with hand hygiene recommendations
- Adequate skin antisepsis
- Choosing proper CL insertion sites
- Performing adequate hub/access port disinfection
- Providing education on CL maintenance and insertion

* Not part of 2002 HICPAC Guidelines for the Prevention of Intravascular Catheter-Related Infections





Prevention Strategies: Core Removing Unnecessary CL



- In one study, 9% of CLs outside of ICU deemed inappropriate
- Perform daily assessment of the need for the CL and promptly discontinue CLs that are no longer required
- Nursing staff should be encouraged to notify physicians of CLs that are unnecessary
- Use peripheral catheters instead
 - These generally have lower rates of BSIs than CL

Trick et al. Infect Control Hospital Epidemiol 2004;25:266-8.



Prevention Strategies: Core Proper Insertion Practices



- Ensure utilization of insertion bundle:
 - Chlorhexidine for skin antisepsis
 - Maximal sterile barrier precautions (e.g., mask, cap [i.e., similar to those worn in the O.R.], gown, sterile gloves, and large sterile drape)
 - Hand hygiene
- Many CLs in patients on non-ICU hospital wards are placed outside those wards (Emergency room, ICU, Operating room, or Pre-operative areas)
- In one study, 49% of CLs were present on admission to the ward. Rates of BSI in this study were higher in CLs placed in Emergency Room
- Define where placement occurs and review technique in those areas

Trick et al. Am J Infect Control 2006;34:636-41.



Prevention Strategies: Core

Facilitating Proper Insertion Practices*

- “Bundling” all needed supplies in one area (e.g., a cart or a kit) helps ensure items are available for use
- Use of a “checklist” to ensure all insertion practices are followed may be beneficial
- Empowering staff to stop a non-emergent CL insertion if proper procedures are not followed
- Promoting safety culture

* Not part of 2002 HICPAC Guidelines for the Prevention of Intravascular Catheter-Related Infections



Prevention Strategies: Core Hand Hygiene

- Hand hygiene should be a cornerstone of CLABSI prevention efforts
 - For both insertion and maintenance
- As part of a hand hygiene intervention, consider:
 - Ensuring easy access to soap and water and alcohol-based hand gels
 - Education for HCP and patients
 - Observation of practices - particularly around high-risk procedures (before and after contact with CL)
 - Feedback – “Just in time” feedback if failure to perform hand hygiene observed



Prevention Strategies: Core Chlorhexidine Skin Cleansing

- Chlorhexidine is the preferred agent for skin cleansing for both CL insertion and maintenance
 - Tincture of iodine, an iodophor, or 70% alcohol are alternatives
 - Recommended application methods and contact time should be followed for maximal effect
- Prior to use should ensure agent is compatible with catheter
 - Alcohol may interact with some polyurethane catheters
 - Some iodine-based compounds may interact with silicone catheters



Prevention Strategies: Core CL Site Choice



- For adult patients receiving non-tunneled CL, femoral site should be avoided due to an increased risk of infection and deep venous thrombosis
- Note:
 - In patients with renal failure, subclavian site should be avoided to minimize stenosis which may limit future vascular access options



Prevention Strategies: Core Hub/access port cleansing



- BSI “outbreaks” have been associated with failure to adequately decontaminate catheter hubs or failure to change them at appropriate intervals
- Cleanse hubs prior to use with an appropriate antiseptic (e.g., 70% alcohol)
- Manufacturer recommendations regarding cleansing and changing connectors should be followed



Prevention Strategies: Core

CL Maintenance and Insertion: Education

- Personnel responsible for insertion and maintenance of catheters should be trained and demonstrate competence
- Recurrent educational sessions for staff who care and/or insert CLs



Prevention Strategies: Supplemental



- Supplemental strategies include:
 - Chlorhexidine bathing*
 - Antimicrobial-impregnated catheters
 - Chlorhexidine-impregnated dressings*

* Not part of 2002 HICPAC Guidelines for the Prevention of Intravascular Catheter-Related Infections



Prevention Strategies: Supplemental Chlorhexidine Bathing*



- In an ICU at a single center, daily bathing with 2% chlorhexidine-impregnated cloths decreased the rate of BSIs compared to soap and water
- No data outside the ICU

Bleasdale, et al. Arch Intern Med 2007;167:2073-9.

* Not part of 2002 HICPAC Guidelines for the Prevention of Intravascular Catheter-Related Infections



Prevention Strategies: Supplemental Antimicrobial-Impregnated Catheters

- 2 types with most supporting evidence:
 - Minocycline-Rifampin
 - Chlorhexidine–Silver Sulfadiazine
- Platinum-Silver catheter available but less evidence to support use
- These may be appropriate for patients whose catheter is expected to be used for more than 5 days and when Core strategies have not decreased rates of CLABSI to established goals.



Prevention Strategies: Supplemental Chlorhexidine Dressings*



- Chlorhexidine-impregnated sponge dressings have been shown to decrease rates of CLABSIs in some studies and not in others.
- These dressings may be an option when Core interventions have not decreased rates of CLABSI to established goals

* Not part of 2002 HICPAC Guidelines for the Prevention of Intravascular Catheter-Related Infections



Summary of Prevention Strategies*

Core Measures

- Removing unnecessary CL
- Following proper insertion practices
- Facilitating proper insertion practices*
- Complying with hand hygiene recommendations
- Performing adequate skin cleaning
- Choosing proper CL insertion sites
- Performing adequate hub/access port cleaning
- Providing education on CL maintenance and insertion

Supplemental Measures

- Implementing chlorhexidine bathing*
- Using antimicrobial-impregnated catheters
- Applying chlorhexidine site dressings*

* Not part of 2002 HICPAC Guidelines for the Prevention of Intravascular Catheter-Related Infections



Measurement

- With CLABSI measurement it is important to
 - Have a definition that is consistent between sites
 - Collecting blood cultures in a similar fashion
 - For recommended indications
 - Via a peripheral venipuncture vs. via a CL



Measurement: Process Measures

- Process measures can help determine if interventions are being fully implemented
 - Ensuring interventions are being performed is itself a “core” intervention
- Potentially important process measures to consider are:
 - Hand hygiene adherence
 - Proportion of patients with CLs, and/or duration of CL use
 - Proportion of CL insertions in which maximal barrier precautions were used
- Consider using NHSN Central Line Insertion Practices (CLIP) option



Measurement: Outcome Calculating CLABSI Rates



$$\text{CLABSI Rate}^* = \frac{\text{\# CLABSIs identified}}{\text{\# central line-days}} \times 1000$$

- * Stratify by:
- Type of ICU/Other Location
 - For special care areas
 - Catheter type (temporary or permanent)
 - For neonatal intensive care units
 - Birthweight category
 - Catheter type (umbilical or central)



Measurement: Outcome Device Utilization (DU) Ratio

$$\text{CL DU Ratio} = \frac{\# \text{ central line-days}}{\# \text{ patient-days}}$$

DU Ratio measures the proportion of total patient-days in which central lines were used.



Measurement: Process CLIP Adherence Rates



- **Using NHSN, adherence rates can be calculated for:**
 - Hand hygiene
 - Barrier precautions used including masks, sterile drape, gowns and sterile gloves
 - Skin preparation including type of agent and whether agent was allowed to dry
- **Other measures collected in the NHSN CLIP option that can be summarized include:**
 - CL type, location, and number of lumens
 - Antiseptic ointment applied to site



Measurement: Process

Calculating CLIP Adherence Rates

$$\text{Hand Hygiene Adherence Rate} = \frac{\text{\# hand hygiene performed for CL insertion}}{\text{\# CL insertions records completed}}$$

Adherence rates can also be measured for each of the barrier and prevention practices by using the number of CLIP records completed as the denominator.



Tools for Implementation

NHSN CLIP Option: Insertion Practices

Event Information [HELP](#)

Event Type*:

Location*:

Date of Insertion*:

Person recording insertion practice data >: Inserter Observer

Central Line Inserter ID:

Last Name: First Name:

Occupation of inserter >:

Insertion Details [HELP](#)

Reason for insertion >:

Inserter performed hand hygiene prior to central line insertion >:

Maximal sterile barrier precautions used >:

- Mask
- Sterile gown
- Large sterile drape
- Sterile gloves
- Cap

Skin Preparation (check all that apply) >: Chlorohexidine gluconate Povidone iodine Alcohol Other

Was skin preparation agent completely dry at the time of first skin puncture? >:

Insertion site >:

Antimicrobial coated catheter used:

Central line catheter type >:

Number of lumens >:

Central line exchanged over a guidewire >:

Antiseptic ointment applied to site >:



Evaluation Considerations

- **Assess baseline policies and procedures**
- **Areas to consider**
 - **Surveillance**
 - **Prevention strategies**
 - **Measurement**
- **Coordinator should track new policies/practices implemented during collaboration**



References

- Bleasdale SC, Trick WE, Gonzalez IM, et al. Effectiveness of chlorhexidine bathing to reduce catheter-associated bloodstream infections in medical intensive care unit patients. Arch Intern Med 2007; 67:2073-9.
- Burton DC, Edwards JR, Horan TC, et al. Methicillin-resistant *Staphylococcus aureus* central line-associated bloodstream infections in US intensive care units, 1997-2007. JAMA 2009;301:727-36.
- CDC. Reduction in central line-associated bloodstream infections among patients in intensive care units— Pennsylvania, April 2001-March 2005. MMWR 2005;54:1013-6.



References

- Climo M, Diekema D, Warren DK, et al. Prevalence of the use of central venous access devices within and outside of the intensive care unit: results of a survey among hospitals in the prevention epicenter program of the Centers for Disease Control and Prevention. *ICHE* 2003;24:942-5.
- Edwards, JR, Peterson KD, Mu Y, et al. National Healthcare Safety Network (NHSN) report: Data summary for 2006 through 2008, issued December 2009. *Am J Infect Control* 2009;37:783-805.



- Klevens RM, Edwards JR, Richards CI, et al. Estimating health care-associated infections and deaths in U.S. hospitals, 2002. Public Health Reports 2007;122:160-6.
- Pittet D, Tarara D, Wenzel RP. Nosocomial bloodstream infection in critically ill patients. Excess length of stay extra costs, and attributable mortality. JAMA 1994;271:1598-1601.



References

- Marschall J, Leone C, Jones M, et al. Catheter-associated bloodstream infections in general medical patients outside the intensive care unit : a surveillance study. *ICHE* 2007; 28:905-9.
- Pronovost P, Needham D, Berenholtz S, et al. An intervention to decrease catheter-related bloodstream infections in the ICU. *NEJM* 2006;355:2725-32.
- Trick WE, Vernon MO, Welbel SF, et al. Unnecessary use of central venous catheters: the need to look outside the intensive care unit. *Infect Control Hospital Epidemiol* 2004; 25:266-8.



References

- Trick WE, Miranda J, Evans AT, et al. Prospective cohort study of central venous catheters among internal medicine ward patients. Am J Infect Control 2006;34:636-41.



Guideline for Disinfection and Sterilization in Healthcare Facilities, 2008

William A. Rutala, Ph.D., M.P.H.^{1,2}, David J. Weber, M.D., M.P.H.^{1,2}, and the Healthcare
Infection Control Practices Advisory Committee (HICPAC)³

¹Hospital Epidemiology
University of North Carolina Health Care System
Chapel Hill, NC 27514

²Division of Infectious Diseases
University of North Carolina School of Medicine
Chapel Hill, NC 27599-7030

³HICPAC Members

Robert A. Weinstein, MD (Chair)
Cook County Hospital
Chicago, IL

Jane D. Siegel, MD (Co-Chair)
University of Texas Southwestern Medical Center
Dallas, TX

Michele L. Pearson, MD
(Executive Secretary)
Centers for Disease Control and Prevention
Atlanta, GA

Raymond Y.W. Chinn, MD
Sharp Memorial Hospital
San Diego, CA

Alfred DeMaria, Jr, MD
Massachusetts Department of Public Health
Jamaica Plain, MA

James T. Lee, MD, PhD
University of Minnesota
Minneapolis, MN

William A. Rutala, PhD, MPH
University of North Carolina Health Care System
Chapel Hill, NC

William E. Scheckler, MD
University of Wisconsin
Madison, WI

Beth H. Stover, RN
Kosair Children's Hospital
Louisville, KY

Marjorie A. Underwood, RN, BSN CIC
Mt. Diablo Medical Center
Concord, CA

This guideline discusses use of products by healthcare personnel in healthcare settings such as hospitals, ambulatory care and home care; the recommendations are not intended for consumer use of the products discussed.

Disinfection and Sterilization in Healthcare Facilities

Executive Summary

Introduction

Methods

Definition of Terms

Approach to Disinfection and Sterilization

 Critical Items

 Semicritical Items

 Noncritical Items

 Changes in Disinfection and Sterilization Since 1981

Disinfection of Healthcare Equipment

 Concerns with Implementing the Spaulding Scheme

 Reprocessing of Endoscopes

 Laparoscopes and Arthroscopes

 Tonometers, Cervical Diaphragm Fitting Rings, Cryosurgical Instruments, Endocavitary Probes

 Dental Instruments

 Disinfection of HBV, HCV, HIV or Tuberculosis-Contaminated Devices

 Disinfection in the Hemodialysis Unit

 Inactivation of *Clostridium difficile*

 OSHA Bloodborne Pathogen Standard

 Emerging Pathogens (*Cryptosporidium*, *Helicobacter pylori*, *E. coli* O157:H7, Rotavirus, Human

 Papilloma Virus, Norovirus, Severe Acute Respiratory Syndrome Coronavirus)

 Inactivation of Bioterrorist Agents

 Toxicological, Environmental, and Occupational Concerns

 Disinfection in Ambulatory Care, Home Care, and the Home

 Susceptibility of Antibiotic-Resistant Bacteria to Disinfectants

 Surface Disinfection: Should We Do It?

 Contact Time for Surface Disinfectants

 Air Disinfection

 Microbial Contamination of Disinfectants

Factors Affecting the Efficacy of Disinfection and Sterilization

 Number and Location of Microorganisms

 Innate Resistance of Microorganisms

 Concentration and Potency of Disinfectants

 Physical and Chemical Factors

 Organic and Inorganic Matter

 Duration of Exposure

 Biofilms

Cleaning

Disinfection

 Chemical Disinfectants

 Alcohol

 Overview

 Mode of Action

 Microbicidal Activity

 Uses

 Chlorine and Chlorine Compounds

 Overview

 Mode of Action

 Microbicidal Activity

- Uses
- Formaldehyde
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Glutaraldehyde
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Hydrogen Peroxide
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Iodophors
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Ortho-phthalaldehyde
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Peracetic Acid
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Peracetic Acid and Hydrogen Peroxide
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Phenolics
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Quaternary Ammonium Compounds
 - Overview
 - Mode of Action
 - Microbicidal Activity
 - Uses
- Miscellaneous Inactivating Agents
 - Other Germicides
 - Ultraviolet Radiation
 - Pasteurization
 - Flushing- and Washer-Disinfectors
- Regulatory Framework for Disinfectants and Sterilants
- Neutralization of Germicides

Sterilization

Steam Sterilization

Overview

Mode of Action

Microbicidal Activity

Uses

Flash Sterilization

Overview

Uses

Low-Temperature Sterilization Technologies

Ethylene Oxide “Gas” Sterilization

Overview

Mode of Action

Microbicidal Activity

Uses

Hydrogen Peroxide Gas Plasma

Overview

Mode of Action

Microbicidal Activity

Uses

Peracetic Acid Sterilization

Overview

Mode of Action

Microbicidal Activity

Uses

Microbicidal Activity of Low-Temperature Sterilization Technology

Bioburden of Surgical Devices

Effect of Cleaning on Sterilization Efficacy

Other Sterilization Methods

Ionizing Radiation

Dry-Heat Sterilizers

Liquid Chemicals

Performic Acid

Filtration

Microwave

Glass Bead “Sterilizer”

Vaporized Hydrogen Peroxide

Ozone

Formaldehyde Steam

Gaseous Chlorine Dioxide

Vaporized Peracetic Acid

Infrared radiation

Sterilizing Practices

Overview

Sterilization Cycle Validation

Physical Facilities

Cleaning

Packaging

Loading

Storage

Monitoring (Mechanical, Chemical, Biological Indicators)

Reuse of Single-Use Medical Devices

Conclusion

Web-Based Disinfection and Sterilization Resources

Recommendations (Category IA, IB, IC, II)

Performance Indicators

Acknowledgements

Glossary

Tables and Figure

References

EXECUTIVE SUMMARY

The Guideline for Disinfection and Sterilization in Healthcare Facilities, 2008, presents evidence-based recommendations on the preferred methods for cleaning, disinfection and sterilization of patient-care medical devices and for cleaning and disinfecting the healthcare environment. This document supercedes the relevant sections contained in the 1985 Centers for Disease Control (CDC) Guideline for Handwashing and Environmental Control.¹ Because maximum effectiveness from disinfection and sterilization results from first cleaning and removing organic and inorganic materials, this document also reviews cleaning methods. The chemical disinfectants discussed for patient-care equipment include alcohols, glutaraldehyde, formaldehyde, hydrogen peroxide, iodophors, *ortho*-phthalaldehyde, peracetic acid, phenolics, quaternary ammonium compounds, and chlorine. The choice of disinfectant, concentration, and exposure time is based on the risk for infection associated with use of the equipment and other factors discussed in this guideline. The sterilization methods discussed include steam sterilization, ethylene oxide (ETO), hydrogen peroxide gas plasma, and liquid peracetic acid. When properly used, these cleaning, disinfection, and sterilization processes can reduce the risk for infection associated with use of invasive and noninvasive medical and surgical devices. However, for these processes to be effective, health-care workers should adhere strictly to the cleaning, disinfection, and sterilization recommendations in this document and to instructions on product labels.

In addition to updated recommendations, new topics addressed in this guideline include 1) inactivation of antibiotic-resistant bacteria, bioterrorist agents, emerging pathogens, and bloodborne pathogens; 2) toxicologic, environmental, and occupational concerns associated with disinfection and sterilization practices; 3) disinfection of patient-care equipment used in ambulatory settings and home care; 4) new sterilization processes, such as hydrogen peroxide gas plasma and liquid peracetic acid; and 5) disinfection of complex medical instruments (e.g., endoscopes).

INTRODUCTION

In the United States, approximately 46.5 million surgical procedures and even more invasive medical procedures—including approximately 5 million gastrointestinal endoscopies—are performed each year.² Each procedure involves contact by a medical device or surgical instrument with a patient's sterile tissue or mucous membranes. A major risk of all such procedures is the introduction of pathogens that can lead to infection. Failure to properly disinfect or sterilize equipment carries not only risk associated with breach of host barriers but also risk for person-to-person transmission (e.g., hepatitis B virus) and transmission of environmental pathogens (e.g., *Pseudomonas aeruginosa*).

Disinfection and sterilization are essential for ensuring that medical and surgical instruments do not transmit infectious pathogens to patients. Because sterilization of all patient-care items is not necessary, health-care policies must identify, primarily on the basis of the items' intended use, whether cleaning, disinfection, or sterilization is indicated.

Multiple studies in many countries have documented lack of compliance with established guidelines for disinfection and sterilization.³⁻⁶ Failure to comply with scientifically-based guidelines has led to numerous outbreaks.⁶⁻¹² This guideline presents a pragmatic approach to the judicious selection and proper use of disinfection and sterilization processes; the approach is based on well-designed studies assessing the efficacy (through laboratory investigations) and effectiveness (through clinical studies) of disinfection and sterilization procedures.

METHODS

This guideline resulted from a review of all MEDLINE articles in English listed under the MeSH headings of *disinfection* or *sterilization* (focusing on health-care equipment and supplies) from January 1980 through August 2006. References listed in these articles also were reviewed. Selected articles published before 1980 were reviewed and, if still relevant, included in the guideline. The three major peer-reviewed journals in infection control—*American Journal of Infection Control*, *Infection Control and Hospital Epidemiology*, and *Journal of Hospital Infection*—were searched for relevant articles published from January 1990 through August 2006. Abstracts presented at the annual meetings of the Society for Healthcare Epidemiology of America and Association for professionals in Infection Control and Epidemiology, Inc. during 1997–2006 also were reviewed; however, abstracts were not used to support the recommendations.

DEFINITION OF TERMS

Sterilization describes a process that destroys or eliminates all forms of microbial life and is carried out in health-care facilities by physical or chemical methods. Steam under pressure, dry heat, EtO gas, hydrogen peroxide gas plasma, and liquid chemicals are the principal sterilizing agents used in health-care facilities. Sterilization is intended to convey an absolute meaning; unfortunately, however, some health professionals and the technical and commercial literature refer to “disinfection” as “sterilization” and items as “partially sterile.” When chemicals are used to destroy all forms of microbiologic life, they can be called chemical sterilants. These same germicides used for shorter exposure periods also can be part of the disinfection process (i.e., high-level disinfection).

Disinfection describes a process that eliminates many or all pathogenic microorganisms, except bacterial spores, on inanimate objects (Tables 1 and 2). In health-care settings, objects usually are disinfected by liquid chemicals or wet pasteurization. Each of the various factors that affect the efficacy of

disinfection can nullify or limit the efficacy of the process.

Factors that affect the efficacy of both disinfection and sterilization include prior cleaning of the object; organic and inorganic load present; type and level of microbial contamination; concentration of and exposure time to the germicide; physical nature of the object (e.g., crevices, hinges, and lumens); presence of biofilms; temperature and pH of the disinfection process; and in some cases, relative humidity of the sterilization process (e.g., ethylene oxide).

Unlike sterilization, disinfection is not sporicidal. A few disinfectants will kill spores with prolonged exposure times (3–12 hours); these are called *chemical sterilants*. At similar concentrations but with shorter exposure periods (e.g., 20 minutes for 2% glutaraldehyde), these same disinfectants will kill all microorganisms except large numbers of bacterial spores; they are called *high-level disinfectants*. *Low-level disinfectants* can kill most vegetative bacteria, some fungi, and some viruses in a practical period of time (≤ 10 minutes). *Intermediate-level disinfectants* might be cidal for mycobacteria, vegetative bacteria, most viruses, and most fungi but do not necessarily kill bacterial spores. Germicides differ markedly, primarily in their antimicrobial spectrum and rapidity of action.

Cleaning is the removal of visible soil (e.g., organic and inorganic material) from objects and surfaces and normally is accomplished manually or mechanically using water with detergents or enzymatic products. Thorough cleaning is essential before high-level disinfection and sterilization because inorganic and organic materials that remain on the surfaces of instruments interfere with the effectiveness of these processes. *Decontamination* removes pathogenic microorganisms from objects so they are safe to handle, use, or discard.

Terms with the suffix *cide* or *cidal* for killing action also are commonly used. For example, a germicide is an agent that can kill microorganisms, particularly pathogenic organisms (“germs”). The term *germicide* includes both antiseptics and disinfectants. *Antiseptics* are germicides applied to living tissue and skin; *disinfectants* are antimicrobials applied only to inanimate objects. In general, antiseptics are used only on the skin and not for surface disinfection, and disinfectants are not used for skin antisepsis because they can injure skin and other tissues. Virucide, fungicide, bactericide, sporicide, and tuberculocide can kill the type of microorganism identified by the prefix. For example, a bactericide is an agent that kills bacteria. ¹³⁻¹⁸

A RATIONAL APPROACH TO DISINFECTION AND STERILIZATION

More than 30 years ago, Earle H. Spaulding devised a rational approach to disinfection and sterilization of patient-care items and equipment.¹⁴ This classification scheme is so clear and logical that it has been retained, refined, and successfully used by infection control professionals and others when planning methods for disinfection or sterilization.^{1, 13, 15, 17, 19, 20} Spaulding believed the nature of disinfection could be understood readily if instruments and items for patient care were categorized as critical, semicritical, and noncritical according to the degree of risk for infection involved in use of the items. The CDC *Guideline for Handwashing and Hospital Environmental Control*²¹, *Guidelines for the Prevention of Transmission of Human Immunodeficiency Virus (HIV) and Hepatitis B Virus (HBV) to Health-Care and Public-Safety Workers*²², and *Guideline for Environmental Infection Control in Health-Care Facilities*²³ employ this terminology.

Critical Items

Critical items confer a high risk for infection if they are contaminated with any microorganism. Thus, objects that enter sterile tissue or the vascular system must be sterile because any microbial contamination could transmit disease. This category includes surgical instruments, cardiac and urinary catheters, implants, and ultrasound probes used in sterile body cavities. Most of the items in this category should be purchased as sterile or be sterilized with steam if possible. Heat-sensitive objects can be treated with EtO, hydrogen peroxide gas plasma; or if other methods are unsuitable, by liquid chemical sterilants. Germicides categorized as chemical sterilants include $\geq 2.4\%$ glutaraldehyde-based formulations, 0.95% glutaraldehyde with 1.64% phenol/phenate, 7.5% stabilized hydrogen peroxide, 7.35% hydrogen peroxide with 0.23% peracetic acid, 0.2% peracetic acid, and 0.08% peracetic acid with 1.0% hydrogen peroxide. Liquid chemical sterilants reliably produce sterility only if cleaning precedes treatment and if proper guidelines are followed regarding concentration, contact time, temperature, and pH.

Semicritical Items

Semicritical items contact mucous membranes or nonintact skin. This category includes respiratory therapy and anesthesia equipment, some endoscopes, laryngoscope blades²⁴, esophageal manometry probes, cystoscopes²⁵, anorectal manometry catheters, and diaphragm fitting rings. These medical devices should be free from all microorganisms; however, small numbers of bacterial spores are permissible. Intact mucous membranes, such as those of the lungs and the gastrointestinal tract, generally are resistant to infection by common bacterial spores but susceptible to other organisms, such as bacteria, mycobacteria, and viruses. Semicritical items minimally require high-level disinfection using chemical disinfectants. Glutaraldehyde, hydrogen peroxide, *ortho*-phthalaldehyde, and peracetic acid with hydrogen peroxide are cleared by the Food and Drug Administration (FDA) and are dependable high-level disinfectants provided the factors influencing germicidal procedures are met (Table 1). When a disinfectant is selected for use with certain patient-care items, the chemical compatibility after extended use with the items to be disinfected also must be considered.

High-level disinfection traditionally is defined as complete elimination of all microorganisms in or on an instrument, except for small numbers of bacterial spores. The FDA definition of high-level disinfection is a sterilant used for a shorter contact time to achieve a 6-log₁₀ kill of an appropriate *Mycobacterium* species. Cleaning followed by high-level disinfection should eliminate enough pathogens to prevent transmission of infection.^{26, 27}

Laparoscopes and arthroscopes entering sterile tissue ideally should be sterilized between patients. However, in the United States, this equipment sometimes undergoes only high-level disinfection between patients.²⁸⁻³⁰ As with flexible endoscopes, these devices can be difficult to clean and high-level disinfect or sterilize because of intricate device design (e.g., long narrow lumens, hinges). Meticulous

cleaning must precede any high-level disinfection or sterilization process. Although sterilization is preferred, no reports have been published of outbreaks resulting from high-level disinfection of these scopes when they are properly cleaned and high-level disinfected. Newer models of these instruments can withstand steam sterilization that for critical items would be preferable to high-level disinfection.

Rinsing endoscopes and flushing channels with sterile water, filtered water, or tap water will prevent adverse effects associated with disinfectant retained in the endoscope (e.g., disinfectant-induced colitis). Items can be rinsed and flushed using sterile water after high-level disinfection to prevent contamination with organisms in tap water, such as nontuberculous mycobacteria,^{10, 31, 32} *Legionella*,³³⁻³⁵ or gram-negative bacilli such as *Pseudomonas*.^{1, 17, 36-38} Alternatively, a tapwater or filtered water (0.2µ filter) rinse should be followed by an alcohol rinse and forced air drying.^{28, 38-40} Forced-air drying markedly reduces bacterial contamination of stored endoscopes, most likely by removing the wet environment favorable for bacterial growth.³⁹ After rinsing, items should be dried and stored (e.g., packaged) in a manner that protects them from recontamination.

Some items that may come in contact with nonintact skin for a brief period of time (i.e., hydrotherapy tanks, bed side rails) are usually considered noncritical surfaces and are disinfected with intermediate-level disinfectants (i.e., phenolic, iodophor, alcohol, chlorine)²³. Since hydrotherapy tanks have been associated with spread of infection, some facilities have chosen to disinfect them with recommended levels of chlorine^{23, 41}.

In the past, high-level disinfection was recommended for mouthpieces and spirometry tubing (e.g., glutaraldehyde) but cleaning the interior surfaces of the spirometers was considered unnecessary.⁴² This was based on a study that showed that mouthpieces and spirometry tubing become contaminated with microorganisms but there was no bacterial contamination of the surfaces inside the spirometers. Filters have been used to prevent contamination of this equipment distal to the filter; such filters and the proximal mouthpiece are changed between patients.

Noncritical Items

Noncritical items are those that come in contact with intact skin but not mucous membranes. Intact skin acts as an effective barrier to most microorganisms; therefore, the sterility of items coming in contact with intact skin is "not critical." In this guideline, noncritical items are divided into noncritical patient care items and noncritical environmental surfaces^{43, 44}. Examples of noncritical patient-care items are bedpans, blood pressure cuffs, crutches and computers⁴⁵. In contrast to critical and some semicritical items, most noncritical reusable items may be decontaminated where they are used and do not need to be transported to a central processing area. Virtually no risk has been documented for transmission of infectious agents to patients through noncritical items³⁷ when they are used as noncritical items and do not contact non-intact skin and/or mucous membranes. Table 1 lists several low-level disinfectants that may be used for noncritical items. Most Environmental Protection Agency (EPA)-registered disinfectants have a 10-minute label claim. However, multiple investigators have demonstrated the effectiveness of these disinfectants against vegetative bacteria (e.g., *Listeria*, *Escherichia coli*, *Salmonella*, vancomycin-resistant Enterococci, methicillin-resistant *Staphylococcus aureus*), yeasts (e.g., *Candida*), mycobacteria (e.g., *Mycobacterium tuberculosis*), and viruses (e.g. poliovirus) at exposure times of 30–60 seconds⁴⁶⁻⁶⁴. Federal law requires all applicable label instructions on EPA-registered products to be followed (e.g., use-dilution, shelf life, storage, material compatibility, safe use, and disposal). If the user selects exposure conditions (e.g., exposure time) that differ from those on the EPA-registered products label, the user assumes liability for any injuries resulting from off-label use and is potentially subject to enforcement action under Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA)⁶⁵.

Noncritical environmental surfaces include bed rails, some food utensils, bedside tables, patient furniture and floors. Noncritical environmental surfaces frequently touched by hand (e.g., bedside tables,

bed rails) potentially could contribute to secondary transmission by contaminating hands of health-care workers or by contacting medical equipment that subsequently contacts patients^{13, 46-48, 51, 66, 67}. Mops and reusable cleaning cloths are regularly used to achieve low-level disinfection on environmental surfaces. However, they often are not adequately cleaned and disinfected, and if the water-disinfectant mixture is not changed regularly (e.g., after every three to four rooms, at no longer than 60-minute intervals), the mopping procedure actually can spread heavy microbial contamination throughout the health-care facility⁶⁸. In one study, standard laundering provided acceptable decontamination of heavily contaminated mopheads but chemical disinfection with a phenolic was less effective.⁶⁸ Frequent laundering of mops (e.g., daily), therefore, is recommended. Single-use disposable towels impregnated with a disinfectant also can be used for low-level disinfection when spot-cleaning of noncritical surfaces is needed⁴⁵.

Changes in Disinfection and Sterilization Since 1981

The Table in the CDC *Guideline for Environmental Control* prepared in 1981 as a guide to the appropriate selection and use of disinfectants has undergone several important changes (Table 1).¹⁵ First, formaldehyde-alcohol has been deleted as a recommended chemical sterilant or high-level disinfectant because it is irritating and toxic and not commonly used. Second, several new chemical sterilants have been added, including hydrogen peroxide, peracetic acid^{58, 69, 70}, and peracetic acid and hydrogen peroxide in combination. Third, 3% phenolics and iodophors have been deleted as high-level disinfectants because of their unproven efficacy against bacterial spores, *M. tuberculosis*, and/or some fungi.^{55, 71} Fourth, isopropyl alcohol and ethyl alcohol have been excluded as high-level disinfectants¹⁵ because of their inability to inactivate bacterial spores and because of the inability of isopropyl alcohol to inactivate hydrophilic viruses (i.e., poliovirus, coxsackie virus).⁷² Fifth, a 1:16 dilution of 2.0% glutaraldehyde-7.05% phenol-1.20% sodium phenate (which contained 0.125% glutaraldehyde, 0.440% phenol, and 0.075% sodium phenate when diluted) has been deleted as a high-level disinfectant because this product was removed from the marketplace in December 1991 because of a lack of bactericidal activity in the presence of organic matter; a lack of fungicidal, tuberculocidal and sporicidal activity; and reduced virucidal activity.^{49, 55, 56, 71, 73-79} Sixth, the exposure time required to achieve high-level disinfection has been changed from 10-30 minutes to 12 minutes or more depending on the FDA-cleared label claim and the scientific literature.^{27, 55, 69, 76, 80-84} A glutaraldehyde and an ortho-phthalaldehyde have an FDA-cleared label claim of 5 minutes when used at 35°C and 25°C, respectively, in an automated endoscope reprocessor with FDA-cleared capability to maintain the solution at the appropriate temperature.⁸⁵

In addition, many new subjects have been added to the guideline. These include inactivation of emerging pathogens, bioterrorist agents, and bloodborne pathogens; toxicologic, environmental, and occupational concerns associated with disinfection and sterilization practices; disinfection of patient-care equipment used in ambulatory and home care; inactivation of antibiotic-resistant bacteria; new sterilization processes, such as hydrogen peroxide gas plasma and liquid peracetic acid; and disinfection of complex medical instruments (e.g., endoscopes).

DISINFECTION OF HEALTHCARE EQUIPMENT

Concerns about Implementing the Spaulding Scheme

One problem with implementing the aforementioned scheme is oversimplification. For example, the scheme does not consider problems with reprocessing of complicated medical equipment that often is heat-sensitive or problems of inactivating certain types of infectious agents (e.g., prions, such as Creutzfeldt-Jakob disease [CJD] agent). Thus, in some situations, choosing a method of disinfection remains difficult, even after consideration of the categories of risk to patients. This is true particularly for a few medical devices (e.g., arthroscopes, laparoscopes) in the critical category because of controversy about whether they should be sterilized or high-level disinfected.^{28, 86} Heat-stable scopes (e.g., many rigid scopes) should be steam sterilized. Some of these items cannot be steam sterilized because they are heat-sensitive; additionally, sterilization using ethylene oxide (EtO) can be too time-consuming for routine use between patients (new technologies, such as hydrogen peroxide gas plasma and peracetic acid reprocessor, provide faster cycle times). However, evidence that sterilization of these items improves patient care by reducing the infection risk is lacking^{29, 87-91}. Many newer models of these instruments can withstand steam sterilization, which for critical items is the preferred method.

Another problem with implementing the Spaulding scheme is processing of an instrument in the semicritical category (e.g., endoscope) that would be used in conjunction with a critical instrument that contacts sterile body tissues. For example, is an endoscope used for upper gastrointestinal tract investigation still a semicritical item when used with sterile biopsy forceps or in a patient who is bleeding heavily from esophageal varices? Provided that high-level disinfection is achieved, and all microorganisms except bacterial spores have been removed from the endoscope, the device should not represent an infection risk and should remain in the semicritical category⁹²⁻⁹⁴. Infection with spore-forming bacteria has not been reported from appropriately high-level disinfected endoscopes.

An additional problem with implementation of the Spaulding system is that the optimal contact time for high-level disinfection has not been defined or varies among professional organizations, resulting in different strategies for disinfecting different types of semicritical items (e.g., endoscopes, applanation tonometers, endocavitary transducers, cryosurgical instruments, and diaphragm fitting rings). Until simpler and effective alternatives are identified for device disinfection in clinical settings, following this guideline, other CDC guidelines^{1, 22, 95, 96} and FDA-cleared instructions for the liquid chemical sterilants/high-level disinfectants would be prudent.

Reprocessing of Endoscopes

Physicians use endoscopes to diagnose and treat numerous medical disorders. Even though endoscopes represent a valuable diagnostic and therapeutic tool in modern medicine and the incidence of infection associated with their use reportedly is very low (about 1 in 1.8 million procedures)⁹⁷, more healthcare-associated outbreaks have been linked to contaminated endoscopes than to any other medical device^{6-8, 12, 98}. To prevent the spread of health-care-associated infections, all heat-sensitive endoscopes (e.g., gastrointestinal endoscopes, bronchoscopes, nasopharygoscopes) must be properly cleaned and, at a minimum, subjected to high-level disinfection after each use. High-level disinfection can be expected to destroy all microorganisms, although when high numbers of bacterial spores are present, a few spores might survive.

Because of the types of body cavities they enter, flexible endoscopes acquire high levels of microbial contamination (bioburden) during each use⁹⁹. For example, the bioburden found on flexible gastrointestinal endoscopes after use has ranged from 10^5 colony forming units (CFU)/mL to 10^{10} CFU/mL, with the highest levels found in the suction channels⁹⁹⁻¹⁰². The average load on bronchoscopes before cleaning was 6.4×10^4 CFU/mL. Cleaning reduces the level of microbial contamination by 4–6 \log_{10} ^{83, 103}. Using human immunovirus (HIV)-contaminated endoscopes, several investigators have shown that cleaning completely eliminates the microbial contamination on the scopes^{104, 105}. Similarly, other investigators found that EtO sterilization or soaking in 2% glutaraldehyde for 20 minutes was effective only when the device first was properly cleaned¹⁰⁶.

FDA maintains a list of cleared liquid chemical sterilants and high-level disinfectants that can be used to reprocess heat-sensitive medical devices, such as flexible endoscopes (<http://www.fda.gov/cdrh/ode/germlab.html>). At this time, the FDA-cleared and marketed formulations include: $\geq 2.4\%$ glutaraldehyde, 0.55% *ortho*-phthalaldehyde (OPA), 0.95% glutaraldehyde with 1.64% phenol/phenate, 7.35% hydrogen peroxide with 0.23% peracetic acid, 1.0% hydrogen peroxide with 0.08% peracetic acid, and 7.5% hydrogen peroxide⁸⁵. These products have excellent antimicrobial activity; however, some oxidizing chemicals (e.g., 7.5% hydrogen peroxide, and 1.0% hydrogen peroxide with 0.08% peracetic acid [latter product is no longer marketed]) reportedly have caused cosmetic and functional damage to endoscopes⁶⁹. Users should check with device manufacturers for information about germicide compatibility with their device. If the germicide is FDA-cleared, then it is safe when used according to label directions; however, professionals should review the scientific literature for newly available data regarding human safety or materials compatibility. EtO sterilization of flexible endoscopes is infrequent because it requires a lengthy processing and aeration time (e.g., 12 hours) and is a potential hazard to staff and patients. The two products most commonly used for reprocessing endoscopes in the United States are glutaraldehyde and an automated, liquid chemical sterilization process that uses peracetic acid¹⁰⁷. The American Society for Gastrointestinal Endoscopy (ASGE) recommends glutaraldehyde solutions that do not contain surfactants because the soapy residues of surfactants are difficult to remove during rinsing¹⁰⁸. *ortho*-phthalaldehyde has begun to replace glutaraldehyde in many health-care facilities because it has several potential advantages over glutaraldehyde: is not known to irritate the eyes and nasal passages, does not require activation or exposure monitoring, and has a 12-minute high-level disinfection claim in the United States⁶⁹. Disinfectants that are not FDA-cleared and should not be used for reprocessing endoscopes include iodophors, chlorine solutions, alcohols, quaternary ammonium compounds, and phenolics. These solutions might still be in use outside the United States, but their use should be strongly discouraged because of lack of proven efficacy against all microorganisms or materials incompatibility.

FDA clearance of the contact conditions listed on germicide labeling is based on the manufacturer's test results (<http://www.fda.gov/cdrh/ode/germlab.html>). Manufacturers test the product under worst-case conditions for germicide formulation (i.e., minimum recommended concentration of the active ingredient), and include organic soil. Typically manufacturers use 5% serum as the organic soil and hard water as examples of organic and inorganic challenges. The soil represents the organic loading to which the device is exposed during actual use and that would remain on the device in the absence of cleaning. This method ensures that the contact conditions completely eliminate the test mycobacteria (e.g., 10^5 to 10^6 *Mycobacteria tuberculosis* in organic soil and dried on a scope) if inoculated in the most difficult areas for the disinfectant to penetrate and contact in the absence of cleaning and thus provides a margin of safety¹⁰⁹. For 2.4% glutaraldehyde that requires a 45-minute immersion at 25°C to achieve high-level disinfection (i.e., 100% kill of *M. tuberculosis*). FDA itself does not conduct testing but relies solely on the disinfectant manufacturer's data. Data suggest that *M. tuberculosis* levels can be reduced by at least 8 log₁₀ with cleaning (4 log₁₀)^{83, 101, 102, 110}, followed by chemical disinfection for 20 minutes at 20°C (4 to 6 log₁₀)^{83, 93, 111, 112}. On the basis of these data, APIC¹¹³, the Society of Gastroenterology Nurses and Associates (SGNA)^{38, 114, 115}, the ASGE¹⁰⁸, American College of Chest Physicians¹², and a multi-society guideline¹¹⁶ recommend alternative contact conditions with 2% glutaraldehyde to achieve high-level disinfection (e.g., that equipment be immersed in 2% glutaraldehyde at 20°C for at least 20 minutes for high-level disinfection). Federal regulations are to follow the FDA-cleared label claim for high-level disinfectants. The FDA-cleared labels for high-level disinfection with $>2\%$ glutaraldehyde at 25°C range from 20-90 minutes, depending upon the product based on three tier testing which includes AOAC sporicidal tests, simulated use testing with mycobacterial and in-use testing. The studies supporting the efficacy of $>2\%$ glutaraldehyde for 20 minutes at 20°C assume adequate cleaning prior to disinfection, whereas the FDA-cleared label claim incorporates an added margin of safety to accommodate possible lapses in cleaning practices. Facilities that have chosen to apply the 20 minute duration at 20°C have done so based on the IA recommendation in the July 2003 SHEA position paper, "Multi-society Guideline for Reprocessing Flexible Gastrointestinal Endoscopes"^{19, 57, 83, 94, 108, 111, 116-121}.

Flexible endoscopes are particularly difficult to disinfect¹²² and easy to damage because of their intricate design and delicate materials.¹²³ Meticulous cleaning must precede any sterilization or high-level disinfection of these instruments. Failure to perform good cleaning can result in sterilization or disinfection failure, and outbreaks of infection can occur. Several studies have demonstrated the importance of cleaning in experimental studies with the duck hepatitis B virus (HBV)^{106, 124}, HIV¹²⁵ and *Helicobacter pylori*.¹²⁶

An examination of health-care-associated infections related only to endoscopes through July 1992 found 281 infections transmitted by gastrointestinal endoscopy and 96 transmitted by bronchoscopy. The clinical spectrum ranged from asymptomatic colonization to death. *Salmonella* species and *Pseudomonas aeruginosa* repeatedly were identified as causative agents of infections transmitted by gastrointestinal endoscopy, and *M. tuberculosis*, atypical mycobacteria, and *P. aeruginosa* were the most common causes of infections transmitted by bronchoscopy¹². Major reasons for transmission were inadequate cleaning, improper selection of a disinfecting agent, and failure to follow recommended cleaning and disinfection procedures^{6, 8, 37, 98}, and flaws in endoscope design^{127, 128} or automated endoscope reprocessors.^{7, 98} Failure to follow established guidelines has continued to result in infections associated with gastrointestinal endoscopes⁸ and bronchoscopes^{7, 12}. Potential device-associated problems should be reported to the FDA Center for Devices and Radiologic Health. One multistate investigation found that 23.9% of the bacterial cultures from the internal channels of 71 gastrointestinal endoscopes grew $\geq 100,000$ colonies of bacteria after completion of all disinfection and sterilization procedures (nine of 25 facilities were using a product that has been removed from the marketplace [six facilities using 1:16 glutaraldehyde phenate], is not FDA-cleared as a high-level disinfectant [an iodophor] or no disinfecting agent) and before use on the next patient¹²⁹. The incidence of postendoscopic procedure infections from an improperly processed endoscope has not been rigorously assessed.

Automated endoscope reprocessors (AER) offer several advantages over manual reprocessing: they automate and standardize several important reprocessing steps¹³⁰⁻¹³², reduce the likelihood that an essential reprocessing step will be skipped, and reduce personnel exposure to high-level disinfectants or chemical sterilants. Failure of AERs has been linked to outbreaks of infections¹³³ or colonization^{7, 134}, and the AER water filtration system might not be able to reliably provide “sterile” or bacteria-free rinse water^{135, 136}. Establishment of correct connectors between the AER and the device is critical to ensure complete flow of disinfectants and rinse water^{7, 137}. In addition, some endoscopes such as the duodenoscopes (e.g., endoscopic retrograde cholangiopancreatography [ERCP]) contain features (e.g., elevator-wire channel) that require a flushing pressure that is not achieved by most AERs and must be reprocessed manually using a 2- to 5-mL syringe, until new duodenoscopes equipped with a wider elevator-channel that AERs can reliably reprocess become available¹³². Outbreaks involving removable endoscope parts^{138, 139} such as suction valves and endoscopic accessories designed to be inserted through flexible endoscopes such as biopsy forceps emphasize the importance of cleaning to remove all foreign matter before high-level disinfection or sterilization.¹⁴⁰ Some types of valves are now available as single-use, disposable products (e.g., bronchoscope valves) or steam sterilizable products (e.g., gastrointestinal endoscope valves).

AERs need further development and redesign^{7, 141}, as do endoscopes^{123, 142}, so that they do not represent a potential source of infectious agents. Endoscopes employing disposable components (e.g., protective barrier devices or sheaths) might provide an alternative to conventional liquid chemical high-level disinfection/sterilization^{143, 144}. Another new technology is a swallowable camera-in-a-capsule that travels through the digestive tract and transmits color pictures of the small intestine to a receiver worn outside the body. This capsule currently does not replace colonoscopies.

Published recommendations for cleaning and disinfecting endoscopic equipment should be strictly followed^{12, 38, 108, 113-116, 145-148}. Unfortunately, audits have shown that personnel do not consistently adhere to guidelines on reprocessing¹⁴⁹⁻¹⁵¹ and outbreaks of infection continue to occur.¹⁵²⁻¹⁵⁴ To ensure

reprocessing personnel are properly trained, each person who reprocesses endoscopic instruments should receive initial and annual competency testing^{38, 155}.

In general, endoscope disinfection or sterilization with a liquid chemical sterilant involves five steps after leak testing:

1. Clean: mechanically clean internal and external surfaces, including brushing internal channels and flushing each internal channel with water and a detergent or enzymatic cleaners (leak testing is recommended for endoscopes before immersion).
2. Disinfect: immerse endoscope in high-level disinfectant (or chemical sterilant) and perfuse (eliminates air pockets and ensures contact of the germicide with the internal channels) disinfectant into all accessible channels, such as the suction/biopsy channel and air/water channel and expose for a time recommended for specific products.
3. Rinse: rinse the endoscope and all channels with sterile water, filtered water (commonly used with AERs) or tap water (i.e., high-quality potable water that meets federal clean water standards at the point of use).
4. Dry: rinse the insertion tube and inner channels with alcohol, and dry with forced air after disinfection and before storage.

Store: store the endoscope in a way that prevents recontamination and promotes drying (e.g., hung vertically). Drying the endoscope (steps 3 and 4) is essential to greatly reduce the chance of recontamination of the endoscope by microorganisms that can be present in the rinse water^{116, 156}. One study demonstrated that reprocessed endoscopes (i.e., air/water channel, suction/biopsy channel) generally were negative (100% after 24 hours; 90% after 7 days [1 CFU of coagulase-negative *Staphylococcus* in one channel]) for bacterial growth when stored by hanging vertically in a ventilated cabinet¹⁵⁷. Other investigators found all endoscopes were bacteria-free immediately after high-level disinfection, and only four of 135 scopes were positive during the subsequent 5-day assessment (skin bacteria cultured from endoscope surfaces). All flush-through samples remained sterile¹⁵⁸. Because tapwater can contain low levels of microorganisms¹⁵⁹, some researchers have suggested that only sterile water (which can be prohibitively expensive)¹⁶⁰ or AER filtered water be used. The suggestion to use only sterile water or filtered water is not consistent with published guidelines that allow tapwater with an alcohol rinse and forced air-drying^{38, 108, 113} or the scientific literature.^{39, 93} In addition, no evidence of disease transmission has been found when a tap water rinse is followed by an alcohol rinse and forced-air drying. AERs produce filtered water by passage through a bacterial filter (e.g., 0.2 μ). Filtered rinse water was identified as a source of bacterial contamination in a study that cultured the accessory and suction channels of endoscopes and the internal chambers of AERs during 1996–2001 and reported 8.7% of samples collected during 1996–1998 had bacterial growth, with 54% being *Pseudomonas* species. After a system of hot water flushing of the piping (60°C for 60 minutes daily) was introduced, the frequency of positive cultures fell to approximately 2% with only rare isolation of >10 CFU/mL¹⁶¹. In addition to the endoscope reprocessing steps, a protocol should be developed that ensures the user knows whether an endoscope has been appropriately cleaned and disinfected (e.g., using a room or cabinet for processed endoscopes only) or has not been reprocessed. When users leave endoscopes on movable carts, confusion can result about whether the endoscope has been processed. Although one guideline recommended endoscopes (e.g., duodenoscopes) be reprocessed immediately before use¹⁴⁷, other guidelines do not require this activity^{38, 108, 115} and except for the Association of periOperative Registered Nurses (AORN), professional organizations do not recommend that reprocessing be repeated as long as the original processing is done correctly. As part of a quality assurance program, healthcare facility personnel can consider random bacterial surveillance cultures of processed endoscopes to ensure high-level disinfection or sterilization^{7, 162-164}. Reprocessed endoscopes should be free of microbial pathogens except for small numbers of relatively avirulent microbes that represent exogenous environmental contamination (e.g., coagulase-negative *Staphylococcus*, *Bacillus* species, diphtheroids). Although recommendations exist for the final rinse water used during endoscope reprocessing to be microbiologically cultured at least monthly¹⁶⁵, a microbiologic standard has not been

set, and the value of routine endoscope cultures has not been shown¹⁶⁶. In addition, neither the routine culture of reprocessed endoscopes nor the final rinse water has been validated by correlating viable counts on an endoscope to infection after an endoscopic procedure. If reprocessed endoscopes were cultured, sampling the endoscope would assess water quality and other important steps (e.g., disinfectant effectiveness, exposure time, cleaning) in the reprocessing procedure. A number of methods for sampling endoscopes and water have been described^{23, 157, 161, 163, 167, 168}. Novel approaches (e.g., detection of adenosine triphosphate [ATP]) to evaluate the effectiveness of endoscope cleaning^{169, 170} or endoscope reprocessing¹⁷¹ also have been evaluated, but no method has been established as a standard for assessing the outcome of endoscope reprocessing.

The carrying case used to transport clean and reprocessed endoscopes outside the health-care environment should not be used to store an endoscope or to transport the instrument within the health-care facility. A contaminated endoscope should never be placed in the carrying case because the case can also become contaminated. When the endoscope is removed from the case, properly reprocessed, and put back in the case, the case could recontaminate the endoscope. A contaminated carrying case should be discarded (Olympus America, June 2002, written communication).

Infection-control professionals should ensure that institutional policies are consistent with national guidelines and conduct infection-control rounds periodically (e.g., at least annually) in areas where endoscopes are reprocessed to ensure policy compliance. Breaches in policy should be documented and corrective action instituted. In incidents in which endoscopes were not exposed to a high-level disinfection process, patients exposed to potentially contaminated endoscopes have been assessed for possible acquisition of HIV, HBV, and hepatitis C virus (HCV). A 14-step method for managing a failure incident associated with high-level disinfection or sterilization has been described [Rutala WA, 2006 #12512]. The possible transmission of bloodborne and other infectious agents highlights the importance of rigorous infection control^{172, 173}.

Laparoscopes and Arthroscopes

Although high-level disinfection appears to be the minimum standard for processing laparoscopes and arthroscopes between patients^{28, 86, 174, 175}, this practice continues to be debated^{89, 90, 176}. However, neither side in the high-level disinfection versus sterilization debate has sufficient data on which to base its conclusions. Proponents of high-level disinfection refer to membership surveys²⁹ or institutional experiences⁸⁷ involving more than 117,000 and 10,000 laparoscopic procedures, respectively, that cite a low risk for infection (<0.3%) when high-level disinfection is used for gynecologic laparoscopic equipment. Only one infection in the membership survey was linked to spores. In addition, growth of common skin microorganisms (e.g., *Staphylococcus epidermidis*, diphtheroids) has been documented from the umbilical area even after skin preparation with povidone-iodine and ethyl alcohol. Similar organisms were recovered in some instances from the pelvic serosal surfaces or from the laparoscopic telescopes, suggesting that the microorganisms probably were carried from the skin into the peritoneal cavity^{177, 178}. Proponents of sterilization focus on the possibility of transmitting infection by spore-forming organisms. Researchers have proposed several reasons why sterility was not necessary for all laparoscopic equipment: only a limited number of organisms (usually ≤ 10) are introduced into the peritoneal cavity during laparoscopy; minimal damage is done to inner abdominal structures with little devitalized tissue; the peritoneal cavity tolerates small numbers of spore-forming bacteria; equipment is simple to clean and disinfect; surgical sterility is relative; the natural bioburden on rigid lumened devices is low¹⁷⁹; and no evidence exists that high-level disinfection instead of sterilization increases the risk for infection^{87, 89, 90}. With the advent of laparoscopic cholecystectomy, concern about high-level disinfection is justifiable because the degree of tissue damage and bacterial contamination is greater than with laparoscopic procedures in gynecology. Failure to completely disassemble, clean, and high-level disinfect laparoscope parts has led to infections in patients¹⁸⁰. Data from one study suggested that disassembly, cleaning, and proper reassembly of laparoscopic equipment used in gynecologic procedures before steam sterilization presents no risk for infection¹⁸¹.

As with laparoscopes and other equipment that enter sterile body sites, arthroscopes ideally should be sterilized before used. Older studies demonstrated that these instruments were commonly (57%) only high-level disinfected in the United States^{28, 86}. A later survey (with a response rate of only 5%) reported that high-level disinfection was used by 31% and a sterilization process in the remainder of the health-care facilities³⁰. High-level disinfection rather than sterilization presumably has been used because the incidence of infection is low and the few infections identified probably are unrelated to the use of high-level disinfection rather than sterilization. A retrospective study of 12,505 arthroscopic procedures found an infection rate of 0.04% (five infections) when arthroscopes were soaked in 2% glutaraldehyde for 15–20 minutes. Four infections were caused by *S. aureus*; the fifth was an anaerobic streptococcal infection⁸⁸. Because these organisms are very susceptible to high-level disinfectants, such as 2% glutaraldehyde, the infections most likely originated from the patient's skin. Two cases of *Clostridium perfringens* arthritis have been reported when the arthroscope was disinfected with glutaraldehyde for an exposure time that is not effective against spores^{182, 183}.

Although only limited data are available, the evidence does not demonstrate that high-level disinfection of arthroscopes and laparoscopes poses an infection risk to the patient. For example, a prospective study that compared the reprocessing of arthroscopes and laparoscopes (per 1,000 procedures) with EtO sterilization to high-level disinfection with glutaraldehyde found no statistically significant difference in infection risk between the two methods (i.e., EtO, 7.5/1,000 procedures; glutaraldehyde, 2.5/1,000 procedures)⁸⁹. Although the debate for high-level disinfection versus sterilization of laparoscopes and arthroscopes will go unsettled until well-designed, randomized clinical trials are published, this guideline should be followed^{1, 17}. That is, laparoscopes, arthroscopes, and other scopes that enter normally sterile tissue should be sterilized before each use; if this is not feasible, they should receive at least high-level disinfection.

Tonometers, Cervical Diaphragm Fitting Rings, Cryosurgical Instruments, and Endocavitary Probes

Disinfection strategies vary widely for other semicritical items (e.g., applanation tonometers, rectal/vaginal probes, cryosurgical instruments, and diaphragm fitting rings). FDA requests that device manufacturers include at least one validated cleaning and disinfection/sterilization protocol in the labeling for their devices. As with all medications and devices, users should be familiar with the label instructions. One study revealed that no uniform technique was in use for disinfection of applanation tonometers, with disinfectant contact times varying from <15 sec to 20 minutes²⁸. In view of the potential for transmission of viruses (e.g., herpes simplex virus [HSV], adenovirus 8, or HIV)¹⁸⁴ by tonometer tips, CDC recommended that the tonometer tips be wiped clean and disinfected for 5–10 minutes with either 3% hydrogen peroxide, 5000 ppm chlorine, 70% ethyl alcohol, or 70% isopropyl alcohol⁹⁵. However, more recent data suggest that 3% hydrogen peroxide and 70% isopropyl alcohol are not effective against adenovirus capable of causing epidemic keratoconjunctivitis and similar viruses and should not be used for disinfecting applanation tonometers^{49, 185, 186}. Structural damage to Schiottz tonometers has been observed with a 1:10 sodium hypochlorite (5,000 ppm chlorine) and 3% hydrogen peroxide¹⁸⁷. After disinfection, the tonometer should be thoroughly rinsed in tapwater and air dried before use. Although these disinfectants and exposure times should kill pathogens that can infect the eyes, no studies directly support this^{188, 189}. The guidelines of the American Academy of Ophthalmology for preventing infections in ophthalmology focus on only one potential pathogen: HIV.¹⁹⁰ Because a short and simple decontamination procedure is desirable in the clinical setting, swabbing the tonometer tip with a 70% isopropyl alcohol wipe sometimes is practiced.¹⁸⁹ Preliminary reports suggest that wiping the tonometer tip with an alcohol swab and then allowing the alcohol to evaporate might be effective in eliminating HSV, HIV, and adenovirus^{189, 191, 192}. However, because these studies involved only a few replicates and were conducted in a controlled laboratory setting, further studies are needed before this technique can be recommended. In addition, two reports have found that disinfection of pneumotonometer tips between uses with a 70% isopropyl alcohol wipe contributed to outbreaks of epidemic keratoconjunctivitis caused

by adenovirus type 8^{193, 194}.

Limited studies have evaluated disinfection techniques for other items that contact mucous membranes, such as diaphragm fitting rings, cryosurgical probes, transesophageal echocardiography probes¹⁹⁵, flexible cystoscopes¹⁹⁶ or vaginal/rectal probes used in sonographic scanning. Lettau, Bond, and McDougal of CDC supported the recommendation of a diaphragm fitting ring manufacturer that involved using a soap-and-water wash followed by a 15-minute immersion in 70% alcohol⁹⁶. This disinfection method should be adequate to inactivate HIV, HBV, and HSV even though alcohols are not classified as high-level disinfectants because their activity against picornaviruses is somewhat limited⁷². No data are available regarding inactivation of human papillomavirus (HPV) by alcohol or other disinfectants because *in vitro* replication of complete virions has not been achieved. Thus, even though alcohol for 15 minutes should kill pathogens of relevance in gynecology, no clinical studies directly support this practice.

Vaginal probes are used in sonographic scanning. A vaginal probe and all endocavitary probes without a probe cover are semicritical devices because they have direct contact with mucous membranes (e.g., vagina, rectum, pharynx). While use of the probe cover could be considered as changing the category, this guideline proposes use of a new condom/probe cover for the probe for each patient, and because condoms/probe covers can fail^{195, 197-199}, the probe also should be high-level disinfected. The relevance of this recommendation is reinforced with the findings that sterile transvaginal ultrasound probe covers have a very high rate of perforations even before use (0%, 25%, and 65% perforations from three suppliers).¹⁹⁹ One study found, after oocyte retrieval use, a very high rate of perforations in used endovaginal probe covers from two suppliers (75% and 81%)¹⁹⁹, other studies demonstrated a lower rate of perforations after use of condoms (2.0% and 0.9%)^{197 200}. Condoms have been found superior to commercially available probe covers for covering the ultrasound probe (1.7% for condoms versus 8.3% leakage for probe covers)²⁰¹. These studies underscore the need for routine probe disinfection between examinations. Although most ultrasound manufacturers recommend use of 2% glutaraldehyde for high-level disinfection of contaminated transvaginal transducers, this agent has been questioned²⁰² because it might shorten the life of the transducer and might have toxic effects on the gametes and embryos²⁰³. An alternative procedure for disinfecting the vaginal transducer involves the mechanical removal of the gel from the transducer, cleaning the transducer in soap and water, wiping the transducer with 70% alcohol or soaking it for 2 minutes in 500 ppm chlorine, and rinsing with tap water and air drying²⁰⁴. The effectiveness of this and other methods²⁰⁰ has not been validated in either rigorous laboratory experiments or in clinical use. High-level disinfection with a product (e.g., hydrogen peroxide) that is not toxic to staff, patients, probes, and retrieved cells should be used until the effectiveness of alternative procedures against microbes of importance at the cavitory site is demonstrated by well-designed experimental scientific studies. Other probes such as rectal, cryosurgical, and transesophageal probes or devices also should be high-level disinfected between patients.

Ultrasound probes used during surgical procedures also can contact sterile body sites. These probes can be covered with a sterile sheath to reduce the level of contamination on the probe and reduce the risk for infection. However, because the sheath does not completely protect the probe, the probes should be sterilized between each patient use as with other critical items. If this is not possible, at a minimum the probe should be high-level disinfected and covered with a sterile probe cover.

Some cryosurgical probes are not fully immersible. During reprocessing, the tip of the probe should be immersed in a high-level disinfectant for the appropriate time; any other portion of the probe that could have mucous membrane contact can be disinfected by immersion or by wrapping with a cloth soaked in a high-level disinfectant to allow the recommended contact time. After disinfection, the probe should be rinsed with tap water and dried before use. Health-care facilities that use nonimmersible probes should replace them as soon as possible with fully immersible probes.

As with other high-level disinfection procedures, proper cleaning of probes is necessary to ensure the success of the subsequent disinfection²⁰⁵. One study demonstrated that vegetative bacteria

inoculated on vaginal ultrasound probes decreased when the probes were cleaned with a towel²⁰⁶. No information is available about either the level of contamination of such probes by potential viral pathogens such as HBV and HPV or their removal by cleaning (such as with a towel). Because these pathogens might be present in vaginal and rectal secretions and contaminate probes during use, high-level disinfection of the probes after such use is recommended.

Dental Instruments

Scientific articles and increased publicity about the potential for transmitting infectious agents in dentistry have focused attention on dental instruments as possible agents for pathogen transmission^{207, 208}. The American Dental Association recommends that surgical and other instruments that normally penetrate soft tissue or bone (e.g., extraction forceps, scalpel blades, bone chisels, periodontal scalers, and surgical burs) be classified as critical devices that should be sterilized after each use or discarded. Instruments not intended to penetrate oral soft tissues or bone (e.g., amalgam condensers, and air/water syringes) but that could contact oral tissues are classified as semicritical, but sterilization after each use is recommended if the instruments are heat-tolerant^{43, 209}. If a semicritical item is heat-sensitive, it should, at a minimum, be processed with high-level disinfection^{43, 210}. Handpieces can be contaminated internally with patient material and should be heat sterilized after each patient. Handpieces that cannot be heat sterilized should not be used.²¹¹ Methods of sterilization that can be used for critical or semicritical dental instruments and materials that are heat-stable include steam under pressure (autoclave), chemical (formaldehyde) vapor, and dry heat (e.g., 320°F for 2 hours). Dental professionals most commonly use the steam sterilizer²¹². All three sterilization procedures can damage some dental instruments, including steam-sterilized hand pieces²¹³. Heat-tolerant alternatives are available for most clinical dental applications and are preferred⁴³.

CDC has divided noncritical surfaces in dental offices into clinical contact and housekeeping surfaces⁴³. Clinical contact surfaces are surfaces that might be touched frequently with gloved hands during patient care or that might become contaminated with blood or other potentially infectious material and subsequently contact instruments, hands, gloves, or devices (e.g., light handles, switches, dental X-ray equipment, chair-side computers). Barrier protective coverings (e.g., clear plastic wraps) can be used for these surfaces, particularly those that are difficult to clean (e.g., light handles, chair switches). The coverings should be changed when visibly soiled or damaged and routinely (e.g., between patients). Protected surfaces should be disinfected at the end of each day or if contamination is evident. If not barrier-protected, these surfaces should be disinfected between patients with an intermediate-disinfectant (i.e., EPA-registered hospital disinfectant with tuberculocidal claim) or low-level disinfectant (i.e., EPA-registered hospital disinfectant with an HBV and HIV label claim)^{43, 214, 215}.

Most housekeeping surfaces need to be cleaned only with a detergent and water or an EPA-registered hospital disinfectant, depending of the nature of the surface and the type and degree of contamination. When housekeeping surfaces are visibly contaminated by blood or body substances, however, prompt removal and surface disinfection is a sound infection control practice and required by the Occupational Safety and Health Administration (OSHA)^{43, 214}.

Several studies have demonstrated variability among dental practices while trying to meet these recommendations^{216, 217}. For example, 68% of respondents believed they were sterilizing their instruments but did not use appropriate chemical sterilants or exposure times and 49% of respondents did not challenge autoclaves with biological indicators²¹⁶. Other investigators using biologic indicators have found a high proportion (15%–65%) of positive spore tests after assessing the efficacy of sterilizers used in dental offices. In one study of Minnesota dental offices, operator error, rather than mechanical malfunction²¹⁸, caused 87% of sterilization failures. Common factors in the improper use of sterilizers include chamber overload, low temperature setting, inadequate exposure time, failure to preheat the sterilizer, and interruption of the cycle.

Mail-return sterilization monitoring services use spore strips to test sterilizers in dental clinics, but

delay caused by mailing to the test laboratory could potentially cause false-negative results. Studies revealed, however, that the post-sterilization time and temperature after a 7-day delay had no influence on the test results²¹⁹. Delays (7 days at 27°C and 37°C, 3-day mail delay) did not cause any predictable pattern of inaccurate spore tests²²⁰.

Disinfection of HBV-, HCV-, HIV- or TB-Contaminated Devices

The CDC recommendation for high-level disinfection of HBV-, HCV-, HIV- or TB-contaminated devices is appropriate because experiments have demonstrated the effectiveness of high-level disinfectants to inactivate these and other pathogens that might contaminate semicritical devices^{61, 62, 73, 81, 105, 121, 125, 221-238}. Nonetheless, some healthcare facilities have modified their disinfection procedures when endoscopes are used with a patient known or suspected to be infected with HBV, HIV, or *M. tuberculosis*^{28, 239}. This is inconsistent with the concept of Standard Precautions that presumes all patients are potentially infected with bloodborne pathogens²²⁸. Several studies have highlighted the inability to distinguish HBV- or HIV-infected patients from noninfected patients on clinical grounds²⁴⁰⁻²⁴². In addition, mycobacterial infection is unlikely to be clinically apparent in many patients. In most instances, hospitals that altered their disinfection procedure used EtO sterilization on the endoscopic instruments because they believed this practice reduced the risk for infection^{28, 239}. EtO is not routinely used for endoscope sterilization because of the lengthy processing time. Endoscopes and other semicritical devices should be managed the same way regardless of whether the patient is known to be infected with HBV, HCV, HIV or *M. tuberculosis*.

An evaluation of a manual disinfection procedure to eliminate HCV from experimentally contaminated endoscopes provided some evidence that cleaning and 2% glutaraldehyde for 20 minutes should prevent transmission²³⁶. A study that used experimentally contaminated hysteroscopes detected HCV by polymerase chain reaction (PCR) in one (3%) of 34 samples after cleaning with a detergent, but no samples were positive after treatment with a 2% glutaraldehyde solution for 20 minutes¹²⁰. Another study demonstrated complete elimination of HCV (as detected by PCR) from endoscopes used on chronically infected patients after cleaning and disinfection for 3–5 minutes in glutaraldehyde¹¹⁸. Similarly, PCR was used to demonstrate complete elimination of HCV after standard disinfection of experimentally contaminated endoscopes²³⁶ and endoscopes used on HCV-antibody-positive patients had no detectable HCV RNA after high-level disinfection²⁴³. The inhibitory activity of a phenolic and a chlorine compound on HCV showed that the phenolic inhibited the binding and replication of HCV, but the chlorine was ineffective, probably because of its low concentration and its neutralization in the presence of organic matter²⁴⁴.

Disinfection in the Hemodialysis Unit

Hemodialysis systems include hemodialysis machines, water supply, water-treatment systems, and distribution systems. During hemodialysis, patients have acquired bloodborne viruses and pathogenic bacteria²⁴⁵⁻²⁴⁷. Cleaning and disinfection are important components of infection control in a hemodialysis center. EPA and FDA regulate disinfectants used to reprocess hemodialyzers, hemodialysis machines, and water-treatment systems.

Noncritical surfaces (e.g., dialysis bed or chair, countertops, external surfaces of dialysis machines, and equipment [scissors, hemostats, clamps, blood pressure cuffs, stethoscopes]) should be disinfected with an EPA-registered disinfectant unless the item is visibly contaminated with blood; in that case a tuberculocidal agent (or a disinfectant with specific label claims for HBV and HIV) or a 1:100 dilution of a hypochlorite solution (500–600 ppm free chlorine) should be used^{246, 248}. This procedure accomplishes two goals: it removes soil on a regular basis and maintains an environment that is consistent with good patient care. Hemodialyzers are disinfected with peracetic acid, formaldehyde, glutaraldehyde, heat pasteurization with citric acid, and chlorine-containing compounds²⁴⁹. Hemodialysis systems usually are disinfected by chlorine-based disinfectants (e.g., sodium hypochlorite), aqueous

formaldehyde, heat pasteurization, ozone, or peracetic acid^{250, 251}. All products must be used according to the manufacturers' recommendations. Some dialysis systems use hot-water disinfection to control microbial contamination.

At its high point, 82% of U.S. chronic hemodialysis centers were reprocessing (i.e., reusing) dialyzers for the same patient using high-level disinfection²⁴⁹. However, one of the large dialysis organizations has decided to phase out reuse and, by 2002 the percentage of dialysis facilities reprocessing hemodialyzers had decreased to 63%²⁵². The two commonly used disinfectants to reprocess dialyzers were peracetic acid and formaldehyde; 72% used peracetic acid and 20% used formaldehyde to disinfect hemodialyzers. Another 4% of the facilities used either glutaraldehyde or heat pasteurization in combination with citric acid²⁵². Infection-control recommendations, including disinfection and sterilization and the use of dedicated machines for hepatitis B surface antigen (HBsAg)-positive patients, in the hemodialysis setting were detailed in two reviews^{245, 246}. The Association for the Advancement of Medical Instrumentation (AAMI) has published recommendations for the reuse of hemodialyzers²⁵³.

Inactivation of *Clostridium difficile*

The source of health-care-associated acquisition of *Clostridium difficile* in nonepidemic settings has not been determined. The environment and carriage on the hands of health-care personnel have been considered possible sources of infection^{66, 254}. Carpeted rooms occupied by a patient with *C. difficile* were more heavily contaminated with *C. difficile* than were noncarpeted rooms²⁵⁵. Because *C. difficile* spore-production can increase when exposed to nonchlorine-based cleaning agents and the spores are more resistant than vegetative cells to commonly used surface disinfectants²⁵⁶, some investigators have recommended use of dilute solutions of hypochlorite (1,600 ppm available chlorine) for routine environmental disinfection of rooms of patients with *C. difficile*-associated diarrhea or colitis²⁵⁷, to reduce the incidence of *C. difficile* diarrhea²⁵⁸, or in units with high *C. difficile* rates.²⁵⁹ Stool samples of patients with symptomatic *C. difficile* colitis contain spores of the organism, as demonstrated by ethanol treatment of the stool to reduce the overgrowth of fecal flora when isolating *C. difficile* in the laboratory^{260, 261}. *C. difficile*-associated diarrhea rates were shown to have decreased markedly in a bone-marrow transplant unit (from 8.6 to 3.3 cases per 1,000 patient-days) during a period of bleach disinfection (1:10 dilution) of environmental surfaces compared with cleaning with a quaternary ammonium compound. Because no EPA-registered products exist that are specific for inactivating *C. difficile* spores, use of diluted hypochlorite should be considered in units with high *C. difficile* rates. Acidified bleach and regular bleach (5000 ppm chlorine) can inactivate 10^6 *C. difficile* spores in ≤ 10 minutes²⁶². However, studies have shown that asymptomatic patients constitute an important reservoir within the health-care facility and that person-to-person transmission is the principal means of transmission between patients. Thus, combined use of hand washing, barrier precautions, and meticulous environmental cleaning with an EPA-registered disinfectant (e.g., germicidal detergent) should effectively prevent spread of the organism²⁶³.

Contaminated medical devices, such as colonoscopes and thermometers, can be vehicles for transmission of *C. difficile* spores²⁶⁴. For this reason, investigators have studied commonly used disinfectants and exposure times to assess whether current practices can place patients at risk. Data demonstrate that 2% glutaraldehyde^{79, 265-267} and peracetic acid^{267, 268} reliably kill *C. difficile* spores using exposure times of 5–20 minutes. *ortho*-Phthalaldehyde and $\geq 0.2\%$ peracetic acid (WA Rutala, personal communication, April 2006) also can inactivate $\geq 10^4$ *C. difficile* spores in 10–12 minutes at 20°C²⁶⁸. Sodium dichloroisocyanurate at a concentration of 1000 ppm available chlorine achieved lower \log_{10} reduction factors against *C. difficile* spores at 10 min, ranging from 0.7 to 1.5, than 0.26% peracetic acid with \log_{10} reduction factors ranging from 2.7 to 6.0²⁶⁸.

OSHA Bloodborne Pathogen Standard

In December 1991, OSHA promulgated a standard entitled "Occupational Exposure to

Bloodborne Pathogens” to eliminate or minimize occupational exposure to bloodborne pathogens²¹⁴. One component of this requirement is that all equipment and environmental and working surfaces be cleaned and decontaminated with an appropriate disinfectant after contact with blood or other potentially infectious materials. Even though the OSHA standard does not specify the type of disinfectant or procedure, the OSHA original compliance document²⁶⁹ suggested that a germicide must be tuberculocidal to kill the HBV. To follow the OSHA compliance document a tuberculocidal disinfectant (e.g., phenolic, and chlorine) would be needed to clean a blood spill. However, in February 1997, OSHA amended its policy and stated that EPA-registered disinfectants labeled as effective against HIV and HBV would be considered as appropriate disinfectants “. . . provided such surfaces have not become contaminated with agent(s) or volumes of or concentrations of agent(s) for which higher level disinfection is recommended.” When bloodborne pathogens other than HBV or HIV are of concern, OSHA continues to require use of EPA-registered tuberculocidal disinfectants or hypochlorite solution (diluted 1:10 or 1:100 with water)^{215, 228}. Studies demonstrate that, in the presence of large blood spills, a 1:10 final dilution of EPA-registered hypochlorite solution initially should be used to inactivate bloodborne viruses^{63, 235} to minimize risk for infection to health-care personnel from percutaneous injury during cleanup.

Emerging Pathogens (*Cryptosporidium*, *Helicobacter pylori*, *Escherichia coli* O157:H7, Rotavirus, Human Papilloma Virus, Norovirus, Severe Acute Respiratory Syndrome [SARS] Coronavirus)

Emerging pathogens are of growing concern to the general public and infection-control professionals. Relevant pathogens include *Cryptosporidium parvum*, *Helicobacter pylori*, *E. coli* O157:H7, HIV, HCV, rotavirus, norovirus, severe acute respiratory syndrome (SARS) coronavirus, multidrug-resistant *M. tuberculosis*, and nontuberculous mycobacteria (e.g., *M. chelonae*). The susceptibility of each of these pathogens to chemical disinfectants and sterilants has been studied. With the exceptions discussed below, all of these emerging pathogens are susceptible to currently available chemical disinfectants and sterilants²⁷⁰.

Cryptosporidium is resistant to chlorine at concentrations used in potable water. *C. parvum* is not completely inactivated by most disinfectants used in healthcare including ethyl alcohol²⁷¹, glutaraldehyde^{271, 272}, 5.25% hypochlorite²⁷¹, peracetic acid²⁷¹, ortho-phthalaldehyde²⁷¹, phenol^{271, 272}, povidone-iodine^{271, 272}, and quaternary ammonium compounds²⁷¹. The only chemical disinfectants and sterilants able to inactivate greater than 3 log₁₀ of *C. parvum* were 6% and 7.5% hydrogen peroxide²⁷¹. Sterilization methods will fully inactivate *C. parvum*, including steam²⁷¹, EtO^{271, 273}, and hydrogen peroxide gas plasma²⁷¹. Although most disinfectants are ineffective against *C. parvum*, current cleaning and disinfection practices appear satisfactory to prevent healthcare-associated transmission. For example, endoscopes are unlikely to be an important vehicle for transmitting *C. parvum* because the results of bacterial studies indicate mechanical cleaning will remove approximately 10⁴ organisms, and drying results in rapid loss of *C. parvum* viability (e.g., 30 minutes, 2.9 log₁₀ decrease; and 60 minutes, 3.8 log₁₀ decrease)²⁷¹.

Chlorine at ~1 ppm has been found capable of eliminating approximately 4 log₁₀ of *E. coli* O157:H7 within 1 minute in a suspension test⁶⁴. Electrolyzed oxidizing water at 23°C was effective in 10 minutes in producing a 5-log₁₀ decrease in *E. coli* O157:H7 inoculated onto kitchen cutting boards²⁷⁴. The following disinfectants eliminated >5 log₁₀ of *E. coli* O157:H7 within 30 seconds: a quaternary ammonium compound, a phenolic, a hypochlorite (1:10 dilution of 5.25% bleach), and ethanol⁵³. Disinfectants including chlorine compounds can reduce *E. coli* O157:H7 experimentally inoculated onto alfalfa seeds or sprouts^{275, 276} or beef carcass surfaces²⁷⁷.

Data are limited on the susceptibility of *H. pylori* to disinfectants. Using a suspension test, one study assessed the effectiveness of a variety of disinfectants against nine strains of *H. pylori*⁶⁰. Ethanol (80%) and glutaraldehyde (0.5%) killed all strains within 15 seconds; chlorhexidine gluconate (0.05%, 1.0%), benzalkonium chloride (0.025%, 0.1%), alkyldiaminoethylglycine hydrochloride (0.1%), povidone-iodine (0.1%), and sodium hypochlorite (150 ppm) killed all strains within 30 seconds. Both ethanol

(80%) and glutaraldehyde (0.5%) retained similar bactericidal activity in the presence of organic matter; the other disinfectants showed reduced bactericidal activity. In particular, the bactericidal activity of povidone-iodine (0.1%) and sodium hypochlorite (150 ppm) markedly decreased in the presence of dried yeast solution with killing times increased to 5 - 10 minutes and 5 - 30 minutes, respectively.

Immersing biopsy forceps in formalin before obtaining a specimen does not affect the ability to culture *H. pylori* from the biopsy specimen²⁷⁸. The following methods are ineffective for eliminating *H. pylori* from endoscopes: cleaning with soap and water^{119, 279}, immersion in 70% ethanol for 3 minutes²⁸⁰, instillation of 70% ethanol¹²⁶, instillation of 30 ml of 83% methanol²⁷⁹, and instillation of 0.2% Hyamine solution²⁸¹. The differing results with regard to the efficacy of ethyl alcohol against *Helicobacter* are unexplained. Cleaning followed by use of 2% alkaline glutaraldehyde (or automated peracetic acid) has been demonstrated by culture to be effective in eliminating *H. pylori*^{119, 279, 282}. Epidemiologic investigations of patients who had undergone endoscopy with endoscopes mechanically washed and disinfected with 2.0%–2.3% glutaraldehyde have revealed no evidence of person-to-person transmission of *H. pylori*^{126, 283}. Disinfection of experimentally contaminated endoscopes using 2% glutaraldehyde (10-minute, 20-minute, 45-minute exposure times) or the peracetic acid system (with and without active peracetic acid) has been demonstrated to be effective in eliminating *H. pylori*¹¹⁹. *H. pylori* DNA has been detected by PCR in fluid flushed from endoscope channels after cleaning and disinfection with 2% glutaraldehyde²⁸⁴. The clinical significance of this finding is unclear. *In vitro* experiments have demonstrated a $>3.5\text{-log}_{10}$ reduction in *H. pylori* after exposure to 0.5 mg/L of free chlorine for 80 seconds²⁸⁵.

An outbreak of healthcare-associated rotavirus gastroenteritis on a pediatric unit has been reported²⁸⁶. Person to person through the hands of health-care workers was proposed as the mechanism of transmission. Prolonged survival of rotavirus on environmental surfaces (90 minutes to >10 days at room temperature) and hands (>4 hours) has been demonstrated. Rotavirus suspended in feces can survive longer^{287, 288}. Vectors have included hands, fomites, air, water, and food^{288, 289}. Products with demonstrated efficacy ($>3\text{ log}_{10}$ reduction in virus) against rotavirus within 1 minute include: 95% ethanol, 70% isopropanol, some phenolics, 2% glutaraldehyde, 0.35% peracetic acid, and some quaternary ammonium compounds^{59, 290-293}. In a human challenge study, a disinfectant spray (0.1% ortho-phenylphenol and 79% ethanol), sodium hypochlorite (800 ppm free chlorine), and a phenol-based product (14.7% phenol diluted 1:256 in tapwater) when sprayed onto contaminated stainless steel disks, were effective in interrupting transfer of a human rotavirus from stainless steel disk to fingerpads of volunteers after an exposure time of 3- 10 minutes. A quaternary ammonium product (7.05% quaternary ammonium compound diluted 1:128 in tapwater) and tapwater allowed transfer of virus⁵².

No data exist on the inactivation of HPV by alcohol or other disinfectants because *in vitro* replication of complete virions has not been achieved. Similarly, little is known about inactivation of noroviruses (members of the family *Caliciviridae* and important causes of gastroenteritis in humans) because they cannot be grown in tissue culture. Improper disinfection of environmental surfaces contaminated by feces or vomitus of infected patients is believed to play a role in the spread of noroviruses in some settings²⁹⁴⁻²⁹⁶. Prolonged survival of a norovirus surrogate (i.e., feline calicivirus virus [FCV], a closely related cultivable virus) has been demonstrated (e.g., at room temperature, FCV in a dried state survived for 21–18 days)²⁹⁷. Inactivation studies with FCV have shown the effectiveness of chlorine, glutaraldehyde, and iodine-based products whereas the quaternary ammonium compound, detergent, and ethanol failed to inactivate the virus completely.²⁹⁷ An evaluation of the effectiveness of several disinfectants against the feline calicivirus found that bleach diluted to 1000 ppm of available chlorine reduced infectivity of FCV by 4.5 logs in 1 minute. Other effective (log_{10} reduction factor of >4 in virus) disinfectants included accelerated hydrogen peroxide, 5,000 ppm (3 min); chlorine dioxide, 1,000 ppm chlorine (1 min); a mixture of four quaternary ammonium compounds, 2,470 ppm (10 min); 79% ethanol with 0.1% quaternary ammonium compound (3 min); and 75% ethanol (10 min)²⁹⁸. A quaternary ammonium compound exhibited activity against feline calicivirus suspensions dried on hard surface carriers in 10 minutes²⁹⁹. Seventy percent ethanol and 70% 1-propanol reduced FCV by a 3–4- log_{10}

reduction in 30 seconds³⁰⁰.

CDC announced that a previously unrecognized human virus from the coronavirus family is the leading hypothesis for the cause of a described syndrome of SARS³⁰¹. Two coronaviruses that are known to infect humans cause one third of common colds and can cause gastroenteritis. The virucidal efficacy of chemical germicides against coronavirus has been investigated. A study of disinfectants against coronavirus 229E found several that were effective after a 1-minute contact time; these included sodium hypochlorite (at a free chlorine concentration of 1,000 ppm and 5,000 ppm), 70% ethyl alcohol, and povidone-iodine (1% iodine)¹⁸⁶. In another study, 70% ethanol, 50% isopropanol, 0.05% benzalkonium chloride, 50 ppm iodine in iodophor, 0.23% sodium chlorite, 1% cresol soap and 0.7% formaldehyde inactivated >3 logs of two animal coronaviruses (mouse hepatitis virus, canine coronavirus) after a 10-minute exposure time³⁰². The activity of povidone-iodine has been demonstrated against human coronaviruses 229E and OC43³⁰³. A study also showed complete inactivation of the SARS coronavirus by 70% ethanol and povidone-iodine with an exposure times of 1 minute and 2.5% glutaraldehyde with an exposure time of 5 minute³⁰⁴. Because the SARS coronavirus is stable in feces and urine at room temperature for at least 1–2 days (WHO, 2003; http://www.who.int/csr/sars/survival_2003_05_04/en/index.html), surfaces might be a possible source of contamination and lead to infection with the SARS coronavirus and should be disinfected. Until more precise information is available, environments in which SARS patients are housed should be considered heavily contaminated, and rooms and equipment should be thoroughly disinfected daily and after the patient is discharged. EPA-registered disinfectants or 1:100 dilution of household bleach and water should be used for surface disinfection and disinfection on noncritical patient-care equipment. High-level disinfection and sterilization of semicritical and critical medical devices, respectively, does not need to be altered for patients with known or suspected SARS.

Free-living amoeba can be pathogenic and can harbor agents of pneumonia such as *Legionella pneumophila*. Limited studies have shown that 2% glutaraldehyde and peracetic acid do not completely inactivate *Acanthamoeba polyphaga* in a 20-minute exposure time for high-level disinfection. If amoeba are found to contaminate instruments and facilitate infection, longer immersion times or other disinfectants may need to be considered³⁰⁵.

Inactivation of Bioterrorist Agents

Publications have highlighted concerns about the potential for biological terrorism^{306, 307}. CDC has categorized several agents as “high priority” because they can be easily disseminated or transmitted from person to person, cause high mortality, and are likely to cause public panic and social disruption³⁰⁸. These agents include *Bacillus anthracis* (the cause of anthrax), *Yersinia pestis* (plague), variola major (smallpox), *Clostridium botulinum* toxin (botulism), *Francisella tularensis* (tularemia), filoviruses (Ebola hemorrhagic fever, Marburg hemorrhagic fever); and arenaviruses (Lassa [Lassa fever], Junin [Argentine hemorrhagic fever]), and related viruses³⁰⁸.

A few comments can be made regarding the role of sterilization and disinfection of potential agents of bioterrorism³⁰⁹. First, the susceptibility of these agents to germicides *in vitro* is similar to that of other related pathogens. For example, variola is similar to vaccinia^{72, 310, 311} and *B. anthracis* is similar to *B. atropaueus* (formerly *B. subtilis*)^{312, 313}. *B. subtilis* spores, for instance, proved as resistant as, if not more resistant than, *B. anthracis* spores (>6 log₁₀ reduction of *B. anthracis* spores in 5 minutes with acidified bleach [5,250 ppm chlorine])³¹³. Thus, one can extrapolate from the larger database available on the susceptibility of genetically similar organisms³¹⁴. Second, many of the potential bioterrorist agents are stable enough in the environment that contaminated environmental surfaces or fomites could lead to transmission of agents such as *B. anthracis*, *F. tularensis*, variola major, *C. botulinum* toxin, and *C. burnetti*³¹⁵. Third, data suggest that current disinfection and sterilization practices are appropriate for managing patient-care equipment and environmental surfaces when potentially contaminated patients are evaluated and/or admitted in a health-care facility after exposure to a bioterrorist agent. For example,

sodium hypochlorite can be used for surface disinfection (see <http://www.epa.gov/pesticides/factsheets/chemicals/bleachfactsheet.htm>). In instances where the health-care facility is the site of a bioterrorist attack, environmental decontamination might require special decontamination procedures (e.g., chlorine dioxide gas for *B. anthracis* spores). Because no antimicrobial products are registered for decontamination of biologic agents after a bioterrorist attack, EPA has granted a crises exemption for each product (see <http://www.epa.gov/pesticides/factsheets/chemicals/bleachfactsheet.htm>). Of only theoretical concern is the possibility that a bioterrorist agent could be engineered to be less susceptible to disinfection and sterilization processes³⁰⁹.

Toxicological, Environmental and Occupational Concerns

Health hazards associated with the use of germicides in healthcare vary from mucous membrane irritation to death, with the latter involving accidental injection by mentally disturbed patients³¹⁶. Although their degrees of toxicity vary³¹⁷⁻³²⁰, all disinfectants should be used with the proper safety precautions³²¹ and only for the intended purpose.

Key factors associated with assessing the health risk of a chemical exposure include the duration, intensity (i.e., how much chemical is involved), and route (e.g., skin, mucous membranes, and inhalation) of exposure. Toxicity can be acute or chronic. Acute toxicity usually results from an accidental spill of a chemical substance. Exposure is sudden and often produces an emergency situation. Chronic toxicity results from repeated exposure to low levels of the chemical over a prolonged period. Employers are responsible for informing workers about the chemical hazards in the workplace and implementing control measures. The OSHA Hazard Communication Standard (29 CFR 1910.1200, 1915.99, 1917.28, 1918.90, 1926.59, and 1928.21) requires manufacturers and importers of hazardous chemicals to develop Material Safety Data Sheets (MSDS) for each chemical or mixture of chemicals. Employers must have these data sheets readily available to employees who work with the products to which they could be exposed.

Exposure limits have been published for many chemicals used in health care to help provide a safe environment and, as relevant, are discussed in each section of this guideline. Only the exposure limits published by OSHA carry the legal force of regulations. OSHA publishes a limit as a time-weighted average (TWA), that is, the average concentration for a normal 8-hour work day and a 40-hour work week to which nearly all workers can be repeatedly exposed to a chemical without adverse health effects. For example, the permissible exposure limit (PEL) for EtO is 1.0 ppm, 8 hour TWA. The CDC National Institute for Occupational Safety and Health (NIOSH) develops recommended exposure limits (RELs). RELs are occupational exposure limits recommended by NIOSH as being protective of worker health and safety over a working lifetime. This limit is frequently expressed as a 40-hour TWA exposure for up to 10 hours per day during a 40-hour work week. These exposure limits are designed for inhalation exposures. Irritant and allergic effects can occur below the exposure limits, and skin contact can result in dermal effects or systemic absorption without inhalation. The American Conference on Governmental Industrial Hygienists (ACGIH) also provides guidelines on exposure limits³²². Information about workplace exposures and methods to reduce them (e.g., work practices, engineering controls, PPE) is available on the OSHA (<http://www.osha.gov>) and NIOSH (<http://www.cdc.gov/niosh>) websites.

Some states have excluded or limited concentrations of certain chemical germicides (e.g., glutaraldehyde, formaldehyde, and some phenols) from disposal through the sewer system. These rules are intended to minimize environmental harm. If health-care facilities exceed the maximum allowable concentration of a chemical (e.g., ≥ 5.0 mg/L), they have three options. First, they can switch to alternative products; for example, they can change from glutaraldehyde to another disinfectant for high-level disinfection or from phenolics to quaternary ammonium compounds for low-level disinfection. Second, the health-care facility can collect the disinfectant and dispose of it as a hazardous chemical. Third, the

facility can use a commercially available small-scale treatment method (e.g., neutralize glutaraldehyde with glycine).

Safe disposal of regulated chemicals is important throughout the medical community. For disposal of large volumes of spent solutions, users might decide to neutralize the microbicidal activity before disposal (e.g., glutaraldehyde). Solutions can be neutralized by reaction with chemicals such as sodium bisulfite^{323, 324} or glycine³²⁵.

European authors have suggested that instruments and ventilation therapy equipment should be disinfected by heat rather than by chemicals. The concerns for chemical disinfection include toxic side effects for the patient caused by chemical residues on the instrument or object, occupational exposure to toxic chemicals, and recontamination by rinsing the disinfectant with microbially contaminated tap water³²⁶.

Disinfection in Ambulatory Care, Home Care, and the Home

With the advent of managed healthcare, increasing numbers of patients are now being cared for in ambulatory-care and home settings. Many patients in these settings might have communicable diseases, immunocompromising conditions, or invasive devices. Therefore, adequate disinfection in these settings is necessary to provide a safe patient environment. Because the ambulatory-care setting (i.e., outpatient facility) provides the same risk for infection as the hospital, the Spaulding classification scheme described in this guideline should be followed (Table 1)¹⁷.

The home environment should be much safer than hospitals or ambulatory care. Epidemics should not be a problem, and cross-infection should be rare. The healthcare provider is responsible for providing the responsible family member information about infection-control procedures to follow in the home, including hand hygiene, proper cleaning and disinfection of equipment, and safe storage of cleaned and disinfected devices. Among the products recommended for home disinfection of reusable objects are bleach, alcohol, and hydrogen peroxide. APIC recommends that reusable objects (e.g., tracheostomy tubes) that touch mucous membranes be disinfected by immersion in 70% isopropyl alcohol for 5 minutes or in 3% hydrogen peroxide for 30 minutes. Additionally, a 1:50 dilution of 5.25%–6.15% sodium hypochlorite (household bleach) for 5 minutes should be effective³²⁷⁻³²⁹. Noncritical items (e.g., blood pressure cuffs, crutches) can be cleaned with a detergent. Blood spills should be handled according to OSHA regulations as previously described (see section on OSHA Bloodborne Pathogen Standard). In general, sterilization of critical items is not practical in homes but theoretically could be accomplished by chemical sterilants or boiling. Single-use disposable items can be used or reusable items sterilized in a hospital^{330, 331}.

Some environmental groups advocate “environmentally safe” products as alternatives to commercial germicides in the home-care setting. These alternatives (e.g., ammonia, baking soda, vinegar, Borax, liquid detergent) are not registered with EPA and should not be used for disinfecting because they are ineffective against *S. aureus*. Borax, baking soda, and detergents also are ineffective against *Salmonella* Typhi and *E. coli*; however, undiluted vinegar and ammonia are effective against *S. Typhi* and *E. coli*^{53, 332, 333}. Common commercial disinfectants designed for home use also are effective against selected antibiotic-resistant bacteria⁵³.

Public concerns have been raised that the use of antimicrobials in the home can promote development of antibiotic-resistant bacteria^{334, 335}. This issue is unresolved and needs to be considered further through scientific and clinical investigations. The public health benefits of using disinfectants in the home are unknown. However, some facts are known: many sites in the home kitchen and bathroom are microbially contaminated³³⁶, use of hypochlorites markedly reduces bacteria³³⁷, and good standards of hygiene (e.g., food hygiene, hand hygiene) can help reduce infections in the home^{338, 339}. In addition, laboratory studies indicate that many commercially prepared household disinfectants are effective against common pathogens⁵³ and can interrupt surface-to-human transmission of pathogens⁴⁸. The “targeted

hygiene concept”—which means identifying situations and areas (e.g., food-preparation surfaces and bathroom) where risk exists for transmission of pathogens—may be a reasonable way to identify when disinfection might be appropriate³⁴⁰.

Susceptibility of Antibiotic-Resistant Bacteria to Disinfectants

As with antibiotics, reduced susceptibility (or acquired “resistance”) of bacteria to disinfectants can arise by either chromosomal gene mutation or acquisition of genetic material in the form of plasmids or transposons^{338, 341-343, 344, 345, 346}. When changes occur in bacterial susceptibility that renders an antibiotic ineffective against an infection previously treatable by that antibiotic, the bacteria are referred to as “resistant.” In contrast, reduced susceptibility to disinfectants does not correlate with failure of the disinfectant because concentrations used in disinfection still greatly exceed the cidal level. Thus, the word “resistance” when applied to these changes is incorrect, and the preferred term is “reduced susceptibility” or “increased tolerance”^{344, 347}. No data are available that show that antibiotic-resistant bacteria are less sensitive to the liquid chemical germicides than antibiotic-sensitive bacteria at currently used germicide contact conditions and concentrations.

MRSA and vancomycin-resistant *Enterococcus* (VRE) are important health-care-associated agents. Some antiseptics and disinfectants have been known for years to be, because of MICs, somewhat less inhibitory to *S. aureus* strains that contain a plasmid-carrying gene encoding resistance to the antibiotic gentamicin³⁴⁴. For example, gentamicin resistance has been shown to also encode reduced susceptibility to propamidine, quaternary ammonium compounds, and ethidium bromide³⁴⁸, and MRSA strains have been found to be less susceptible than methicillin-sensitive *S. aureus* (MSSA) strains to chlorhexidine, propamidine, and the quaternary ammonium compound cetrimide³⁴⁹. In other studies, MRSA and MSSA strains have been equally sensitive to phenols and chlorhexidine, but MRSA strains were slightly more tolerant to quaternary ammonium compounds³⁵⁰. Two gene families (*qacCD* [now referred to as *smr*] and *qacAB*) are involved in providing protection against agents that are components of disinfectant formulations such as quaternary ammonium compounds. Staphylococci have been proposed to evade destruction because the protein specified by the *qacA* determinant is a cytoplasmic-membrane-associated protein involved in an efflux system that actively reduces intracellular accumulation of toxicants, such as quaternary ammonium compounds, to intracellular targets³⁵¹.

Other studies demonstrated that plasmid-mediated formaldehyde tolerance is transferable from *Serratia marcescens* to *E. coli*³⁵² and plasmid-mediated quaternary ammonium tolerance is transferable from *S. aureus* to *E. coli*³⁵³. Tolerance to mercury and silver also is plasmid borne^{341, 343-346}.

Because the concentrations of disinfectants used in practice are much higher than the MICs observed, even for the more tolerant strains, the clinical relevance of these observations is questionable. Several studies have found antibiotic-resistant hospital strains of common healthcare-associated pathogens (i.e., *Enterococcus*, *P. aeruginosa*, *Klebsiella pneumoniae*, *E. coli*, *S. aureus*, and *S. epidermidis*) to be equally susceptible to disinfectants as antibiotic-sensitive strains^{53, 354-356}. The susceptibility of glycopeptide-intermediate *S. aureus* was similar to vancomycin-susceptible, MRSA³⁵⁷. On the basis of these data, routine disinfection and housekeeping protocols do not need to be altered because of antibiotic resistance provided the disinfection method is effective^{358, 359}. A study that evaluated the efficacy of selected cleaning methods (e.g., QUAT-sprayed cloth, and QUAT-immersed cloth) for eliminating VRE found that currently used disinfection processes most likely are highly effective in eliminating VRE. However, surface disinfection must involve contact with all contaminated surfaces³⁵⁸. A new method using an invisible fluorescent marker to objectively evaluate the thoroughness of cleaning activities in patient rooms might lead to improvement in cleaning of all objects and surfaces but needs further evaluation³⁶⁰.

Lastly, does the use of antiseptics or disinfectants facilitate the development of disinfectant-tolerant organisms? Evidence and reviews indicate enhanced tolerance to disinfectants can be

developed in response to disinfectant exposure^{334, 335, 346, 347, 361}. However, the level of tolerance is not important in clinical terms because it is low and unlikely to compromise the effectiveness of disinfectants of which much higher concentrations are used^{347, 362}.

The issue of whether low-level tolerance to germicides selects for antibiotic-resistant strains is unsettled but might depend on the mechanism by which tolerance is attained. For example, changes in the permeability barrier or efflux mechanisms might affect susceptibility to both antibiotics and germicides, but specific changes to a target site might not. Some researchers have suggested that use of disinfectants or antiseptics (e.g., triclosan) could facilitate development of antibiotic-resistant microorganisms^{334, 335, 363}. Although evidence in laboratory studies indicates low-level resistance to triclosan, the concentrations of triclosan in these studies were low (generally <1 µg/mL) and dissimilar from the higher levels used in antimicrobial products (2,000–20,000 µg/mL)^{364, 365}. Thus, researchers can create laboratory-derived mutants that demonstrate reduced susceptibility to antiseptics or disinfectants. In some experiments, such bacteria have demonstrated reduced susceptibility to certain antibiotics³³⁵. There is no evidence that using antiseptics or disinfectants selects for antibiotic-resistant organisms in nature or that such mutants survive in nature³⁶⁶. In addition, the action of antibiotics and the action of disinfectants differ fundamentally. Antibiotics are selectively toxic and generally have a single target site in bacteria, thereby inhibiting a specific biosynthetic process. Germicides generally are considered nonspecific antimicrobials because of a multiplicity of toxic-effect mechanisms or target sites and are broader spectrum in the types of microorganisms against which they are effective^{344, 347}.

The rotational use of disinfectants in some environments (e.g., pharmacy production units) has been recommended and practiced in an attempt to prevent development of resistant microbes^{367, 368}. There have been only rare case reports that appropriately used disinfectants have resulted in a clinical problem arising from the selection or development of nonsusceptible microorganisms³⁶⁹.

Surface Disinfection

Is Surface Disinfection Necessary?

The effective use of disinfectants is part of a multibarrier strategy to prevent health-care–associated infections. Surfaces are considered noncritical items because they contact intact skin. Use of noncritical items or contact with noncritical surfaces carries little risk of causing an infection in patients or staff. Thus, the routine use of germicidal chemicals to disinfect hospital floors and other noncritical items is controversial³⁷⁰⁻³⁷⁵. A 1991 study expanded the Spaulding scheme by dividing the noncritical environmental surfaces into housekeeping surfaces and medical equipment surfaces³⁷⁶. The classes of disinfectants used on housekeeping and medical equipment surfaces can be similar. However, the frequency of decontaminating can vary (see Recommendations). Medical equipment surfaces (e.g., blood pressure cuffs, stethoscopes, hemodialysis machines, and X-ray machines) can become contaminated with infectious agents and contribute to the spread of health-care–associated infections^{248, 375}. For this reason, noncritical medical equipment surfaces should be disinfected with an EPA-registered low- or intermediate-level disinfectant. Use of a disinfectant will provide antimicrobial activity that is likely to be achieved with minimal additional cost or work.

Environmental surfaces (e.g., bedside table) also could potentially contribute to cross-transmission by contamination of health-care personnel from hand contact with contaminated surfaces, medical equipment, or patients^{50, 375, 377}. A paper reviews the epidemiologic and microbiologic data (Table 3) regarding the use of disinfectants on noncritical surfaces³⁷⁸.

Of the seven reasons to use a disinfectant on noncritical surfaces, five are particularly noteworthy and support the use of a germicidal detergent. First, hospital floors become contaminated with microorganisms from settling airborne bacteria: by contact with shoes, wheels, and other objects; and occasionally by spills. The removal of microbes is a component in controlling health-care–associated infections. In an investigation of the cleaning of hospital floors, the use of soap and water (80% reduction) was less effective in reducing the numbers of bacteria than was a phenolic disinfectant (94%–99.9%

reduction)³⁷⁹. However, a few hours after floor disinfection, the bacterial count was nearly back to the pretreatment level. Second, detergents become contaminated and result in seeding the patient's environment with bacteria. Investigators have shown that mop water becomes increasingly dirty during cleaning and becomes contaminated if soap and water is used rather than a disinfectant. For example, in one study, bacterial contamination in soap and water without a disinfectant increased from 10 CFU/mL to 34,000 CFU/mL after cleaning a ward, whereas contamination in a disinfectant solution did not change (20 CFU/mL)³⁸⁰. Contamination of surfaces close to the patient that are frequently touched by the patient or staff (e.g., bed rails) could result in patient exposures³⁸¹. In a study, using of detergents on floors and patient room furniture, increased bacterial contamination of the patients' environmental surfaces was found after cleaning (average increase = 103.6 CFU/24cm²)³⁸². In addition, a *P. aeruginosa* outbreak was reported in a hematology-oncology unit associated with contamination of the surface cleaning equipment when nongermicidal cleaning solutions instead of disinfectants were used to decontaminate the patients' environment³⁸³ and another study demonstrated the role of environmental cleaning in controlling an outbreak of *Acinetobacter baumannii*³⁸⁴. Studies also have shown that, in situations where the cleaning procedure failed to eliminate contamination from the surface and the cloth is used to wipe another surface, the contamination is transferred to that surface and the hands of the person holding the cloth^{381, 385}. Third, the CDC Isolation Guideline recommends that noncritical equipment contaminated with blood, body fluids, secretions, or excretions be cleaned and disinfected after use. The same guideline recommends that, in addition to cleaning, disinfection of the bedside equipment and environmental surfaces (e.g., bedrails, bedside tables, carts, commodes, door-knobs, and faucet handles) is indicated for certain pathogens, e.g., enterococci, which can survive in the inanimate environment for prolonged periods³⁸⁶. Fourth, OSHA requires that surfaces contaminated with blood and other potentially infectious materials (e.g., amniotic, pleural fluid) be disinfected. Fifth, using a single product throughout the facility can simplify both training and appropriate practice.

Reasons also exist for using a detergent alone on floors because noncritical surfaces contribute minimally to endemic health-care-associated infections³⁸⁷, and no differences have been found in health-care-associated infections rates when floors are cleaned with detergent rather than disinfectant^{382, 388, 389}. However, these studies have been small and of short duration and suffer from low statistical power because the outcome—health-care-associated infections—is of low frequency. The low rate of infections makes the efficacy of an intervention statistically difficult to demonstrate. Because housekeeping surfaces are associated with the lowest risk for disease transmission, some researchers have suggested that either detergents or a disinfectant/detergent could be used³⁷⁶. No data exist that show reduced health-care-associated infection rates with use of surface disinfection of floors, but some data demonstrate reduced microbial load associated with the use of disinfectants. Given this information; other information showing that environmental surfaces (e.g., bedside table, bed rails) close to the patient and in outpatient settings³⁹⁰ can be contaminated with epidemiologically important microbes (such as VRE and MRSA)^{47, 390-394}, and data showing these organisms survive on various hospital surfaces^{395, 396}, some researchers have suggested that such surfaces should be disinfected on a regular schedule³⁷⁸. Spot decontamination on fabrics that remain in hospitals or clinic rooms while patients move in and out (e.g., privacy curtains) also should be considered. One study demonstrated the effectiveness of spraying the fabric with 3% hydrogen peroxide³⁹⁷. Future studies should evaluate the level of contamination on noncritical environmental surfaces as a function of high and low hand contact and whether some surfaces (e.g., bed rails) near the patient with high contact frequencies require more frequent disinfection. Regardless of whether a detergent or disinfectant is used on surfaces in a health-care facility, surfaces should be cleaned routinely and when dirty or soiled to provide an aesthetically pleasing environment and to prevent potentially contaminated objects from serving as a source for health-care-associated infections³⁹⁸. The value of designing surfaces (e.g. hexyl-polyvinylpyridine) that kill bacteria on contact³⁹⁹ or have sustained antimicrobial activity⁴⁰⁰ should be further evaluated.

Several investigators have recognized heavy microbial contamination of wet mops and cleaning cloths and the potential for spread of such contamination^{68, 401}. They have shown that wiping hard surfaces with contaminated cloths can contaminate hands, equipment, and other surfaces^{68, 402}. Data

have been published that can be used to formulate effective policies for decontamination and maintenance of reusable cleaning cloths. For example, heat was the most reliable treatment of cleaning cloths as a detergent washing followed by drying at 80°C for 2 hours produced elimination of contamination. However, the dry heating process might be a fire hazard if the mop head contains petroleum-based products or lint builds up within the equipment or vent hose (American Health Care Association, personal communication, March 2003). Alternatively, immersing the cloth in hypochlorite (4,000 ppm) for 2 minutes produced no detectable surviving organisms in 10 of 13 cloths⁴⁰³. If reusable cleaning cloths or mops are used, they should be decontaminated regularly to prevent surface contamination during cleaning with subsequent transfer of organisms from these surfaces to patients or equipment by the hands of health-care workers. Some hospitals have begun using a new mopping technique involving microfiber materials to clean floors. Microfibers are densely constructed, polyester and polyamide (nylon) fibers, that are approximately 1/16 the thickness of a human hair. The positively charged microfibers attract dust (which has a negative charge) and are more absorbent than a conventional, cotton-loop mop. Microfiber materials also can be wet with disinfectants, such as quaternary ammonium compounds. In one study, the microfiber system tested demonstrated superior microbial removal compared with conventional string mops when used with a detergent cleaner (94% vs 68%). The use of a disinfectant did not improve the microbial elimination demonstrated by the microfiber system (95% vs 94%). However, use of disinfectant significantly improved microbial removal when a conventional string mop was used (95% vs 68%) (WA Rutala, unpublished data, August 2006). The microfiber system also prevents the possibility of transferring microbes from room to room because a new microfiber pad is used in each room.

Contact Times for Surface Disinfectants

An important issue concerning use of disinfectants for noncritical surfaces in health-care settings is that the contact time specified on the label of the product is often too long to be practically followed. The labels of most products registered by EPA for use against HBV, HIV, or *M. tuberculosis* specify a contact time of 10 minutes. Such a long contact time is not practical for disinfection of environmental surfaces in a health-care setting because most health-care facilities apply a disinfectant and allow it to dry (~1 minute). Multiple scientific papers have demonstrated significant microbial reduction with contact times of 30 to 60 seconds^{46-56, 58-64}. In addition, EPA will approve a shortened contact time for any product for which the manufacturers will submit confirmatory efficacy data.

Currently, some EPA-registered disinfectants have contact times of one to three minutes. By law, users must follow all applicable label instructions for EPA-registered products. Ideally, product users should consider and use products that have the shortened contact time. However, disinfectant manufacturers also need to obtain EPA approval for shortened contact times so these products will be used correctly and effectively in the health-care environment.

Air Disinfection

Disinfectant spray-fog techniques for antimicrobial control in hospital rooms has been used. This technique of spraying of disinfectants is an unsatisfactory method of decontaminating air and surfaces and is not recommended for general infection control in routine patient-care areas³⁸⁶. Disinfectant fogging is rarely, if ever, used in U.S. healthcare facilities for air and surface disinfection in patient-care areas. Methods (e.g., filtration, ultraviolet germicidal irradiation, chlorine dioxide) to reduce air contamination in the healthcare setting are discussed in another guideline²³.

Microbial Contamination of Disinfectants

Contaminated disinfectants and antiseptics have been occasional vehicles of health-care infections and pseudoepidemics for more than 50 years. Published reports describing contaminated disinfectants and antiseptic solutions leading to health-care-associated infections have been summarized

⁴⁰⁴. Since this summary additional reports have been published ⁴⁰⁵⁻⁴⁰⁸. An examination of reports of disinfectants contaminated with microorganisms revealed noteworthy observations. Perhaps most importantly, high-level disinfectants/liquid chemical sterilants have not been associated with outbreaks due to intrinsic or extrinsic contamination. Members of the genus *Pseudomonas* (e.g., *P. aeruginosa*) are the most frequent isolates from contaminated disinfectants—recovered from 80% of contaminated products. Their ability to remain viable or grow in use-dilutions of disinfectants is unparalleled. This survival advantage for *Pseudomonas* results presumably from their nutritional versatility, their unique outer membrane that constitutes an effective barrier to the passage of germicides, and/or efflux systems ⁴⁰⁹. Although the concentrated solutions of the disinfectants have not been demonstrated to be contaminated at the point of manufacture, an undiluted phenolic can be contaminated by a *Pseudomonas* sp. during use ⁴¹⁰. In most of the reports that describe illness associated with contaminated disinfectants, the product was used to disinfect patient-care equipment, such as cystoscopes, cardiac catheters, and thermometers. Germicides used as disinfectants that were reported to have been contaminated include chlorhexidine, quaternary ammonium compounds, phenolics, and pine oil.

The following control measures should be instituted to reduce the frequency of bacterial growth in disinfectants and the threat of serious healthcare-associated infections from the use of such contaminated products ⁴⁰⁴. First, some disinfectants should not be diluted; those that are diluted must be prepared correctly to achieve the manufacturers' recommended use-dilution. Second, infection-control professionals must learn from the literature what inappropriate activities result in extrinsic contamination (i.e., at the point of use) of germicides and train users to prevent recurrence. Common sources of extrinsic contamination of germicides in the reviewed literature are the water to make working dilutions, contaminated containers, and general contamination of the hospital areas where the germicides are prepared and/or used. Third, stock solutions of germicides must be stored as indicated on the product label. EPA verifies manufacturers' efficacy claims against microorganisms. These measures should provide assurance that products meeting the EPA registration requirements can achieve a certain level of antimicrobial activity when used as directed.

FACTORS AFFECTING THE EFFICACY OF DISINFECTION AND STERILIZATION

The activity of germicides against microorganisms depends on a number of factors, some of which are intrinsic qualities of the organism, others of which are the chemical and external physical environment. Awareness of these factors should lead to better use of disinfection and sterilization processes and will be briefly reviewed. More extensive consideration of these and other factors is available elsewhere^{13, 14, 16, 411-413}.

Number and Location of Microorganisms

All other conditions remaining constant, the larger the number of microbes, the more time a germicide needs to destroy all of them. Spaulding illustrated this relation when he employed identical test conditions and demonstrated that it took 30 minutes to kill 10 *B. atrophaeus* (formerly *Bacillus subtilis*) spores but 3 hours to kill 100,000 *Bacillus atrophaeus* spores. This reinforces the need for scrupulous cleaning of medical instruments before disinfection and sterilization. Reducing the number of microorganisms that must be inactivated through meticulous cleaning, increases the margin of safety when the germicide is used according to the labeling and shortens the exposure time required to kill the entire microbial load. Researchers also have shown that aggregated or clumped cells are more difficult to inactivate than monodispersed cells⁴¹⁴.

The location of microorganisms also must be considered when factors affecting the efficacy of germicides are assessed. Medical instruments with multiple pieces must be disassembled and equipment such as endoscopes that have crevices, joints, and channels are more difficult to disinfect than are flat-surface equipment because penetration of the disinfectant of all parts of the equipment is more difficult. Only surfaces that directly contact the germicide will be disinfected, so there must be no air pockets and the equipment must be completely immersed for the entire exposure period. Manufacturers should be encouraged to produce equipment engineered for ease of cleaning and disinfection.

Innate Resistance of Microorganisms

Microorganisms vary greatly in their resistance to chemical germicides and sterilization processes (Figure 1)³⁴². Intrinsic resistance mechanisms in microorganisms to disinfectants vary. For example, spores are resistant to disinfectants because the spore coat and cortex act as a barrier, mycobacteria have a waxy cell wall that prevents disinfectant entry, and gram-negative bacteria possess an outer membrane that acts as a barrier to the uptake of disinfectants^{341, 343-345}. Implicit in all disinfection strategies is the consideration that the most resistant microbial subpopulation controls the sterilization or disinfection time. That is, to destroy the most resistant types of microorganisms (i.e., bacterial spores), the user needs to employ exposure times and a concentration of germicide needed to achieve complete destruction. Except for prions, bacterial spores possess the highest innate resistance to chemical germicides, followed by coccidia (e.g., *Cryptosporidium*), mycobacteria (e.g., *M. tuberculosis*), nonlipid or small viruses (e.g., poliovirus, and coxsackievirus), fungi (e.g., *Aspergillus*, and *Candida*), vegetative bacteria (e.g., *Staphylococcus*, and *Pseudomonas*) and lipid or medium-size viruses (e.g., herpes, and HIV). The germicidal resistance exhibited by the gram-positive and gram-negative bacteria is similar with some exceptions (e.g., *P. aeruginosa* which shows greater resistance to some disinfectants)^{369, 415, 416}. *P. aeruginosa* also is significantly more resistant to a variety of disinfectants in its "naturally occurring" state than are cells subcultured on laboratory media^{415, 417}. *Rickettsiae*, *Chlamydiae*, and mycoplasma cannot be placed in this scale of relative resistance because information about the efficacy of germicides against these agents is limited⁴¹⁸. Because these microorganisms contain lipid and are similar in structure and composition to other bacteria, they can be predicted to be inactivated by the same germicides that destroy lipid viruses and vegetative bacteria. A known exception to this supposition is *Coxiella burnetti*, which has demonstrated resistance to disinfectants⁴¹⁹.

Concentration and Potency of Disinfectants

With other variables constant, and with one exception (iodophors), the more concentrated the

disinfectant, the greater its efficacy and the shorter the time necessary to achieve microbial kill. Generally not recognized, however, is that all disinfectants are not similarly affected by concentration adjustments. For example, quaternary ammonium compounds and phenol have a concentration exponent of 1 and 6, respectively; thus, halving the concentration of a quaternary ammonium compound requires doubling its disinfecting time, but halving the concentration of a phenol solution requires a 64-fold (i.e., 2^6) increase in its disinfecting time^{365, 413, 420}.

Considering the length of the disinfection time, which depends on the potency of the germicide, also is important. This was illustrated by Spaulding who demonstrated using the mucin-loop test that 70% isopropyl alcohol destroyed 10^4 *M. tuberculosis* in 5 minutes, whereas a simultaneous test with 3% phenolic required 2–3 hours to achieve the same level of microbial kill¹⁴.

Physical and Chemical Factors

Several physical and chemical factors also influence disinfectant procedures: temperature, pH, relative humidity, and water hardness. For example, the activity of most disinfectants increases as the temperature increases, but some exceptions exist. Furthermore, too great an increase in temperature causes the disinfectant to degrade and weakens its germicidal activity and thus might produce a potential health hazard.

An increase in pH improves the antimicrobial activity of some disinfectants (e.g., glutaraldehyde, quaternary ammonium compounds) but decreases the antimicrobial activity of others (e.g., phenols, hypochlorites, and iodine). The pH influences the antimicrobial activity by altering the disinfectant molecule or the cell surface⁴¹³.

Relative humidity is the single most important factor influencing the activity of gaseous disinfectants/sterilants, such as EtO, chlorine dioxide, and formaldehyde.

Water hardness (i.e., high concentration of divalent cations) reduces the rate of kill of certain disinfectants because divalent cations (e.g., magnesium, calcium) in the hard water interact with the disinfectant to form insoluble precipitates^{13, 421}.

Organic and Inorganic Matter

Organic matter in the form of serum, blood, pus, or fecal or lubricant material can interfere with the antimicrobial activity of disinfectants in at least two ways. Most commonly, interference occurs by a chemical reaction between the germicide and the organic matter resulting in a complex that is less germicidal or nongermicidal, leaving less of the active germicide available for attacking microorganisms. Chlorine and iodine disinfectants, in particular, are prone to such interaction. Alternatively, organic material can protect microorganisms from attack by acting as a physical barrier^{422, 423}.

The effects of inorganic contaminants on the sterilization process were studied during the 1950s and 1960s^{424, 425}. These and other studies show the protection by inorganic contaminants of microorganisms to all sterilization processes results from occlusion in salt crystals^{426, 427}. This further emphasizes the importance of meticulous cleaning of medical devices before any sterilization or disinfection procedure because both organic and inorganic soils are easily removed by washing⁴²⁶.

Duration of Exposure

Items must be exposed to the germicide for the appropriate minimum contact time. Multiple investigators have demonstrated the effectiveness of low-level disinfectants against vegetative bacteria (e.g., *Listeria*, *E. coli*, *Salmonella*, VRE, MRSA), yeasts (e.g., *Candida*), mycobacteria (e.g., *M. tuberculosis*), and viruses (e.g., poliovirus) at exposure times of 30–60 seconds⁴⁶⁻⁶⁴. By law, all applicable label instructions on EPA-registered products must be followed. If the user selects exposure conditions that differ from those on the EPA-registered product label, the user assumes liability for any injuries resulting from off-label use and is potentially subject to enforcement action under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA)

All lumens and channels of endoscopic instruments must contact the disinfectant. Air pockets interfere with the disinfection process, and items that float on the disinfectant will not be disinfected. The disinfectant must be introduced reliably into the internal channels of the device. The exact times for disinfecting medical items are somewhat elusive because of the effect of the aforementioned factors on disinfection efficacy. Certain contact times have proved reliable (Table 1), but, in general, longer contact times are more effective than shorter contact times.

Biofilms

Microorganisms may be protected from disinfectants by production of thick masses of cells⁴²⁸ and extracellular materials, or biofilms⁴²⁹⁻⁴³⁵. Biofilms are microbial communities that are tightly attached to surfaces and cannot be easily removed. Once these masses form, microbes within them can be resistant to disinfectants by multiple mechanisms, including physical characteristics of older biofilms, genotypic variation of the bacteria, microbial production of neutralizing enzymes, and physiologic gradients within the biofilm (e.g., pH). Bacteria within biofilms are up to 1,000 times more resistant to antimicrobials than are the same bacteria in suspension⁴³⁶. Although new decontamination methods⁴³⁷ are being investigated for removing biofilms, chlorine and monochloramines can effectively inactivate biofilm bacteria^{431 438}. Investigators have hypothesized that the glycocalyx-like cellular masses on the interior walls of polyvinyl chloride pipe would protect embedded organisms from some disinfectants and be a reservoir for continuous contamination^{429, 430, 439}. Biofilms have been found in whirlpools⁴⁴⁰, dental unit waterlines⁴⁴¹, and numerous medical devices (e.g., contact lenses, pacemakers, hemodialysis systems, urinary catheters, central venous catheters, endoscopes)^{434, 436, 438, 442}. Their presence can have serious implications for immunocompromised patients and patients who have indwelling medical devices. Some enzymes^{436, 443, 444} and detergents⁴³⁶ can degrade biofilms or reduce numbers of viable bacteria within a biofilm, but no products are EPA-registered or FDA-cleared for this purpose.

CLEANING

Cleaning is the removal of foreign material (e.g., soil, and organic material) from objects and is normally accomplished using water with detergents or enzymatic products. Thorough cleaning is required before high-level disinfection and sterilization because inorganic and organic materials that remain on the surfaces of instruments interfere with the effectiveness of these processes. Also, if soiled materials dry or bake onto the instruments, the removal process becomes more difficult and the disinfection or sterilization process less effective or ineffective. Surgical instruments should be presoaked or rinsed to prevent drying of blood and to soften or remove blood from the instruments.

Cleaning is done manually in use areas without mechanical units (e.g., ultrasonic cleaners or washer-disinfectors) or for fragile or difficult-to-clean instruments. With manual cleaning, the two essential components are friction and fluidics. Friction (e.g., rubbing/scrubbing the soiled area with a brush) is an old and dependable method. Fluidics (i.e., fluids under pressure) is used to remove soil and debris from internal channels after brushing and when the design does not allow passage of a brush through a channel⁴⁴⁵. When a washer-disinfector is used, care should be taken in loading instruments: hinged instruments should be opened fully to allow adequate contact with the detergent solution; stacking of instruments in washers should be avoided; and instruments should be disassembled as much as possible.

The most common types of mechanical or automatic cleaners are ultrasonic cleaners, washer-decontaminators, washer-disinfectors, and washer-sterilizers. Ultrasonic cleaning removes soil by cavitation and implosion in which waves of acoustic energy are propagated in aqueous solutions to disrupt the bonds that hold particulate matter to surfaces. Bacterial contamination can be present in used ultrasonic cleaning solutions (and other used detergent solutions) because these solutions generally do not make antibacterial label claims⁴⁴⁶. Even though ultrasound alone does not significantly inactivate bacteria, sonication can act synergistically to increase the cidal efficacy of a disinfectant⁴⁴⁷. Users of ultrasonic cleaners should be aware that the cleaning fluid could result in endotoxin contamination of surgical instruments, which could cause severe inflammatory reactions⁴⁴⁸. Washer-sterilizers are modified steam sterilizers that clean by filling the chamber with water and detergent through which steam passes to provide agitation. Instruments are subsequently rinsed and subjected to a short steam-sterilization cycle. Another washer-sterilizer employs rotating spray arms for a wash cycle followed by a steam sterilization cycle at 285°F^{449, 450}. Washer-decontaminators/disinfectors act like a dishwasher that uses a combination of water circulation and detergents to remove soil. These units sometimes have a cycle that subjects the instruments to a heat process (e.g., 93°C for 10 minutes)⁴⁵¹. Washer-disinfectors are generally computer-controlled units for cleaning, disinfecting, and drying solid and hollow surgical and medical equipment. In one study, cleaning (measured as 5–6 log₁₀ reduction) was achieved on surfaces that had adequate contact with the water flow in the machine⁴⁵². Detailed information about cleaning and preparing supplies for terminal sterilization is provided by professional organizations^{453, 454} and books⁴⁵⁵. Studies have shown that manual and mechanical cleaning of endoscopes achieves approximately a 4-log₁₀ reduction of contaminating organisms^{83, 104, 456, 457}. Thus, cleaning alone effectively reduces the number of microorganisms on contaminated equipment. In a quantitative analysis of residual protein contamination of reprocessed surgical instruments, median levels of residual protein contamination per instrument for five trays were 267, 260, 163, 456, and 756 µg⁴⁵⁸. In another study, the median amount of protein from reprocessed surgical instruments from different hospitals ranged from 8 µg to 91 µg⁴⁵⁹. When manual methods were compared with automated methods for cleaning reusable accessory devices used for minimally invasive surgical procedures, the automated method was more efficient for cleaning biopsy forceps and ported and nonported laparoscopic devices and achieved a >99% reduction in soil parameters (i.e., protein, carbohydrate, hemoglobin) in the ported and nonported laparoscopic devices^{460, 461}.

For instrument cleaning, a neutral or near-neutral pH detergent solution commonly is used because such solutions generally provide the best material compatibility profile and good soil removal.

Enzymes, usually proteases, sometimes are added to neutral pH solutions to assist in removing organic material. Enzymes in these formulations attack proteins that make up a large portion of common soil (e.g., blood, pus). Cleaning solutions also can contain lipases (enzymes active on fats) and amylases (enzymes active on starches). Enzymatic cleaners are not disinfectants, and proteinaceous enzymes can be inactivated by germicides. As with all chemicals, enzymes must be rinsed from the equipment or adverse reactions (e.g., fever, residual amounts of high-level disinfectants, proteinaceous residue) could result^{462, 463}. Enzyme solutions should be used in accordance with manufacturer's instructions, which include proper dilution of the enzymatic detergent and contact with equipment for the amount of time specified on the label⁴⁶³. Detergent enzymes can result in asthma or other allergic effects in users. Neutral pH detergent solutions that contain enzymes are compatible with metals and other materials used in medical instruments and are the best choice for cleaning delicate medical instruments, especially flexible endoscopes⁴⁵⁷. Alkaline-based cleaning agents are used for processing medical devices because they efficiently dissolve protein and fat residues⁴⁶⁴; however, they can be corrosive⁴⁵⁷. Some data demonstrate that enzymatic cleaners are more effective than neutral detergents^{465, 466} in removing microorganisms from surfaces but two more recent studies found no difference in cleaning efficiency between enzymatic and alkaline-based cleaners^{443, 464}. Another study found no significant difference between enzymatic and non-enzymatic cleaners in terms of microbial cleaning efficacy⁴⁶⁷. A new non-enzyme, hydrogen peroxide-based formulation (not FDA-cleared) was as effective as enzymatic cleaners in removing protein, blood, carbohydrate, and endotoxin from surface test carriers⁴⁶⁸. In addition, this product effected a 5- \log_{10} reduction in microbial loads with a 3-minute exposure at room temperature⁴⁶⁸.

Although the effectiveness of high-level disinfection and sterilization mandates effective cleaning, no "real-time" tests exist that can be employed in a clinical setting to verify cleaning. If such tests were commercially available they could be used to ensure an adequate level of cleaning⁴⁶⁹⁻⁴⁷². The only way to ensure adequate cleaning is to conduct a reprocessing verification test (e.g., microbiologic sampling), but this is not routinely recommended⁴⁷³. Validation of the cleaning processes in a laboratory-testing program is possible by microorganism detection, chemical detection for organic contaminants, radionuclide tagging, and chemical detection for specific ions^{426, 471}. During the past few years, data have been published describing use of an artificial soil, protein, endotoxin, X-ray contrast medium, or blood to verify the manual or automated cleaning process^{169, 452, 474-478} and adenosine triphosphate bioluminescence and microbiologic sampling to evaluate the effectiveness of environmental surface cleaning^{170, 479}. At a minimum, all instruments should be individually inspected and be visibly clean.

DISINFECTION

Many disinfectants are used alone or in combinations (e.g., hydrogen peroxide and peracetic acid) in the health-care setting. These include alcohols, chlorine and chlorine compounds, formaldehyde, glutaraldehyde, *ortho*-phthalaldehyde, hydrogen peroxide, iodophors, peracetic acid, phenolics, and quaternary ammonium compounds. Commercial formulations based on these chemicals are considered unique products and must be registered with EPA or cleared by FDA. In most instances, a given product is designed for a specific purpose and is to be used in a certain manner. Therefore, users should read labels carefully to ensure the correct product is selected for the intended use and applied efficiently.

Disinfectants are not interchangeable, and incorrect concentrations and inappropriate disinfectants can result in excessive costs. Because occupational diseases among cleaning personnel have been associated with use of several disinfectants (e.g., formaldehyde, glutaraldehyde, and chlorine), precautions (e.g., gloves and proper ventilation) should be used to minimize exposure^{318, 480, 481}. Asthma and reactive airway disease can occur in sensitized persons exposed to any airborne chemical, including germicides. Clinically important asthma can occur at levels below ceiling levels regulated by OSHA or recommended by NIOSH. The preferred method of control is elimination of the chemical (through engineering controls or substitution) or relocation of the worker.

The following overview of the performance characteristics of each provides users with sufficient information to select an appropriate disinfectant for any item and use it in the most efficient way.

Chemical Disinfectants

Alcohol

Overview. In the healthcare setting, “alcohol” refers to two water-soluble chemical compounds—ethyl alcohol and isopropyl alcohol—that have generally underrated germicidal characteristics⁴⁸². FDA has not cleared any liquid chemical sterilant or high-level disinfectant with alcohol as the main active ingredient. These alcohols are rapidly bactericidal rather than bacteriostatic against vegetative forms of bacteria; they also are tuberculocidal, fungicidal, and virucidal but do not destroy bacterial spores. Their cidal activity drops sharply when diluted below 50% concentration, and the optimum bactericidal concentration is 60%–90% solutions in water (volume/volume)^{483, 484}.

Mode of Action. The most feasible explanation for the antimicrobial action of alcohol is denaturation of proteins. This mechanism is supported by the observation that absolute ethyl alcohol, a dehydrating agent, is less bactericidal than mixtures of alcohol and water because proteins are denatured more quickly in the presence of water^{484, 485}. Protein denaturation also is consistent with observations that alcohol destroys the dehydrogenases of *Escherichia coli*⁴⁸⁶, and that ethyl alcohol increases the lag phase of *Enterobacter aerogenes*⁴⁸⁷ and that the lag phase effect could be reversed by adding certain amino acids. The bacteriostatic action was believed caused by inhibition of the production of metabolites essential for rapid cell division.

Microbicidal Activity. Methyl alcohol (methanol) has the weakest bactericidal action of the alcohols and thus seldom is used in healthcare⁴⁸⁸. The bactericidal activity of various concentrations of ethyl alcohol (ethanol) was examined against a variety of microorganisms in exposure periods ranging from 10 seconds to 1 hour⁴⁸³. *Pseudomonas aeruginosa* was killed in 10 seconds by all concentrations of ethanol from 30% to 100% (v/v), and *Serratia marcescens*, *E. coli* and *Salmonella typhosa* were killed in 10 seconds by all concentrations of ethanol from 40% to 100%. The gram-positive organisms *Staphylococcus aureus* and *Streptococcus pyogenes* were slightly more resistant, being killed in 10 seconds by ethyl alcohol concentrations of 60%–95%. Isopropyl alcohol (isopropanol) was slightly more bactericidal than ethyl alcohol for *E. coli* and *S. aureus*⁴⁸⁹.

Ethyl alcohol, at concentrations of 60%–80%, is a potent virucidal agent inactivating all of the lipophilic viruses (e.g., herpes, vaccinia, and influenza virus) and many hydrophilic viruses (e.g.,

adenovirus, enterovirus, rhinovirus, and rotaviruses but not hepatitis A virus (HAV)⁵⁸ or poliovirus)⁴⁹. Isopropyl alcohol is not active against the nonlipid enteroviruses but is fully active against the lipid viruses⁷². Studies also have demonstrated the ability of ethyl and isopropyl alcohol to inactivate the hepatitis B virus (HBV)^{224, 225} and the herpes virus,⁴⁹⁰ and ethyl alcohol to inactivate human immunodeficiency virus (HIV)²²⁷, rotavirus, echovirus, and astrovirus⁴⁹¹.

In tests of the effect of ethyl alcohol against *M. tuberculosis*, 95% ethanol killed the tubercle bacilli in sputum or water suspension within 15 seconds⁴⁹². In 1964, Spaulding stated that alcohols were the germicide of choice for tuberculocidal activity, and they should be the standard by which all other tuberculocides are compared. For example, he compared the tuberculocidal activity of iodophor (450 ppm), a substituted phenol (3%), and isopropanol (70%/volume) using the mucin-loop test (10⁶ *M. tuberculosis* per loop) and determined the contact times needed for complete destruction were 120–180 minutes, 45–60 minutes, and 5 minutes, respectively. The mucin-loop test is a severe test developed to produce long survival times. Thus, these figures should not be extrapolated to the exposure times needed when these germicides are used on medical or surgical material⁴⁸².

Ethyl alcohol (70%) was the most effective concentration for killing the tissue phase of *Cryptococcus neoformans*, *Blastomyces dermatitidis*, *Coccidioides immitis*, and *Histoplasma capsulatum* and the culture phases of the latter three organisms aerosolized onto various surfaces. The culture phase was more resistant to the action of ethyl alcohol and required about 20 minutes to disinfect the contaminated surface, compared with <1 minute for the tissue phase^{493, 494}.

Isopropyl alcohol (20%) is effective in killing the cysts of *Acanthamoeba culbertsoni* (560) as are chlorhexidine, hydrogen peroxide, and thimerosal⁴⁹⁶.

Uses. Alcohols are not recommended for sterilizing medical and surgical materials principally because they lack sporicidal action and they cannot penetrate protein-rich materials. Fatal postoperative wound infections with *Clostridium* have occurred when alcohols were used to sterilize surgical instruments contaminated with bacterial spores⁴⁹⁷. Alcohols have been used effectively to disinfect oral and rectal thermometers^{498, 499}, hospital pagers⁵⁰⁰, scissors⁵⁰¹, and stethoscopes⁵⁰². Alcohols have been used to disinfect fiberoptic endoscopes^{503, 504} but failure of this disinfectant have lead to infection^{280, 505}. Alcohol towelettes have been used for years to disinfect small surfaces such as rubber stoppers of multiple-dose medication vials or vaccine bottles. Furthermore, alcohol occasionally is used to disinfect external surfaces of equipment (e.g., stethoscopes, ventilators, manual ventilation bags)⁵⁰⁶, CPR manikins⁵⁰⁷, ultrasound instruments⁵⁰⁸ or medication preparation areas. Two studies demonstrated the effectiveness of 70% isopropyl alcohol to disinfect reusable transducer heads in a controlled environment^{509, 510}. In contrast, three bloodstream infection outbreaks have been described when alcohol was used to disinfect transducer heads in an intensive-care setting⁵¹¹.

The documented shortcomings of alcohols on equipment are that they damage the shellac mountings of lensed instruments, tend to swell and harden rubber and certain plastic tubing after prolonged and repeated use, bleach rubber and plastic tiles⁴⁸² and damage tonometer tips (by deterioration of the glue) after the equivalent of 1 working year of routine use⁵¹². Tonometer biprisms soaked in alcohol for 4 days developed rough front surfaces that potentially could cause corneal damage; this appeared to be caused by weakening of the cementing substances used to fabricate the biprisms⁵¹³. Corneal opacification has been reported when tonometer tips were swabbed with alcohol immediately before measurement of intraocular pressure⁵¹⁴. Alcohols are flammable and consequently must be stored in a cool, well-ventilated area. They also evaporate rapidly, making extended exposure time difficult to achieve unless the items are immersed.

Chlorine and Chlorine Compounds

Overview. Hypochlorites, the most widely used of the chlorine disinfectants, are available as liquid (e.g., sodium hypochlorite) or solid (e.g., calcium hypochlorite). The most prevalent chlorine

products in the United States are aqueous solutions of 5.25%–6.15% sodium hypochlorite (see glossary), usually called household bleach. They have a broad spectrum of antimicrobial activity, do not leave toxic residues, are unaffected by water hardness, are inexpensive and fast acting³²⁸, remove dried or fixed organisms and biofilms from surfaces⁴⁶⁵, and have a low incidence of serious toxicity⁵¹⁵⁻⁵¹⁷. Sodium hypochlorite at the concentration used in household bleach (5.25-6.15%) can produce ocular irritation or oropharyngeal, esophageal, and gastric burns^{318, 518-522}. Other disadvantages of hypochlorites include corrosiveness to metals in high concentrations (>500 ppm), inactivation by organic matter, discoloring or “bleaching” of fabrics, release of toxic chlorine gas when mixed with ammonia or acid (e.g., household cleaning agents)⁵²³⁻⁵²⁵, and relative stability³²⁷. The microbicidal activity of chlorine is attributed largely to undissociated hypochlorous acid (HOCl). The dissociation of HOCl to the less microbicidal form (hypochlorite ion OCl⁻) depends on pH. The disinfecting efficacy of chlorine decreases with an increase in pH that parallels the conversion of undissociated HOCl to OCl⁻^{329, 526}. A potential hazard is production of the carcinogen bis(chloromethyl) ether when hypochlorite solutions contact formaldehyde⁵²⁷ and the production of the animal carcinogen trihalomethane when hot water is hyperchlorinated⁵²⁸. After reviewing environmental fate and ecologic data, EPA has determined the currently registered uses of hypochlorites will not result in unreasonable adverse effects to the environment⁵²⁹.

Alternative compounds that release chlorine and are used in the health-care setting include demand-release chlorine dioxide, sodium dichloroisocyanurate, and chloramine-T. The advantage of these compounds over the hypochlorites is that they retain chlorine longer and so exert a more prolonged bactericidal effect. Sodium dichloroisocyanurate tablets are stable, and for two reasons, the microbicidal activity of solutions prepared from sodium dichloroisocyanurate tablets might be greater than that of sodium hypochlorite solutions containing the same total available chlorine. First, with sodium dichloroisocyanurate, only 50% of the total available chlorine is free (HOCl and OCl⁻), whereas the remainder is combined (monochloroisocyanurate or dichloroisocyanurate), and as free available chlorine is used up, the latter is released to restore the equilibrium. Second, solutions of sodium dichloroisocyanurate are acidic, whereas sodium hypochlorite solutions are alkaline, and the more microbicidal type of chlorine (HOCl) is believed to predominate⁵³⁰⁻⁵³³. Chlorine dioxide-based disinfectants are prepared fresh as required by mixing the two components (base solution [citric acid with preservatives and corrosion inhibitors] and the activator solution [sodium chlorite]). In vitro suspension tests showed that solutions containing about 140 ppm chlorine dioxide achieved a reduction factor exceeding 10⁶ of *S. aureus* in 1 minute and of *Bacillus atrophaeus* spores in 2.5 minutes in the presence of 3 g/L bovine albumin. The potential for damaging equipment requires consideration because long-term use can damage the outer plastic coat of the insertion tube⁵³⁴. In another study, chlorine dioxide solutions at either 600 ppm or 30 ppm killed *Mycobacterium avium-intracellulare* within 60 seconds after contact but contamination by organic material significantly affected the microbicidal properties⁵³⁵.

The microbicidal activity of a new disinfectant, “superoxidized water,” has been examined. The concept of electrolyzing saline to create a disinfectant or antiseptics is appealing because the basic materials of saline and electricity are inexpensive and the end product (i.e., water) does not damage the environment. The main products of this water are hypochlorous acid (e.g., at a concentration of about 144 mg/L) and chlorine. As with any germicide, the antimicrobial activity of superoxidized water is strongly affected by the concentration of the active ingredient (available free chlorine)⁵³⁶. One manufacturer generates the disinfectant at the point of use by passing a saline solution over coated titanium electrodes at 9 amps. The product generated has a pH of 5.0–6.5 and an oxidation-reduction potential (redox) of >950 mV. Although superoxidized water is intended to be generated fresh at the point of use, when tested under clean conditions the disinfectant was effective within 5 minutes when 48 hours old⁵³⁷. Unfortunately, the equipment required to produce the product can be expensive because parameters such as pH, current, and redox potential must be closely monitored. The solution is nontoxic to biologic tissues. Although the United Kingdom manufacturer claims the solution is noncorrosive and nondamaging to endoscopes and processing equipment, one flexible endoscope manufacturer (Olympus Key-Med, United Kingdom) has voided the warranty on the endoscopes if superoxidized water is used to disinfect them⁵³⁸. As with any germicide formulation, the user should check with the device manufacturer for

compatibility with the germicide. Additional studies are needed to determine whether this solution could be used as an alternative to other disinfectants or antiseptics for hand washing, skin antiseptics, room cleaning, or equipment disinfection (e.g., endoscopes, dialyzers)^{400, 539, 540}. In October 2002, the FDA cleared superoxidized water as a high-level disinfectant (FDA, personal communication, September 18, 2002).

Mode of Action. The exact mechanism by which free chlorine destroys microorganisms has not been elucidated. Inactivation by chlorine can result from a number of factors: oxidation of sulfhydryl enzymes and amino acids; ring chlorination of amino acids; loss of intracellular contents; decreased uptake of nutrients; inhibition of protein synthesis; decreased oxygen uptake; oxidation of respiratory components; decreased adenosine triphosphate production; breaks in DNA; and depressed DNA synthesis^{329, 347}. The actual microbicidal mechanism of chlorine might involve a combination of these factors or the effect of chlorine on critical sites³⁴⁷.

Microbicidal Activity. Low concentrations of free available chlorine (e.g., HOCl, OCl⁻, and elemental chlorine-Cl₂) have a biocidal effect on mycoplasma (25 ppm) and vegetative bacteria (<5 ppm) in seconds in the absence of an organic load^{329, 418}. Higher concentrations (1,000 ppm) of chlorine are required to kill *M. tuberculosis* using the Association of Official Analytical Chemists (AOAC) tuberculocidal test⁷³. A concentration of 100 ppm will kill ≥99.9% of *B. atrophaeus* spores within 5 minutes^{541, 542} and destroy mycotic agents in <1 hour³²⁹. Acidified bleach and regular bleach (5,000 ppm chlorine) can inactivate 10⁶ *Clostridium difficile* spores in <10 minutes²⁶². One study reported that 25 different viruses were inactivated in 10 minutes with 200 ppm available chlorine⁷². Several studies have demonstrated the effectiveness of diluted sodium hypochlorite and other disinfectants to inactivate HIV⁶¹. Chlorine (500 ppm) showed inhibition of *Candida* after 30 seconds of exposure⁵⁴. In experiments using the AOAC Use-Dilution Method, 100 ppm of free chlorine killed 10⁶–10⁷ *S. aureus*, *Salmonella choleraesuis*, and *P. aeruginosa* in <10 minutes³²⁷. Because household bleach contains 5.25%–6.15% sodium hypochlorite, or 52,500–61,500 ppm available chlorine, a 1:1,000 dilution provides about 53–62 ppm available chlorine, and a 1:10 dilution of household bleach provides about 5250–6150 ppm.

Data are available for chlorine dioxide that support manufacturers' bactericidal, fungicidal, sporicidal, tuberculocidal, and virucidal label claims⁵⁴³⁻⁵⁴⁶. A chlorine dioxide generator has been shown effective for decontaminating flexible endoscopes⁵³⁴ but it is not currently FDA-cleared for use as a high-level disinfectant⁸⁵. Chlorine dioxide can be produced by mixing solutions, such as a solution of chlorine with a solution of sodium chlorite³²⁹. In 1986, a chlorine dioxide product was voluntarily removed from the market when its use caused leakage of cellulose-based dialyzer membranes, which allowed bacteria to migrate from the dialysis fluid side of the dialyzer to the blood side⁵⁴⁷.

Sodium dichloroisocyanurate at 2,500 ppm available chlorine is effective against bacteria in the presence of up to 20% plasma, compared with 10% plasma for sodium hypochlorite at 2,500 ppm⁵⁴⁸.

“Superoxidized water” has been tested against bacteria, mycobacteria, viruses, fungi, and spores^{537, 539, 549}. Freshly generated superoxidized water is rapidly effective (<2 minutes) in achieving a 5-log₁₀ reduction of pathogenic microorganisms (i.e., *M. tuberculosis*, *M. chelonae*, poliovirus, HIV, multidrug-resistant *S. aureus*, *E. coli*, *Candida albicans*, *Enterococcus faecalis*, *P. aeruginosa*) in the absence of organic loading. However, the biocidal activity of this disinfectant decreased substantially in the presence of organic material (e.g., 5% horse serum)^{537, 549, 550}. No bacteria or viruses were detected on artificially contaminated endoscopes after a 5-minute exposure to superoxidized water⁵⁵¹ and HBV-DNA was not detected from any endoscope experimentally contaminated with HBV-positive mixed sera after a disinfectant exposure time of 7 minutes⁵⁵².

Uses. Hypochlorites are widely used in healthcare facilities in a variety of settings.³²⁸ Inorganic chlorine solution is used for disinfecting tonometer heads¹⁸⁸ and for spot-disinfection of countertops and floors. A 1:10–1:100 dilution of 5.25%–6.15% sodium hypochlorite (i.e., household bleach)^{22, 228, 553, 554} or

an EPA-registered tuberculocidal disinfectant¹⁷ has been recommended for decontaminating blood spills. For small spills of blood (i.e., drops of blood) on noncritical surfaces, the area can be disinfected with a 1:100 dilution of 5.25%–6.15% sodium hypochlorite or an EPA-registered tuberculocidal disinfectant. Because hypochlorites and other germicides are substantially inactivated in the presence of blood^{63, 548, 555, 556}, large spills of blood require that the surface be cleaned before an EPA-registered disinfectant or a 1:10 (final concentration) solution of household bleach is applied⁵⁵⁷. If a sharps injury is possible, the surface initially should be decontaminated^{69, 318}, then cleaned and disinfected (1:10 final concentration)⁶³. Extreme care always should be taken to prevent percutaneous injury. At least 500 ppm available chlorine for 10 minutes is recommended for decontaminating CPR training manikins⁵⁵⁸. Full-strength bleach has been recommended for self-disinfection of needles and syringes used for illicit-drug injection when needle-exchange programs are not available. The difference in the recommended concentrations of bleach reflects the difficulty of cleaning the interior of needles and syringes and the use of needles and syringes for parenteral injection⁵⁵⁹. Clinicians should not alter their use of chlorine on environmental surfaces on the basis of testing methodologies that do not simulate actual disinfection practices^{560, 561}. Other uses in healthcare include as an irrigating agent in endodontic treatment⁵⁶² and as a disinfectant for manikins, laundry, dental appliances, hydrotherapy tanks^{23, 41}, regulated medical waste before disposal³²⁸, and the water distribution system in hemodialysis centers and hemodialysis machines⁵⁶³.

Chlorine long has been used as the disinfectant in water treatment. Hyperchlorination of a *Legionella*-contaminated hospital water system²³ resulted in a dramatic decrease (from 30% to 1.5%) in the isolation of *L. pneumophila* from water outlets and a cessation of healthcare-associated Legionnaires' disease in an affected unit^{528, 564}. Water disinfection with monochloramine by municipal water-treatment plants substantially reduced the risk for healthcare-associated Legionnaires disease^{565, 566}. Chlorine dioxide also has been used to control *Legionella* in a hospital water supply.⁵⁶⁷ Chloramine T⁵⁶⁸ and hypochlorites⁴¹ have been used to disinfect hydrotherapy equipment.

Hypochlorite solutions in tap water at a pH >8 stored at room temperature (23°C) in closed, opaque plastic containers can lose up to 40%–50% of their free available chlorine level over 1 month. Thus, if a user wished to have a solution containing 500 ppm of available chlorine at day 30, he or she should prepare a solution containing 1,000 ppm of chlorine at time 0. Sodium hypochlorite solution does not decompose after 30 days when stored in a closed brown bottle³²⁷.

The use of powders, composed of a mixture of a chlorine-releasing agent with highly absorbent resin, for disinfecting spills of body fluids has been evaluated by laboratory tests and hospital ward trials. The inclusion of acrylic resin particles in formulations markedly increases the volume of fluid that can be soaked up because the resin can absorb 200–300 times its own weight of fluid, depending on the fluid consistency. When experimental formulations containing 1%, 5%, and 10% available chlorine were evaluated by a standardized surface test, those containing 10% demonstrated bactericidal activity. One problem with chlorine-releasing granules is that they can generate chlorine fumes when applied to urine⁵⁶⁹.

Formaldehyde

Overview. Formaldehyde is used as a disinfectant and sterilant in both its liquid and gaseous states. Liquid formaldehyde will be considered briefly in this section, and the gaseous form is reviewed elsewhere⁵⁷⁰. Formaldehyde is sold and used principally as a water-based solution called formalin, which is 37% formaldehyde by weight. The aqueous solution is a bactericide, tuberculocide, fungicide, virucide and sporicide^{72, 82, 571-573}. OSHA indicated that formaldehyde should be handled in the workplace as a potential carcinogen and set an employee exposure standard for formaldehyde that limits an 8-hour time-weighted average exposure concentration of 0.75 ppm^{574, 575}. The standard includes a second permissible exposure limit in the form of a short-term exposure limit (STEL) of 2 ppm that is the maximum exposure allowed during a 15-minute period⁵⁷⁶. Ingestion of formaldehyde can be fatal, and long-term exposure to low levels in the air or on the skin can cause asthma-like respiratory problems and skin irritation, such as dermatitis and itching. For these reasons, employees should have limited direct contact

with formaldehyde, and these considerations limit its role in sterilization and disinfection processes. Key provisions of the OSHA standard that protects workers from exposure to formaldehyde appear in Title 29 of the Code of Federal Regulations (CFR) Part 1910.1048 (and equivalent regulations in states with OSHA-approved state plans)⁵⁷⁷.

Mode of Action. Formaldehyde inactivates microorganisms by alkylating the amino and sulfhydryl groups of proteins and ring nitrogen atoms of purine bases³⁷⁶.

Microbicidal Activity. Varying concentrations of aqueous formaldehyde solutions destroy a wide range of microorganisms. Inactivation of poliovirus in 10 minutes required an 8% concentration of formalin, but all other viruses tested were inactivated with 2% formalin⁷². Four percent formaldehyde is a tuberculocidal agent, inactivating 10^4 *M. tuberculosis* in 2 minutes⁸², and 2.5% formaldehyde inactivated about 10^7 *Salmonella* Typhi in 10 minutes in the presence of organic matter⁵⁷². The sporicidal action of formaldehyde was slower than that of glutaraldehyde in comparative tests with 4% aqueous formaldehyde and 2% glutaraldehyde against the spores of *B. anthracis*⁸². The formaldehyde solution required 2 hours of contact to achieve an inactivation factor of 10^4 , whereas glutaraldehyde required only 15 minutes.

Uses. Although formaldehyde-alcohol is a chemical sterilant and formaldehyde is a high-level disinfectant, the health-care uses of formaldehyde are limited by its irritating fumes and its pungent odor even at very low levels (<1 ppm). For these reasons and others—such as its role as a suspected human carcinogen linked to nasal cancer and lung cancer⁵⁷⁸, this germicide is excluded from Table 1. When it is used, direct exposure to employees generally is limited; however, excessive exposures to formaldehyde have been documented for employees of renal transplant units^{574, 579}, and students in a gross anatomy laboratory⁵⁸⁰. Formaldehyde is used in the health-care setting to prepare viral vaccines (e.g., poliovirus and influenza); as an embalming agent; and to preserve anatomic specimens; and historically has been used to sterilize surgical instruments, especially when mixed with ethanol. A 1997 survey found that formaldehyde was used for reprocessing hemodialyzers by 34% of U.S. hemodialysis centers—a 60% decrease from 1983^{249, 581}. If used at room temperature, a concentration of 4% with a minimum exposure of 24 hours is required to disinfect disposable hemodialyzers reused on the same patient^{582, 583}. Aqueous formaldehyde solutions (1%–2%) also have been used to disinfect the internal fluid pathways of dialysis machines⁵⁸³. To minimize a potential health hazard to dialysis patients, the dialysis equipment must be thoroughly rinsed and tested for residual formaldehyde before use.

Paraformaldehyde, a solid polymer of formaldehyde, can be vaporized by heat for the gaseous decontamination of laminar flow biologic safety cabinets when maintenance work or filter changes require access to the sealed portion of the cabinet.

Glutaraldehyde

Overview. Glutaraldehyde is a saturated dialdehyde that has gained wide acceptance as a high-level disinfectant and chemical sterilant¹⁰⁷. Aqueous solutions of glutaraldehyde are acidic and generally in this state are not sporicidal. Only when the solution is “activated” (made alkaline) by use of alkalinizing agents to pH 7.5–8.5 does the solution become sporicidal. Once activated, these solutions have a shelf-life of minimally 14 days because of the polymerization of the glutaraldehyde molecules at alkaline pH levels. This polymerization blocks the active sites (aldehyde groups) of the glutaraldehyde molecules that are responsible for its biocidal activity.

Novel glutaraldehyde formulations (e.g., glutaraldehyde-phenol-sodium phenate, potentiated acid glutaraldehyde, stabilized alkaline glutaraldehyde) produced in the past 30 years have overcome the problem of rapid loss of activity (e.g., use-life 28–30 days) while generally maintaining excellent microbicidal activity^{584–588}. However, antimicrobial activity depends not only on age but also on use conditions, such as dilution and organic stress. Manufacturers' literature for these preparations suggests the neutral or alkaline glutaraldehydes possess microbicidal and anticorrosion properties superior to

those of acid glutaraldehydes, and a few published reports substantiate these claims^{542, 589, 590}. However, two studies found no difference in the microbicidal activity of alkaline and acid glutaraldehydes^{73, 591}. The use of glutaraldehyde-based solutions in health-care facilities is widespread because of their advantages, including excellent biocidal properties; activity in the presence of organic matter (20% bovine serum); and noncorrosive action to endoscopic equipment, thermometers, rubber, or plastic equipment (Tables 4 and 5).

Mode of Action. The biocidal activity of glutaraldehyde results from its alkylation of sulfhydryl, hydroxyl, carboxyl, and amino groups of microorganisms, which alters RNA, DNA, and protein synthesis. The mechanism of action of glutaraldehydes are reviewed extensively elsewhere^{592, 593}.

Microbicidal Activity. The in vitro inactivation of microorganisms by glutaraldehydes has been extensively investigated and reviewed^{592, 593}. Several investigators showed that $\geq 2\%$ aqueous solutions of glutaraldehyde, buffered to pH 7.5–8.5 with sodium bicarbonate effectively killed vegetative bacteria in <2 minutes; *M. tuberculosis*, fungi, and viruses in <10 minutes; and spores of *Bacillus* and *Clostridium* species in 3 hours^{542, 592-597}. Spores of *C. difficile* are more rapidly killed by 2% glutaraldehyde than are spores of other species of *Clostridium* and *Bacillus*^{79, 265, 266}. Microorganisms with substantial resistance to glutaraldehyde have been reported, including some mycobacteria (*M. chelonae*, *Mycobacterium avium-intracellulare*, *M. xenopi*)⁵⁹⁸⁻⁶⁰¹, *Methylobacterium mesophilicum*⁶⁰², *Trichosporon*, fungal ascospores (e.g., *Microascus cinereus*, *Cheatomium globosum*), and *Cryptosporidium*^{271, 603}. *M. chelonae* persisted in a 0.2% glutaraldehyde solution used to store porcine prosthetic heart valves⁶⁰⁴.

Two percent alkaline glutaraldehyde solution inactivated 10^5 *M. tuberculosis* cells on the surface of penicylinders within 5 minutes at 18°C⁵⁸⁹. However, subsequent studies⁸² questioned the mycobactericidal prowess of glutaraldehydes. Two percent alkaline glutaraldehyde has slow action (20 to >30 minutes) against *M. tuberculosis* and compares unfavorably with alcohols, formaldehydes, iodine, and phenol⁸². Suspensions of *M. avium*, *M. intracellulare*, and *M. gordonae* were more resistant to inactivation by a 2% alkaline glutaraldehyde (estimated time to complete inactivation: ~60 minutes) than were virulent *M. tuberculosis* (estimated time to complete inactivation ~25 minutes)⁶⁰⁵. The rate of kill was directly proportional to the temperature, and a standardized suspension of *M. tuberculosis* could not be sterilized within 10 minutes⁸⁴. An FDA-cleared chemical sterilant containing 2.5% glutaraldehyde uses increased temperature (35°C) to reduce the time required to achieve high-level disinfection (5 minutes)^{85, 606}, but its use is limited to automatic endoscope reprocessors equipped with a heater. In another study employing membrane filters for measurement of mycobactericidal activity of 2% alkaline glutaraldehyde, complete inactivation was achieved within 20 minutes at 20°C when the test inoculum was 10^6 *M. tuberculosis* per membrane⁸¹. Several investigators^{55, 57, 73, 76, 80, 81, 84, 605} have demonstrated that glutaraldehyde solutions inactivate 2.4 to >5.0 log₁₀ of *M. tuberculosis* in 10 minutes (including multidrug-resistant *M. tuberculosis*) and 4.0–6.4 log₁₀ of *M. tuberculosis* in 20 minutes. On the basis of these data and other studies, 20 minutes at room temperature is considered the minimum exposure time needed to reliably kill *Mycobacteria* and other vegetative bacteria with $\geq 2\%$ glutaraldehyde^{17, 19, 27, 57, 83, 94, 108, 111, 117-121, 607}.

Glutaraldehyde is commonly diluted during use, and studies showed a glutaraldehyde concentration decline after a few days of use in an automatic endoscope washer^{608, 609}. The decline occurs because instruments are not thoroughly dried and water is carried in with the instrument, which increases the solution's volume and dilutes its effective concentration⁶¹⁰. This emphasizes the need to ensure that semicritical equipment is disinfected with an acceptable concentration of glutaraldehyde. Data suggest that 1.0%–1.5% glutaraldehyde is the minimum effective concentration for $>2\%$ glutaraldehyde solutions when used as a high-level disinfectant^{76, 589, 590, 609}. Chemical test strips or liquid chemical monitors^{610, 611} are available for determining whether an effective concentration of glutaraldehyde is present despite repeated use and dilution. The frequency of testing should be based on how frequently the solutions are used (e.g., used daily, test daily; used weekly, test before use; used 30 times per day, test each 10th use), but the strips should not be used to extend the use life beyond the expiration date. Data suggest the chemicals in the test strip deteriorate with time⁶¹² and a

manufacturer's expiration date should be placed on the bottles. The bottle of test strips should be dated when opened and used for the period of time indicated on the bottle (e.g., 120 days). The results of test strip monitoring should be documented. The glutaraldehyde test kits have been preliminarily evaluated for accuracy and range⁶¹² but the reliability has been questioned⁶¹³. To ensure the presence of minimum effective concentration of the high-level disinfectant, manufacturers of some chemical test strips recommend the use of quality-control procedures to ensure the strips perform properly. If the manufacturer of the chemical test strip recommends a quality-control procedure, users should comply with the manufacturer's recommendations. The concentration should be considered unacceptable or unsafe when the test indicates a dilution below the product's minimum effective concentration (MEC) (generally to $\leq 1.0\%$ – 1.5% glutaraldehyde) by the indicator not changing color.

A 2.0% glutaraldehyde–7.05% phenol–1.20% sodium phenate product that contained 0.125% glutaraldehyde–0.44% phenol–0.075% sodium phenate when diluted 1:16 is not recommended as a high-level disinfectant because it lacks bactericidal activity in the presence of organic matter and lacks tuberculocidal, fungicidal, virucidal, and sporicidal activity^{49, 55, 56, 71, 73-79, 614}. In December 1991, EPA issued an order to stop the sale of all batches of this product because of efficacy data showing the product is not effective against spores and possibly other microorganisms or inanimate objects as claimed on the label⁶¹⁵. FDA has cleared a glutaraldehyde–phenol/phenate concentrate as a high-level disinfectant that contains 1.12% glutaraldehyde with 1.93% phenol/phenate at its use concentration. Other FDA cleared glutaraldehyde sterilants that contain 2.4%–3.4% glutaraldehyde are used undiluted⁶⁰⁶.

Uses. Glutaraldehyde is used most commonly as a high-level disinfectant for medical equipment such as endoscopes^{69, 107, 504}, spirometry tubing, dialyzers⁶¹⁶, transducers, anesthesia and respiratory therapy equipment⁶¹⁷, hemodialysis proportioning and dialysate delivery systems^{249, 618}, and reuse of laparoscopic disposable plastic trocars⁶¹⁹. Glutaraldehyde is noncorrosive to metal and does not damage lensed instruments, rubber, or plastics. Glutaraldehyde should not be used for cleaning noncritical surfaces because it is too toxic and expensive.

Colitis believed caused by glutaraldehyde exposure from residual disinfecting solution in endoscope solution channels has been reported and is preventable by careful endoscope rinsing^{318, 620-630}. One study found that residual glutaraldehyde levels were higher and more variable after manual disinfection (<0.2 mg/L to 159.5 mg/L) than after automatic disinfection (0.2–6.3 mg/L)⁶³¹. Similarly, keratopathy and corneal decompensation were caused by ophthalmic instruments that were inadequately rinsed after soaking in 2% glutaraldehyde^{632, 633}.

Healthcare personnel can be exposed to elevated levels of glutaraldehyde vapor when equipment is processed in poorly ventilated rooms, when spills occur, when glutaraldehyde solutions are activated or changed,⁶³⁴ or when open immersion baths are used. Acute or chronic exposure can result in skin irritation or dermatitis, mucous membrane irritation (eye, nose, mouth), or pulmonary symptoms^{318, 635-639}. Epistaxis, allergic contact dermatitis, asthma, and rhinitis also have been reported in healthcare workers exposed to glutaraldehyde^{636, 640-647}.

Glutaraldehyde exposure should be monitored to ensure a safe work environment. Testing can be done by four techniques: a silica gel tube/gas chromatography with a flame ionization detector, dinitrophenylhydrazine (DNPH)-impregnated filter cassette/high-performance liquid chromatography (HPLC) with an ultraviolet (UV) detector, a passive badge/HPLC, or a handheld glutaraldehyde air monitor⁶⁴⁸. The silica gel tube and the DNPH-impregnated cassette are suitable for monitoring the 0.05 ppm ceiling limit. The passive badge, with a 0.02 ppm limit of detection, is considered marginal at the American Council of Governmental Industrial Hygienists (ACGIH) ceiling level. The ceiling level is considered too close to the glutaraldehyde meter's 0.03 ppm limit of detection to provide confidence in the readings⁶⁴⁸. ACGIH does not require a specific monitoring schedule for glutaraldehyde; however, a monitoring schedule is needed to ensure the level is less than the ceiling limit. For example, monitoring

should be done initially to determine glutaraldehyde levels, after procedural or equipment changes, and in response to worker complaints⁶⁴⁹. In the absence of an OSHA permissible exposure limit, if the glutaraldehyde level is higher than the ACGIH ceiling limit of 0.05 ppm, corrective action and repeat monitoring would be prudent⁶⁴⁹.

Engineering and work-practice controls that can be used to resolve these problems include ducted exhaust hoods, air systems that provide 7–15 air exchanges per hour, ductless fume hoods with absorbents for the glutaraldehyde vapor, tight-fitting lids on immersion baths, personal protection (e.g., nitrile or butyl rubber gloves but not natural latex gloves, goggles) to minimize skin or mucous membrane contact, and automated endoscope processors^{7, 650}. If engineering controls fail to maintain levels below the ceiling limit, institutions can consider the use of respirators (e.g., a half-face respirator with organic vapor cartridge⁶⁴⁰ or a type "C" supplied air respirator with a full facepiece operated in a positive pressure mode)⁶⁵¹. In general, engineering controls are preferred over work-practice and administrative controls because they do not require active participation by the health-care worker. Even though enforcement of the OSHA ceiling limit was suspended in 1993 by the U.S. Court of Appeals⁵⁷⁷, limiting employee exposure to 0.05 ppm (according to ACGIH) is prudent because, at this level, glutaraldehyde can irritate the eyes, throat, and nose^{318, 577, 639, 652}. If glutaraldehyde disposal through the sanitary sewer system is restricted, sodium bisulfate can be used to neutralize the glutaraldehyde and make it safe for disposal.

Hydrogen Peroxide

Overview. The literature contains several accounts of the properties, germicidal effectiveness, and potential uses for stabilized hydrogen peroxide in the health-care setting. Published reports ascribe good germicidal activity to hydrogen peroxide and attest to its bactericidal, virucidal, sporicidal, and fungicidal properties⁶⁵³⁻⁶⁵⁵. (Tables 4 and 5) The FDA website lists cleared liquid chemical sterilants and high-level disinfectants containing hydrogen peroxide and their cleared contact conditions.

Mode of Action. Hydrogen peroxide works by producing destructive hydroxyl free radicals that can attack membrane lipids, DNA, and other essential cell components. Catalase, produced by aerobic organisms and facultative anaerobes that possess cytochrome systems, can protect cells from metabolically produced hydrogen peroxide by degrading hydrogen peroxide to water and oxygen. This defense is overwhelmed by the concentrations used for disinfection^{653, 654}.

Microbicidal Activity. Hydrogen peroxide is active against a wide range of microorganisms, including bacteria, yeasts, fungi, viruses, and spores^{78, 654}. A 0.5% accelerated hydrogen peroxide demonstrated bactericidal and virucidal activity in 1 minute and mycobactericidal and fungicidal activity in 5 minutes⁶⁵⁶. Bactericidal effectiveness and stability of hydrogen peroxide in urine has been demonstrated against a variety of health-care-associated pathogens; organisms with high cellular catalase activity (e.g., *S. aureus*, *S. marcescens*, and *Proteus mirabilis*) required 30–60 minutes of exposure to 0.6% hydrogen peroxide for a 10⁸ reduction in cell counts, whereas organisms with lower catalase activity (e.g., *E. coli*, *Streptococcus* species, and *Pseudomonas* species) required only 15 minutes' exposure⁶⁵⁷. In an investigation of 3%, 10%, and 15% hydrogen peroxide for reducing spacecraft bacterial populations, a complete kill of 10⁶ spores (i.e., *Bacillus* species) occurred with a 10% concentration and a 60-minute exposure time. A 3% concentration for 150 minutes killed 10⁶ spores in six of seven exposure trials⁶⁵⁸. A 10% hydrogen peroxide solution resulted in a 10³ decrease in *B. atrophaeus* spores, and a $\geq 10^5$ decrease when tested against 13 other pathogens in 30 minutes at 20°C^{659, 660}. A 3.0% hydrogen peroxide solution was ineffective against VRE after 3 and 10 minutes exposure times⁶⁶¹ and caused only a 2-log₁₀ reduction in the number of *Acanthamoeba* cysts in approximately 2 hours⁶⁶². A 7% stabilized hydrogen peroxide proved to be sporicidal (6 hours of exposure), mycobactericidal (20 minutes), fungicidal (5 minutes) at full strength, virucidal (5 minutes) and bactericidal (3 minutes) at a 1:16 dilution when a quantitative carrier test was used⁶⁵⁵. The 7% solution of hydrogen peroxide, tested after 14 days of stress (in the form of germ-loaded carriers and respiratory therapy equipment), was sporicidal (>7 log₁₀ reduction in 6 hours), mycobactericidal (>6.5 log₁₀ reduction in 25

minutes), fungicidal (>5 log₁₀ reduction in 20 minutes), bactericidal (>6 log₁₀ reduction in 5 minutes) and virucidal (5 log₁₀ reduction in 5 minutes)⁶⁶³. Synergistic sporicidal effects were observed when spores were exposed to a combination of hydrogen peroxide (5.9%–23.6%) and peracetic acid⁶⁶⁴. Other studies demonstrated the antiviral activity of hydrogen peroxide against rhinovirus⁶⁶⁵. The time required for inactivating three serotypes of rhinovirus using a 3% hydrogen peroxide solution was 6–8 minutes; this time increased with decreasing concentrations (18–20 minutes at 1.5%, 50–60 minutes at 0.75%).

Concentrations of hydrogen peroxide from 6% to 25% show promise as chemical sterilants. The product marketed as a sterilant is a premixed, ready-to-use chemical that contains 7.5% hydrogen peroxide and 0.85% phosphoric acid (to maintain a low pH)⁶⁹. The mycobactericidal activity of 7.5% hydrogen peroxide has been corroborated in a study showing the inactivation of >10⁵ multidrug-resistant *M. tuberculosis* after a 10-minute exposure⁶⁶⁶. Thirty minutes were required for >99.9% inactivation of poliovirus and HAV⁶⁶⁷. Three percent and 6% hydrogen peroxide were unable to inactivate HAV in 1 minute in a carrier test⁵⁸. When the effectiveness of 7.5% hydrogen peroxide at 10 minutes was compared with 2% alkaline glutaraldehyde at 20 minutes in manual disinfection of endoscopes, no significant difference in germicidal activity was observed⁶⁶⁸. No complaints were received from the nursing or medical staff regarding odor or toxicity. In one study, 6% hydrogen peroxide (unused product was 7.5%) was more effective in the high-level disinfection of flexible endoscopes than was the 2% glutaraldehyde solution⁴⁵⁶. A new, rapid-acting 13.4% hydrogen peroxide formulation (that is not yet FDA-cleared) has demonstrated sporicidal, mycobactericidal, fungicidal, and virucidal efficacy. Manufacturer data demonstrate that this solution sterilizes in 30 minutes and provides high-level disinfection in 5 minutes⁶⁶⁹. This product has not been used long enough to evaluate material compatibility to endoscopes and other semicritical devices, and further assessment by instrument manufacturers is needed.

Under normal conditions, hydrogen peroxide is extremely stable when properly stored (e.g., in dark containers). The decomposition or loss of potency in small containers is less than 2% per year at ambient temperatures⁶⁷⁰.

Uses. Commercially available 3% hydrogen peroxide is a stable and effective disinfectant when used on inanimate surfaces. It has been used in concentrations from 3% to 6% for disinfecting soft contact lenses (e.g., 3% for 2–3 hrs)^{653, 671, 672}, tonometer biphisms⁵¹³, ventilators⁶⁷³, fabrics³⁹⁷, and endoscopes⁴⁵⁶. Hydrogen peroxide was effective in spot-disinfecting fabrics in patients' rooms³⁹⁷. Corneal damage from a hydrogen peroxide-soaked tonometer tip that was not properly rinsed has been reported⁶⁷⁴. Hydrogen peroxide also has been instilled into urinary drainage bags in an attempt to eliminate the bag as a source of bladder bacteriuria and environmental contamination⁶⁷⁵. Although the instillation of hydrogen peroxide into the bag reduced microbial contamination of the bag, this procedure did not reduce the incidence of catheter-associated bacteriuria⁶⁷⁵.

A chemical irritation resembling pseudomembranous colitis caused by either 3% hydrogen peroxide or a 2% glutaraldehyde has been reported⁶²¹. An epidemic of pseudomembrane-like enteritis and colitis in seven patients in a gastrointestinal endoscopy unit also has been associated with inadequate rinsing of 3% hydrogen peroxide from the endoscope⁶⁷⁶.

As with other chemical sterilants, dilution of the hydrogen peroxide must be monitored by regularly testing the minimum effective concentration (i.e., 7.5%–6.0%). Compatibility testing by Olympus America of the 7.5% hydrogen peroxide found both cosmetic changes (e.g., discoloration of black anodized metal finishes)⁶⁹ and functional changes with the tested endoscopes (Olympus, written communication, October 15, 1999).

Iodophors

Overview. Iodine solutions or tinctures long have been used by health professionals primarily as antiseptics on skin or tissue. Iodophors, on the other hand, have been used both as antiseptics and

disinfectants. FDA has not cleared any liquid chemical sterilant or high-level disinfectants with iodophors as the main active ingredient. An iodophor is a combination of iodine and a solubilizing agent or carrier; the resulting complex provides a sustained-release reservoir of iodine and releases small amounts of free iodine in aqueous solution. The best-known and most widely used iodophor is povidone-iodine, a compound of polyvinylpyrrolidone with iodine. This product and other iodophors retain the germicidal efficacy of iodine but unlike iodine generally are nonstaining and relatively free of toxicity and irritancy^{677, 678}.

Several reports that documented intrinsic microbial contamination of antiseptic formulations of povidone-iodine and poloxamer-iodine⁶⁷⁹⁻⁶⁸¹ caused a reappraisal of the chemistry and use of iodophors⁶⁸². “Free” iodine (I₂) contributes to the bactericidal activity of iodophors and dilutions of iodophors demonstrate more rapid bactericidal action than does a full-strength povidone-iodine solution. The reason for the observation that dilution increases bactericidal activity is unclear, but dilution of povidone-iodine might weaken the iodine linkage to the carrier polymer with an accompanying increase of free iodine in solution⁶⁸⁰. Therefore, iodophors must be diluted according to the manufacturers' directions to achieve antimicrobial activity.

Mode of Action. Iodine can penetrate the cell wall of microorganisms quickly, and the lethal effects are believed to result from disruption of protein and nucleic acid structure and synthesis.

Microbicidal Activity. Published reports on the in vitro antimicrobial efficacy of iodophors demonstrate that iodophors are bactericidal, mycobactericidal, and virucidal but can require prolonged contact times to kill certain fungi and bacterial spores^{14, 71-73, 290, 683-686}. Three brands of povidone-iodine solution have demonstrated more rapid kill (seconds to minutes) of *S. aureus* and *M. chelonae* at a 1:100 dilution than did the stock solution⁶⁸³. The virucidal activity of 75–150 ppm available iodine was demonstrated against seven viruses⁷². Other investigators have questioned the efficacy of iodophors against poliovirus in the presence of organic matter⁶⁸⁵ and rotavirus SA-11 in distilled or tapwater²⁹⁰. Manufacturers' data demonstrate that commercial iodophors are not sporicidal, but they are tuberculocidal, fungicidal, virucidal, and bactericidal at their recommended use-dilution.

Uses. Besides their use as an antiseptic, iodophors have been used for disinfecting blood culture bottles and medical equipment, such as hydrotherapy tanks, thermometers, and endoscopes. Antiseptic iodophors are not suitable for use as hard-surface disinfectants because of concentration differences. Iodophors formulated as antiseptics contain less free iodine than do those formulated as disinfectants³⁷⁶. Iodine or iodine-based antiseptics should not be used on silicone catheters because they can adversely affect the silicone tubing⁶⁸⁷.

Ortho-phthalaldehyde (OPA)

Overview. Ortho-phthalaldehyde is a high-level disinfectant that received FDA clearance in October 1999. It contains 0.55% 1,2-benzenedicarboxaldehyde (OPA). OPA solution is a clear, pale-blue liquid with a pH of 7.5. (Tables 4 and 5)

Mode of Action. Preliminary studies on the mode of action of OPA suggest that both OPA and glutaraldehyde interact with amino acids, proteins, and microorganisms. However, OPA is a less potent cross-linking agent. This is compensated for by the lipophilic aromatic nature of OPA that is likely to assist its uptake through the outer layers of mycobacteria and gram-negative bacteria⁶⁸⁸⁻⁶⁹⁰. OPA appears to kill spores by blocking the spore germination process⁶⁹¹.

Microbicidal Activity. Studies have demonstrated excellent microbicidal activity in vitro^{69, 100, 271, 400, 692-703}. For example, OPA has superior mycobactericidal activity (5-log₁₀ reduction in 5 minutes) to glutaraldehyde. The mean times required to produce a 6-log₁₀ reduction for *M. bovis* using 0.21% OPA was 6 minutes, compared with 32 minutes using 1.5% glutaraldehyde⁶⁹³. OPA showed good activity against the mycobacteria tested, including the glutaraldehyde-resistant strains, but 0.5% OPA was not sporicidal with 270 minutes of exposure. Increasing the pH from its unadjusted level (about 6.5) to pH 8 improved the sporicidal activity of OPA⁶⁹⁴. The level of biocidal activity was directly related to the

temperature. A greater than 5- \log_{10} reduction of *B. atrophaeus* spores was observed in 3 hours at 35°C, than in 24 hours at 20°C. Also, with an exposure time \leq 5 minutes, biocidal activity decreased with increasing serum concentration. However, efficacy did not differ when the exposure time was \geq 10 minutes⁶⁹⁷. In addition, OPA is effective ($>$ 5- \log_{10} reduction) against a wide range of microorganisms, including glutaraldehyde-resistant mycobacteria and *B. atrophaeus* spores⁶⁹⁴.

The influence of laboratory adaptation of test strains, such as *P. aeruginosa*, to 0.55% OPA has been evaluated. Resistant and multiresistant strains increased substantially in susceptibility to OPA after laboratory adaptation (\log_{10} reduction factors increased by 0.54 and 0.91 for resistant and multiresistant strains, respectively)⁷⁰⁴. Other studies have found naturally occurring cells of *P. aeruginosa* were more resistant to a variety of disinfectants than were subcultured cells⁷⁰⁵.

Uses. OPA has several potential advantages over glutaraldehyde. It has excellent stability over a wide pH range (pH 3–9), is not a known irritant to the eyes and nasal passages⁷⁰⁶, does not require exposure monitoring, has a barely perceptible odor, and requires no activation. OPA, like glutaraldehyde, has excellent material compatibility. A potential disadvantage of OPA is that it stains proteins gray (including unprotected skin) and thus must be handled with caution⁶⁹. However, skin staining would indicate improper handling that requires additional training and/or personal protective equipment (e.g., gloves, eye and mouth protection, and fluid-resistant gowns). OPA residues remaining on inadequately water-rinsed transesophageal echo probes can stain the patient's mouth⁷⁰⁷. Meticulous cleaning, using the correct OPA exposure time (e.g., 12 minutes) and copious rinsing of the probe with water should eliminate this problem. The results of one study provided a basis for a recommendation that rinsing of instruments disinfected with OPA will require at least 250 mL of water per channel to reduce the chemical residue to a level that will not compromise patient or staff safety ($<$ 1 ppm)⁷⁰⁸. Personal protective equipment should be worn when contaminated instruments, equipment, and chemicals are handled⁴⁰⁰. In addition, equipment must be thoroughly rinsed to prevent discoloration of a patient's skin or mucous membrane.

In April 2004, the manufacturer of OPA disseminated information to users about patients who reportedly experienced an anaphylaxis-like reaction after cystoscopy where the scope had been reprocessed using OPA. Of approximately 1 million urologic procedures performed using instruments reprocessed using OPA, 24 cases (17 cases in the United States, six in Japan, one in the United Kingdom) of anaphylaxis-like reactions have been reported after repeated cystoscopy (typically after four to nine treatments). Preventive measures include removal of OPA residues by thorough rinsing and not using OPA for reprocessing urologic instrumentation used to treat patients with a history of bladder cancer (Nevine Erian, personal communication, June 4, 2004; Product Notification, Advanced Sterilization Products, April 23, 2004)⁷⁰⁹.

A few OPA clinical studies are available. In a clinical-use study, OPA exposure of 100 endoscopes for 5 minutes resulted in a $>$ 5- \log_{10} reduction in bacterial load. Furthermore, OPA was effective over a 14-day use cycle¹⁰⁰. Manufacturer data show that OPA will last longer in an automatic endoscope reprocessor before reaching its MEC limit (MEC after 82 cycles) than will glutaraldehyde (MEC after 40 cycles)⁴⁰⁰. High-pressure liquid chromatography confirmed that OPA levels are maintained above 0.3% for at least 50 cycles^{706, 710}. OPA must be disposed in accordance with local and state regulations. If OPA disposal through the sanitary sewer system is restricted, glycine (25 grams/gallon) can be used to neutralize the OPA and make it safe for disposal.

The high-level disinfectant label claims for OPA solution at 20°C vary worldwide (e.g., 5 minutes in Europe, Asia, and Latin America; 10 minutes in Canada and Australia; and 12 minutes in the United States). These label claims differ worldwide because of differences in the test methodology and requirements for licensure. In an automated endoscope reprocessor with an FDA-cleared capability to maintain solution temperatures at 25°C, the contact time for OPA is 5 minutes.

Peracetic Acid

Overview. Peracetic, or peroxyacetic, acid is characterized by rapid action against all microorganisms. Special advantages of peracetic acid are that it lacks harmful decomposition products (i.e., acetic acid, water, oxygen, hydrogen peroxide), enhances removal of organic material⁷¹¹, and leaves no residue. It remains effective in the presence of organic matter and is sporicidal even at low temperatures (Tables 4 and 5). Peracetic acid can corrode copper, brass, bronze, plain steel, and galvanized iron but these effects can be reduced by additives and pH modifications. It is considered unstable, particularly when diluted; for example, a 1% solution loses half its strength through hydrolysis in 6 days, whereas 40% peracetic acid loses 1%–2% of its active ingredients per month⁶⁵⁴.

Mode of Action. Little is known about the mechanism of action of peracetic acid, but it is believed to function similarly to other oxidizing agents—that is, it denatures proteins, disrupts the cell wall permeability, and oxidizes sulfhydryl and sulfur bonds in proteins, enzymes, and other metabolites⁶⁵⁴.

Microbicidal Activity. Peracetic acid will inactivate gram-positive and gram-negative bacteria, fungi, and yeasts in <5 minutes at <100 ppm. In the presence of organic matter, 200–500 ppm is required. For viruses, the dosage range is wide (12–2250 ppm), with poliovirus inactivated in yeast extract in 15 minutes with 1,500–2,250 ppm. In one study, 3.5% peracetic acid was ineffective against HAV after 1-minute exposure using a carrier test⁵⁸. Peracetic acid (0.26%) was effective (log₁₀ reduction factor >5) against all test strains of mycobacteria (*M. tuberculosis*, *M. avium-intracellulare*, *M. chelonae*, and *M. fortuitum*) within 20–30 minutes in the presence or absence of an organic load^{607, 712}. With bacterial spores, 500–10,000 ppm (0.05%–1%) inactivates spores in 15 seconds to 30 minutes using a spore suspension test^{654, 659, 713-715}.

Uses. An automated machine using peracetic acid to chemically sterilize medical (e.g., endoscopes, arthroscopes), surgical, and dental instruments is used in the United States⁷¹⁶⁻⁷¹⁸. As previously noted, dental handpieces should be steam sterilized. The sterilant, 35% peracetic acid, is diluted to 0.2% with filtered water at 50°C. Simulated-use trials have demonstrated excellent microbicidal activity^{111, 718-722}, and three clinical trials have demonstrated both excellent microbial killing and no clinical failures leading to infection^{90, 723, 724}. The high efficacy of the system was demonstrated in a comparison of the efficacies of the system with that of ethylene oxide. Only the peracetic acid system completely killed 6 log₁₀ of *M. chelonae*, *E. faecalis*, and *B. atrophaeus* spores with both an organic and inorganic challenge⁷²². An investigation that compared the costs, performance, and maintenance of urologic endoscopic equipment processed by high-level disinfection (with glutaraldehyde) with those of the peracetic acid system reported no clinical differences between the two systems. However, the use of this system led to higher costs than the high-level disinfection, including costs for processing (\$6.11 vs. \$0.45 per cycle), purchasing and training (\$24,845 vs. \$16), installation (\$5,800 vs. \$0), and endoscope repairs (\$6,037 vs. \$445)⁹⁰. Furthermore, three clusters of infection using the peracetic acid automated endoscope reprocessor were linked to inadequately processed bronchoscopes when inappropriate channel connectors were used with the system⁷²⁵. These clusters highlight the importance of training, proper model-specific endoscope connector systems, and quality-control procedures to ensure compliance with endoscope manufacturer recommendations and professional organization guidelines. An alternative high-level disinfectant available in the United Kingdom contains 0.35% peracetic acid. Although this product is rapidly effective against a broad range of microorganisms^{466, 726, 727}, it tarnishes the metal of endoscopes and is unstable, resulting in only a 24-hour use life⁷²⁷.

Peracetic Acid and Hydrogen Peroxide

Overview. Two chemical sterilants are available that contain peracetic acid plus hydrogen peroxide (i.e., 0.08% peracetic acid plus 1.0% hydrogen peroxide [no longer marketed]; and 0.23% peracetic acid plus 7.35% hydrogen peroxide (Tables 4 and 5).

Microbicidal Activity. The bactericidal properties of peracetic acid and hydrogen peroxide have been demonstrated⁷²⁸. Manufacturer data demonstrated this combination of peracetic acid and

hydrogen peroxide inactivated all microorganisms except bacterial spores within 20 minutes. The 0.08% peracetic acid plus 1.0% hydrogen peroxide product effectively inactivated glutaraldehyde-resistant mycobacteria⁷²⁹.

Uses. The combination of peracetic acid and hydrogen peroxide has been used for disinfecting hemodialyzers⁷³⁰. The percentage of dialysis centers using a peracetic acid-hydrogen peroxide-based disinfectant for reprocessing dialyzers increased from 5% in 1983 to 56% in 1997²⁴⁹. Olympus America does not endorse use of 0.08% peracetic acid plus 1.0% hydrogen peroxide (Olympus America, personal communication, April 15, 1998) on any Olympus endoscope because of cosmetic and functional damage and will not assume liability for chemical damage resulting from use of this product. This product is not currently available. FDA has cleared a newer chemical sterilant with 0.23% peracetic acid and 7.35% hydrogen peroxide (Tables 4 and 5). After testing the 7.35% hydrogen peroxide and 0.23% peracetic acid product, Olympus America concluded it was not compatible with the company's flexible gastrointestinal endoscopes; this conclusion was based on immersion studies where the test insertion tubes had failed because of swelling and loosening of the black polymer layer of the tube (Olympus America, personal communication, September 13, 2000).

Phenolics

Overview. Phenol has occupied a prominent place in the field of hospital disinfection since its initial use as a germicide by Lister in his pioneering work on antiseptic surgery. In the past 30 years, however, work has concentrated on the numerous phenol derivatives or phenolics and their antimicrobial properties. Phenol derivatives originate when a functional group (e.g., alkyl, phenyl, benzyl, halogen) replaces one of the hydrogen atoms on the aromatic ring. Two phenol derivatives commonly found as constituents of hospital disinfectants are *ortho*-phenylphenol and *ortho*-benzyl-*para*-chlorophenol. The antimicrobial properties of these compounds and many other phenol derivatives are much improved over those of the parent chemical. Phenolics are absorbed by porous materials, and the residual disinfectant can irritate tissue. In 1970, depigmentation of the skin was reported to be caused by phenolic germicidal detergents containing *para*-tertiary butylphenol and *para*-tertiary amyphenol⁷³¹.

Mode of Action. In high concentrations, phenol acts as a gross protoplasmic poison, penetrating and disrupting the cell wall and precipitating the cell proteins. Low concentrations of phenol and higher molecular-weight phenol derivatives cause bacterial death by inactivation of essential enzyme systems and leakage of essential metabolites from the cell wall⁷³².

Microbicidal Activity. Published reports on the antimicrobial efficacy of commonly used phenolics showed they were bactericidal, fungicidal, virucidal, and tuberculocidal^{14, 61, 71, 73, 227, 416, 573, 732-738}. One study demonstrated little or no virucidal effect of a phenolic against coxsackie B4, echovirus 11, and poliovirus 1⁷³⁶. Similarly, 12% *ortho*-phenylphenol failed to inactivate any of the three hydrophilic viruses after a 10-minute exposure time, although 5% phenol was lethal for these viruses⁷². A 0.5% dilution of a phenolic (2.8% *ortho*-phenylphenol and 2.7% *ortho*-benzyl-*para*-chlorophenol) inactivated HIV²²⁷ and a 2% solution of a phenolic (15% *ortho*-phenylphenol and 6.3% *para*-tertiary-amyphenol) inactivated all but one of 11 fungi tested⁷¹.

Manufacturers' data using the standardized AOAC methods demonstrate that commercial phenolics are not sporicidal but are tuberculocidal, fungicidal, virucidal, and bactericidal at their recommended use-dilution. Attempts to substantiate the bactericidal label claims of phenolics using the AOAC Use-Dilution Method occasionally have failed^{416, 737}. However, results from these same studies have varied dramatically among laboratories testing identical products.

Uses. Many phenolic germicides are EPA-registered as disinfectants for use on environmental surfaces (e.g., bedside tables, bedrails, and laboratory surfaces) and noncritical medical devices. Phenolics are not FDA-cleared as high-level disinfectants for use with semicritical items but could be used to preclean or decontaminate critical and semicritical devices before terminal sterilization or high-

level disinfection.

The use of phenolics in nurseries has been questioned because of hyperbilirubinemia in infants placed in bassinets where phenolic detergents were used⁷³⁹. In addition, bilirubin levels were reported to increase in phenolic-exposed infants, compared with nonphenolic-exposed infants, when the phenolic was prepared according to the manufacturers' recommended dilution⁷⁴⁰. If phenolics are used to clean nursery floors, they must be diluted as recommended on the product label. Phenolics (and other disinfectants) should not be used to clean infant bassinets and incubators while occupied. If phenolics are used to terminally clean infant bassinets and incubators, the surfaces should be rinsed thoroughly with water and dried before reuse of infant bassinets and incubators¹⁷.

Quaternary Ammonium Compounds

Overview. The quaternary ammonium compounds are widely used as disinfectants. Health-care-associated infections have been reported from contaminated quaternary ammonium compounds used to disinfect patient-care supplies or equipment, such as cystoscopes or cardiac catheters^{741, 742}. The quaternaries are good cleaning agents, but high water hardness⁷⁴³ and materials such as cotton and gauze pads can make them less microbicidal because of insoluble precipitates or cotton and gauze pads absorb the active ingredients, respectively. One study showed a significant decline (~40%–50% lower at 1 hour) in the concentration of quaternaries released when cotton rags or cellulose-based wipers were used in the open-bucket system, compared with the nonwoven spunlace wipers in the closed-bucket system⁷⁴⁴. As with several other disinfectants (e.g., phenolics, iodophors) gram-negative bacteria can survive or grow in them⁴⁰⁴.

Chemically, the quaternaries are organically substituted ammonium compounds in which the nitrogen atom has a valence of 5, four of the substituent radicals (R1-R4) are alkyl or heterocyclic radicals of a given size or chain length, and the fifth (X⁻) is a halide, sulfate, or similar radical⁷⁴⁵. Each compound exhibits its own antimicrobial characteristics, hence the search for one compound with outstanding antimicrobial properties. Some of the chemical names of quaternary ammonium compounds used in healthcare are alkyl dimethyl benzyl ammonium chloride, alkyl didecyl dimethyl ammonium chloride, and dialkyl dimethyl ammonium chloride. The newer quaternary ammonium compounds (i.e., fourth generation), referred to as twin-chain or dialkyl quaternaries (e.g. didecyl dimethyl ammonium bromide and dioctyl dimethyl ammonium bromide), purportedly remain active in hard water and are tolerant of anionic residues⁷⁴⁶.

A few case reports have documented occupational asthma as a result of exposure to benzalkonium chloride⁷⁴⁷.

Mode of Action. The bactericidal action of the quaternaries has been attributed to the inactivation of energy-producing enzymes, denaturation of essential cell proteins, and disruption of the cell membrane⁷⁴⁶. Evidence exists that supports these and other possibilities^{745, 748}.

Microbicidal Activity. Results from manufacturers' data sheets and from published scientific literature indicate that the quaternaries sold as hospital disinfectants are generally fungicidal, bactericidal, and virucidal against lipophilic (enveloped) viruses; they are not sporicidal and generally not tuberculocidal or virucidal against hydrophilic (nonenveloped) viruses^{14, 54-56, 58, 59, 61, 71, 73, 186, 297, 748, 749}. The poor mycobactericidal activities of quaternary ammonium compounds have been demonstrated^{55, 73}. Quaternary ammonium compounds (as well as 70% isopropyl alcohol, phenolic, and a chlorine-containing wipe [80 ppm]) effectively (>95%) remove and/or inactivate contaminants (i.e., multidrug-resistant *S. aureus*, vancomycin-resistant *Enterococcus*, *P. aeruginosa*) from computer keyboards with a 5-second application time. No functional damage or cosmetic changes occurred to the computer keyboards after 300 applications of the disinfectants⁴⁵.

Attempts to reproduce the manufacturers' bactericidal and tuberculocidal claims using the AOAC

tests with a limited number of quaternary ammonium compounds occasionally have failed^{73, 416, 737}. However, test results have varied extensively among laboratories testing identical products^{416, 737}.

Uses. The quaternaries commonly are used in ordinary environmental sanitation of noncritical surfaces, such as floors, furniture, and walls. EPA-registered quaternary ammonium compounds are appropriate to use for disinfecting medical equipment that contacts intact skin (e.g., blood pressure cuffs).

MISCELLANEOUS INACTIVATING AGENTS

Other Germicides

Several compounds have antimicrobial activity but for various reasons have not been incorporated into the armamentarium of health-care disinfectants. These include mercurials, sodium hydroxide, β -propiolactone, chlorhexidine gluconate, cetrimide-chlorhexidine, glycols (triethylene and propylene), and the Tego disinfectants. Two authoritative references examine these agents in detail^{16,412}.

A peroxygen-containing formulation had marked bactericidal action when used as a 1% weight/volume solution and virucidal activity at 3%⁴⁹, but did not have mycobactericidal activity at concentrations of 2.3% and 4% and exposure times ranging from 30 to 120 minutes⁷⁵⁰. It also required 20 hours to kill *B. atrophaeus* spores⁷⁵¹. A powder-based peroxygen compound for disinfecting contaminated spill was strongly and rapidly bactericidal⁷⁵².

In preliminary studies, nanoemulsions (composed of detergents and lipids in water) showed activity against vegetative bacteria, enveloped viruses and *Candida*. This product represents a potential agent for use as a topical biocidal agent.⁷⁵³⁻⁷⁵⁵

New disinfectants that require further evaluation include glucoprotamin⁷⁵⁶, tertiary amines⁷⁰³, and a light-activated antimicrobial coating⁷⁵⁷. Several other disinfection technologies might have potential applications in the healthcare setting⁷⁵⁸.

Metals as Microbicides

Comprehensive reviews of antiseptics⁷⁵⁹, disinfection⁴²¹, and anti-infective chemotherapy⁷⁶⁰ barely mention the antimicrobial activity of heavy metals^{761,762}. Nevertheless, the anti-infective activity of some heavy metals has been known since antiquity. Heavy metals such as silver have been used for prophylaxis of conjunctivitis of the newborn, topical therapy for burn wounds, and bonding to indwelling catheters, and the use of heavy metals as antiseptics or disinfectants is again being explored⁷⁶³. Inactivation of bacteria on stainless steel surfaces by zeolite ceramic coatings containing silver and zinc ions has also been demonstrated^{764,765}.

Metals such as silver, iron, and copper could be used for environmental control, disinfection of water, or reusable medical devices or incorporated into medical devices (e.g., intravascular catheters)^{400,761-763,766-770}. A comparative evaluation of six disinfectant formulations for residual antimicrobial activity demonstrated that only the silver disinfectant demonstrated significant residual activity against *S. aureus* and *P. aeruginosa*⁷⁶³. Preliminary data suggest metals are effective against a wide variety of microorganisms.

Clinical uses of other heavy metals include copper-8-quinolinolate as a fungicide against *Aspergillus*, copper-silver ionization for *Legionella* disinfection⁷⁷¹⁻⁷⁷⁴, organic mercurials as an antiseptic (e.g., mercurochrome) and preservative/disinfectant (e.g., thimerosal [currently being removed from vaccines]) in pharmaceuticals and cosmetics⁷⁶².

Ultraviolet Radiation (UV)

The wavelength of UV radiation ranges from 328 nm to 210 nm (3280 Å to 2100 Å). Its maximum bactericidal effect occurs at 240–280 nm. Mercury vapor lamps emit more than 90% of their radiation at 253.7 nm, which is near the maximum microbicidal activity⁷⁷⁵. Inactivation of microorganisms results from destruction of nucleic acid through induction of thymine dimers. UV radiation has been employed in the disinfection of drinking water⁷⁷⁶, air⁷⁷⁵, titanium implants⁷⁷⁷, and contact lenses⁷⁷⁸. Bacteria and viruses are more easily killed by UV light than are bacterial spores⁷⁷⁵. UV radiation has several potential applications, but unfortunately its germicidal effectiveness and use is influenced by organic matter; wavelength; type of suspension; temperature; type of microorganism; and UV intensity, which is affected by distance and dirty tubes⁷⁷⁹. The application of UV radiation in the health-care environment (i.e.,

operating rooms, isolation rooms, and biologic safety cabinets) is limited to destruction of airborne organisms or inactivation of microorganisms on surfaces. The effect of UV radiation on postoperative wound infections was investigated in a double-blind, randomized study in five university medical centers. After following 14,854 patients over a 2-year period, the investigators reported the overall wound infection rate was unaffected by UV radiation, although postoperative infection in the “refined clean” surgical procedures decreased significantly (3.8%–2.9%)⁷⁸⁰. No data support the use of UV lamps in isolation rooms, and this practice has caused at least one epidemic of UV-induced skin erythema and keratoconjunctivitis in hospital patients and visitors⁷⁸¹.

Pasteurization

Pasteurization is not a sterilization process; its purpose is to destroy all pathogenic microorganisms. However, pasteurization does not destroy bacterial spores. The time-temperature relation for hot-water pasteurization is generally ~70°C (158°F) for 30 minutes. The water temperature and time should be monitored as part of a quality-assurance program⁷⁸². Pasteurization of respiratory therapy^{783, 784} and anesthesia equipment⁷⁸⁵ is a recognized alternative to chemical disinfection. The efficacy of this process has been tested using an inoculum that the authors believed might simulate contamination by an infected patient. Use of a large inoculum (10^7) of *P. aeruginosa* or *Acinetobacter calcoaceticus* in sets of respiratory tubing before processing demonstrated that machine-assisted chemical processing was more efficient than machine-assisted pasteurization with a disinfection failure rate of 6% and 83%, respectively⁷⁸³. Other investigators found hot water disinfection to be effective (inactivation factor $>5 \log_{10}$) against multiple bacteria, including multidrug-resistant bacteria, for disinfecting reusable anesthesia or respiratory therapy equipment⁷⁸⁴⁻⁷⁸⁶.

Flushing- and Washer-Disinfectors

Flushing- and washer-disinfectors are automated and closed equipment that clean and disinfect objects from bedpans and washbowls to surgical instruments and anesthesia tubes. Items such as bedpans and urinals can be cleaned and disinfected in flushing-disinfectors. They have a short cycle of a few minutes. They clean by flushing with warm water, possibly with a detergent, and then disinfect by flushing the items with hot water or with steam. Because this machine empties, cleans, and disinfects, manual cleaning is eliminated, fewer disposable items are needed, and fewer chemical germicides are used. A microbiologic evaluation of one washer/disinfector demonstrated complete inactivation of suspensions of *E. faecalis* or poliovirus⁷⁸⁷. Other studies have shown that strains of *Enterococcus faecium* can survive the British Standard for heat disinfection of bedpans (80°C for 1 minute). The significance of this finding with reference to the potential for enterococci to survive and disseminate in the health-care environment is debatable⁷⁸⁸⁻⁷⁹⁰. These machines are available and used in many European countries.

Surgical instruments and anesthesia equipment are more difficult to clean. They are run in washer-disinfectors on a longer cycle of approximately 20–30 minutes with a detergent. These machines also disinfect by hot water at approximately 90°C⁷⁹¹.

THE REGULATORY FRAMEWORK FOR DISINFECTANTS AND STERILANTS

Before using the guidance provided in this document, health-care workers should be aware of the federal laws and regulations that govern the sale, distribution, and use of disinfectants and sterilants. In particular, health-care workers need to know what requirements pertain to them when they apply these products. Finally, they should understand the relative roles of EPA, FDA, and CDC so the context for the guidance provided in this document is clear.

EPA and FDA

In the United States, chemical germicides formulated as sanitizers, disinfectants, or sterilants are regulated in interstate commerce by the Antimicrobials Division, Office of Pesticides Program, EPA, under the authority of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) of 1947, as amended⁷⁹². Under FIFRA, any substance or mixture of substances intended to prevent, destroy, repel, or mitigate any pest (including microorganisms but excluding those in or on living humans or animals) must be registered before sale or distribution. To obtain a registration, a manufacturer must submit specific data about the safety and effectiveness of each product. For example, EPA requires manufacturers of sanitizers, disinfectants, or chemical sterilants to test formulations by using accepted methods for microbiocidal activity, stability, and toxicity to animals and humans. The manufacturers submit these data to EPA along with proposed labeling. If EPA concludes the product can be used without causing “unreasonable adverse effects,” then the product and its labeling are registered, and the manufacturer can sell and distribute the product in the United States.

FIFRA also requires users of products to follow explicitly the labeling directions on each product. The following standard statement appears on all labels under the “Directions for Use” heading: “It is a violation of federal law to use this product in a manner inconsistent with its labeling.” This statement means a health-care worker must follow the safety precautions and use directions on the labeling of each registered product. Failure to follow the specified use-dilution, contact time, method of application, or any other condition of use is considered a misuse of the product and potentially subject to enforcement action under FIFRA.

In general, EPA regulates disinfectants and sterilants used on environmental surfaces, and not those used on critical or semicritical medical devices; the latter are regulated by FDA. In June 1993, FDA and EPA issued a “Memorandum of Understanding” that divided responsibility for review and surveillance of chemical germicides between the two agencies. Under the agreement, FDA regulates liquid chemical sterilants used on critical and semicritical devices, and EPA regulates disinfectants used on noncritical surfaces and gaseous sterilants⁷⁹³. In 1996, Congress passed the Food Quality Protection Act (FQPA). This act amended FIFRA in regard to several types of products regulated by both EPA and FDA. One provision of FQPA removed regulation of liquid chemical sterilants used on critical and semicritical medical devices from EPA’s jurisdiction, and it now rests solely with FDA^{792, 794}. EPA continues to register nonmedical chemical sterilants. FDA and EPA have considered the impact of FQPA, and in January 2000, FDA published its final guidance document on product submissions and labeling. Antiseptics are considered antimicrobial drugs used on living tissue and thus are regulated by FDA under the Food, Drug and Cosmetic Act. FDA regulates liquid chemical sterilants and high-level disinfectants intended to process critical and semicritical devices. FDA has published recommendations on the types of test methods that manufacturers should submit to FDA for 510[k] clearance for such agents.

CDC

At CDC, the mission of the Coordinating Center for Infections Diseases is to guide the public on how to prevent and respond to infectious diseases in both health-care settings and at home. With respect to disinfectants and sterilants, part of CDC’s role is to inform the public (in this case healthcare personnel) of current scientific evidence pertaining to these products, to comment about their safety and efficacy, and to recommend which chemicals might be most appropriate or effective for specific microorganisms and settings.

Test Methods

The methods EPA has used for registration are standardized by the AOAC International; however, a survey of scientific literature reveals a number of problems with these tests that were reported during 1987–1990^{58, 76, 80, 428, 736, 737, 795-800} that cause them to be neither accurate nor reproducible^{416, 737}.

As part of their regulatory authority, EPA and FDA support development and validation of methods for assessing disinfection claims⁸⁰¹⁻⁸⁰³. For example, EPA has supported the work of Dr. Syed Sattar and coworkers who have developed a two-tier quantitative carrier test to assess sporicidal, mycobactericidal, bactericidal, fungicidal, virucidal, and protozoacidal activity of chemical germicides^{701, 803}. EPA is accepting label claims against hepatitis B virus (HBV) using a surrogate organism, the duck HBV, to quantify disinfectant activity^{124, 804}. EPA also is accepting labeling claims against hepatitis C virus using the bovine viral diarrhea virus as a surrogate.

For nearly 30 years, EPA also performed intramural preregistration and postregistration efficacy testing of some chemical disinfectants in its own laboratories. In 1982, this was stopped, reportedly for budgetary reasons. At that time, manufacturers did not need to have microbiologic activity claims verified by EPA or an independent testing laboratory when registering a disinfectant or chemical sterilant⁸⁰⁵. This occurred when the frequency of contaminated germicides and infections secondary to their use had increased⁴⁰⁴. Investigations demonstrating that interlaboratory reproducibility of test results was poor and manufacturers' label claims were not verifiable^{416, 737} and symposia sponsored by the American Society for Microbiology⁸⁰⁰ heightened awareness of these problems and reconfirmed the need to improve the AOAC methods and reinstate a microbiologic activity verification program. A General Accounting Office report entitled *Disinfectants: EPA Lacks Assurance They Work*⁸⁰⁶ seemed to provide the necessary impetus for EPA to initiate corrective measures, including cooperative agreements to improve the AOAC methods and independent verification testing for all products labeled as sporicidal and disinfectants labeled as tuberculocidal. For example, of 26 sterilant products tested by EPA, 15 were canceled because of product failure. A list of products registered with EPA and labeled for use as sterilants or tuberculocides or against HIV and/or HBV is available through EPA's website at <http://www.epa.gov/oppad001/chemregindex.htm>. Organizations (e.g., Organization for Economic Cooperation and Development) are working to standardize requirements for germicide testing and registration.

Neutralization of Germicides

One of the difficulties associated with evaluating the bactericidal activity of disinfectants is prevention of bacteriostasis from disinfectant residues carried over into the subculture media. Likewise, small amounts of disinfectants on environmental surfaces can make an accurate bacterial count difficult to get when sampling of the health-care environment as part of an epidemiologic or research investigation. One way these problems may be overcome is by employing neutralizers that inactivate residual disinfectants⁸⁰⁷⁻⁸⁰⁹. Two commonly used neutralizing media for chemical disinfectants are Lethen Media and D/E Neutralizing Media. The former contains lecithin to neutralize quaternaries and polysorbate 80 (Tween 80) to neutralize phenolics, hexachlorophene, formalin, and, with lecithin, ethanol. The D/E Neutralizing media will neutralize a broad spectrum of antiseptic and disinfectant chemicals, including quaternary ammonium compounds, phenols, iodine and chlorine compounds, mercurials, formaldehyde, and glutaraldehyde⁸¹⁰. A review of neutralizers used in germicide testing has been published⁸⁰⁸.

STERILIZATION

Most medical and surgical devices used in healthcare facilities are made of materials that are heat stable and therefore undergo heat, primarily steam, sterilization. However, since 1950, there has been an increase in medical devices and instruments made of materials (e.g., plastics) that require low-temperature sterilization. Ethylene oxide gas has been used since the 1950s for heat- and moisture-sensitive medical devices. Within the past 15 years, a number of new, low-temperature sterilization systems (e.g., hydrogen peroxide gas plasma, peracetic acid immersion, ozone) have been developed and are being used to sterilize medical devices. This section reviews sterilization technologies used in healthcare and makes recommendations for their optimum performance in the processing of medical devices^{1, 18, 811-820}.

Sterilization destroys all microorganisms on the surface of an article or in a fluid to prevent disease transmission associated with the use of that item. While the use of inadequately sterilized critical items represents a high risk of transmitting pathogens, documented transmission of pathogens associated with an inadequately sterilized critical item is exceedingly rare^{821, 822}. This is likely due to the wide margin of safety associated with the sterilization processes used in healthcare facilities. The concept of what constitutes "sterile" is measured as a probability of sterility for each item to be sterilized. This probability is commonly referred to as the sterility assurance level (SAL) of the product and is defined as the probability of a single viable microorganism occurring on a product after sterilization. SAL is normally expressed as a 10^{-n} . For example, if the probability of a spore surviving were one in one million, the SAL would be 10^{-6} ^{823, 824}. In short, a SAL is an estimate of lethality of the entire sterilization process and is a conservative calculation. Dual SALs (e.g., 10^{-3} SAL for blood culture tubes, drainage bags; 10^{-6} SAL for scalpels, implants) have been used in the United States for many years and the choice of a 10^{-6} SAL was strictly arbitrary and not associated with any adverse outcomes (e.g., patient infections)⁸²³.

Medical devices that have contact with sterile body tissues or fluids are considered critical items. These items should be sterile when used because any microbial contamination could result in disease transmission. Such items include surgical instruments, biopsy forceps, and implanted medical devices. If these items are heat resistant, the recommended sterilization process is steam sterilization, because it has the largest margin of safety due to its reliability, consistency, and lethality. However, reprocessing heat- and moisture-sensitive items requires use of a low-temperature sterilization technology (e.g., ethylene oxide, hydrogen peroxide gas plasma, peracetic acid)⁸²⁵. A summary of the advantages and disadvantages for commonly used sterilization technologies is presented in Table 6.

Steam Sterilization

Overview. Of all the methods available for sterilization, moist heat in the form of saturated steam under pressure is the most widely used and the most dependable. Steam sterilization is nontoxic, inexpensive⁸²⁶, rapidly microbicidal, sporicidal, and rapidly heats and penetrates fabrics (Table 6)⁸²⁷. Like all sterilization processes, steam sterilization has some deleterious effects on some materials, including corrosion and combustion of lubricants associated with dental handpieces²¹²; reduction in ability to transmit light associated with laryngoscopes⁸²⁸; and increased hardening time (5.6 fold) with plaster-cast⁸²⁹.

The basic principle of steam sterilization, as accomplished in an autoclave, is to expose each item to direct steam contact at the required temperature and pressure for the specified time. Thus, there are four parameters of steam sterilization: steam, pressure, temperature, and time. The ideal steam for sterilization is dry saturated steam and entrained water (dryness fraction $\geq 97\%$)^{813, 819}. Pressure serves as a means to obtain the high temperatures necessary to quickly kill microorganisms. Specific temperatures must be obtained to ensure the microbicidal activity. The two common steam-sterilizing temperatures are 121°C (250°F) and 132°C (270°F). These temperatures (and other high temperatures)⁸³⁰ must be maintained for a minimal time to kill microorganisms. Recognized minimum exposure periods for sterilization of wrapped healthcare supplies are 30 minutes at 121°C (250°F) in a gravity displacement

sterilizer or 4 minutes at 132°C (270°C) in a prevacuum sterilizer (Table 7). At constant temperatures, sterilization times vary depending on the type of item (e.g., metal versus rubber, plastic, items with lumens), whether the item is wrapped or unwrapped, and the sterilizer type.

The two basic types of steam sterilizers (autoclaves) are the gravity displacement autoclave and the high-speed prevacuum sterilizer. In the former, steam is admitted at the top or the sides of the sterilizing chamber and, because the steam is lighter than air, forces air out the bottom of the chamber through the drain vent. The gravity displacement autoclaves are primarily used to process laboratory media, water, pharmaceutical products, regulated medical waste, and nonporous articles whose surfaces have direct steam contact. For gravity displacement sterilizers the penetration time into porous items is prolonged because of incomplete air elimination. This point is illustrated with the decontamination of 10 lbs of microbiological waste, which requires at least 45 minutes at 121°C because the entrapped air remaining in a load of waste greatly retards steam permeation and heating efficiency^{831, 832}. The high-speed prevacuum sterilizers are similar to the gravity displacement sterilizers except they are fitted with a vacuum pump (or ejector) to ensure air removal from the sterilizing chamber and load before the steam is admitted. The advantage of using a vacuum pump is that there is nearly instantaneous steam penetration even into porous loads. The Bowie-Dick test is used to detect air leaks and inadequate air removal and consists of folded 100% cotton surgical towels that are clean and preconditioned. A commercially available Bowie-Dick-type test sheet should be placed in the center of the pack. The test pack should be placed horizontally in the front, bottom section of the sterilizer rack, near the door and over the drain, in an otherwise empty chamber and run at 134°C for 3.5 minutes^{813, 819}. The test is used each day the vacuum-type steam sterilizer is used, before the first processed load. Air that is not removed from the chamber will interfere with steam contact. Smaller disposable test packs (or process challenge devices) have been devised to replace the stack of folded surgical towels for testing the efficacy of the vacuum system in a prevacuum sterilizer.⁸³³ These devices are “designed to simulate product to be sterilized and to constitute a defined challenge to the sterilization process”^{819, 834}. They should be representative of the load and simulate the greatest challenge to the load⁸³⁵. Sterilizer vacuum performance is acceptable if the sheet inside the test pack shows a uniform color change. Entrapped air will cause a spot to appear on the test sheet, due to the inability of the steam to reach the chemical indicator. If the sterilizer fails the Bowie-Dick test, do not use the sterilizer until it is inspected by the sterilizer maintenance personnel and passes the Bowie-Dick test^{813, 819, 836}.

Another design in steam sterilization is a steam flush-pressure pulsing process, which removes air rapidly by repeatedly alternating a steam flush and a pressure pulse above atmospheric pressure. Air is rapidly removed from the load as with the prevacuum sterilizer, but air leaks do not affect this process because the steam in the sterilizing chamber is always above atmospheric pressure. Typical sterilization temperatures and times are 132°C to 135°C with 3 to 4 minutes exposure time for porous loads and instruments^{827, 837}.

Like other sterilization systems, the steam cycle is monitored by mechanical, chemical, and biological monitors. Steam sterilizers usually are monitored using a printout (or graphically) by measuring temperature, the time at the temperature, and pressure. Typically, chemical indicators are affixed to the outside and incorporated into the pack to monitor the temperature or time and temperature. The effectiveness of steam sterilization is monitored with a biological indicator containing spores of *Geobacillus stearothermophilus* (formerly *Bacillus stearothermophilus*). Positive spore test results are a relatively rare event⁸³⁸ and can be attributed to operator error, inadequate steam delivery⁸³⁹, or equipment malfunction.

Portable (table-top) steam sterilizers are used in outpatient, dental, and rural clinics⁸⁴⁰. These sterilizers are designed for small instruments, such as hypodermic syringes and needles and dental instruments. The ability of the sterilizer to reach physical parameters necessary to achieve sterilization should be monitored by mechanical, chemical, and biological indicators.

Microbicidal Activity. The oldest and most recognized agent for inactivation of microorganisms is heat. D-values (time to reduce the surviving population by 90% or 1 log₁₀) allow a direct comparison of the heat resistance of microorganisms. Because a D-value can be determined at various temperatures, a subscript is used to designate the exposure temperature (i.e., D_{121C}). D_{121C}-values for *Geobacillus stearothermophilus* used to monitor the steam sterilization process range from 1 to 2 minutes. Heat-resistant nonspore-forming bacteria, yeasts, and fungi have such low D_{121C} values that they cannot be experimentally measured⁸⁴¹.

Mode of Action. Moist heat destroys microorganisms by the irreversible coagulation and denaturation of enzymes and structural proteins. In support of this fact, it has been found that the presence of moisture significantly affects the coagulation temperature of proteins and the temperature at which microorganisms are destroyed.

Uses. Steam sterilization should be used whenever possible on all critical and semicritical items that are heat and moisture resistant (e.g., steam sterilizable respiratory therapy and anesthesia equipment), even when not essential to prevent pathogen transmission. Steam sterilizers also are used in healthcare facilities to decontaminate microbiological waste and sharps containers^{831, 832, 842} but additional exposure time is required in the gravity displacement sterilizer for these items.

Flash Sterilization

Overview. “Flash” steam sterilization was originally defined by Underwood and Perkins as sterilization of an unwrapped object at 132°C for 3 minutes at 27-28 lbs. of pressure in a gravity displacement sterilizer⁸⁴³. Currently, the time required for flash sterilization depends on the type of sterilizer and the type of item (i.e., porous vs non-porous items)(see Table 8). Although the wrapped method of sterilization is preferred for the reasons listed below, correctly performed flash sterilization is an effective process for the sterilization of critical medical devices^{844, 845}. Flash sterilization is a modification of conventional steam sterilization (either gravity, prevacuum, or steam-flush pressure-pulse) in which the flashed item is placed in an open tray or is placed in a specially designed, covered, rigid container to allow for rapid penetration of steam. Historically, it is not recommended as a routine sterilization method because of the lack of timely biological indicators to monitor performance, absence of protective packaging following sterilization, possibility for contamination of processed items during transportation to the operating rooms, and the sterilization cycle parameters (i.e., time, temperature, pressure) are minimal. To address some of these concerns, many healthcare facilities have done the following: placed equipment for flash sterilization in close proximity to operating rooms to facilitate aseptic delivery to the point of use (usually the sterile field in an ongoing surgical procedure); extended the exposure time to ensure lethality comparable to sterilized wrapped items (e.g., 4 minutes at 132°C)^{846, 847}; used biological indicators that provide results in 1 hour for flash-sterilized items^{846, 847}; and used protective packaging that permits steam penetration^{812, 817-819, 845, 848}. Further, some rigid, reusable sterilization container systems have been designed and validated by the container manufacturer for use with flash cycles. When sterile items are open to air, they will eventually become contaminated. Thus, the longer a sterile item is exposed to air, the greater the number of microorganisms that will settle on it. Sterilization cycle parameters for flash sterilization are shown in Table 8.

A few adverse events have been associated with flash sterilization. When evaluating an increased incidence of neurosurgical infections, the investigators noted that surgical instruments were flash sterilized between cases and 2 of 3 craniotomy infections involved plate implants that were flash sterilized⁸⁴⁹. A report of two patients who received burns during surgery from instruments that had been flash sterilized reinforced the need to develop policies and educate staff to prevent the use of instruments hot enough to cause clinical burns⁸⁵⁰. Staff should use precautions to prevent burns with potentially hot instruments (e.g., transport tray using heat-protective gloves). Patient burns may be prevented by either air-cooling the instruments or immersion in sterile liquid (e.g., saline).

Uses. Flash sterilization is considered acceptable for processing cleaned patient-care items that

cannot be packaged, sterilized, and stored before use. It also is used when there is insufficient time to sterilize an item by the preferred package method. Flash sterilization should not be used for reasons of convenience, as an alternative to purchasing additional instrument sets, or to save time⁸¹⁷. Because of the potential for serious infections, flash sterilization is not recommended for implantable devices (i.e., devices placed into a surgically or naturally formed cavity of the human body); however, flash sterilization may be unavoidable for some devices (e.g., orthopedic screw, plates). If flash sterilization of an implantable device is unavoidable, recordkeeping (i.e., load identification, patient's name/hospital identifier, and biological indicator result) is essential for epidemiological tracking (e.g., of surgical site infection, tracing results of biological indicators to patients who received the item to document sterility), and for an assessment of the reliability of the sterilization process (e.g., evaluation of biological monitoring records and sterilization maintenance records noting preventive maintenance and repairs with dates).

Low-Temperature Sterilization Technologies

Ethylene oxide (ETO) has been widely used as a low-temperature sterilant since the 1950s. It has been the most commonly used process for sterilizing temperature- and moisture-sensitive medical devices and supplies in healthcare institutions in the United States. Two types of ETO sterilizers are available, mixed gas and 100% ETO. Until 1995, ethylene oxide sterilizers combined ETO with a chlorofluorocarbon (CFC) stabilizing agent, most commonly in a ratio of 12% ETO mixed with 88% CFC (referred to as 12/88 ETO).

For several reasons, healthcare personnel have been exploring the use of new low-temperature sterilization technologies^{825, 851}. First, CFCs were phased out in December 1995 under provisions of the Clean Air Act⁸⁵². CFCs were classified as a Class I substance under the Clean Air Act because of scientific evidence linking them to destruction of the earth's ozone layer. Second, some states (e.g., California, New York, Michigan) require the use of ETO abatement technology to reduce the amount of ETO being released into ambient air from 90 to 99.9% depending on the state. Third, OSHA regulates the acceptable vapor levels of ETO (i.e., 1 ppm averaged over 8 hours) due to concerns that ETO exposure represents an occupational hazard³¹⁸. These constraints have led to the development of alternative technologies for low-temperature sterilization in the healthcare setting.

Alternative technologies to ETO with chlorofluorocarbon that are currently available and cleared by the FDA for medical equipment include 100% ETO; ETO with a different stabilizing gas, such as carbon dioxide or hydrochlorofluorocarbons (HCFC); immersion in peracetic acid; hydrogen peroxide gas plasma; and ozone. Technologies under development for use in healthcare facilities, but not cleared by the FDA, include vaporized hydrogen peroxide, vapor phase peracetic acid, gaseous chlorine dioxide, ionizing radiation, or pulsed light^{400, 758, 853}. However, there is no guarantee that these new sterilization technologies will receive FDA clearance for use in healthcare facilities.

These new technologies should be compared against the characteristics of an ideal low-temperature (<60°C) sterilant (Table 9).⁸⁵¹ While it is apparent that all technologies will have limitations (Table 9), understanding the limitations imposed by restrictive device designs (e.g., long, narrow lumens) is critical for proper application of new sterilization technology⁸⁵⁴. For example, the development of increasingly small and complex endoscopes presents a difficult challenge for current sterilization processes. This occurs because microorganisms must be in direct contact with the sterilant for inactivation to occur. Several peer-reviewed scientific publications have data demonstrating concerns about the efficacy of several of the low-temperature sterilization processes (i.e., gas plasma, vaporized hydrogen peroxide, ETO, peracetic acid), particularly when the test organisms are challenged in the presence of serum and salt and a narrow lumen vehicle^{469, 721, 825, 855, 856}. Factors shown to affect the efficacy of sterilization are shown in Table 10.

Ethylene Oxide "Gas" Sterilization

Overview. ETO is a colorless gas that is flammable and explosive. The four essential

parameters (operational ranges) are: gas concentration (450 to 1200 mg/l); temperature (37 to 63°C); relative humidity (40 to 80%)(water molecules carry ETO to reactive sites); and exposure time (1 to 6 hours). These influence the effectiveness of ETO sterilization^{814, 857, 858}. Within certain limitations, an increase in gas concentration and temperature may shorten the time necessary for achieving sterilization.

The main disadvantages associated with ETO are the lengthy cycle time, the cost, and its potential hazards to patients and staff; the main advantage is that it can sterilize heat- or moisture-sensitive medical equipment without deleterious effects on the material used in the medical devices (Table 6). Acute exposure to ETO may result in irritation (e.g., to skin, eyes, gastrointestinal or respiratory tracts) and central nervous system depression⁸⁵⁹⁻⁸⁶². Chronic inhalation has been linked to the formation of cataracts, cognitive impairment, neurologic dysfunction, and disabling polyneuropathies^{860, 861, 863-866}. Occupational exposure in healthcare facilities has been linked to hematologic changes⁸⁶⁷ and an increased risk of spontaneous abortions and various cancers^{318, 868-870}. ETO should be considered a known human carcinogen⁸⁷¹.

The basic ETO sterilization cycle consists of five stages (i.e., preconditioning and humidification, gas introduction, exposure, evacuation, and air washes) and takes approximately 2 1/2 hrs excluding aeration time. Mechanical aeration for 8 to 12 hours at 50 to 60°C allows desorption of the toxic ETO residual contained in exposed absorbent materials. Most modern ETO sterilizers combine sterilization and aeration in the same chamber as a continuous process. These ETO models minimize potential ETO exposure during door opening and load transfer to the aerator. Ambient room aeration also will achieve desorption of the toxic ETO but requires 7 days at 20°C. There are no federal regulations for ETO sterilizer emission; however, many states have promulgated emission-control regulations⁸¹⁴.

The use of ETO evolved when few alternatives existed for sterilizing heat- and moisture-sensitive medical devices; however, favorable properties (Table 6) account for its continued widespread use⁸⁷². Two ETO gas mixtures are available to replace ETO-chlorofluorocarbon (CFC) mixtures for large capacity, tank-supplied sterilizers. The ETO-carbon dioxide (CO₂) mixture consists of 8.5% ETO and 91.5% CO₂. This mixture is less expensive than ETO-hydrochlorofluorocarbons (HCFC), but a disadvantage is the need for pressure vessels rated for steam sterilization, because higher pressures (28-psi gauge) are required. The other mixture, which is a drop-in CFC replacement, is ETO mixed with HCFC. HCFCs are approximately 50-fold less damaging to the earth's ozone layer than are CFCs. The EPA will begin regulation of HCFC in the year 2015 and will terminate production in the year 2030. Two companies provide ETO-HCFC mixtures as drop-in replacement for CFC-12; one mixture consists of 8.6% ETO and 91.4% HCFC, and the other mixture is composed of 10% ETO and 90% HCFC⁸⁷². An alternative to the pressurized mixed gas ETO systems is 100% ETO. The 100% ETO sterilizers using unit-dose cartridges eliminate the need for external tanks.

ETO is absorbed by many materials. For this reason, following sterilization the item must undergo aeration to remove residual ETO. Guidelines have been promulgated regarding allowable ETO limits for devices that depend on how the device is used, how often, and how long in order to pose a minimal risk to patients in normal product use⁸¹⁴.

ETO toxicity has been established in a variety of animals. Exposure to ETO can cause eye pain, sore throat, difficulty breathing and blurred vision. Exposure can also cause dizziness, nausea, headache, convulsions, blisters and vomiting and coughing⁸⁷³. In a variety of *in vitro* and animal studies, ETO has been demonstrated to be carcinogenic. ETO has been linked to spontaneous abortion, genetic damage, nerve damage, peripheral paralysis, muscle weakness, and impaired thinking and memory⁸⁷³. Occupational exposure in healthcare facilities has been linked to an increased risk of spontaneous abortions and various cancers³¹⁸. Injuries (e.g., tissue burns) to patients have been associated with ETO residues in implants used in surgical procedures⁸⁷⁴. Residual ETO in capillary flow dialysis membranes has been shown to be neurotoxic *in vitro*⁸⁷⁵. OSHA has established a PEL of 1 ppm airborne ETO in the workplace, expressed as a TWA for an 8-hour work shift in a 40-hour work week. The "action level" for ETO is 0.5 ppm, expressed as an 8-hour TWA, and the short-term excursion limit is 5 ppm, expressed as

a 15-minute TWA⁸¹⁴. For details of the requirements in OSHA's ETO standard for occupational exposures, see Title 29 of the Code of Federal Regulations (CFR) Part 1910.1047⁸⁷³. Several personnel monitoring methods (e.g., charcoal tubes and passive sampling devices) are in use⁸¹⁴. OSHA has established a PEL of 5 ppm for ethylene chlorohydrin (a toxic by-product of ETO) in the workplace⁸⁷⁶. Additional information regarding use of ETO in health care facilities is available from NIOSH.

Mode of Action. The microbicidal activity of ETO is considered to be the result of alkylation of protein, DNA, and RNA. Alkylation, or the replacement of a hydrogen atom with an alkyl group, within cells prevents normal cellular metabolism and replication⁸⁷⁷.

Microbicidal Activity. The excellent microbicidal activity of ETO has been demonstrated in several studies^{469, 721, 722, 856, 878, 879} and summarized in published reports⁸⁷⁷. ETO inactivates all microorganisms although bacterial spores (especially *B. atrophaeus*) are more resistant than other microorganisms. For this reason *B. atrophaeus* is the recommended biological indicator.

Like all sterilization processes, the effectiveness of ETO sterilization can be altered by lumen length, lumen diameter, inorganic salts, and organic materials^{469, 721, 722, 855, 856, 879}. For example, although ETO is not used commonly for reprocessing endoscopes²⁸, several studies have shown failure of ETO in inactivating contaminating spores in endoscope channels⁸⁵⁵ or lumen test units^{469, 721, 879} and residual ETO levels averaging 66.2 ppm even after the standard degassing time⁴⁵⁶. Failure of ETO also has been observed when dental handpieces were contaminated with *Streptococcus mutans* and exposed to ETO⁸⁸⁰. It is recommended that dental handpieces be steam sterilized.

Uses. ETO is used in healthcare facilities to sterilize critical items (and sometimes semicritical items) that are moisture or heat sensitive and cannot be sterilized by steam sterilization.

Hydrogen Peroxide Gas Plasma

Overview. New sterilization technology based on plasma was patented in 1987 and marketed in the United States in 1993. Gas plasmas have been referred to as the fourth state of matter (i.e., liquids, solids, gases, and gas plasmas). Gas plasmas are generated in an enclosed chamber under deep vacuum using radio frequency or microwave energy to excite the gas molecules and produce charged particles, many of which are in the form of free radicals. A free radical is an atom with an unpaired electron and is a highly reactive species. The proposed mechanism of action of this device is the production of free radicals within a plasma field that are capable of interacting with essential cell components (e.g., enzymes, nucleic acids) and thereby disrupt the metabolism of microorganisms. The type of seed gas used and the depth of the vacuum are two important variables that can determine the effectiveness of this process.

In the late 1980s the first hydrogen peroxide gas plasma system for sterilization of medical and surgical devices was field-tested. According to the manufacturer, the sterilization chamber is evacuated and hydrogen peroxide solution is injected from a cassette and is vaporized in the sterilization chamber to a concentration of 6 mg/l. The hydrogen peroxide vapor diffuses through the chamber (50 minutes), exposes all surfaces of the load to the sterilant, and initiates the inactivation of microorganisms. An electrical field created by a radio frequency is applied to the chamber to create a gas plasma. Microbicidal free radicals (e.g., hydroxyl and hydroperoxyl) are generated in the plasma. The excess gas is removed and in the final stage (i.e., vent) of the process the sterilization chamber is returned to atmospheric pressure by introduction of high-efficiency filtered air. The by-products of the cycle (e.g., water vapor, oxygen) are nontoxic and eliminate the need for aeration. Thus, the sterilized materials can be handled safely, either for immediate use or storage. The process operates in the range of 37-44°C and has a cycle time of 75 minutes. If any moisture is present on the objects the vacuum will not be achieved and the cycle aborts^{856, 881-883}.

A newer version of the unit improves sterilizer efficacy by using two cycles with a hydrogen

peroxide diffusion stage and a plasma stage per sterilization cycle. This revision, which is achieved by a software modification, reduces total processing time from 73 to 52 minutes. The manufacturer believes that the enhanced activity obtained with this system is due in part to the pressure changes that occur during the injection and diffusion phases of the process and to the fact that the process consists of two equal and consecutive half cycles, each with a separate injection of hydrogen peroxide.^{856, 884, 885} This system and a smaller version^{400, 882} have received FDA 510[k] clearance with limited application for sterilization of medical devices (Table 6). The biological indicator used with this system is *Bacillus atrophaeus* spores⁸⁵¹. The newest version of the unit, which employs a new vaporization system that removes most of the water from the hydrogen peroxide, has a cycle time from 28-38 minutes (see manufacturer's literature for device dimension restrictions).

Penetration of hydrogen peroxide vapor into long or narrow lumens has been addressed outside the United States by the use of a diffusion enhancer. This is a small, breakable glass ampoule of concentrated hydrogen peroxide (50%) with an elastic connector that is inserted into the device lumen and crushed immediately before sterilization^{470, 885}. The diffusion enhancer has been shown to sterilize bronchoscopes contaminated with *Mycobacteria tuberculosis*⁸⁸⁶. At the present time, the diffusion enhancer is not FDA cleared.

Another gas plasma system, which differs from the above in several important ways, including the use of peracetic acid-acetic acid-hydrogen peroxide vapor, was removed from the marketplace because of reports of corneal destruction to patients when ophthalmic surgery instruments had been processed in the sterilizer^{887, 888}. In this investigation, exposure of potentially wet ophthalmologic surgical instruments with small bores and brass components to the plasma gas led to degradation of the brass to copper and zinc^{888, 889}. The experimenters showed that when rabbit eyes were exposed to the rinsates of the gas plasma-sterilized instruments, corneal decompensation was documented. This toxicity is highly unlikely with the hydrogen peroxide gas plasma process since a toxic, soluble form of copper would not form (LA Feldman, written communication, April 1998).

Mode of Action. This process inactivates microorganisms primarily by the combined use of hydrogen peroxide gas and the generation of free radicals (hydroxyl and hydroperoxyl free radicals) during the plasma phase of the cycle.

Microbicidal Activity. This process has the ability to inactivate a broad range of microorganisms, including resistant bacterial spores. Studies have been conducted against vegetative bacteria (including mycobacteria), yeasts, fungi, viruses, and bacterial spores^{469, 721, 856, 881-883, 890-893}. Like all sterilization processes, the effectiveness can be altered by lumen length, lumen diameter, inorganic salts, and organic materials^{469, 721, 855, 856, 890, 891, 893}.

Uses. Materials and devices that cannot tolerate high temperatures and humidity, such as some plastics, electrical devices, and corrosion-susceptible metal alloys, can be sterilized by hydrogen peroxide gas plasma. This method has been compatible with most (>95%) medical devices and materials tested^{884, 894, 895}.

Peracetic Acid Sterilization

Overview. Peracetic acid is a highly biocidal oxidizer that maintains its efficacy in the presence of organic soil. Peracetic acid removes surface contaminants (primarily protein) on endoscopic tubing^{711, 717}. An automated machine using peracetic acid to sterilize medical, surgical, and dental instruments chemically (e.g., endoscopes, arthroscopes) was introduced in 1988. This microprocessor-controlled, low-temperature sterilization method is commonly used in the United States¹⁰⁷. The sterilant, 35% peracetic acid, and an anticorrosive agent are supplied in a single-dose container. The container is punctured at the time of use, immediately prior to closing the lid and initiating the cycle. The concentrated peracetic acid is diluted to 0.2% with filtered water (0.2 μm) at a temperature of approximately 50°C. The diluted peracetic acid is circulated within the chamber of the machine and

pumped through the channels of the endoscope for 12 minutes, decontaminating exterior surfaces, lumens, and accessories. Interchangeable trays are available to permit the processing of up to three rigid endoscopes or one flexible endoscope. Connectors are available for most types of flexible endoscopes for the irrigation of all channels by directed flow. Rigid endoscopes are placed within a lidded container, and the sterilant fills the lumens either by immersion in the circulating sterilant or by use of channel connectors to direct flow into the lumen(s) (see below for the importance of channel connectors). The peracetic acid is discarded via the sewer and the instrument rinsed four times with filtered water. Concern has been raised that filtered water may be inadequate to maintain sterility⁸⁹⁶. Limited data have shown that low-level bacterial contamination may follow the use of filtered water in an AER but no data has been published on AERs using the peracetic acid system¹⁶¹. Clean filtered air is passed through the chamber of the machine and endoscope channels to remove excess water⁷¹⁹. As with any sterilization process, the system can only sterilize surfaces that can be contacted by the sterilant. For example, bronchoscopy-related infections occurred when bronchoscopes were processed using the wrong connector^{155, 725}. Investigation of these incidents revealed that bronchoscopes were inadequately reprocessed when inappropriate channel connectors were used and when there were inconsistencies between the reprocessing instructions provided by the manufacturer of the bronchoscope and the manufacturer of the automatic endoscope reprocessor¹⁵⁵. The importance of channel connectors to achieve sterilization was also shown for rigid lumen devices^{137, 856}.

The manufacturers suggest the use of biological monitors (*G. stearothermophilus* spore strips) both at the time of installation and routinely to ensure effectiveness of the process. The manufacturer's clip must be used to hold the strip in the designated spot in the machine as a broader clamp will not allow the sterilant to reach the spores trapped under it⁸⁹⁷. One investigator reported a 3% failure rate when the appropriate clips were used to hold the spore strip within the machine⁷¹⁸. The use of biological monitors designed to monitor either steam sterilization or ETO for a liquid chemical sterilizer has been questioned for several reasons including spore wash-off from the filter paper strips which may cause less valid monitoring⁸⁹⁸⁻⁹⁰¹. The processor is equipped with a conductivity probe that will automatically abort the cycle if the buffer system is not detected in a fresh container of the peracetic acid solution. A chemical monitoring strip that detects that the active ingredient is >1500 ppm is available for routine use as an additional process control.

Mode of Action. Only limited information is available regarding the mechanism of action of peracetic acid, but it is thought to function as other oxidizing agents, i.e., it denatures proteins, disrupts cell wall permeability, and oxidizes sulfhydryl and sulfur bonds in proteins, enzymes, and other metabolites^{654, 726}.

Microbicidal Activity. Peracetic acid will inactivate gram-positive and gram-negative bacteria, fungi, and yeasts in <5 minutes at <100 ppm. In the presence of organic matter, 200-500 ppm is required. For viruses, the dosage range is wide (12-2250 ppm), with poliovirus inactivated in yeast extract in 15 minutes with 1500 to 2250 ppm. Bacterial spores in suspension are inactivated in 15 seconds to 30 minutes with 500 to 10,000 ppm (0.05 to 1%)⁶⁵⁴.

Simulated-use trials have demonstrated microbicidal activity^{111, 718-722} and three clinical trials have demonstrated both microbial killing and no clinical failures leading to infection^{90, 723, 724}. Alfa and co-workers, who compared the peracetic acid system with ETO, demonstrated the high efficacy of the system. Only the peracetic acid system was able to completely kill 6-log₁₀ of *Mycobacterium chelonae*, *Enterococcus faecalis*, and *B. atrophaeus* spores with both an organic and inorganic challenge⁷²². Like other sterilization processes, the efficacy of the process can be diminished by soil challenges⁹⁰² and test conditions⁸⁵⁶.

Uses. This automated machine is used to chemically sterilize medical (e.g., GI endoscopes) and surgical (e.g., flexible endoscopes) instruments in the United States. Lumened endoscopes must be connected to an appropriate channel connector to ensure that the sterilant has direct contact with the contaminated lumen.^{137, 856, 903} Olympus America has not listed this system as a compatible product for

use in reprocessing Olympus bronchoscopes and gastrointestinal endoscopes (Olympus America, January 30, 2002, written communication).

Microbicidal Activity of Low-Temperature Sterilization Technologies

Sterilization processes used in the United States must be cleared by FDA, and they require that sterilizer microbicidal performance be tested under simulated-use conditions⁹⁰⁴. FDA requires that the test article be inoculated with 10^6 colony-forming units of the most resistant test organism and prepared with organic and inorganic test loads as would occur after actual use. FDA requires manufacturers to use organic soil (e.g., 5% fetal calf serum), dried onto the device with the inoculum, to represent soil remaining on the device following marginal cleaning. However, 5% fetal calf serum as a measure of marginal cleaning has not been validated by measurements of protein load on devices following use and the level of protein removal by various cleaning methods. The inocula must be placed in various locations of the test articles, including those least favorable to penetration and contact with the sterilant (e.g., lumens). Cleaning before sterilization is not allowed in the demonstration of sterilization efficacy⁹⁰⁴. Several studies have evaluated the relative microbicidal efficacy of these low-temperature sterilization technologies (Table 11). These studies have either tested the activity of a sterilization process against specific microorganisms^{892, 905, 906}, evaluated the microbicidal activity of a singular technology^{711, 719, 724, 855, 879, 882-884, 890, 891, 907}, or evaluated the comparative effectiveness of several sterilization technologies^{271, 426, 469, 721, 722, 856, 908, 909}. Several test methodologies use stainless steel or porcelain carriers that are inoculated with a test organism. Commonly used test organisms include vegetative bacteria, mycobacteria, and spores of *Bacillus* species. The available data demonstrate that low-temperature sterilization technologies are able to provide a 6- \log_{10} reduction of microbes when inoculated onto carriers in the absence of salt and serum. However, tests can be constructed such that all of the available sterilization technologies are unable to reliably achieve complete inactivation of a microbial load.^{425, 426, 469, 721, 856, 909} For example, almost all of the sterilization processes will fail to reliably inactivate the microbial load in the presence of salt and serum^{469, 721, 909}.

The effect of salts and serums on the sterilization process were studied initially in the 1950s and 1960s^{424, 910}. These studies showed that a high concentration of crystalline-type materials and a low protein content provided greater protection to spores than did serum with a high protein content⁴²⁶. A study by Doyle and Ernst demonstrated resistance of spores by crystalline material applied not only to low-temperature sterilization technology but also to steam and dry heat⁴²⁵. These studies showed that occlusion of *Bacillus atrophaeus* spores in calcium carbonate crystals dramatically increased the time required for inactivation as follows: 10 seconds to 150 minutes for steam (121°C), 3.5 hours to 50 hours for dry heat (121°C), 30 seconds to >2 weeks for ETO (54°C). Investigators have corroborated and extended these findings^{469, 470, 721, 855, 908, 909}. While soils containing both organic and inorganic materials impair microbial killing, soils that contain a high inorganic salt-to-protein ratio favor crystal formation and impair sterilization by occlusion of organisms^{425, 426, 881}.

Alfa and colleagues demonstrated a 6- \log_{10} reduction of the microbial inoculum of porcelain penicylinders using a variety of vegetative and spore-forming organisms (Table 11)⁴⁶⁹. However, if the bacterial inoculum was in tissue-culture medium supplemented with 10% serum, only the ETO 12/88 and ETO-HCFC sterilization mixtures could sterilize 95% to 97% of the penicylinder carriers. The plasma and 100% ETO sterilizer demonstrated significantly reduced activity (Table 11). For all sterilizers evaluated using penicylinder carriers (i.e., ETO 12/88, 100% ETO, hydrogen peroxide gas plasma), there was a 3- to 6- \log_{10} reduction of inoculated bacteria even in the presence of serum and salt. For each sterilizer evaluated, the ability to inactivate microorganisms in the presence of salt and serum was reduced even further when the inoculum was placed in a narrow-lumen test object (3 mm diameter by 125 cm long). Although there was a 2- to 4- \log_{10} reduction in microbial kill, less than 50% of the lumen test objects were sterile when processed using any of the sterilization methods evaluated except the peracetic acid immersion system (Table 11)⁷²¹. Complete killing (or removal) of 6- \log_{10} of *Enterococcus faecalis*, *Mycobacterium chelonae*, and *Bacillus atrophaeus* spores in the presence of salt and serum and lumen test objects was observed only for the peracetic acid immersion system.

With respect to the results by Alfa and coworkers⁴⁶⁹, Jacobs showed that the use of the tissue culture media created a technique-induced sterilization failure⁴²⁶. Jacobs et al. showed that microorganisms mixed with tissue culture media, used as a surrogate body fluid, formed physical crystals that protected the microorganisms used as a challenge. If the carriers were exposed for 60 sec to nonflowing water, the salts dissolved and the protective effect disappeared. Since any device would be exposed to water for a short period of time during the washing procedure, these protective effects would have little clinical relevance⁴²⁶.

Narrow lumens provide a challenge to some low-temperature sterilization processes. For example, Rutala and colleagues showed that, as lumen size decreased, increased failures occurred with some low-temperature sterilization technologies. However, some low-temperature processes such as ETO-HCFC and the hydrogen peroxide gas plasma process remained effective even when challenged by a lumen as small as 1 mm in the absence of salt and serum⁸⁵⁶.

The importance of allowing the sterilant to come into contact with the inoculated carrier is demonstrated by comparing the results of two investigators who studied the peracetic acid immersion system. Alfa and coworkers demonstrated excellent activity of the peracetic acid immersion system against three test organisms using a narrow-lumen device. In these experiments, the lumen test object was connected to channel irrigators, which ensured that the sterilant had direct contact with the contaminated carriers⁷²². This effectiveness was achieved through a combination of organism wash-off and peracetic acid sterilant killing the test organisms⁷²². The data reported by Rutala et al. demonstrated failure of the peracetic acid immersion system to eliminate *Geobacillus stearothermophilus* spores from a carrier placed in a lumen test object. In these experiments, the lumen test unit was not connected to channel irrigators. The authors attributed the failure of the peracetic acid immersion system to eliminate the high levels of spores from the center of the test unit to the inability of the peracetic acid to diffuse into the center of 40-cm long, 3-mm diameter tubes. This may be caused by an air lock or air bubbles formed in the lumen, impeding the flow of the sterilant through the long and narrow lumen and limiting complete access to the *Bacillus* spores^{137, 856}. Experiments using a channel connector specifically designed for 1-, 2-, and 3-mm lumen test units with the peracetic acid immersion system were completely effective in eliminating an inoculum of 10^6 *Geobacillus stearothermophilus* spores⁷. The restricted diffusion environment that exists in the test conditions would not exist with flexible scopes processed in the peracetic acid immersion system, because the scopes are connected to channel irrigators to ensure that the sterilant has direct contact with contaminated surfaces. Alfa and associates attributed the efficacy of the peracetic acid immersion system to the ability of the liquid chemical process to dissolve salts and remove protein and bacteria due to the flushing action of the fluid⁷²².

Bioburden of Surgical Devices

In general, used medical devices are contaminated with a relatively low bioburden of organisms^{179, 911, 912}. Nystrom evaluated medical instruments used in general surgical, gynecological, orthopedic, and ear-nose-throat operations and found that 62% of the instruments were contaminated with $<10^1$ organisms after use, 82% with $<10^2$, and 91% with $<10^3$. After being washed in an instrument washer, more than 98% of the instruments had $<10^1$ organisms, and none $>10^2$ organisms⁹¹¹. Other investigators have published similar findings^{179, 912}. For example, after a standard cleaning procedure, 72% of 50 surgical instruments contained $<10^1$ organisms, 86% $<10^2$, and only 6% had $>3 \times 10^{2912}$. In another study of rigid-lumen medical devices, the bioburden on both the inner and outer surface of the lumen ranged from 10^1 to 10^4 organisms per device. After cleaning, 83% of the devices had a bioburden $\leq 10^2$ organisms¹⁷⁹. In all of these studies, the contaminating microflora consisted mainly of vegetative bacteria, usually of low pathogenicity (e.g., coagulase-negative *Staphylococcus*)^{179, 911, 912}.

An evaluation of the microbial load on used critical medical devices such as spinal anesthesia needles and angiographic catheters and sheaths demonstrated that mesophilic microorganisms were detected at levels of 10^1 to 10^2 in only two of five needles. The bioburden on used angiographic

catheters and sheath introducers exceeded 10^3 CFUs on 14% (3 of 21) and 21% (6 of 28), respectively⁹⁰⁷.

Effect of Cleaning on Sterilization Efficacy

The effect of salt and serum on the efficacy of low-temperature sterilization technologies has raised concern regarding the margin of safety of these technologies. Experiments have shown that salts have the greatest impact on protecting microorganisms from killing^{426, 469}. However, other studies have suggested that these concerns may not be clinically relevant. One study evaluated the relative rate of removal of inorganic salts, organic soil, and microorganisms from medical devices to better understand the dynamics of the cleaning process⁴²⁶. These tests were conducted by inoculating Alfa soil (tissue-culture media and 10% fetal bovine serum)⁴⁶⁹ containing 10^6 *G. stearothermophilus* spores onto the surface of a stainless-steel scalpel blade. After drying for 30 minutes at 35°C followed by 30 minutes at room temperature, the samples were placed in water at room temperature. The blades were removed at specified times, and the concentration of total protein and chloride ion was measured. The results showed that soaking in deionized water for 60 seconds resulted in a >95% release rate of chloride ion from NaCl solution in 20 seconds, Alfa soil in 30 seconds, and fetal bovine serum in 120 seconds. Thus, contact with water for short periods, even in the presence of protein, rapidly leads to dissolution of salt crystals and complete inactivation of spores by a low-temperature sterilization process (Table 10). Based on these experimental data, cleaning procedures would eliminate the detrimental effect of high salt content on a low-temperature sterilization process.

These articles^{426, 469, 721} assessing low-temperature sterilization technology reinforce the importance of meticulous cleaning before sterilization. These data support the critical need for healthcare facilities to develop rigid protocols for cleaning contaminated objects before sterilization⁴⁷². Sterilization of instruments and medical devices is compromised if the process is not preceded by meticulous cleaning.

The cleaning of any narrow-lumen medical device used in patient care presents a major challenge to reprocessing areas. While attention has been focused on flexible endoscopes, cleaning issues related to other narrow-lumen medical devices such as sphinctertomes have been investigated⁹¹³. This study compared manual cleaning with that of automated cleaning with a narrow-lumen cleaner and found that only retro-flushing with the narrow lumen cleaner provided adequate cleaning of the three channels. If reprocessing was delayed for more than 24 hours, retro-flush cleaning was no longer effective and ETO sterilization failure was detected when devices were held for 7 days⁹¹³. In another study involving simulated-use cleaning of laparoscopic devices, Alfa found that minimally the use of retro-flushing should be used during cleaning of non-ported laparoscopic devices⁹¹⁴.

Other Sterilization Methods

Ionizing Radiation. Sterilization by ionizing radiation, primarily by cobalt 60 gamma rays or electron accelerators, is a low-temperature sterilization method that has been used for a number of medical products (e.g., tissue for transplantation, pharmaceuticals, medical devices). There are no FDA-cleared ionizing radiation sterilization processes for use in healthcare facilities. Because of high sterilization costs, this method is an unfavorable alternative to ETO and plasma sterilization in healthcare facilities but is suitable for large-scale sterilization. Some deleterious effects on patient-care equipment associated with gamma radiation include induced oxidation in polyethylene⁹¹⁵ and delamination and cracking in polyethylene knee bearings⁹¹⁶. Several reviews^{917, 918} dealing with the sources, effects, and application of ionizing radiation may be referred to for more detail.

Dry-Heat Sterilizers. This method should be used only for materials that might be damaged by moist heat or that are impenetrable to moist heat (e.g., powders, petroleum products, sharp instruments). The advantages for dry heat include the following: it is nontoxic and does not harm the environment; a dry heat cabinet is easy to install and has relatively low operating costs; it penetrates materials; and it is noncorrosive for metal and sharp instruments. The disadvantages for dry heat are the slow rate of heat penetration and microbial killing makes this a time-consuming method. In addition, the high temperatures

are not suitable for most materials⁹¹⁹. The most common time-temperature relationships for sterilization with hot air sterilizers are 170°C (340°F) for 60 minutes, 160°C (320°F) for 120 minutes, and 150°C (300°F) for 150 minutes. *B. atrophaeus* spores should be used to monitor the sterilization process for dry heat because they are more resistant to dry heat than are *G. stearothermophilus* spores. The primary lethal process is considered to be oxidation of cell constituents.

There are two types of dry-heat sterilizers: the static-air type and the forced-air type. The static-air type is referred to as the oven-type sterilizer as heating coils in the bottom of the unit cause the hot air to rise inside the chamber via gravity convection. This type of dry-heat sterilizer is much slower in heating, requires longer time to reach sterilizing temperature, and is less uniform in temperature control throughout the chamber than is the forced-air type. The forced-air or mechanical convection sterilizer is equipped with a motor-driven blower that circulates heated air throughout the chamber at a high velocity, permitting a more rapid transfer of energy from the air to the instruments⁹²⁰.

Liquid Chemicals. Several FDA-cleared liquid chemical sterilants include indications for sterilization of medical devices (Tables 4 and 5)⁶⁹. The indicated contact times range from 3 hours to 12 hours. However, except for a few of the products, the contact time is based only on the conditions to pass the AOAC Sporicidal Test as a sterilant and not on simulated use testing with devices. These solutions are commonly used as high-level disinfectants when a shorter processing time is required. Generally, chemical liquid sterilants cannot be monitored using a biological indicator to verify sterility^{899, 900}.

The survival kinetics for thermal sterilization methods, such as steam and dry heat, have been studied and characterized extensively, whereas the kinetics for sterilization with liquid sterilants are less well understood⁹²¹. The information that is available in the literature suggests that sterilization processes based on liquid chemical sterilants, in general, may not convey the same sterility assurance level as sterilization achieved using thermal or physical methods⁸²³. The data indicate that the survival curves for liquid chemical sterilants may not exhibit log-linear kinetics and the shape of the survivor curve may vary depending of the formulation, chemical nature and stability of the liquid chemical sterilant. In addition, the design of the AOAC Sporicidal Test does not provide quantification of the microbial challenge. Therefore, sterilization with a liquid chemical sterilant may not convey the same sterility assurance as other sterilization methods.

One of the differences between thermal and liquid chemical processes for sterilization of devices is the accessibility of microorganisms to the sterilant. Heat can penetrate barriers, such as biofilm, tissue, and blood, to attain organism kill, whereas liquids cannot adequately penetrate these barriers. In addition, the viscosity of some liquid chemical sterilants impedes their access to organisms in the narrow lumens and mated surfaces of devices⁹²². Another limitation to sterilization of devices with liquid chemical germicides is the post-processing environment of the device. Devices cannot be wrapped or adequately contained during processing in a liquid chemical sterilant to maintain sterility following processing and during storage. Furthermore, devices may require rinsing following exposure to the liquid chemical sterilant with water that typically is not sterile. Therefore, due to the inherent limitations of using liquid chemical sterilants, their use should be restricted to reprocessing critical devices that are heat-sensitive and incompatible with other sterilization methods.

Several published studies compare the sporicidal effect of liquid chemical germicides against spores of *Bacillus* and *Clostridium*^{78, 659, 660, 715}.

Performic Acid. Performic acid is a fast-acting sporicide that was incorporated into an automated endoscope reprocessing system⁴⁰⁰. Systems using performic acid are not currently FDA cleared.

Filtration. Although filtration is not a lethality-based process and is not an FDA-cleared sterilization method, this technology is used to remove bacteria from thermolabile pharmaceutical fluids

that cannot be purified by any other means. In order to remove bacteria, the membrane pore size (e.g., 0.22 μm) must be smaller than the bacteria and uniform throughout⁹²³. Some investigators have appropriately questioned whether the removal of microorganisms by filtration really is a sterilization method because of slight bacterial passage through filters, viral passage through filters, and transference of the sterile filtrate into the final container under aseptic conditions entail a risk of contamination⁹²⁴.

Microwave. Microwaves are used in medicine for disinfection of soft contact lenses, dental instruments, dentures, milk, and urinary catheters for intermittent self-catheterization⁹²⁵⁻⁹³¹. However, microwaves must only be used with products that are compatible (e.g., do not melt)⁹³¹. Microwaves are radio-frequency waves, which are usually used at a frequency of 2450 MHz. The microwaves produce friction of water molecules in an alternating electrical field. The intermolecular friction derived from the vibrations generates heat and some authors believe that the effect of microwaves depends on the heat produced while others postulate a nonthermal lethal effect⁹³²⁻⁹³⁴. The initial reports showed microwaves to be an effective microbicide. The microwaves produced by a "home-type" microwave oven (2.45 GHz) completely inactivate bacterial cultures, mycobacteria, viruses, and *G. stearothermophilus* spores within 60 seconds to 5 minutes depending on the challenge organism^{933, 935-937}. Another study confirmed these results but also found that higher power microwaves in the presence of water may be needed for sterilization⁹³². Complete destruction of *Mycobacterium bovis* was obtained with 4 minutes of microwave exposure (600W, 2450 MHz)⁹³⁷. The effectiveness of microwave ovens for different sterilization and disinfection purposes should be tested and demonstrated as test conditions affect the results (e.g., presence of water, microwave power). Sterilization of metal instruments can be accomplished but requires certain precautions.⁹²⁶ Of concern is that home-type microwave ovens may not have even distribution of microwave energy over the entire dry device (there may be hot and cold spots on solid medical devices); hence there may be areas that are not sterilized or disinfected. The use of microwave ovens to disinfect intermittent-use catheters also has been suggested. Researchers found that test bacteria (e.g., *E. coli*, *Klebsiella pneumoniae*, *Candida albicans*) were eliminated from red rubber catheters within 5 minutes⁹³¹. Microwaves used for sterilization of medical devices have not been FDA cleared.

Glass Bead "Sterilizer". Glass bead "sterilization" uses small glass beads (1.2-1.5 mm diameter) and high temperature (217 °C -232°C) for brief exposure times (e.g., 45 seconds) to inactivate microorganisms. These devices have been used for several years in the dental profession⁹³⁸⁻⁹⁴⁰. FDA believes there is a risk of infection with this device because of potential failure to sterilize dental instruments and their use should be discontinued until the device has received FDA clearance.

Vaporized Hydrogen Peroxide (VHP®). Hydrogen peroxide solutions have been used as chemical sterilants for many years. However, the VHP® was not developed for the sterilization of medical equipment until the mid-1980s. One method for delivering VHP to the reaction site uses a deep vacuum to pull liquid hydrogen peroxide (30-35% concentration) from a disposable cartridge through a heated vaporizer and then, following vaporization, into the sterilization chamber. A second approach to VHP delivery is the flow-through approach in which the VHP is carried into the sterilization chamber by a carrier gas such as air using either a slight negative pressure (vacuum) or slight positive pressure. Applications of this technology include vacuum systems for industrial sterilization of medical devices and atmospheric systems for decontaminating for large and small areas⁸⁵³. VHP offers several appealing features that include rapid cycle time (e.g., 30-45 minutes); low temperature; environmentally safe by-products (H_2O , oxygen [O_2]); good material compatibility; and ease of operation, installation and monitoring. VHP has limitations including that cellulose cannot be processed; nylon becomes brittle; and VHP penetration capabilities are less than those of ETO. VHP has not been cleared by FDA for sterilization of medical devices in healthcare facilities.

The feasibility of utilizing vapor-phase hydrogen peroxide as a surface decontaminant and sterilizer was evaluated in a centrifuge decontamination application. In this study, vapor-phase hydrogen peroxide was shown to possess significant sporicidal activity⁹⁴¹. In preliminary studies, hydrogen

peroxide vapor decontamination has been found to be a highly effective method of eradicating MRSA, *Serratia marcescens*, *Clostridium botulinum* spores and *Clostridium difficile* from rooms, furniture, surfaces and/or equipment; however, further investigation of this method to demonstrate both safety and effectiveness in reducing infection rates are required⁹⁴²⁻⁹⁴⁵.

Ozone. Ozone has been used for years as a drinking water disinfectant. Ozone is produced when O₂ is energized and split into two monatomic (O₁) molecules. The monatomic oxygen molecules then collide with O₂ molecules to form ozone, which is O₃. Thus, ozone consists of O₂ with a loosely bonded third oxygen atom that is readily available to attach to, and oxidize, other molecules. This additional oxygen atom makes ozone a powerful oxidant that destroys microorganisms but is highly unstable (i.e., half-life of 22 minutes at room temperature).

A new sterilization process, which uses ozone as the sterilant, was cleared by FDA in August 2003 for processing reusable medical devices. The sterilizer creates its own sterilant internally from USP grade oxygen, steam-quality water and electricity; the sterilant is converted back to oxygen and water vapor at the end of the cycle by a passing through a catalyst before being exhausted into the room. The duration of the sterilization cycle is about 4 h and 15 m, and it occurs at 30-35°C. Microbial efficacy has been demonstrated by achieving a SAL of 10⁻⁶ with a variety of microorganisms to include the most resistant microorganism, *Geobacillus stearothermophilus*.

The ozone process is compatible with a wide range of commonly used materials including stainless steel, titanium, anodized aluminum, ceramic, glass, silica, PVC, Teflon, silicone, polypropylene, polyethylene and acrylic. In addition, rigid lumen devices of the following diameter and length can be processed: internal diameter (ID): > 2 mm, length ≤ 25 cm; ID > 3 mm, length ≤ 47 cm; and ID > 4 mm, length ≤ 60 cm.

The process should be safe for use by the operator because there is no handling of the sterilant, no toxic emissions, no residue to aerate, and low operating temperature means there is no danger of an accidental burn. The cycle is monitored using a self-contained biological indicator and a chemical indicator. The sterilization chamber is small, about 4 ft³ (Written communication, S Dufresne, July 2004).

A gaseous ozone generator was investigated for decontamination of rooms used to house patients colonized with MRSA. The results demonstrated that the device tested would be inadequate for the decontamination of a hospital room⁹⁴⁶.

Formaldehyde Steam. Low-temperature steam with formaldehyde is used as a low-temperature sterilization method in many countries, particularly in Scandinavia, Germany, and the United Kingdom. The process involves the use of formalin, which is vaporized into a formaldehyde gas that is admitted into the sterilization chamber. A formaldehyde concentration of 8-16 mg/l is generated at an operating temperature of 70-75°C. The sterilization cycle consists of a series of stages that include an initial vacuum to remove air from the chamber and load, followed by steam admission to the chamber with the vacuum pump running to purge the chamber of air and to heat the load, followed by a series of pulses of formaldehyde gas, followed by steam. Formaldehyde is removed from the sterilizer and load by repeated alternate evacuations and flushing with steam and air. This system has some advantages, e.g., the cycle time for formaldehyde gas is faster than that for ETO and the cost per cycle is relatively low. However, ETO is more penetrating and operates at lower temperatures than do steam/formaldehyde sterilizers. Low-temperature steam formaldehyde sterilization has been found effective against vegetative bacteria, mycobacteria, *B. atrophaeus* and *G. stearothermophilus* spores and *Candida albicans*⁹⁴⁷⁻⁹⁴⁹.

Formaldehyde vapor cabinets also may be used in healthcare facilities to sterilize heat-sensitive medical equipment⁹⁵⁰. Commonly, there is no circulation of formaldehyde and no temperature and humidity controls. The release of gas from paraformaldehyde tablets (placed on the lower tray) is slow and produces a low partial pressure of gas. The microbicidal quality of this procedure is unknown⁹⁵¹.

Reliable sterilization using formaldehyde is achieved when performed with a high concentration of gas, at a temperature between 60° and 80°C and with a relative humidity of 75 to 100%.

Studies indicate that formaldehyde is a mutagen and a potential human carcinogen, and OSHA regulates formaldehyde. The permissible exposure limit for formaldehyde in work areas is 0.75 ppm measured as a 8-hour TWA. The OSHA standard includes a 2 ppm STEL (i.e., maximum exposure allowed during a 15-minute period). As with the ETO standard, the formaldehyde standard requires that the employer conduct initial monitoring to identify employees who are exposed to formaldehyde at or above the action level or STEL. If this exposure level is maintained, employers may discontinue exposure monitoring until there is a change that could affect exposure levels or an employee reports formaldehyde-related signs and symptoms^{269, 578}. The formaldehyde steam sterilization system has not been FDA cleared for use in healthcare facilities.

Gaseous chlorine dioxide. A gaseous chlorine dioxide system for sterilization of healthcare products was developed in the late 1980s^{853, 952, 953}. Chlorine dioxide is not mutagenic or carcinogenic in humans. As the chlorine dioxide concentration increases, the time required to achieve sterilization becomes progressively shorter. For example, only 30 minutes were required at 40 mg/l to sterilize the 10⁶ *B. atrophaeus* spores at 30° to 32°C⁹⁵⁴. Currently, no gaseous chlorine dioxide system is FDA cleared.

Vaporized Peracetic Acid. The sporicidal activity of peracetic acid vapor at 20, 40, 60, and 80% relative humidity and 25°C was determined on *Bacillus atrophaeus* spores on paper and glass surfaces. Appreciable activity occurred within 10 minutes of exposure to 1 mg of peracetic acid per liter at 40% or higher relative humidity⁹⁵⁵. No vaporized peracetic acid system is FDA cleared.

Infrared radiation. An infrared radiation prototype sterilizer was investigated and found to destroy *B. atrophaeus* spores. Some of the possible advantages of infrared technology include short cycle time, low energy consumption, no cycle residuals, and no toxicologic or environmental effects. This may provide an alternative technology for sterilization of selected heat-resistant instruments but there are no FDA-cleared systems for use in healthcare facilities⁹⁵⁶.

The other sterilization technologies mentioned above may be used for sterilization of critical medical items if cleared by the FDA and ideally, the microbicidal effectiveness of the technology has been published in the scientific literature. The selection and use of disinfectants, chemical sterilants and sterilization processes in the healthcare field is dynamic, and products may become available that are not in existence when this guideline was written. As newer disinfectants and sterilization processes become available, persons or committees responsible for selecting disinfectants and sterilization processes should be guided by products cleared by FDA and EPA as well as information in the scientific literature.

Sterilizing Practices

Overview. The delivery of sterile products for use in patient care depends not only on the effectiveness of the sterilization process but also on the unit design, decontamination, disassembling and packaging of the device, loading the sterilizer, monitoring, sterilant quality and quantity, and the appropriateness of the cycle for the load contents, and other aspects of device reprocessing. Healthcare personnel should perform most cleaning, disinfecting, and sterilizing of patient-care supplies in a central processing department in order to more easily control quality. The aim of central processing is the orderly processing of medical and surgical instruments to protect patients from infections while minimizing risks to staff and preserving the value of the items being reprocessed⁹⁵⁷. Healthcare facilities should promote the same level of efficiency and safety in the preparation of supplies in other areas (e.g., operating room, respiratory therapy) as is practiced in central processing.

Ensuring consistency of sterilization practices requires a comprehensive program that ensures operator competence and proper methods of cleaning and wrapping instruments, loading the sterilizer,

operating the sterilizer, and monitoring of the entire process. Furthermore, care must be consistent from an infection prevention standpoint in all patient-care settings, such as hospital and outpatient facilities.

Sterilization Cycle Verification. A sterilization process should be verified before it is put into use in healthcare settings. All steam, ETO, and other low-temperature sterilizers are tested with biological and chemical indicators upon installation, when the sterilizer is relocated, redesigned, after major repair and after a sterilization failure has occurred to ensure they are functioning prior to placing them into routine use. Three consecutive empty steam cycles are run with a biological and chemical indicator in an appropriate test package or tray. Each type of steam cycle used for sterilization (e.g., vacuum-assisted, gravity) is tested separately. In a prevacuum steam sterilizer three consecutive empty cycles are also run with a Bowie-Dick test. The sterilizer is not put back into use until all biological indicators are negative and chemical indicators show a correct end-point response^{811-814, 819, 958}.

Biological and chemical indicator testing is also done for ongoing quality assurance testing of representative samples of actual products being sterilized and product testing when major changes are made in packaging, wraps, or load configuration. Biological and chemical indicators are placed in products, which are processed in a full load. When three consecutive cycles show negative biological indicators and chemical indicators with a correct end point response, you can put the change made into routine use^{811-814, 958}. Items processed during the three evaluation cycles should be quarantined until the test results are negative.

Physical Facilities. The central processing area(s) ideally should be divided into at least three areas: decontamination, packaging, and sterilization and storage. Physical barriers should separate the decontamination area from the other sections to contain contamination on used items. In the decontamination area reusable contaminated supplies (and possibly disposable items that are reused) are received, sorted, and decontaminated. The recommended airflow pattern should contain contaminants within the decontamination area and minimize the flow of contaminants to the clean areas. The American Institute of Architects⁹⁵⁹ recommends negative pressure and no fewer than six air exchanges per hour in the decontamination area (AAMI recommends 10 air changes per hour) and 10 air changes per hour with positive pressure in the sterilizer equipment room. The packaging area is for inspecting, assembling, and packaging clean, but not sterile, material. The sterile storage area should be a limited access area with a controlled temperature (may be as high as 75°F) and relative humidity (30-60% in all works areas except sterile storage, where the relative humidity should not exceed 70%)⁸¹⁹. The floors and walls should be constructed of materials capable of withstanding chemical agents used for cleaning or disinfecting. Ceilings and wall surfaces should be constructed of non-shedding materials. Physical arrangements of processing areas are presented schematically in four references^{811, 819, 920, 957}.

Cleaning. As repeatedly mentioned, items must be cleaned using water with detergents or enzymatic cleaners^{465, 466, 468} before processing. Cleaning reduces the bioburden and removes foreign material (i.e., organic residue and inorganic salts) that interferes with the sterilization process by acting as a barrier to the sterilization agent^{179, 426, 457, 911, 912}. Surgical instruments are generally presoaked or prerinsed to prevent drying of blood and tissue. Precleaning in patient-care areas may be needed on items that are heavily soiled with feces, sputum, blood, or other material. Items sent to central processing without removing gross soil may be difficult to clean because of dried secretions and excretions. Cleaning and decontamination should be done as soon as possible after items have been used.

Several types of mechanical cleaning machines (e.g., utensil washer-sanitizer, ultrasonic cleaner, washer-sterilizer, dishwasher, washer-disinfector) may facilitate cleaning and decontamination of most items. This equipment often is automated and may increase productivity, improve cleaning effectiveness, and decrease worker exposure to blood and body fluids. Delicate and intricate objects and heat- or moisture-sensitive articles may require careful cleaning by hand. All used items sent to the central processing area should be considered contaminated (unless decontaminated in the area of origin), handled with gloves (forceps or tongs are sometimes needed to avoid exposure to sharps), and decontaminated by one of the aforementioned methods to render them safer to handle. Items composed

of more than one removable part should be disassembled. Care should be taken to ensure that all parts are kept together, so that reassembly can be accomplished efficiently⁸¹¹.

Investigators have described the degree of cleanliness by visual and microscopic examination. One study found 91% of the instruments to be clean visually but, when examined microscopically, 84% of the instruments had residual debris. Sites that contained residual debris included junctions between insulating sheaths and activating mechanisms of laparoscopic instruments and articulations and grooves of forceps. More research is needed to understand the clinical significance of these findings⁹⁶⁰ and how to ensure proper cleaning.

Personnel working in the decontamination area should wear household-cleaning-type rubber or plastic gloves when handling or cleaning contaminated instruments and devices. Face masks, eye protection such as goggles or full-length faceshields, and appropriate gowns should be worn when exposure to blood and contaminated fluids may occur (e.g., when manually cleaning contaminated devices)⁹⁶¹. Contaminated instruments are a source of microorganisms that could inoculate personnel through nonintact skin on the hands or through contact with the mucous membranes of eyes, nose, or mouth^{214, 811, 813}. Reusable sharps that have been in contact with blood present a special hazard. Employees must not reach with their gloved hands into trays or containers that hold these sharps to retrieve them²¹⁴. Rather, employees should use engineering controls (e.g., forceps) to retrieve these devices.

Packaging. Once items are cleaned, dried, and inspected, those requiring sterilization must be wrapped or placed in rigid containers and should be arranged in instrument trays/baskets according to the guidelines provided by the AAMI and other professional organizations^{454, 811-814, 819, 836, 962}. These guidelines state that hinged instruments should be opened; items with removable parts should be disassembled unless the device manufacturer or researchers provide specific instructions or test data to the contrary¹⁸¹; complex instruments should be prepared and sterilized according to device manufacturer's instructions and test data; devices with concave surfaces should be positioned to facilitate drainage of water; heavy items should be positioned not to damage delicate items; and the weight of the instrument set should be based on the design and density of the instruments and the distribution of metal mass^{811, 962}. While there is no longer a specified sterilization weight limit for surgical sets, heavy metal mass is a cause of wet packs (i.e., moisture inside the case and tray after completion of the sterilization cycle)⁹⁶³. Other parameters that may influence drying are the density of the wraps and the design of the set⁹⁶⁴.

There are several choices in methods to maintain sterility of surgical instruments, including rigid containers, peel-open pouches (e.g., self-sealed or heat-sealed plastic and paper pouches), roll stock or reels (i.e., paper-plastic combinations of tubing designed to allow the user to cut and seal the ends to form a pouch)⁴⁵⁴ and sterilization wraps (woven and nonwoven). Healthcare facilities may use all of these packaging options. The packaging material must allow penetration of the sterilant, provide protection against contact contamination during handling, provide an effective barrier to microbial penetration, and maintain the sterility of the processed item after sterilization⁹⁶⁵. An ideal sterilization wrap would successfully address barrier effectiveness, penetrability (i.e., allows sterilant to penetrate), aeration (e.g., allows ETO to dissipate), ease of use, drapeability, flexibility, puncture resistance, tear strength, toxicity, odor, waste disposal, linting, cost, and transparency⁹⁶⁶. Unacceptable packaging for use with ETO (e.g., foil, polyvinylchloride, and polyvinylidene chloride [kitchen-type transparent wrap])⁸¹⁴ or hydrogen peroxide gas plasma (e.g., linens and paper) should not be used to wrap medical items.

In central processing, double wrapping can be done sequentially or nonsequentially (i.e., simultaneous wrapping). Wrapping should be done in such a manner to avoid tenting and gapping. The sequential wrap uses two sheets of the standard sterilization wrap, one wrapped after the other. This procedure creates a package within a package. The nonsequential process uses two sheets wrapped at the same time so that the wrapping needs to be performed only once. This latter method provides

multiple layers of protection of surgical instruments from contamination and saves time since wrapping is done only once. Multiple layers are still common practice due to the rigors of handling within the facility even though the barrier efficacy of a single sheet of wrap has improved over the years⁹⁶⁶. Written and illustrated procedures for preparation of items to be packaged should be readily available and used by personnel when packaging procedures are performed⁴⁵⁴.

Loading. All items to be sterilized should be arranged so all surfaces will be directly exposed to the sterilizing agent. Thus, loading procedures must allow for free circulation of steam (or another sterilant) around each item. Historically, it was recommended that muslin fabric packs should not exceed the maximal dimensions, weight, and density of 12 inches wide x 12 inches high x 20 inches long, 12 lbs, and 7.2 lbs per cubic foot, respectively. Due to the variety of textiles and metal/plastic containers on the market, the textile and metal/plastic container manufacturer and the sterilizer manufacturers should be consulted for instructions on pack preparation and density parameters⁸¹⁹.

There are several important basic principles for loading a sterilizer: allow for proper sterilant circulation; perforated trays should be placed so the tray is parallel to the shelf; nonperforated containers should be placed on their edge (e.g., basins); small items should be loosely placed in wire baskets; and peel packs should be placed on edge in perforated or mesh bottom racks or baskets^{454, 811, 836}.

Storage. Studies in the early 1970s suggested that wrapped surgical trays remained sterile for varying periods depending on the type of material used to wrap the trays. Safe storage times for sterile packs vary with the porosity of the wrapper and storage conditions (e.g., open versus closed cabinets). Heat-sealed, plastic peel-down pouches and wrapped packs sealed in 3-mil (3/1000 inch) polyethylene overwrap have been reported to be sterile for as long as 9 months after sterilization. The 3-mil polyethylene is applied after sterilization to extend the shelf life for infrequently used items⁹⁶⁷. Supplies wrapped in double-thickness muslin comprising four layers, or equivalent, remain sterile for at least 30 days. Any item that has been sterilized should not be used after the expiration date has been exceeded or if the sterilized package is wet, torn, or punctured.

Although some hospitals continue to date every sterilized product and use the time-related shelf-life practice, many hospitals have switched to an event-related shelf-life practice. This latter practice recognizes that the product should remain sterile until some event causes the item to become contaminated (e.g., tear in packaging, packaging becomes wet, seal is broken)⁹⁶⁸. Event-related factors that contribute to the contamination of a product include bioburden (i.e., the amount of contamination in the environment), air movement, traffic, location, humidity, insects, vermin, flooding, storage area space, open/closed shelving, temperature, and the properties of the wrap material^{966, 969}. There are data that support the event-related shelf-life practice⁹⁷⁰⁻⁹⁷². One study examined the effect of time on the sterile integrity of paper envelopes, peel pouches, and nylon sleeves. The most important finding was the absence of a trend toward an increased rate of contamination over time for any pack when placed in covered storage⁹⁷¹. Another evaluated the effectiveness of event-related outdating by microbiologically testing sterilized items. During the 2-year study period, all of the items tested were sterile⁹⁷². Thus, contamination of a sterile item is event-related and the probability of contamination increases with increased handling⁹⁷³.

Following the sterilization process, medical and surgical devices must be handled using aseptic technique in order to prevent contamination. Sterile supplies should be stored far enough from the floor (8 to 10 inches), the ceiling (5 inches unless near a sprinkler head [18 inches from sprinkler head]), and the outside walls (2 inches) to allow for adequate air circulation, ease of cleaning, and compliance with local fire codes (e.g., supplies must be at least 18 inches from sprinkler heads). Medical and surgical supplies should not be stored under sinks or in other locations where they can become wet. Sterile items that become wet are considered contaminated because moisture brings with it microorganisms from the air and surfaces. Closed or covered cabinets are ideal but open shelving may be used for storage. Any package that has fallen or been dropped on the floor must be inspected for damage to the packaging and

contents (if the items are breakable). If the package is heat-sealed in impervious plastic and the seal is still intact, the package should be considered not contaminated. If undamaged, items packaged in plastic need not be reprocessed.

Monitoring. The sterilization procedure should be monitored routinely by using a combination of mechanical, chemical, and biological indicators to evaluate the sterilizing conditions and indirectly the microbiologic status of the processed items. The mechanical monitors for steam sterilization include the daily assessment of cycle time and temperature by examining the temperature record chart (or computer printout) and an assessment of pressure via the pressure gauge. The mechanical monitors for ETO include time, temperature, and pressure recorders that provide data via computer printouts, gauges, and/or displays⁸¹⁴. Generally, two essential elements for ETO sterilization (i.e., the gas concentration and humidity) cannot be monitored in healthcare ETO sterilizers.

Chemical indicators are convenient, are inexpensive, and indicate that the item has been exposed to the sterilization process. In one study, chemical indicators were more likely than biological indicators to inaccurately indicate sterilization at marginal sterilization times (e.g., 2 minutes)⁸⁴⁷. Chemical indicators should be used in conjunction with biological indicators, but based on current studies should not replace them because they indicate sterilization at marginal sterilization time and because only a biological indicator consisting of resistant spores can measure the microbial killing power of the sterilization process.^{847, 974} Chemical indicators are affixed on the outside of each pack to show that the package has been processed through a sterilization cycle, but these indicators do not prove sterilization has been achieved. Preferably, a chemical indicator also should be placed on the inside of each pack to verify sterilant penetration. Chemical indicators usually are either heat-or chemical-sensitive inks that change color when one or more sterilization parameters (e.g., steam-time, temperature, and/or saturated steam; ETO-time, temperature, relative humidity and/or ETO concentration) are present. Chemical indicators have been grouped into five classes based on their ability to monitor one or multiple sterilization parameters^{813, 819}. If the internal and/or external indicator suggests inadequate processing, the item should not be used⁸¹⁵. An air-removal test (Bowie-Dick Test) must be performed daily in an empty dynamic-air-removal sterilizer (e.g., prevacuum steam sterilizer) to ensure air removal.

Biological indicators are recognized by most authorities as being closest to the ideal monitors of the sterilization process^{974, 975} because they measure the sterilization process directly by using the most resistant microorganisms (i.e., *Bacillus* spores), and not by merely testing the physical and chemical conditions necessary for sterilization. Since the *Bacillus* spores used in biological indicators are more resistant and present in greater numbers than are the common microbial contaminants found on patient-care equipment, the demonstration that the biological indicator has been inactivated strongly implies that other potential pathogens in the load have been killed⁸⁴⁴.

An ideal biological monitor of the sterilization process should be easy to use, be inexpensive, not be subject to exogenous contamination, provide positive results as soon as possible after the cycle so that corrective action may be accomplished, and provide positive results only when the sterilization parameters (e.g., steam-time, temperature, and/or saturated steam; ETO-time, temperature, relative humidity and/or ETO concentration) are inadequate to kill microbial contaminants⁸⁴⁷.

Biological indicators are the only process indicators that directly monitor the lethality of a given sterilization process. Spores used to monitor a sterilization process have demonstrated resistance to the sterilizing agent and are more resistant than the bioburden found on medical devices^{179, 911, 912}. *B. atropheus* spores (10^6) are used to monitor ETO and dry heat, and *G. stearothermophilus* spores (10^5) are used to monitor steam sterilization, hydrogen peroxide gas plasma, and liquid peracetic acid sterilizers. *G. stearothermophilus* is incubated at 55-60°C, and *B. atropheus* is incubated at 35-37°C. Steam and low temperature sterilizers (e.g., hydrogen peroxide gas plasma, peracetic acid) should be monitored at least weekly with the appropriate commercial preparation of spores. If a sterilizer is used frequently (e.g., several loads per day), daily use of biological indicators allows earlier discovery of

equipment malfunctions or procedural errors and thus minimizes the extent of patient surveillance and product recall needed in the event of a positive biological indicator⁸¹¹. Each load should be monitored if it contains implantable objects. If feasible, implantable items should not be used until the results of spore tests are known to be negative.

Originally, spore-strip biological indicators required up to 7 days of incubation to detect viable spores from marginal cycles (i.e., when few spores remained viable). The next generation of biological indicator was self-contained in plastic vials containing a spore-coated paper strip and a growth media in a crushable glass ampoule. This indicator had a maximum incubation of 48 hours but significant failures could be detected in ≤ 24 hours. A rapid-readout biological indicator that detects the presence of enzymes of *G. stearothermophilus* by reading a fluorescent product produced by the enzymatic breakdown of a nonfluorescent substrate has been marketed for the more than 10 years. Studies demonstrate that the sensitivity of rapid-readout tests for steam sterilization (1 hour for 132°C gravity sterilizers, 3 hrs for 121°C gravity and 132°C vacuum sterilizers) parallels that of the conventional sterilization-specific biological indicators^{846, 847, 976, 977} and the fluorescent rapid readout results reliably predict 24- and 48-hour and 7-day growth⁹⁷⁸. The rapid-readout biological indicator is a dual indicator system as it also detects acid metabolites produced during growth of the *G. stearothermophilus* spores. This system is different from the indicator system consisting of an enzyme system of bacterial origin without spores. Independent comparative data using suboptimal sterilization cycles (e.g., reduced time or temperature) with the enzyme-based indicator system have not been published⁹⁷⁹.

A new rapid-readout ETO biological indicator has been designed for rapid and reliable monitoring of ETO sterilization processes. The indicator has been cleared by the FDA for use in the United States⁴⁰⁰. The rapid-readout ETO biological indicator detects the presence of *B. atrophaeus* by detecting a fluorescent signal indicating the activity of an enzyme present within the *B. atrophaeus* organism, beta-glucosidase. The fluorescence indicates the presence of an active spore-associated enzyme and a sterilization process failure. This indicator also detects acid metabolites produced during growth of the *B. atrophaeus* spore. Per manufacturer's data, the enzyme always was detected whenever viable spores were present. This was expected because the enzyme is relatively ETO resistant and is inactivated at a slightly longer exposure time than the spore. The rapid-readout ETO biological indicator can be used to monitor 100% ETO, and ETO-HCFC mixture sterilization cycles. It has not been tested in ETO-CO₂ mixture sterilization cycles.

The standard biological indicator used for monitoring full-cycle steam sterilizers does not provide reliable monitoring flash sterilizers⁹⁸⁰. Biological indicators specifically designed for monitoring flash sterilization are now available, and studies comparing them have been published^{846, 847, 981}.

Since sterilization failure can occur (about 1% for steam)⁹⁸², a procedure to follow in the event of positive spore tests with steam sterilization has been provided by CDC and the Association of periOperative Registered Nurses (AORN). The 1981 CDC recommendation is that "objects, other than implantable objects, do not need to be recalled because of a single positive spore test unless the steam sterilizer or the sterilization procedure is defective." The rationale for this recommendation is that single positive spore tests in sterilizers occur sporadically. They may occur for reasons such as slight variation in the resistance of the spores⁹⁸³, improper use of the sterilizer, and laboratory contamination during culture (uncommon with self-contained spore tests). If the mechanical (e.g., time, temperature, pressure in the steam sterilizer) and chemical (internal and/or external) indicators suggest that the sterilizer was functioning properly, a single positive spore test probably does not indicate sterilizer malfunction but the spore test should be repeated immediately⁹⁸³. If the spore tests remain positive, use of the sterilizer should be discontinued until it is serviced¹. Similarly, AORN states that a single positive spore test does not necessarily indicate a sterilizer failure. If the test is positive, the sterilizer should immediately be rechallenged for proper use and function. Items, other than implantable ones, do not necessarily need to be recalled unless a sterilizer malfunction is found. If a sterilizer malfunction is discovered, the items must be considered nonsterile, and the items from the suspect load(s) should be recalled, insofar as

possible, and reprocessed⁹⁸⁴. A suggested protocol for management of positive biological indicators is shown in Table 12⁸³⁹. A more conservative approach also has been recommended⁸¹³ in which any positive spore test is assumed to represent sterilizer malfunction and requires that all materials processed in that sterilizer, dating from the sterilization cycle having the last negative biologic indicator to the next cycle showing satisfactory biologic indicator challenge results, must be considered nonsterile and retrieved, if possible, and reprocessed. This more conservative approach should be used for sterilization methods other than steam (e.g., ETO, hydrogen peroxide gas plasma). However, no action is necessary if there is strong evidence for the biological indicator being defective⁹⁸³ or the growth medium contained a *Bacillus* contaminant⁹⁸⁵.

If patient-care items were used before retrieval, the infection control professional should assess the risk of infection in collaboration with central processing, surgical services, and risk management staff.

The factors that should be considered include the chemical indicator result (e.g., nonreactive chemical indicator may indicate temperature not achieved); the results of other biological indicators that followed the positive biological indicator (e.g., positive on Tuesday, negative on Wednesday); the parameters of the sterilizer associated with the positive biological indicator (e.g., reduced time at correct temperature); the time-temperature chart (or printout); and the microbial load associated with decontaminated surgical instruments (e.g., 85% of decontaminated surgical instruments have less than 100 CFU). The margin of safety in steam sterilization is sufficiently large that there is minimal infection risk associated with items in a load that show spore growth, especially if the item was properly cleaned and the temperature was achieved (e.g., as shown by acceptable chemical indicator or temperature chart). There are no published studies that document disease transmission via a nonretrieved surgical instrument following a sterilization cycle with a positive biological indicator.

False-positive biological indicators may occur from improper testing or faulty indicators. The latter may occur from improper storage, processing, product contamination, material failure, or variation in resistance of spores. Gram stain and subculture of a positive biological indicator may determine if a contaminant has created a false-positive result^{839, 986}. However, in one incident, the broth used as growth medium contained a contaminant, *B. coagulans*, which resulted in broth turbidity at 55°C⁹⁸⁵. Testing of paired biological indicators from different manufacturers can assist in assessing a product defect⁸³⁹. False-positive biological indicators due to extrinsic contamination when using self-contained biological indicators should be uncommon. A biological indicator should not be considered a false-positive indicator until a thorough analysis of the entire sterilization process shows this to be likely.

The size and composition of the biological indicator test pack should be standardized to create a significant challenge to air removal and sterilant penetration and to obtain interpretable results. There is a standard 16-towel pack recommended by AAMI for steam sterilization^{813, 819, 987} consisting of 16 clean, preconditioned, reusable huck or absorbent surgical towels each of which is approximately 16 inches by 26 inches. Each towel is folded lengthwise into thirds and then folded widthwise in the middle. One or more biological indicators are placed between the eight and ninth towels in the approximate geometric center of the pack. When the towels are folded and placed one on top of another, to form a stack (approximately 6 inch height) it should weigh approximately 3 pounds and should have a density of approximately 11.3 pounds per cubic foot⁸¹³. This test pack has not gained universal use as a standard pack that simulates the actual in-use conditions of steam sterilizers. Commercially available disposable test packs that have been shown to be equivalent to the AAMI 16 towel test pack also may be used. The test pack should be placed flat in an otherwise fully loaded sterilizer chamber, in the area least favorable to sterilization (i.e., the area representing the greatest challenge to the biological indicator). This area is normally in the front, bottom section of the sterilizer, near the drain^{811, 813}. A control biological indicator from the lot used for testing should be left unexposed to the sterilant, and then incubated to verify the presterilization viability of the test spores and proper incubation. The most conservative approach would be to use a control for each run; however, less frequent use may be adequate (e.g., weekly). There also is a routine test pack for ETO where a biological indicator is placed in a plastic syringe with plunger, then placed in the folds of a clean surgical towel, and wrapped. Alternatively, commercially available disposal

test packs that have been shown to be equivalent to the AAMI test pack may be used. The test pack is placed in the center of the sterilizer load⁸¹⁴. Sterilization records (mechanical, chemical, and biological) should be retained for a time period in compliance with standards (e.g., Joint Commission for the Accreditation of Healthcare Facilities requests 3 years) and state and federal regulations.

In Europe, biological monitors are not used routinely to monitor the sterilization process. Instead, release of sterilizer items is based on monitoring the physical conditions of the sterilization process that is termed “parametric release.” Parametric release requires that there is a defined quality system in place at the facility performing the sterilization and that the sterilization process be validated for the items being sterilized. At present in Europe, parametric release is accepted for steam, dry heat, and ionizing radiation processes, as the physical conditions are understood and can be monitored directly⁹⁸⁸. For example, with steam sterilizers the load could be monitored with probes that would yield data on temperature, time, and humidity at representative locations in the chamber and compared to the specifications developed during the validation process.

Periodic infection control rounds to areas using sterilizers to standardize the sterilizer’s use may identify correctable variances in operator competence; documentation of sterilization records, including chemical and biological indicator test results; sterilizer maintenance and wrapping; and load numbering of packs. These rounds also may identify improvement activities to ensure that operators are adhering to established standards⁹⁸⁹.

REUSE OF SINGLE-USE MEDICAL DEVICES

The reuse of single-use medical devices began in the late 1970s. Before this time most devices were considered reusable. Reuse of single-use devices increased as a cost-saving measure. Approximately 20 to 30% of U.S. hospitals reported that they reuse at least one type of single-use device. Reuse of single-use devices involves regulatory, ethical, medical, legal and economic issues and has been extremely controversial for more than two decades⁹⁹⁰. The U.S. public has expressed increasing concern regarding the risk of infection and injury when reusing medical devices intended and labeled for single use. Although some investigators have demonstrated it is safe to reuse disposable medical devices such as cardiac electrode catheters,⁹⁹¹⁻⁹⁹³ additional studies are needed to define the risks⁹⁹⁴ and document the benefits. In August 2000, FDA released a guidance document on single-use devices reprocessed by third parties or hospitals⁹⁹⁵. In this guidance document, FDA states that hospitals or third-party reproducers will be considered “manufacturers” and regulated in the same manner. A reused single-use device will have to comply with the same regulatory requirements of the device when it was originally manufactured. This document presents FDA’s intent to enforce premarket submission requirements within 6 months (February 2001) for class III devices (e.g., cardiovascular intra-aortic balloon pump, transluminal coronary angioplasty catheter); 12 months (August 2001) for class II devices (e.g., blood pressure cuff, bronchoscope biopsy forceps); and 18 months (February 2002) for class I devices (e.g., disposable medical scissors, ophthalmic knife). FDA uses two types of premarket requirements for nonexempt class I and II devices, a 510(k) submission that may have to show that the device is as safe and effective as the same device when new, and a premarket approval application. The 510(k) submission must provide scientific evidence that the device is safe and effective for its intended use. FDA allowed hospitals a year to comply with the nonpremarket requirements (registration and listing, reporting adverse events associated with medical devices, quality system regulations, and proper labeling). The options for hospitals are to stop reprocessing single-use devices, comply with the rule, or outsource to a third-party reproducer. FDA guidance document does not apply to permanently implantable pacemakers, hemodialyzers, opened but unused single-use devices, or healthcare settings other than acute-care hospitals. The reuse of single use medical devices continues to be an evolving area of regulations. For this reason, healthcare workers should refer to FDA for the latest guidance (www.fda.gov)⁹⁹⁶.

CONCLUSION

When properly used, disinfection and sterilization can ensure the safe use of invasive and non-invasive medical devices. However, current disinfection and sterilization guidelines must be strictly followed.

WED-BASED DISINFECTION AND STERILIZATION RESOURCES

Additional information about disinfection and sterilization is available at the following dedicated websites:

Food and Drug Administration, Rockville, Maryland
<http://www.fda.gov/dcrh/ode/germlab.html>

Environmental Protection Agency, Washington, D.C.
<http://www.epa.gov/oppad001/chemregindex.htm>

Centers for Disease Control and Prevention, Atlanta, Georgia
<http://www.cdc.gov/ncidod/dhqp/sterile.html>

University of North Carolina, Chapel Hill, North Carolina
<http://www.disinfectionandsterilization.org>

RECOMMENDATIONS FOR DISINFECTION AND STERILIZATION IN HEALTHCARE FACILITIES

A. Rationale

The ultimate goal of the Recommendations for Disinfection and Sterilization in Health-Care Facilities, 2008, is to reduce rates of health-care–associated infections through appropriate use of both disinfection and sterilization. Each recommendation is categorized according to scientific evidence, theoretical rationale, applicability, and federal regulations. Examples are included in some recommendations to aid the reader; however, these examples are not intended to define the only method of implementing the recommendation. The CDC system for categorizing recommendations is defined in the following (Rankings) section.

B. Rankings

Category IA. Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiologic studies.

Category IB. Strongly recommended for implementation and supported by some experimental, clinical, or epidemiologic studies, and by a strong theoretical rationale.

Category IC. Required by state or federal regulations. Because of state differences, readers should not assume that the absence of an *IC* recommendation implies the absence of state regulations.

Category II. Suggested for implementation and supported by suggestive clinical or epidemiologic studies or by a theoretical rationale.

No recommendation. Unresolved issue. These include practices for which insufficient evidence or no consensus exists regarding efficacy.

C. Recommendations

1. **Occupational Health and Exposure**

- a. Inform each worker of the possible health effects of his or her exposure to infectious agents (e.g., hepatitis B virus [HBV], hepatitis C virus, human immunodeficiency virus [HIV]), and/or chemicals (e.g., EtO, formaldehyde). The information should be consistent with Occupational Safety and Health Administration (OSHA) requirements and identify the areas and tasks in which potential exists for exposure. *Category II, IC*^{214, 320, 959, 997, 998}
- b. Educate health-care workers in the selection and proper use of personal protective equipment (PPE). *Category II, IC*
- c. Ensure that workers wear appropriate PPE to preclude exposure to infectious agents or chemicals through the respiratory system, skin, or mucous membranes of the eyes, nose, or mouth. PPE can include gloves, gowns, masks, and eye protection. The exact type of PPE depends on the infectious or chemical agent and the anticipated duration of exposure. The employer is responsible for making such equipment and training available. *Category II, IC.*^{214, 997-999}
- d. Establish a program for monitoring occupational exposure to regulated chemicals (e.g., formaldehyde, EtO) that adheres to state and federal regulations. *Category II, IC.*^{997, 1000, 1001}
- e. Exclude healthcare workers with weeping dermatitis of hands from direct contact with patient-care equipment. *Category IB.*^{1002, 1003}

2. **Cleaning of Patient-Care Devices**

- a. In hospitals, perform most cleaning, disinfection, and sterilization of patient-care devices in a central processing department in order to more easily control quality. *Category II.*^{454, 836, 959}
- b. Meticulously clean patient-care items with water and detergent, or with water and enzymatic cleaners before high-level disinfection or sterilization procedures. *Category IB.*^{6, 83, 101, 104-106, 124, 179, 424-426, 436, 465, 471, 911-913, 1004}
 - i. Remove visible organic residue (e.g., residue of blood and tissue) and inorganic salts with cleaning. Use cleaning agents that are capable of removing visible organic and inorganic residues. *Category IB.*^{424-426, 466, 468, 469, 471, 908, 910}

- ii. Clean medical devices as soon as practical after use (e.g., at the point of use) because soiled materials become dried onto the instruments. Dried or baked materials on the instrument make the removal process more difficult and the disinfection or sterilization process less effective or ineffective. *Category IB.*^{55, 56, 59, 291, 465, 1005, 1006}
 - c. Perform either manual cleaning (i.e., using friction) or mechanical cleaning (e.g., with ultrasonic cleaners, washer-disinfector, washer-sterilizers). *Category IB.*^{426, 456, 471, 999}
 - d. If using an automatic washer/disinfector, ensure that the unit is used in accordance with the manufacturer's recommendations. *Category IB.*^{7, 133, 155, 725}
 - e. Ensure that the detergents or enzymatic cleaners selected are compatible with the metals and other materials used in medical instruments. Ensure that the rinse step is adequate for removing cleaning residues to levels that will not interfere with subsequent disinfection/sterilization processes. *Category II.*^{836, 1004}
 - f. Inspect equipment surfaces for breaks in integrity that would impair either cleaning or disinfection/sterilization. Discard or repair equipment that no longer functions as intended or cannot be properly cleaned, and disinfected or sterilized. *Category II.*⁸⁸⁸
 - g.
- 3. **Indications for Sterilization, High-Level Disinfection, and Low-Level Disinfection**
 - a. Before use on each patient, sterilize critical medical and surgical devices and instruments that enter normally sterile tissue or the vascular system or through which a sterile body fluid flows (e.g., blood). See recommendation 7g for exceptions. *Category IA.*^{179, 497, 821, 822, 907, 911, 912}
 - b. Provide, at a minimum, high-level disinfection for semicritical patient-care equipment (e.g., gastrointestinal endoscopes, endotracheal tubes, anesthesia breathing circuits, and respiratory therapy equipment) that touches either mucous membranes or nonintact skin. *Category IA.*^{6-8, 17, 20, 99, 101, 108, 113-115, 129, 138, 139, 147, 152-154, 471, 1007}
 - c. Perform low-level disinfection for noncritical patient-care surfaces (e.g., bedrails, over-the-bed table) and equipment (e.g., blood pressure cuff) that touch intact skin (see Recommendation 5g). *Category II.*^{17, 46-48, 50-52, 67, 68, 372, 373, 378, 382, 401}
- 4. **Selection and Use of Low-Level Disinfectants for Noncritical Patient-Care Devices**
 - a. Process noncritical patient-care devices using a disinfectant and the concentration of germicide listed in Table 1. *Category IB.*^{17, 46-48, 50-52, 67, 68, 378, 382, 401}
 - b. Disinfect noncritical medical devices (e.g., blood pressure cuff) with an EPA-registered hospital disinfectant using the label's safety precautions and use directions. Most EPA-registered hospital disinfectants have a label contact time of 10 minutes. However, multiple scientific studies have demonstrated the efficacy of hospital disinfectants against pathogens with a contact time of at least 1 minute. By law, all applicable label instructions on EPA-registered products must be followed. If the user selects exposure conditions that differ from those on the EPA-registered product label, the user assumes liability from any injuries resulting from off-label use and is potentially subject to enforcement action under FIFRA. *Category IB.*^{17, 47, 48, 50, 51, 53-57, 59, 60, 62-64, 355, 378, 382}
 - c. Ensure that, at a minimum, noncritical patient-care devices are disinfected when visibly soiled and on a regular basis (such as after use on each patient or once daily or once weekly). *Category II.*^{378, 380, 1008}
 - d. If dedicated, disposable devices are not available, disinfect noncritical patient-care equipment after using it on a patient who is on contact precautions before using this equipment on another patient. *Category IB.*^{47, 67, 391, 1009}
- 5. **Cleaning and Disinfecting Environmental Surfaces in Healthcare Facilities**
 - a. Clean housekeeping surfaces (e.g., floors, tabletops) on a regular basis, when spills occur, and when these surfaces are visibly soiled. *Category II.*^{23, 378, 380, 382, 1008, 1010}
 - b. Disinfect (or clean) environmental surfaces on a regular basis (e.g., daily, three times per week) and when surfaces are visibly soiled. *Category II.*^{378, 380, 402, 1008}
 - c. Follow manufacturers' instructions for proper use of disinfecting (or detergent) products --- such as recommended use-dilution, material compatibility, storage, shelf-life, and safe use and

- disposal. *Category II.* ^{327, 365, 404}
- d. Clean walls, blinds, and window curtains in patient-care areas when these surfaces are visibly contaminated or soiled. *Category II.* ¹⁰¹¹
 - e. Prepare disinfecting (or detergent) solutions as needed and replace these with fresh solution frequently (e.g., replace floor mopping solution every three patient rooms, change no less often than at 60-minute intervals), according to the facility's policy. *Category IB.* ^{68, 379}
 - f. Decontaminate mop heads and cleaning cloths regularly to prevent contamination (e.g., launder and dry at least daily). *Category II.* ^{68, 402, 403}
 - g. Use a one-step process and an EPA-registered hospital disinfectant designed for housekeeping purposes in patient care areas where 1) uncertainty exists about the nature of the soil on the surfaces (e.g., blood or body fluid contamination versus routine dust or dirt); or 2) uncertainty exists about the presence of multidrug resistant organisms on such surfaces. See 5n for recommendations requiring cleaning and disinfecting blood-contaminated surfaces. *Category II.* ^{23, 47, 48, 51, 214, 378, 379, 382, 416, 1012}
 - h. Detergent and water are adequate for cleaning surfaces in nonpatient-care areas (e.g., administrative offices). *Category II.* ²³
 - i. Do not use high-level disinfectants/liquid chemical sterilants for disinfection of non-critical surfaces. *Category IB.* ^{23, 69, 318}
 - j. Wet-dust horizontal surfaces regularly (e.g., daily, three times per week) using clean cloths moistened with an EPA-registered hospital disinfectant (or detergent). Prepare the disinfectant (or detergent) as recommended by the manufacturer. *Category II.* ^{68, 378, 380, 402, 403, 1008}
 - k. Disinfect noncritical surfaces with an EPA-registered hospital disinfectant according to the label's safety precautions and use directions. Most EPA-registered hospital disinfectants have a label contact time of 10 minutes. However, many scientific studies have demonstrated the efficacy of hospital disinfectants against pathogens with a contact time of at least 1 minute. By law, the user must follow all applicable label instructions on EPA-registered products. If the user selects exposure conditions that differ from those on the EPA-registered product label, the user assumes liability for any injuries resulting from off-label use and is potentially subject to enforcement action under FIFRA. *Category II, IC.* ^{17, 47, 48, 50, 51, 53-57, 59, 60, 62-64, 355, 378, 382}
 - l. Do not use disinfectants to clean infant bassinets and incubators while these items are occupied. If disinfectants (e.g., phenolics) are used for the terminal cleaning of infant bassinets and incubators, thoroughly rinse the surfaces of these items with water and dry them before these items are reused. *Category IB.* ^{17, 739, 740}
 - m. Promptly clean and decontaminate spills of blood and other potentially infectious materials. Discard blood-contaminated items in compliance with federal regulations. *Category IB, IC.* ²¹⁴
 - n. For site decontamination of spills of blood or other potentially infectious materials (OPIM), implement the following procedures. Use protective gloves and other PPE (e.g., when sharps are involved use forceps to pick up sharps, and discard these items in a puncture-resistant container) appropriate for this task. Disinfect areas contaminated with blood spills using an EPA-registered tuberculocidal agent, a registered germicide on the EPA Lists D and E (i.e., products with specific label claims for HIV or HBV or freshly diluted hypochlorite solution. *Category II, IC.* ^{214, 215, 557, 1013} If sodium hypochlorite solutions are selected use a 1:100 dilution (e.g., 1:100 dilution of a 5.25-6.15% sodium hypochlorite provides 525-615 ppm available chlorine) to decontaminate nonporous surfaces after a small spill (e.g., <10 mL) of either blood or OPIM. If a spill involves large amounts (e.g., >10 mL) of blood or OPIM, or involves a culture spill in the laboratory, use a 1:10 dilution for the first application of hypochlorite solution before cleaning in order to reduce the risk of infection during the cleaning process in the event of a sharp injury. Follow this decontamination process with a terminal disinfection, using a 1:100 dilution of sodium hypochlorite. *Category IB, IC.* ^{63, 215, 557}
 - o. If the spill contains large amounts of blood or body fluids, clean the visible matter with disposable absorbent material, and discard the contaminated materials in appropriate, labeled containment. *Category II, IC.* ^{44, 214}
 - p. Use protective gloves and other PPE appropriate for this task. *Category II, IC.* ^{44, 214}

- q. In units with high rates of endemic *Clostridium difficile* infection or in an outbreak setting, use dilute solutions of 5.25%–6.15% sodium hypochlorite (e.g., 1:10 dilution of household bleach) for routine environmental disinfection. Currently, no products are EPA-registered specifically for inactivating *C. difficile* spores. *Category II.*²⁵⁷⁻²⁵⁹
- r. If chlorine solution is not prepared fresh daily, it can be stored at room temperature for up to 30 days in a capped, opaque plastic bottle with a 50% reduction in chlorine concentration after 30 days of storage (e.g., 1000 ppm chlorine [approximately a 1:50 dilution] at day 0 decreases to 500 ppm chlorine by day 30). *Category IB.*^{327, 1014}
- s. An EPA-registered sodium hypochlorite product is preferred, but if such products are not available, generic versions of sodium hypochlorite solutions (e.g., household chlorine bleach) can be used. *Category II.*⁴⁴

6. Disinfectant Fogging

- a. Do not perform disinfectant fogging for routine purposes in patient-care areas. *Category II.*^{23, 228}

7. High-Level Disinfection of Endoscopes

- a. To detect damaged endoscopes, test each flexible endoscope for leaks as part of each reprocessing cycle. Remove from clinical use any instrument that fails the leak test, and repair this instrument. *Category II.*^{113, 115, 116}
- b. Immediately after use, meticulously clean the endoscope with an enzymatic cleaner that is compatible with the endoscope. Cleaning is necessary before both automated and manual disinfection. *Category IA.*^{83, 101, 104-106, 113, 115, 116, 124, 126, 456, 465, 466, 471, 1015}
- c. Disconnect and disassemble endoscopic components (e.g., suction valves) as completely as possible and completely immerse all components in the enzymatic cleaner. Steam sterilize these components if they are heat stable. *Category IB.*^{115, 116, 139, 465, 466}
- d. Flush and brush all accessible channels to remove all organic (e.g., blood, tissue) and other residue. Clean the external surfaces and accessories of the devices by using a soft cloth or sponge or brushes. Continue brushing until no debris appears on the brush. *Category IA.*^{6, 17, 108, 113, 115, 116, 137, 145, 147, 725, 856, 903}
- e. Use cleaning brushes appropriate for the size of the endoscope channel or port (e.g., bristles should contact surfaces). Cleaning items (e.g., brushes, cloth) should be disposable or, if they are not disposable, they should be thoroughly cleaned and either high-level disinfected or sterilized after each use. *Category II.*^{113, 115, 116, 1016}
- f. Discard enzymatic cleaners (or detergents) after each use because they are not microbicidal and, therefore, will not retard microbial growth. *Category IB.*^{38, 113, 115, 116, 466}
- g. Process endoscopes (e.g., arthroscopes, cystoscope, laparoscopes) that pass through normally sterile tissues using a sterilization procedure before each use; if this is not feasible, provide at least high-level disinfection. High-level disinfection of arthroscopes, laparoscopes, and cystoscopes should be followed by a sterile water rinse. *Category IB.*^{1, 17, 31, 32, 35, 89, 90, 113, 554}
- h. Phase out endoscopes that are critical items (e.g., arthroscopes, laparoscopes) but cannot be steam sterilized. Replace these endoscopes with steam sterilizable instruments when feasible. *Category II.*
- i. Mechanically clean reusable accessories inserted into endoscopes (e.g., biopsy forceps or other cutting instruments) that break the mucosal barrier (e.g., ultrasonically clean biopsy forceps) and then sterilize these items between each patient. *Category IA.*^{1, 6, 8, 17, 108, 113, 115, 116, 138, 145, 147, 153, 278}
- j. Use ultrasonic cleaning of reusable endoscopic accessories to remove soil and organic material from hard-to-clean areas. *Category II.*^{116, 145, 148}
- k. Process endoscopes and accessories that contact mucous membranes as semicritical items, and use at least high-level disinfection after use on each patient. *Category IA.*^{1, 6, 8, 17, 108, 113, 115, 116, 129, 138, 145-148, 152-154, 278}
- l. Use an FDA-cleared sterilant or high-level disinfectant for sterilization or high-level disinfection (Table 1). *Category IA.*^{1, 6-8, 17, 85, 108, 113, 115, 116, 147}
- m. After cleaning, use formulations containing glutaraldehyde, glutaraldehyde with phenol/phenate,

- ortho-phthalaldehyde, hydrogen peroxide, and both hydrogen peroxide and peracetic acid to achieve high-level disinfection followed by rinsing and drying (see Table 1 for recommended concentrations). *Category IB.* ^{1, 6-8, 17, 38, 85, 108, 113, 145-148}
- n. Extend exposure times beyond the minimum effective time for disinfecting semicritical patient-care equipment cautiously and conservatively because extended exposure to a high-level disinfectant is more likely to damage delicate and intricate instruments such as flexible endoscopes. The exposure times vary among the Food and Drug Administration (FDA)-cleared high-level disinfectants (Table 2). *Category IB.* ^{17, 69, 73, 76, 78, 83}
 - o. Federal regulations are to follow the FDA-cleared label claim for high-level disinfectants. The FDA-cleared labels for high-level disinfection with >2% glutaraldehyde at 25°C range from 20-90 minutes, depending upon the product based on three tier testing which includes AOAC sporicidal tests, simulated use testing with mycobacteria and in-use testing. *Category IC.*
 - p. Several scientific studies and professional organizations support the efficacy of >2% glutaraldehyde for 20 minutes at 20°C; that efficacy assumes adequate cleaning prior to disinfection, whereas the FDA-cleared label claim incorporates an added margin of safety to accommodate possible lapses in cleaning practices. Facilities that have chosen to apply the 20 minute duration at 20°C have done so based on the IA recommendation in the July 2003 SHEA position paper, "Multi-society Guideline for Reprocessing Flexible Gastrointestinal Endoscopes" ^{12, 17, 19, 26, 27, 49, 55, 57, 58, 60, 73, 76, 79-81, 83-85, 93, 94, 104-106, 110, 111, 115-121, 124, 125, 233, 235, 236, 243, 265, 266, 609}
 - q. When using FDA-cleared high-level disinfectants, use manufacturers' recommended exposure conditions. Certain products may require a shorter exposure time (e.g., 0.55% ortho-phthalaldehyde for 12 minutes at 20°C, 7.35% hydrogen peroxide plus 0.23% peracetic acid for 15 minutes at 20°C) than glutaraldehyde at room temperature because of their rapid inactivation of mycobacteria or reduced exposure time because of increased mycobactericidal activity at elevated temperature (e.g., 2.5% glutaraldehyde at 5 minutes at 35°C). *Category IB.* ^{83, 100, 689, 693, 694, 700}
 - r. Select a disinfectant or chemical sterilant that is compatible with the device that is being reprocessed. Avoid using reprocessing chemicals on an endoscope if the endoscope manufacturer warns against using these chemicals because of functional damage (with or without cosmetic damage). *Category IB.* ^{69, 113, 116}
 - s. Completely immerse the endoscope in the high-level disinfectant, and ensure all channels are perfused. As soon as is feasible, phase out nonimmersible endoscopes. *Category IB.* ^{108, 113-116, 137, 725, 856, 882}
 - t. After high-level disinfection, rinse endoscopes and flush channels with sterile water, filtered water, or tapwater to prevent adverse effects on patients associated with disinfectant retained in the endoscope (e.g., disinfectant induced colitis). Follow this water rinse with a rinse with 70% - 90% ethyl or isopropyl alcohol. *Category IB.* ^{17, 31-35, 38, 39, 108, 113, 115, 116, 134, 145-148, 620-622, 624-630, 1017}
 - u. After flushing all channels with alcohol, purge the channels using forced air to reduce the likelihood of contamination of the endoscope by waterborne pathogens and to facilitate drying. *Category IB.* ^{39, 113, 115, 116, 145, 147}
 - v. Hang endoscopes in a vertical position to facilitate drying. *Category II.* ^{17, 108, 113, 115, 116, 145, 815}
 - w. Store endoscopes in a manner that will protect them from damage or contamination. *Category II.* ^{17, 108, 113, 115, 116, 145}
 - x. Sterilize or high-level disinfect both the water bottle used to provide intraprocedural flush solution and its connecting tube at least once daily. After sterilizing or high-level disinfecting the water bottle, fill it with sterile water. *Category IB.* ^{10, 31-35, 113, 116, 1017}
 - y. Maintain a log for each procedure and record the following: patient's name and medical record number (if available), procedure, date, endoscopist, system used to reprocess the endoscope (if more than one system could be used in the reprocessing area), and serial number or other identifier of the endoscope used. *Category II.* ^{108, 113, 115, 116}
 - z. Design facilities where endoscopes are used and disinfected to provide a safe environment for healthcare workers and patients. Use air-exchange equipment (e.g., the ventilation system, out-exhaust ducts) to minimize exposure of all persons to potentially toxic vapors (e.g.,

glutaraldehyde vapor). Do not exceed the allowable limits of the vapor concentration of the chemical sterilant or high-level disinfectant (e.g., those of ACGIH and OSHA). *Category IB, IC.* 116, 145, 318, 322, 577, 652

- aa. Routinely test the liquid sterilant/high-level disinfectant to ensure minimal effective concentration of the active ingredient. Check the solution each day of use (or more frequently) using the appropriate chemical indicator (e.g., glutaraldehyde chemical indicator to test minimal effective concentration of glutaraldehyde) and document the results of this testing. Discard the solution if the chemical indicator shows the concentration is less than the minimum effective concentration. Do not use the liquid sterilant/high-level disinfectant beyond the reuse-life recommended by the manufacturer (e.g., 14 days for *ortho*-phthalaldehyde). *Category IA.* 76, 108, 113, 115, 116, 608, 609
 - bb. Provide personnel assigned to reprocess endoscopes with device-specific reprocessing instructions to ensure proper cleaning and high-level disinfection or sterilization. Require competency testing on a regular basis (e.g., beginning of employment, annually) of all personnel who reprocess endoscopes. *Category IA.* 6-8, 108, 113, 115, 116, 145, 148, 155
 - cc. Educate all personnel who use chemicals about the possible biologic, chemical, and environmental hazards of performing procedures that require disinfectants. *Category IB, IC.* 116, 997, 998, 1018, 1019
 - dd. Make PPE (e.g., gloves, gowns, eyewear, face mask or shields, respiratory protection devices) available and use these items appropriately to protect workers from exposure to both chemicals and microorganisms (e.g., HBV). *Category IB, IC.* 115, 116, 214, 961, 997, 998, 1020, 1021
 - ee. If using an automated endoscope reprocessor (AER), place the endoscope in the reprocessor and attach all channel connectors according to the AER manufacturer's instructions to ensure exposure of all internal surfaces to the high-level disinfectant/chemical sterilant. *Category IB.* 7, 8, 115, 116, 155, 725, 903
 - ff. If using an AER, ensure the endoscope can be effectively reprocessed in the AER. Also, ensure any required manual cleaning/disinfecting steps are performed (e.g., elevator wire channel of duodenoscopes might not be effectively disinfected by most AERs). *Category IB.* 7, 8, 115, 116, 155, 725
 - gg. Review the FDA advisories and the scientific literature for reports of deficiencies that can lead to infection because design flaws and improper operation and practices have compromised the effectiveness of AERs. *Category II.* 7, 98, 133, 134, 155, 725
 - hh. Develop protocols to ensure that users can readily identify an endoscope that has been properly processed and is ready for patient use. *Category II.*
 - ii. Do not use the carrying case designed to transport clean and reprocessed endoscopes outside of the healthcare environment to store an endoscope or to transport the instrument within the healthcare environment. *Category II.*
 - jj. No recommendation is made about routinely performing microbiologic testing of either endoscopes or rinse water for quality assurance purposes. *Unresolved Issue.* 116, 164
 - kk. If environmental microbiologic testing is conducted, use standard microbiologic techniques. *Category II.* 23, 116, 157, 161, 167
 - ll. If a cluster of endoscopy-related infections occurs, investigate potential routes of transmission (e.g., person-to-person, common source) and reservoirs. *Category IA.* 8, 1022
 - mm. Report outbreaks of endoscope-related infections to persons responsible for institutional infection control and risk management and to FDA. *Category IB.* 6, 7, 113, 116, 1023 Notify the local and the state health departments, CDC, and the manufacturer(s). *Category II.*
 - nn. No recommendation is made regarding the reprocessing of an endoscope again immediately before use if that endoscope has been processed after use according to the recommendations in this guideline. *Unresolved issue.* 157
 - oo. Compare the reprocessing instructions provided by both the endoscope's and the AER's manufacturer's instructions and resolve any conflicting recommendations. *Category IB.* 116, 155
- 8. Management of Equipment and Surfaces in Dentistry**
- a. Dental instruments that penetrate soft tissue or bone (e.g., extraction forceps, scalpel blades, bone chisels, periodontal scalers, and surgical burs) are classified as critical and should be

sterilized after each use or discarded. In addition, after each use, sterilize dental instruments that are not intended to penetrate oral soft tissue or bone (e.g., amalgam condensers, air-water syringes) but that might contact oral tissues and are heat-tolerant, although classified as semicritical. Clean and, at a minimum, high-level disinfect heat-sensitive semicritical items.

Category IA. ^{43, 209-211}

- b. Noncritical clinical contact surfaces, such as uncovered operatory surfaces (e.g., countertops, switches, light handles), should be barrier-protected or disinfected between patients with an intermediate-disinfectant (i.e., EPA-registered hospital disinfectant with a tuberculocidal claim) or low-level disinfectant (i.e., EPA-registered hospital disinfectant with HIV and HBV claim).

Category IB. ^{43, 209-211}

- c. Barrier protective coverings can be used for noncritical clinical contact surfaces that are touched frequently with gloved hands during the delivery of patient care, that are likely to become contaminated with blood or body substances, or that are difficult to clean. Change these coverings when they are visibly soiled, when they become damaged, and on a routine basis (e.g., between patients). Disinfect protected surfaces at the end of the day or if visibly soiled. *Category II.* ^{43, 210}

9. Processing Patient-Care Equipment Contaminated with Bloodborne Pathogens (HBV, Hepatitis C Virus, HIV), Antibiotic-Resistant Bacteria (e.g., Vancomycin-Resistant Enterococci, Methicillin-Resistant Staphylococcus aureus, Multidrug Resistant Tuberculosis), or Emerging Pathogens (e.g., Cryptosporidium, Helicobacter pylori, Escherichia coli O157:H7, Clostridium difficile, Mycobacterium tuberculosis, Severe Acute Respiratory Syndrome Coronavirus), or Bioterrorist Agents

- a. Use standard sterilization and disinfection procedures for patient-care equipment (as recommended in this guideline), because these procedures are adequate to sterilize or disinfect instruments or devices contaminated with blood or other body fluids from persons infected with bloodborne pathogens or emerging pathogens, with the exception of prions. No changes in these procedures for cleaning, disinfecting, or sterilizing are necessary for removing bloodborne and emerging pathogens other than prions. *Category IA.* ^{22, 53, 60-62, 73, 79-81, 105, 118-121, 125, 126, 221, 224-234, 236, 244, 265, 266, 271-273, 279, 282, 283, 354-357, 666}

10. Disinfection Strategies for Other Semicritical Devices

- a. Even if probe covers have been used, clean and high-level disinfect other semicritical devices such as rectal probes, vaginal probes, and cryosurgical probes with a product that is not toxic to staff, patients, probes, and retrieved germ cells (if applicable). Use a high-level disinfectant at the FDA-cleared exposure time. (See Recommendations 7o and 11e for exceptions.) *Category IB.* ^{6-8, 17, 69}
- b. When probe covers are available, use a probe cover or condom to reduce the level of microbial contamination. *Category II.* ¹⁹⁷⁻²⁰¹ Do not use a lower category of disinfection or cease to follow the appropriate disinfectant recommendations when using probe covers because these sheaths and condoms can fail. *Category IB.* ¹⁹⁷⁻²⁰¹
- c. After high-level disinfection, rinse all items. Use sterile water, filtered water or tapwater followed by an alcohol rinse for semicritical equipment that will have contact with mucous membranes of the upper respiratory tract (e.g., nose, pharynx, esophagus). *Category II.* ^{10, 31-35, 1017}
- d. There is no recommendation to use sterile or filtered water rather than tapwater for rinsing semicritical equipment that contact the mucous membranes of the rectum (e.g., rectal probes, anoscope) or vagina (e.g., vaginal probes). *Unresolved issue.* ¹¹
- e. Wipe clean tonometer tips and then disinfect them by immersing for 5-10 minutes in either 5000 ppm chlorine or 70% ethyl alcohol. None of these listed disinfectant products are FDA-cleared high-level disinfectants. *Category II.* ^{49, 95, 185, 188, 293}

11. Disinfection by Healthcare Personnel in Ambulatory Care and Home Care

- a. Follow the same classification scheme described above (i.e., that critical devices require sterilization, semicritical devices require high-level disinfection, and noncritical equipment

requires low-level disinfection) in the ambulatory-care (outpatient medical/surgical facilities) setting because risk for infection in this setting is similar to that in the hospital setting (see Table 1). *Category IB.*^{6-8, 17, 330}

- b. When performing care in the home, clean and disinfect reusable objects that touch mucous membranes (e.g., tracheostomy tubes) by immersing these objects in a 1:50 dilution of 5.25%-6.15% sodium hypochlorite (household bleach) (3 minutes), 70% isopropyl alcohol (5 minutes), or 3% hydrogen peroxide (30 minutes) because the home environment is, in most instances, safer than either hospital or ambulatory care settings because person-to-person transmission is less likely. *Category II.*^{327, 328, 330, 331}
- c. Clean noncritical items that would not be shared between patients (e.g., crutches, blood pressure cuffs) in the home setting with a detergent or commercial household disinfectant. *Category II.*^{53, 330}

12. **Microbial Contamination of Disinfectants**

- a. Institute the following control measures to reduce the occurrence of contaminated disinfectants: 1) prepare the disinfectant correctly to achieve the manufacturer's recommended use-dilution; and 2) prevent common sources of extrinsic contamination of germicides (e.g., container contamination or surface contamination of the healthcare environment where the germicide are prepared and/or used). *Category IB.*^{404, 406, 1024}

13. **Flash Sterilization**

- a. Do not flash sterilize implanted surgical devices unless doing so is unavoidable. *Category IB.*^{849, 850}
- b. Do not use flash sterilization for convenience, as an alternative to purchasing additional instrument sets, or to save time. *Category II.*^{817, 962}
- c. When using flash sterilization, make sure the following parameters are met: 1) clean the item before placing it in the sterilizing container (that are FDA cleared for use with flash sterilization) or tray; 2) prevent exogenous contamination of the item during transport from the sterilizer to the patient; and 3) monitor sterilizer function with mechanical, chemical, and biologic monitors. *Category IB.*^{812, 819, 846, 847, 962}
- d. Do not use packaging materials and containers in flash sterilization cycles unless the sterilizer and the packaging material/container are designed for this use. *Category IB.*^{812, 819, 1025}
- e. When necessary, use flash sterilization for patient-care items that will be used immediately (e.g., to reprocess an inadvertently dropped instrument). *Category IB.*^{812, 817, 819, 845}
- f. When necessary, use flash sterilization for processing patient-care items that cannot be packaged, sterilized, and stored before use. *Category IB.*^{812, 819}

14. **Methods of Sterilization**

- a. Steam is the preferred method for sterilizing critical medical and surgical instruments that are not damaged by heat, steam, pressure, or moisture. *Category IA.*^{181, 271, 425, 426, 827, 841, 1026, 1027}
- b. Cool steam- or heat-sterilized items before they are handled or used in the operative setting. *Category IB.*⁸⁵⁰
- c. Follow the sterilization times, temperatures, and other operating parameters (e.g., gas concentration, humidity) recommended by the manufacturers of the instruments, the sterilizer, and the container or wrap used, and that are consistent with guidelines published by government agencies and professional organizations. *Category IB.*^{811-814, 819, 825, 827, 841, 1026-1028}
- d. Use low-temperature sterilization technologies (e.g., EtO, hydrogen peroxide gas plasma) for reprocessing critical patient-care equipment that is heat or moisture sensitive. *Category IA.*^{469, 721, 825, 856, 858, 878, 879, 881, 882, 890, 891, 1027}
- e. Completely aerate surgical and medical items that have been sterilized in the EtO sterilizer (e.g., polyvinylchloride tubing requires 12 hours at 50°C, 8 hours at 60°C) before using these items in patient care. *Category IB.*⁸¹⁴
- f. Sterilization using the peracetic acid immersion system can be used to sterilize heat-sensitive

immersible medical and surgical items. *Category IB.*^{90, 717-719, 721-724}

- g. Critical items that have been sterilized by the peracetic acid immersion process must be used immediately (i.e., items are not completely protected from contamination, making long-term storage unacceptable). *Category II.*^{817, 825}
- h. Dry-heat sterilization (e.g., 340°F for 60 minutes) can be used to sterilize items (e.g., powders, oils) that can sustain high temperatures. *Category IB.*^{815, 827}
- i. Comply with the sterilizer manufacturer's instructions regarding the sterilizer cycle parameters (e.g., time, temperature, concentration). *Category IB.*^{155, 725, 811-814, 819}
- j. Because narrow-lumen devices provide a challenge to all low-temperature sterilization technologies and direct contact is necessary for the sterilant to be effective, ensure that the sterilant has direct contact with contaminated surfaces (e.g., scopes processed in peracetic acid must be connected to channel irrigators). *Category IB.*^{137, 725, 825, 856, 890, 891, 1029}

15. **Packaging**

- a. Ensure that packaging materials are compatible with the sterilization process and have received FDA 510[k] clearance. *Category IB.*^{811-814, 819, 966}
- b. Ensure that packaging is sufficiently strong to resist punctures and tears to provide a barrier to microorganisms and moisture. *Category IB.*^{454, 811-814, 819, 966}

16. **Monitoring of Sterilizers**

- a. Use mechanical, chemical, and biologic monitors to ensure the effectiveness of the sterilization process. *Category IB.*^{811-815, 819, 846, 847, 975-977}
- b. Monitor each load with mechanical (e.g., time, temperature, pressure) and chemical (internal and external) indicators. If the internal chemical indicator is visible, an external indicator is not needed. *Category II.*^{811-815, 819, 846, 847, 975-977, 980}
- c. Do not use processed items if the mechanical (e.g., time, temperature, pressure) or chemical (internal and/or external) indicators suggest inadequate processing. *Category IB.*^{811-814, 819}
- d. Use biologic indicators to monitor the effectiveness of sterilizers at least weekly with an FDA-cleared commercial preparation of spores (e.g., *Geobacillus stearothermophilus* for steam) intended specifically for the type and cycle parameters of the sterilizer. *Category IB.*^{1, 811, 813-815, 819, 846, 847, 976, 977}
- e. After a single positive biologic indicator used with a method other than steam sterilization, treat as nonsterile all items that have been processed in that sterilizer, dating from the sterilization cycle having the last negative biologic indicator to the next cycle showing satisfactory biologic indicator results. These nonsterile items should be retrieved if possible and reprocessed. *Category II.*¹
- f. After a positive biologic indicator with steam sterilization, objects other than implantable objects do not need to be recalled because of a single positive spore test unless the sterilizer or the sterilization procedure is defective as determined by maintenance personnel or inappropriate cycle settings. If additional spore tests remain positive, consider the items nonsterile and recall and reprocess the items from the implicated load(s). *Category II.*¹
- g. Use biologic indicators for every load containing implantable items and quarantine items, whenever possible, until the biologic indicator is negative. *Category IB.*^{811-814, 819}

17. **Load Configuration.**

- a. Place items correctly and loosely into the basket, shelf, or cart of the sterilizer so as not to impede the penetration of the sterilant. *Category IB.*^{445, 454, 811, 813, 819, 836}

18. **Storage of Sterile Items**

- a. Ensure the sterile storage area is a well-ventilated area that provides protection against dust, moisture, insects, and temperature and humidity extremes. *Category II.*^{454, 819, 836, 969}
- b. Store sterile items so the packaging is not compromised (e.g., punctured, bent). *Category II.*^{454, 816, 819, 968, 969, 1030}

- c. Label sterilized items with a load number that indicates the sterilizer used, the cycle or load number, the date of sterilization, and, if applicable, the expiration date. *Category IB.*^{811, 812, 814, 816, 819}
- d. The shelf life of a packaged sterile item depends on the quality of the wrapper, the storage conditions, the conditions during transport, the amount of handling, and other events (moisture) that compromise the integrity of the package. If event-related storage of sterile items is used, then packaged sterile items can be used indefinitely unless the packaging is compromised (see f and g below). *Category IB.*^{816, 819, 836, 968, 973, 1030, 1031}
- e. Evaluate packages before use for loss of integrity (e.g., torn, wet, punctured). The pack can be used unless the integrity of the packaging is compromised. *Category II.*^{819, 968}
- f. If the integrity of the packaging is compromised (e.g., torn, wet, or punctured), repack and reprocess the pack before use. *Category II.*^{819, 1032}
- g. If time-related storage of sterile items is used, label the pack at the time of sterilization with an expiration date. Once this date expires, reprocess the pack. *Category II.*^{819, 968}

19. Quality Control

- a. Provide comprehensive and intensive training for all staff assigned to reprocess semicritical and critical medical/surgical instruments to ensure they understand the importance of reprocessing these instruments. To achieve and maintain competency, train each member of the staff that reprocesses semicritical and/or critical instruments as follows: 1) provide hands-on training according to the institutional policy for reprocessing critical and semicritical devices; 2) supervise all work until competency is documented for each reprocessing task; 3) conduct competency testing at beginning of employment and regularly thereafter (e.g., annually); and 4) review the written reprocessing instructions regularly to ensure they comply with the scientific literature and the manufacturers' instructions. *Category IB.*^{6-8, 108, 114, 129, 155, 725, 813, 819}
- b. Compare the reprocessing instructions (e.g., for the appropriate use of endoscope connectors, the capping/noncapping of specific lumens) provided by the instrument manufacturer and the sterilizer manufacturer and resolve any conflicting recommendations by communicating with both manufacturers. *Category IB.*^{155, 725}
- c. Conduct infection control rounds periodically (e.g., annually) in high-risk reprocessing areas (e.g., the Gastroenterology Clinic, Central Processing); ensure reprocessing instructions are current and accurate and are correctly implemented. Document all deviations from policy. All stakeholders should identify what corrective actions will be implemented. *Category IB.*^{6-8, 129}
- d. Include the following in a quality control program for sterilized items: a sterilizer maintenance contract with records of service; a system of process monitoring; air-removal testing for prevacuum steam sterilizers; visual inspection of packaging materials; and traceability of load contents. *Category II*^{811-814, 819}
- e. For each sterilization cycle, record the type of sterilizer and cycle used; the load identification number; the load contents; the exposure parameters (e.g., time and temperature); the operator's name or initials; and the results of mechanical, chemical, and biological monitoring. *Category II*^{811-814, 819}
- f. Retain sterilization records (mechanical, chemical, and biological) for a time period that complies with standards (e.g., 3 years), statutes of limitations, and state and federal regulations. *Category II, IC.*¹⁰³³
- g. Prepare and package items to be sterilized so that sterility can be achieved and maintained to the point of use. Consult the Association for the Advancement of Medical Instrumentation or the manufacturers of surgical instruments, sterilizers, and container systems for guidelines for the density of wrapped packages. *Category II.*^{811-814, 819}
- h. Periodically review policies and procedures for sterilization. *Category II.*¹⁰³³
- i. Perform preventive maintenance on sterilizers by qualified personnel who are guided by the manufacturer's instruction. *Category II.*^{811-814, 819}

20. Reuse of Single-Use Medical Devices

- a. Adhere to the FDA enforcement document for single-use devices reprocessed by hospitals. FDA considers the hospital that reprocesses a single-use device as the manufacturer of the device and regulates the hospital using the same standards by which it regulates the original equipment manufacturer. *Category II, IC.*⁹⁹⁵

PERFORMANCE INDICATORS

1. Monitor adherence to high-level disinfection and/or sterilization guidelines for endoscopes on a regular basis. This monitoring should include ensuring the proper training of persons performing reprocessing and their adherence to all endoscope reprocessing steps, as demonstrated by competency testing at commencement of employment and annually.
2. Develop a mechanism for the occupational health service to report all adverse health events potentially resulting from exposure to disinfectants and sterilants; review such exposures; and implement engineering, work practice, and PPE to prevent future exposures.
3. Monitor possible sterilization failures that resulted in instrument recall. Assess whether additional training of personnel or equipment maintenance is required.

ACKNOWLEDGEMENTS

The authors gratefully acknowledge Eva P. Clontz, M.S., for her assistance in referencing this guideline. The Healthcare Infection Control Practices Advisory Committee thanks the following experts for reviewing a draft of this guideline: Martin S. Favero, Ph.D., Syed A. Sattar, Ph.D., A. Denver Russell, D.Sc., and Martin Exner, M.D. The opinions of the reviewers might not be reflected in all the recommendations contained in this document.

GLOSSARY

Action level: concentration of a regulated substance (e.g., ethylene oxide, formaldehyde) within the employee breathing zone, above which OSHA requirements apply.

Activation of a sterilant: process of mixing the contents of a chemical sterilant that come in two containers (small vial with the activator solution; container of the chemical) Keeping the two chemicals separate until use extends the shelf life of the chemicals.

Aeration: method by which ethylene oxide (EtO) is removed from EtO-sterilized items by warm air circulation in an enclosed cabinet specifically designed for this purpose.

Antimicrobial agent: any agent that kills or suppresses the growth of microorganisms.

Antiseptic: substance that prevents or arrests the growth or action of microorganisms by inhibiting their activity or by destroying them. The term is used especially for preparations applied topically to living tissue.

Asepsis: prevention of contact with microorganisms.

Autoclave: device that sterilizes instruments or other objects using steam under pressure. The length of time required for sterilization depends on temperature, vacuum, and pressure.

Bacterial count: method of estimating the number of bacteria per unit sample. The term also refers to the estimated number of bacteria per unit sample, usually expressed as number of colony-forming units.

Bactericide: agent that kills bacteria.

Bioburden: number and types of viable microorganisms with which an item is contaminated; also called *bioload* or *microbial load*.

Biofilm: accumulated mass of bacteria and extracellular material that is tightly adhered to a surface and cannot be easily removed.

Biologic indicator: device for monitoring the sterilization process. The device consists of a standardized, viable population of microorganisms (usually bacterial spores) known to be resistant to the sterilization process being monitored. Biologic indicators are intended to demonstrate whether conditions were adequate to achieve sterilization. A negative biologic indicator does not prove that all items in the load are sterile or that they were all exposed to adequate sterilization conditions.

Bleach: Household bleach (5.25% or 6.00%–6.15% sodium hypochlorite depending on manufacturer) usually diluted in water at 1:10 or 1:100. Approximate dilutions are 1.5 cups of bleach in a gallon of water for a 1:10 dilution (~6,000 ppm) and 0.25 cup of bleach in a gallon of water for a 1:100 dilution (~600 ppm). Sodium hypochlorite products that make pesticidal claims, such as sanitization or disinfection, must be registered by EPA and be labeled with an EPA Registration Number.

Bleach Solution	Dilution	Chlorine (ppm)
5.25-6.15%	None	52,500-61,500
	1:10	5,250-6,150
	1:100	525-615
	1:1000	53-62

Bowie-Dick test: diagnostic test of a sterilizer's ability to remove air from the chamber of a prevacuum steam sterilizer. The air-removal or Bowie-Dick test is not a test for sterilization.

Ceiling limit: concentration of an airborne chemical contaminant that should not be exceeded during any part of the workday. If instantaneous monitoring is not feasible, the ceiling must be assessed as a 15-minute time-weighted average exposure.

Centigrade or Celsius: a temperature scale (0°C = freezing point of water; 100°C = boiling point of water at sea level). Equivalents mentioned in the guideline are as follows: $20^{\circ}\text{C} = 68^{\circ}\text{F}$; $25^{\circ}\text{C} = 77^{\circ}\text{F}$; $121^{\circ}\text{C} = 250^{\circ}\text{F}$; $132^{\circ}\text{C} = 270^{\circ}\text{F}$; $134^{\circ}\text{C} = 273^{\circ}\text{F}$. For other temperatures the formula is: $F^{\circ} = (C^{\circ} \times 9/5) + 32$ or $C^{\circ} = (F^{\circ} - 32) \times 5/9$.

Central processing or Central service department: the department within a health-care facility that processes, issues, and controls professional supplies and equipment, both sterile and nonsterile, for some or all patient-care areas of the facility.

Challenge test pack: pack used in installation, qualification, and ongoing quality assurance testing of health-care facility sterilizers.

Chemical indicator: device for monitoring a sterilization process. The device is designed to respond with a characteristic chemical or physical change to one or more of the physical conditions within the sterilizing chamber. Chemical indicators are intended to detect potential sterilization failures that could result from incorrect packaging, incorrect loading of the sterilizer, or malfunctions of the sterilizer. The "pass" response of a chemical indicator does not prove the item accompanied by the indicator is necessarily sterile. The Association for the Advancement of Medical Instrumentation has defined five classes of chemical indicators: Class 1 (process indicator); Class 2 (Bowie-Dick test indicator); Class 3 (single-parameter indicator); Class 4 (multi-parameter indicator); and Class 5 (integrating indicator).

Contact time: time a disinfectant is in direct contact with the surface or item to be disinfected. For surface disinfection, this period is framed by the application to the surface until complete drying has occurred.

Container system, rigid container: sterilization containment device designed to hold medical devices for sterilization, storage, transportation, and aseptic presentation of contents.

Contaminated: state of having actual or potential contact with microorganisms. As used in health care, the term generally refers to the presence of microorganisms that could produce disease or infection.

Control, positive: biologic indicator, from the same lot as a test biologic indicator, that is left unexposed to the sterilization cycle and then incubated to verify the viability of the test biologic indicator.

Cleaning: removal, usually with detergent and water or enzyme cleaner and water, of adherent visible soil, blood, protein substances, microorganisms and other debris from the surfaces, crevices, serrations, joints, and lumens of instruments, devices, and equipment by a manual or mechanical process that prepares the items for safe handling and/or further decontamination.

Culture: growth of microorganisms in or on a nutrient medium; to grow microorganisms in or on such a medium.

Culture medium: substance or preparation used to grow and cultivate microorganisms.

Cup: 8 fluid ounces.

Decontamination: according to OSHA, “the use of physical or chemical means to remove, inactivate, or destroy bloodborne pathogens on a surface or item to the point where they are no longer capable of transmitting infectious particles and the surface or item is rendered safe for handling, use, or disposal” [29 CFR 1910.1030]. In health-care facilities, the term generally refers to all pathogenic organisms.

Decontamination area: area of a health-care facility designated for collection, retention, and cleaning of soiled and/or contaminated items.

Detergent: cleaning agent that makes no antimicrobial claims on the label. They comprise a hydrophilic component and a lipophilic component and can be divided into four types: anionic, cationic, amphoteric, and non-ionic detergents.

Disinfectant: usually a chemical agent (but sometimes a physical agent) that destroys disease-causing pathogens or other harmful microorganisms but might not kill bacterial spores. It refers to substances applied to inanimate objects. EPA groups disinfectants by product label claims of “limited,” “general,” or “hospital” disinfection.

Disinfection: thermal or chemical destruction of pathogenic and other types of microorganisms. Disinfection is less lethal than sterilization because it destroys most recognized pathogenic microorganisms but not necessarily all microbial forms (e.g., bacterial spores).

D value: time or radiation dose required to inactivate 90% of a population of the test microorganism under stated exposure conditions.

Endoscope: an instrument that allows examination and treatment of the interior of the body canals and hollow organs.

Enzyme cleaner: a solution used before disinfecting instruments to improve removal of organic material (e.g., proteases to assist in removing protein).

EPA Registration Number or EPA Reg. No.: a hyphenated, two- or three-part number assigned by EPA to identify each germicidal product registered within the United States. The first number is the company identification number, the second is the specific product number, and the third (when present) is the company identification number for a supplemental registrant.

Exposure time: period in a sterilization process during which items are exposed to the sterilant at the specified sterilization parameters. For example, in a steam sterilization process, exposure time is the period during which items are exposed to saturated steam at the specified temperature.

Flash sterilization: process designed for the steam sterilization of unwrapped patient-care items for immediate use (or placed in a specially designed, covered, rigid container to allow for rapid penetration of steam).

Fungicide: agent that destroys fungi (including yeasts) and/or fungal spores pathogenic to humans or other animals in the inanimate environment.

General disinfectant: EPA-registered disinfectant labeled for use against both gram-negative and gram-positive bacteria. Efficacy is demonstrated against both *Salmonella choleraesuis* and *Staphylococcus aureus*. Also called *broad-spectrum disinfectant*.

Germicide: agent that destroys microorganisms, especially pathogenic organisms.

Germicidal detergent: detergent that also is EPA-registered as a disinfectant.

High-level disinfectant: agent capable of killing bacterial spores when used in sufficient concentration under suitable conditions. It therefore is expected to kill all other microorganisms.

Hospital disinfectant: disinfectant registered for use in hospitals, clinics, dental offices, and any other medical-related facility. Efficacy is demonstrated against *Salmonella choleraesuis*, *Staphylococcus aureus*, and *Pseudomonas aeruginosa*. EPA has registered approximately 1,200 hospital disinfectants.

Huck towel: all-cotton surgical towel with a honey-comb weave; both warp and fill yarns are tightly twisted. Huck towels can be used to prepare biologic indicator challenge test packs.

Implantable device: according to FDA, “device that is placed into a surgically or naturally formed cavity of the human body if it is intended to remain there for a period of 30 days or more” [21 CFR 812.3(d)].

Inanimate surface: nonliving surface (e.g., floors, walls, furniture).

Incubator: apparatus for maintaining a constant and suitable temperature for the growth and cultivation of microorganisms.

Infectious microorganisms: microorganisms capable of producing disease in appropriate hosts.

Inorganic and organic load: naturally occurring or artificially placed inorganic (e.g., metal salts) or organic (e.g., proteins) contaminants on a medical device before exposure to a microbicidal process.

Intermediate-level disinfectant: agent that destroys all vegetative bacteria, including tubercle bacilli, lipid and some nonlipid viruses, and fungi, but not bacterial spores.

Limited disinfectant: disinfectant registered for use against a specific major group of organisms (gram-negative or gram-positive bacteria). Efficacy has been demonstrated in laboratory tests against either *Salmonella choleraesuis* or *Staphylococcus aureus* bacteria.

Lipid virus: virus surrounded by an envelope of lipoprotein in addition to the usual core of nucleic acid surrounded by a coat of protein. This type of virus (e.g., HIV) is generally easily inactivated by many types of disinfectants. Also called *enveloped* or *lipophilic virus*.

Low-level disinfectant: agent that destroys all vegetative bacteria (except tubercle bacilli), lipid viruses, some nonlipid viruses, and some fungi, but not bacterial spores.

Mechanical indicator: devices that monitor the sterilization process (e.g., graphs, gauges, printouts).

Medical device: instrument, apparatus, material, or other article, whether used alone or in combination, including software necessary for its application, intended by the manufacturer to be used for human beings for

- diagnosis, prevention, monitoring treatment, or alleviation of disease;
- diagnosis, monitoring, treatment, or alleviation of or compensation for an injury or handicap;
- investigation, replacement, or modification of the anatomy or of a physiologic process; or
- control of conception

and that does not achieve its primary intended action in or on the human body by pharmacologic, immunologic, or metabolic means but might be assisted in its function by such means.

Microbicide: any substance or mixture of substances that effectively kills microorganisms.

Microorganisms: animals or plants of microscopic size. As used in health care, generally refers to bacteria, fungi, viruses, and bacterial spores.

Minimum effective concentration (MEC): the minimum concentration of a liquid chemical germicide needed to achieve the claimed microbicidal activity as determined by dose-response testing. Sometimes used interchangeably with *minimum recommended concentration*.

Muslin: loosely woven (by convention, 140 threads per square inch), 100% cotton cloth. Formerly used as a wrap for sterile packs or a surgical drape. Fabric wraps used currently consist of a cotton-polyester blend.

Mycobacteria: bacteria with a thick, waxy coat that makes them more resistant to chemical germicides than other types of vegetative bacteria.

Nonlipid viruses: generally considered more resistant to inactivation than lipid viruses. Also called nonenveloped or hydrophilic viruses.

One-step disinfection process: simultaneous cleaning and disinfection of a noncritical surface or item.

Pasteurization: process developed by Louis Pasteur of heating milk, wine, or other liquids to 65–77°C (or the equivalent) for approximately 30 minutes to kill or markedly reduce the number of pathogenic and spoilage organisms other than bacterial spores.

Parametric release: declaration that a product is sterile on the basis of physical and/or chemical process data rather than on sample testing or biologic indicator results.

Penicylinder: carriers inoculated with the test bacteria for in vitro tests of germicides. Can be constructed of stainless steel, porcelain, glass, or other materials and are approximately 8 x 10 mm in diameter.

Permissible exposure limit (PEL): time-weighted average maximum concentration of an air contaminant to which a worker can be exposed, according to OSHA standards. Usually calculated over 8 hours, with exposure considered over a 40-hour work week.

Personal protective equipment (PPE): specialized clothing or equipment worn by an employee for protection against a hazard. General work clothes (e.g., uniforms, pants, shirts) not intended to function as protection against a hazard are not considered to be PPE.

Parts per million (ppm): common measurement for concentrations by volume of trace contaminant gases in the air (or chemicals in a liquid); 1 volume of contaminated gas per 1 million volumes of contaminated air or 1¢ in \$10,000 both equal 1 ppm. Parts per million = µg/mL or mg/L.

Prions: transmissible pathogenic agents that cause a variety of neurodegenerative diseases of humans and animals, including sheep and goats, bovine spongiform encephalopathy in cattle, and Creutzfeldt-Jakob disease in humans. They are unlike any other infectious pathogens because they are composed of an abnormal conformational isoform of a normal cellular protein, the prion protein (PrP). Prions are extremely resistant to inactivation by sterilization processes and disinfecting agents.

Process challenge device (PCD): item designed to simulate product to be sterilized and to constitute a defined challenge to the sterilization process and used to assess the effective performance of the process. A PCD is a challenge test pack or test tray that contains a biologic indicator, a Class 5 integrating indicator, or an enzyme-only indicator.

QUAT: abbreviation for *quaternary ammonium compound*, a surface-active, water-soluble disinfecting

substance that has four carbon atoms linked to a nitrogen atom through covalent bonds.

Recommended exposure limit (REL): occupational exposure limit recommended by NIOSH as being protective of worker health and safety over a working lifetime. Frequently expressed as a 40-hour time-weighted-average exposure for up to 10 hours per day during a 40-work week.

Reprocess: method to ensure proper disinfection or sterilization; can include: cleaning, inspection, wrapping, sterilizing, and storing.

Sanitizer: agent that reduces the number of bacterial contaminants to safe levels as judged by public health requirements. Commonly used with substances applied to inanimate objects. According to the protocol for the official sanitizer test, a sanitizer is a chemical that kills 99.999% of the specific test bacteria in 30 seconds under the conditions of the test.

Shelf life: length of time an undiluted or use dilution of a product can remain active and effective. Also refers to the length of time a sterilized product (e.g., sterile instrument set) is expected to remain sterile.

Spaulding classification: strategy for reprocessing contaminated medical devices. The system classifies a medical device as critical, semicritical, or noncritical on the basis of risk to patient safety from contamination on a device. The system also established three levels of germicidal activity (sterilization, high-level disinfection, and low-level disinfection) for strategies with the three classes of medical devices (critical, semicritical, and noncritical).

Spore: relatively water-poor round or elliptical resting cell consisting of condensed cytoplasm and nucleus surrounded by an impervious cell wall or coat. Spores are relatively resistant to disinfectant and sterilant activity and drying conditions (specifically in the genera *Bacillus* and *Clostridium*).

Spore strip: paper strip impregnated with a known population of spores that meets the definition of biological indicators.

Steam quality: steam characteristic reflecting the dryness fraction (weight of dry steam in a mixture of dry saturated steam and entrained water) and the level of noncondensable gas (air or other gas that will not condense under the conditions of temperature and pressure used during the sterilization process). The dryness fraction (i.e., the proportion of completely dry steam in the steam being considered) should not fall below 97%.

Steam sterilization: sterilization process that uses saturated steam under pressure for a specified exposure time and at a specified temperature, as the sterilizing agent.

Steam sterilization, dynamic air removal type: one of two types of sterilization cycles in which air is removed from the chamber and the load by a series of pressure and vacuum excursions (prevacuum cycle) or by a series of steam flushes and pressure pulses above atmospheric pressure (steam-flush-pressure-pulse cycle).

Sterile or Sterility: state of being free from all living microorganisms. In practice, usually described as a probability function, e.g., as the probability of a microorganism surviving sterilization being one in one million.

Sterility assurance level (SAL): probability of a viable microorganism being present on a product unit after sterilization. Usually expressed as 10^{-6} ; a SAL of 10^{-6} means $\leq 1/1$ million chance that a single viable microorganism is present on a sterilized item. A SAL of 10^{-6} generally is accepted as appropriate for items intended to contact compromised tissue (i.e., tissue that has lost the integrity of the natural body barriers). The sterilizer manufacturer is responsible for ensuring the sterilizer can achieve the desired SAL. The

user is responsible for monitoring the performance of the sterilizer to ensure it is operating in conformance to the manufacturer's recommendations.

Sterilization: validated process used to render a product free of all forms of viable microorganisms. In a sterilization process, the presence of microorganisms on any individual item can be expressed in terms of probability. Although this probability can be reduced to a very low number, it can never be reduced to zero.

Sterilization area: area of a health-care facility designed to house sterilization equipment, such as steam ethylene oxide, hydrogen peroxide gas plasma, or ozone sterilizers.

Sterilizer: apparatus used to sterilize medical devices, equipment, or supplies by direct exposure to the sterilizing agent.

Sterilizer, gravity-displacement type: type of steam sterilizer in which incoming steam displaces residual air through a port or drain in or near the bottom (usually) of the sterilizer chamber. Typical operating temperatures are 121–123°C (250–254°F) and 132–135°C (270–275°F).

Sterilizer, prevacuum type: type of steam sterilizer that depends on one or more pressure and vacuum excursions at the beginning of the cycle to remove air. This method of operation results in shorter cycle times for wrapped items because of the rapid removal of air from the chamber and the load by the vacuum system and because of the usually higher operating temperature (132–135°C [270–275°F]; 141–144°C [285–291°F]). This type of sterilizer generally provides for shorter exposure time and accelerated drying of fabric loads by pulling a further vacuum at the end of the sterilizing cycle.

Sterilizer, steam-flush pressure-pulse type: type of sterilizer in which a repeated sequence consisting of a steam flush and a pressure pulse removes air from the sterilizing chamber and processed materials using steam at above atmospheric pressure (no vacuum is required). Like a prevacuum sterilizer, a steam-flush pressure-pulse sterilizer rapidly removes air from the sterilizing chamber and wrapped items; however, the system is not susceptible to air leaks because air is removed with the sterilizing chamber pressure at above atmospheric pressure. Typical operating temperatures are 121–123°C (250–254°F), 132–135°C (270–275°F), and 141–144°C (285–291°F).

Surfactant: agent that reduces the surface tension of water or the tension at the interface between water and another liquid; a wetting agent found in many sterilants and disinfectants.

Tabletop steam sterilizer: a compact gravity-displacement steam sterilizer that has a chamber volume of not more than 2 cubic feet and that generates its own steam when distilled or deionized water is added.

Time-weighted average (TWA): an average of all the concentrations of a chemical to which a worker has been exposed during a specific sampling time, reported as an average over the sampling time. For example, the permissible exposure limit for ethylene oxide is 1 ppm as an 8-hour TWA. Exposures above the ppm limit are permitted if they are compensated for by equal or longer exposures below the limit during the 8-hour workday as long as they do not exceed the ceiling limit; short-term exposure limit; or, in the case of ethylene oxide, excursion limit of 5 ppm averaged over a 15-minute sampling period.

Tuberculocide: an EPA-classified hospital disinfectant that also kills *Mycobacterium tuberculosis* (tubercle bacilli). EPA has registered approximately 200 tuberculocides. Such agents also are called *mycobactericides*.

Use-life: the length of time a diluted product can remain active and effective. The stability of the chemical and the storage conditions (e.g., temperature and presence of air, light, organic matter, or metals)

determine the use-life of antimicrobial products.

Vegetative bacteria: bacteria that are devoid of spores and usually can be readily inactivated by many types of germicides.

Virucide: an agent that kills viruses to make them noninfective.

Adapted from Association for the Advancement of Medical Instrumentation;^{811-814, 819} Association of periOperating Registered Nurses (AORN),⁸¹⁵ American Hospital Association,³¹⁹ and Block.^{16, 1034}

Table 1. Methods of sterilization and disinfection.

Object	Sterilization		Disinfection		
	Procedure	Exposure time	High-level (semicritical items; [except dental] will come in contact with mucous membrane or nonintact skin)	Intermediate-level (some semicritical items ¹ and noncritical items)	Low-level (noncritical items; will come in contact with intact skin)
			Critical items (will enter tissue or vascular system or blood will flow through them)	Procedure (exposure time 12-30 min at $\geq 20^{\circ}\text{C}$) ^{2,3}	Procedure (exposure time ≥ 1 m) ⁹
Smooth, hard Surface ^{1,4}	A	MR	D	K	K
	B	MR	E	L ⁵	L
	C	MR	F	M	M
	D	10 h at 20-25°C	H	N	N
	F	6 h	I ⁶		O
	G	12 m at 50-56°C	J		
	H	3-8 h			
Rubber tubing and catheters ^{3,4}	A	MR	D		
	B	MR	E		
	C	MR	F		
	D	10 h at 20-25°C	H		
	F	6 h	I ⁶		
	G	12 m at 50-56°C	J		
	H	3-8 h			
Polyethylene tubing and catheters ^{3,4,7}	A	MR	D		
	B	MR	E		
	C	MR	F		
	D	10 h at 20-25°C	H		
	F	6 h	I ⁶		
	G	12 m at 50-56°C	J		
	H	3-8 h			
Lensed instruments ⁴	A	MR	D		
	B	MR	E		
	C	MR	F		
	D	10 h at 20-25°C	H		
	F	6 h	J		
	G	12 m at 50-56°C			
	H	3-8 h			
Thermometers (oral and rectal) ⁸					K ⁸
Hinged instruments ⁴	A	MR	D		
	B	MR	E		
	C	MR	F		
	D	10 h at 20-25°C	H		
	F	6 h	I ⁶		
	G	12 m at 50-56°C	J		
	H	3-8 h			

Modified from Rutala and Simmons.^{15, 17, 18, 421} The selection and use of disinfectants in the healthcare field is dynamic, and products may become available that are not in existence when this guideline was written. As newer disinfectants become available, persons or committees responsible for selecting disinfectants and sterilization processes should be guided by products cleared by the FDA and the EPA as well as information in the scientific literature.

- A, Heat sterilization, including steam or hot air (see manufacturer's recommendations, steam sterilization processing time from 3-30 minutes)
- B, Ethylene oxide gas (see manufacturer's recommendations, generally 1-6 hours processing time plus aeration time of 8-12 hours at 50-60°C)
- C, Hydrogen peroxide gas plasma (see manufacturer's recommendations for internal diameter and length restrictions, processing time between 45-72 minutes).
- D, Glutaraldehyde-based formulations ($\geq 2\%$ glutaraldehyde, caution should be exercised with all glutaraldehyde formulations when further in-use dilution is anticipated); glutaraldehyde (1.12%) and 1.93% phenol/phenate. One glutaraldehyde-based product has a high-level disinfection claim of 5 minutes at 35°C.
- E, Ortho-phthalaldehyde (OPA) 0.55%
- F, Hydrogen peroxide 7.5% (will corrode copper, zinc, and brass)
- G, Peracetic acid, concentration variable but 0.2% or greater is sporicidal. Peracetic acid immersion system operates at 50-56°C.
- H, Hydrogen peroxide (7.35%) and 0.23% peracetic acid; hydrogen peroxide 1% and peracetic acid 0.08% (will corrode metal instruments)
- I, Wet pasteurization at 70°C for 30 minutes with detergent cleaning
- J, Hypochlorite, single use chlorine generated on-site by electrolyzing saline containing >650-675 active free chlorine; (will corrode metal instruments)
- K, Ethyl or isopropyl alcohol (70-90%)
- L, Sodium hypochlorite (5.25-6.15% household bleach diluted 1:500 provides >100 ppm available chlorine)
- M, Phenolic germicidal detergent solution (follow product label for use-dilution)
- N, Iodophor germicidal detergent solution (follow product label for use-dilution)
- O, Quaternary ammonium germicidal detergent solution (follow product label for use-dilution)
- MR, Manufacturer's recommendations
- NA, Not applicable

¹ See text for discussion of hydrotherapy.

² The longer the exposure to a disinfectant, the more likely it is that all microorganisms will be eliminated. Follow the FDA-cleared high-level disinfection claim. Ten-minute exposure is not adequate to disinfect many objects, especially those that are difficult to clean because they have narrow channels or other areas that can harbor organic material and bacteria. Twenty-minute exposure at 20°C is the minimum time needed to reliably kill *M. tuberculosis* and nontuberculous mycobacteria with a 2% glutaraldehyde. Some high-level disinfectants have a reduced exposure time (e.g., ortho-phthalaldehyde at 12 minutes at 20°C) because of their rapid activity against mycobacteria or reduced exposure time due to increased mycobactericidal activity at elevated temperature (e.g., 2.5% glutaraldehyde at 5 minutes at 35°C, 0.55% OPA at 5 min at 25°C in automated endoscope reprocessor).

³ Tubing must be completely filled for high-level disinfection and liquid chemical sterilization; care must be taken to avoid entrapment of air bubbles during immersion.

⁴ Material compatibility should be investigated when appropriate.

⁵ A concentration of 1000 ppm available chlorine should be considered where cultures or concentrated preparations of microorganisms have spilled (5.25% to 6.15% household bleach diluted 1:50 provides > 1000 ppm available chlorine). This solution may corrode some surfaces.

⁶ Pasteurization (washer-disinfector) of respiratory therapy or anesthesia equipment is a recognized alternative to high-level disinfection. Some data challenge the efficacy of some pasteurization units.

⁷ Thermostability should be investigated when appropriate.

⁸ Do not mix rectal and oral thermometers at any stage of handling or processing.

⁹ By law, all applicable label instructions on EPA-registered products must be followed. If the user selects exposure conditions that differ from those on the EPA-registered products label, the user assumes liability from any injuries resulting from off-label use and is potentially subject to enforcement action under FIFRA.

Table 2. Properties of an ideal disinfectant.

Broad spectrum: should have a wide antimicrobial spectrum
Fast acting: should produce a rapid kill
Not affected by environmental factors: should be active in the presence of organic matter (e.g., blood, sputum, feces) and compatible with soaps, detergents, and other chemicals encountered in use
Nontoxic: should not be harmful to the user or patient
Surface compatibility: should not corrode instruments and metallic surfaces and should not cause the deterioration of cloth, rubber, plastics, and other materials
Residual effect on treated surfaces: should leave an antimicrobial film on the treated surface
Easy to use with clear label directions
Odorless: should have a pleasant odor or no odor to facilitate its routine use
Economical: should not be prohibitively high in cost
Solubility: should be soluble in water
Stability: should be stable in concentrate and use-dilution
Cleaner: should have good cleaning properties
Environmentally friendly: should not damage the environment on disposal

Modified from Molinari¹⁰³⁵.

Table 3. Epidemiologic evidence associated with the use of surface disinfectants or detergents on noncritical environmental surfaces.

Justification for Use of Disinfectants for Noncritical Environmental Surfaces

Surfaces may contribute to transmission of epidemiologically important microbes (e.g., vancomycin-resistant Enterococci, methicillin-resistant *S. aureus*, viruses)

Disinfectants are needed for surfaces contaminated by blood and other potentially infective material

Disinfectants are more effective than detergents in reducing microbial load on floors

Detergents become contaminated and result in seeding the patient's environment with bacteria

Disinfection of noncritical equipment and surfaces is recommended for patients on isolation precautions by the Centers for Disease Control and Prevention.

Advantage of using a single product for decontamination of noncritical surfaces, both floors and equipment

Some newer disinfectants have persistent antimicrobial activity

Justification for Using a Detergent on Noncritical Environmental Surfaces

Noncritical surfaces contribute minimally to endemic healthcare-associated infections

No difference in healthcare-associated infection rates when floors are cleaned with detergent versus disinfectant

No environmental impact (aquatic or terrestrial) issues with disposal

No occupational health exposure issues

Lower costs

Use of antiseptics/disinfectants selects for antibiotic-resistant bacteria (?)

More aesthetically pleasing floor

Modified from Rutala³⁷⁸.

Figure 1. Decreasing order of resistance of microorganisms to disinfection and sterilization and the level of disinfection or sterilization.

Resistant	Level
Prions (Creutzfeldt-Jakob Disease)	Prion reprocessing
Bacterial spores (<i>Bacillus atrophaeus</i>)	Sterilization
Coccidia (<i>Cryptosporidium</i>)	
Mycobacteria (<i>M. tuberculosis</i> , <i>M. terrae</i>)	High
Nonlipid or small viruses (polio, coxsackie)	Intermediate
Fungi (<i>Aspergillus</i> , <i>Candida</i>)	
Vegetative bacteria (<i>S. aureus</i> , <i>P. aeruginosa</i>)	Low
↓ Lipid or medium-sized viruses (HIV, herpes, hepatitis B)	

Susceptible

Modified from Russell and Favero^{13, 344}.

Table 4. Comparison of the characteristics of selected chemicals used as high-level disinfectants or chemical sterilants.

	HP (7.5%)	PA (0.2%)	Glut ($\geq 2.0\%$)	OPA (0.55%)	HP/PA (7.35%/0.23%)
HLD Claim	30 m @ 20°C	NA	20-90 m @ 20°-25°C	12 m @ 20°C, 5 m @ 25°C in AER	15m @ 20°C
Sterilization Claim	6 h @ 20°	12m @ 50-56°C	10 h @ 20°-25°C	None	3 h @ 20°C
Activation	No	No	Yes (alkaline glut)	No	No
Reuse Life ¹	21d	Single use	14-30 d	14d	14d
Shelf Life Stability ²	2 y	6 mo	2 y	2 y	2 y
Disposal Restrictions	None	None	Local ³	Local ³	None
Materials Compatibility	Good	Good	Excellent	Excellent	No data
Monitor MEC ⁴	Yes (6%)	No	Yes (1.5% or higher)	Yes (0.3% OPA)	No
Safety	Serious eye damage (safety glasses)	Serious eye and skin damage (conc soln) ⁵	Respiratory	Eye irritant, stains skin	Eye damage
Processing	Manual or automated	Automated	Manual or automated	Manual or automated	Manual
Organic material resistance	Yes	Yes	Yes	Yes	Yes
OSHA exposure limit	1 ppm TWA	None	None ⁶	None	HP-1 ppm TWA
Cost profile (per cycle) ⁷	+ (manual), ++ (automated)	+++++ (automated)	+ (manual), ++ (automated)	++ (manual)	++ (manual)

Modified from Rutala⁶⁹.

Abbreviations: HLD=high-level disinfectant; HP=hydrogen peroxide; PA=peracetic acid; glut=glutaraldehyde; PA/HP=peracetic acid and hydrogen peroxide; OPA =ortho-phthalaldehyde (FDA cleared as a high-level disinfectant, included for comparison to other chemical agents used for high-level disinfection); m=minutes; h=hours; NA=not applicable; TWA=time-weighted average for a conventional 8-hour workday.

¹number of days a product can be reused as determined by re-use protocol

²time a product can remain in storage (unused)

³no U.S. EPA regulations but some states and local authorities have additional restrictions

⁴MEC=minimum effective concentration is the lowest concentration of active ingredients at which the product is still effective

⁵Conc soln=concentrated solution

⁶The ceiling limit recommended by the American Conference of Governmental Industrial Hygienists is 0.05 ppm.

⁷per cycle cost profile considers cost of the processing solution (suggested list price to healthcare facilities in August 2001) and assumes maximum use life (e.g., 21 days for hydrogen peroxide, 14 days for glutaraldehyde), 5 reprocessing cycles per day, 1-gallon basin for manual processing, and 4-gallon tank for automated processing. + = least expensive; +++++ = most expensive

Table 5. Summary of advantages and disadvantages of chemical agents used as chemical sterilants ¹ or as high-level disinfectants.		
Sterilization Method	Advantages	Disadvantages
Peracetic Acid/Hydrogen Peroxide	<ul style="list-style-type: none"> No activation required Odor or irritation not significant 	<ul style="list-style-type: none"> Materials compatibility concerns (lead, brass, copper, zinc) both cosmetic and functional Limited clinical experience Potential for eye and skin damage
Glutaraldehyde	<ul style="list-style-type: none"> Numerous use studies published Relatively inexpensive Excellent materials compatibility 	<ul style="list-style-type: none"> Respiratory irritation from glutaraldehyde vapor Pungent and irritating odor Relatively slow mycobactericidal activity Coagulates blood and fixes tissue to surfaces Allergic contact dermatitis Glutaraldehyde vapor monitoring recommended
Hydrogen Peroxide	<ul style="list-style-type: none"> No activation required May enhance removal of organic matter and organisms No disposal issues No odor or irritation issues Does not coagulate blood or fix tissues to surfaces Inactivates <i>Cryptosporidium</i> Use studies published 	<ul style="list-style-type: none"> Material compatibility concerns (brass, zinc, copper, and nickel/silver plating) both cosmetic and functional Serious eye damage with contact
Ortho-phthalaldehyde	<ul style="list-style-type: none"> Fast acting high-level disinfectant No activation required Odor not significant Excellent materials compatibility claimed Does not coagulate blood or fix tissues to surfaces claimed 	<ul style="list-style-type: none"> Stains skin, mucous membranes, clothing, and environmental surfaces Repeated exposure may result in hypersensitivity in some patients with bladder cancer More expensive than glutaraldehyde Eye irritation with contact Slow sporicidal activity
Peracetic Acid	<ul style="list-style-type: none"> Rapid sterilization cycle time (30-45 minutes) Low temperature (50-55°C) liquid immersion sterilization Environmental friendly by-products (acetic acid, O₂, H₂O) Fully automated Single-use system eliminates need for concentration testing Standardized cycle May enhance removal of organic material and endotoxin No adverse health effects to operators under normal operating conditions Compatible with many materials and instruments Does not coagulate blood or fix tissues to surfaces Sterilant flows through scope facilitating salt, protein, and microbe removal Rapidly sporicidal Provides procedure standardization (constant dilution, perfusion of channel, temperatures, exposure) 	<ul style="list-style-type: none"> Potential material incompatibility (e.g., aluminum anodized coating becomes dull) Used for immersible instruments only Biological indicator may not be suitable for routine monitoring One scope or a small number of instruments can be processed in a cycle More expensive (endoscope repairs, operating costs, purchase costs) than high-level disinfection Serious eye and skin damage (concentrated solution) with contact Point-of-use system, no sterile storage

Modified from Rutala⁶⁹.

¹All products effective in presence of organic soil, relatively easy to use, and have a broad spectrum of antimicrobial activity (bacteria, fungi, viruses, bacterial spores, and mycobacteria). The above characteristics are documented in the literature; contact the manufacturer of the instrument and sterilant for additional information. All products listed above are FDA-cleared as chemical sterilants except OPA, which is an FDA-cleared high-level disinfectant.

Table 6. Summary of advantages and disadvantages of commonly used sterilization technologies.

Sterilization Method	Advantages	Disadvantages
Steam	<ul style="list-style-type: none"> · Nontoxic to patient, staff, environment · Cycle easy to control and monitor · Rapidly microbicidal · Least affected by organic/inorganic soils among sterilization processes listed · Rapid cycle time · Penetrates medical packing, device lumens 	<ul style="list-style-type: none"> · Deleterious for heat-sensitive instruments · Microsurgical instruments damaged by repeated exposure · May leave instruments wet, causing them to rust • Potential for burns
Hydrogen Peroxide Gas Plasma	<ul style="list-style-type: none"> · Safe for the environment · Leaves no toxic residuals · Cycle time is 28-75 minutes (varies with model type) and no aeration necessary · Used for heat- and moisture-sensitive items since process temperature <50°C · Simple to operate, install (208 V outlet), and monitor · Compatible with most medical devices · Only requires electrical outlet 	<ul style="list-style-type: none"> · Cellulose (paper), linens and liquids cannot be processed · Sterilization chamber size from 1.8-9.4 ft³ total volume (varies with model type) · Some endoscopes or medical devices with long or narrow lumens cannot be processed at this time in the United States (see manufacturer's recommendations for internal diameter and length restrictions) · Requires synthetic packaging (polypropylene wraps, polyolefin pouches) and special container tray • Hydrogen peroxide may be toxic at levels greater than 1 ppmTWA
100% Ethylene Oxide (ETO)	<ul style="list-style-type: none"> · Penetrates packaging materials, device lumens · Single-dose cartridge and negative- pressure chamber minimizes the potential for gas leak and ETO exposure · Simple to operate and monitor · Compatible with most medical materials 	<ul style="list-style-type: none"> · Requires aeration time to remove ETO residue · Sterilization chamber size from 4.0-7.9 ft³ total volume (varies with model type) · ETO is toxic, a carcinogen, and flammable · ETO emission regulated by states but catalytic cell removes 99.9% of ETO and converts it to CO₂ and H₂O · ETO cartridges should be stored in flammable liquid storage cabinet · Lengthy cycle/aeration time
ETO Mixtures 8.6% ETO/91.4% HCFC 10% ETO/90% HCFC 8.5% ETO/91.5% CO ₂	<ul style="list-style-type: none"> · Penetrates medical packaging and many plastics · Compatible with most medical materials · Cycle easy to control and monitor 	<ul style="list-style-type: none"> · Some states (e.g., CA, NY, MI) require ETO emission reduction of 90-99.9% · CFC (inert gas that eliminates explosion hazard) banned in 1995 · Potential hazards to staff and patients · Lengthy cycle/aeration time · ETO is toxic, a carcinogen, and flammable
Peracetic Acid	<ul style="list-style-type: none"> · Rapid cycle time (30-45 minutes) · Low temperature (50-55°C liquid immersion sterilization) · Environmental friendly by-products · Sterilant flows through endoscope which facilitates salt, protein and microbe removal 	<ul style="list-style-type: none"> · Point-of-use system, no sterile storage · Biological indicator may not be suitable for routine monitoring · Used for immersible instruments only · Some material incompatibility (e.g., aluminum anodized coating becomes dull) · One scope or a small number of instruments processed in a cycle • Potential for serious eye and skin damage (concentrated solution) with contact

Modified from Rutala.⁸²⁵

Abbreviations: CFC=chlorofluorocarbon, HCFC=hydrochlorofluorocarbon.

Table 7. Minimum cycle times for steam sterilization cycles

Type of sterilizer	Item	Exposure time at 250°F (121°C)	Exposure time at 270°F (132°C)	Drying time
Gravity displacement	Wrapped instruments	30 min	15 min	15-30 min
	Textile packs	30 min	25 min	15 min
	Wrapped utensils	30 min	15 min	15-30 min
Dynamic-air-removal (e.g., prevacuum)	Wrapped instruments		4 min	20-30 min
	Textile packs		4 min	5-20 min
	Wrapped utensils		4 min	20 min

Modified from Association for the Advancement of Medical Instrumentation.^{813, 819}

Table 8. Examples of flash steam sterilization parameters.

Type of sterilizer	Load configuration	Temperature	Time
Gravity displacement	Nonporous items only (i.e., routine metal instruments, no lumens)	132°C (270°F)	3 minutes
	Nonporous and porous items (e.g., rubber or plastic items, items with lumens) sterilized together	132°C (270°F)	10 minutes
Prevacuum	Nonporous items only (i.e., routine metal instruments, no lumens)	132°C (270°F)	3 minutes
	Nonporous and porous items (e.g., rubber or plastic items, items with lumens) sterilized together	132°C (270°F)	4 minutes
Steam-flush pressure-pulse	Nonporous or mixed nonporous/porous items	132° (270°F) Manufacturers' instruction	4 minutes

Modified from Association for the Advancement of Medical Instrumentation. ^{812, 819}

Table 9. Characteristics of an ideal low-temperature sterilization process.

High efficacy: the agent should be virucidal, bactericidal, tuberculocidal, fungicidal and sporicidal
Rapid activity: ability to quickly achieve sterilization
Strong penetrability: ability to penetrate common medical-device packaging materials and penetrate into the interior of device lumens
Material compatibility: produces only negligible changes in the appearance or the function of processed items and packaging materials even after repeated cycling
Nontoxic: presents no toxic health risk to the operator or the patient and poses no hazard to the environment
Organic material resistance: withstands reasonable organic material challenge without loss of efficacy
Adaptability: suitable for large or small (point of use) installations
Monitoring capability: monitored easily and accurately with physical, chemical, and biological process monitors
Cost effectiveness: reasonable cost for installation and for routine operation

Modified from Schneider.⁸⁵¹

Table 10. Factors affecting the efficacy of sterilization.

Factors	Effect
Cleaning ¹	Failure to adequately clean instrument results in higher bioburden, protein load, and salt concentration. These will decrease sterilization efficacy.
Bioburden ¹	The natural bioburden of used surgical devices is 10 ⁰ to 10 ³ organisms (primarily vegetative bacteria), which is substantially below the 10 ⁵ -10 ⁶ spores used with biological indicators.
Pathogen type	Spore-forming organisms are most resistant to sterilization and are the test organisms required for FDA clearance. However, the contaminating microflora on used surgical instruments consists mainly of vegetative bacteria.
Protein ¹	Residual protein decreases efficacy of sterilization. However, cleaning appears to rapidly remove protein load.
Salt ¹	Residual salt decreases efficacy of sterilization more than does protein load. However, cleaning appears to rapidly remove salt load.
Biofilm accumulation ¹	Biofilm accumulation reduces efficacy of sterilization by impairing exposure of the sterilant to the microbial cell.
Lumen length	Increasing lumen length impairs sterilant penetration. May require forced flow through lumen to achieve sterilization.
Lumen diameter	Decreasing lumen diameter impairs sterilant penetration. May require forced flow through lumen to achieve sterilization.
Restricted flow	Sterilant must come into contact with microorganisms. Device designs that prevent or inhibit this contact (e.g., sharp bends, blind lumens) will decrease sterilization efficacy.
Device design and construction	Materials used in construction may affect compatibility with different sterilization processes and affect sterilization efficacy. Design issues (e.g., screws, hinges) will also affect sterilization efficacy.

Modified from Alfa and Rutala.^{470, 825}

¹ Factor only relevant for reused surgical/medical devices

Table 11. Comparative evaluation of the microbicidal activity of low-temperature sterilization technology.

Challenge	Carriers Sterilized by Various Low-Temperature Sterilization Technologies						Reference
	ETO 12/88	100% ETO	HCFC-ETO	HPGP 100	HPGP 100S	PA	
No salt or serum ¹	100%	100%	96%	100%	ND	ND	Alfa ⁷²¹
10% serum and 0.65% salt ²	97%	60%	95%	37%	ND	ND	Alfa ⁷²¹
Lumen (125 cm long x 3 mm wide) without serum or salt ¹	ND	96%	96%	ND	ND	ND	Alfa ⁷²¹
Lumen (125 cm long x 3 mm wide) with 10% serum and 0.65% salt ²	44%	40%	49%	35%	ND	100% ¹	Alfa ⁷²¹
Lumen (40 cm long x 3 mm wide) ³	ND	ND	100%	95%	100%	8%	Rutala ⁸⁵⁶
Lumen (40 cm long x 2 mm wide) ³	ND	ND	100%	93%	100%	ND	Rutala ⁸⁵⁶
Lumen (40 cm long x 1 mm wide) ³	ND	ND	100%	26%	100%	ND	Rutala ⁸⁵⁶
Lumen (40 cm long x 3 mm wide) ⁴	ND	ND	100%	100%	100%	ND	Rutala ⁸⁵⁶

—

Modified from Rutala.⁸²⁵

Abbreviations: ETO=ethylene oxide; HCFC=hydrochlorofluorocarbon; ND=no data; HPGP=hydrogen peroxide gas plasma; PA=peracetic acid.

¹Test organisms included *Enterococcus faecalis*, *Mycobacterium chelonae*, and *Bacillus atrophaeus* spores.

²Test organisms included *E. faecalis*, *P. aeruginosa*, *E. coli*, *M. chelonae*, *B. atrophaeus* spores, *G. stearothermophilus* spores, and *B. circulans* spores.

³Test organism was *G. stearothermophilus* spores. The lumen test units had a removable 5 cm center piece (1.2 cm diameter) of stainless steel sealed to the narrower steel tubing by hard rubber septums.

⁴Test organism was *G. stearothermophilus* spores. The lumen test unit was a straight stainless steel tube.

Table 12. Suggested protocol for management of positive biological indicator in a steam sterilizer.

1. Take the sterilizer out of service. Notify area supervisor and infection control department.
2. Objects, other than implantable objects, do not need to be recalled because of a single positive spore test unless the sterilizer or the sterilization procedure is defective. As soon as possible, repeat biological indicator test in three consecutive sterilizer cycles. If additional spore tests remain positive, the items should be considered nonsterile, and supplies processed since the last acceptable (negative) biological indicator should be recalled. The items from the suspect load(s) should be recalled and reprocessed.
3. Check to ensure the sterilizer was used correctly (e.g., verify correct time and temperature setting). If not, repeat using appropriate settings and recall and reprocess all inadequately processed items.
4. Check with hospital maintenance for irregularities (e.g., electrical) or changes in the hospital steam supply (i.e., from standard $\geq 97\%$ steam, $< 3\%$ moisture). Any abnormalities should be reported to the person who performs sterilizer maintenance (e.g., medical engineering, sterilizer manufacturer).
5. Check to ensure the correct biological indicator was used and appropriately interpreted. If not, repeat using appropriate settings.

If steps 1 through 5 resolve the problem

6. If all three repeat biological indicators from three consecutive sterilizer cycles (step 2 above) are negative, put the sterilizer back in service.

If one or both biological indicators are positive, do one or more of the following until problem is resolved.

7.
 - A. Request an inspection of the equipment by sterilizer maintenance personnel.
 - B. Have hospital maintenance inspect the steam supply lines.
 - C. Discuss the abnormalities with the sterilizer manufacturer.
 - D. Repeat the biological indicator using a different manufacturer's indicator.

If step 7 does not resolve the problem

Close sterilizer down until the manufacturer can assure that it is operating properly. Retest at that time with biological indicators in three consecutive sterilizer cycles.

Modified from Bryce.⁸³⁹

Disclosure of Financial Interests and Relationships (2000- July 2004)

William A. Rutala: Honoraria from Advanced Sterilization Products, Kimberly-Clark; consultation with Advanced Sterilization Products, Aesculap, Clorox, 3M, SC Johnson, Intelligent Biocides, Metrex; and an educational grant from Consumer Specialty Products Association, Kimberly-Clark.

David J. Weber: Honoraria from Consumer Specialty Products Association; consultation with Clorox; and educational grant from Consumer Specialty Products Association.

REFERENCES

1. Garner JS, Favero MS. CDC Guideline for handwashing and hospital environmental control, 1985. *Infect. Control* 1986;7:231-43.
2. Centers for Disease Control. Ambulatory and inpatient procedures in the United States, 1996. Atlanta, GA, 1998:1-39.
3. Uttley AH, Simpson RA. Audit of bronchoscope disinfection: a survey of procedures in England and Wales and incidents of mycobacterial contamination. *J. Hosp. Infect.* 1994;26:301-8.
4. Zaidi M, Angulo M, Sifuentes-Osornio J. Disinfection and sterilization practices in Mexico. *J. Hosp. Infect.* 1995;31:25-32.
5. McCarthy GM, Koval JJ, John MA, MacDonald JK. Infection control practices across Canada: do dentists follow the recommendations? *J. Can. Dent. Assoc.* 1999;65:506-11.
6. Spach DH, Silverstein FE, Stamm WE. Transmission of infection by gastrointestinal endoscopy and bronchoscopy. *Ann. Intern. Med.* 1993;118:117-28.
7. Weber DJ, Rutala WA. Lessons from outbreaks associated with bronchoscopy. *Infect. Control Hosp. Epidemiol.* 2001;22:403-8.
8. Weber DJ, Rutala WA, DiMarino AJ, Jr. The prevention of infection following gastrointestinal endoscopy: the importance of prophylaxis and reprocessing. In: DiMarino AJ, Jr, Benjamin SB, eds. *Gastrointestinal diseases: an endoscopic approach*. Thorofare, NJ: Slack Inc., 2002:87-106.
9. Meyers H, Brown-Elliott BA, Moore D, et al. An outbreak of *Mycobacterium chelonae* infection following liposuction. *Clin. Infect. Dis.* 2002;34:1500-7.
10. Lowry PW, Jarvis WR, Oberle AD, et al. *Mycobacterium chelonae* causing otitis media in an ear-nose-and-throat practice. *N. Engl. J. Med.* 1988;319:978-82.
11. Centers for Disease Control and Prevention. *Pseudomonas aeruginosa* infections associated with transrectal ultrasound-guided prostate biopsies--Georgia, 2005. *MMWR CDC Surveill. Summ.* 2006;55:776-7.
12. Mehta AC, Prakash UBS, Garland R, et al. Prevention of flexible bronchoscopy-associated infection. *Chest* 2006;128:1742-55.
13. Favero MS, Bond WW. Chemical disinfection of medical and surgical materials. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001:881-917.
14. Spaulding EH. Chemical disinfection of medical and surgical materials. In: Lawrence C, Block SS, eds. *Disinfection, sterilization, and preservation*. Philadelphia: Lea & Febiger, 1968:517-31.
15. Simmons BP. CDC guidelines for the prevention and control of nosocomial infections. Guideline for hospital environmental control. *Am. J. Infect. Control* 1983;11:97-120.
16. Block SS. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001.
17. Rutala WA, 1994, 1995, and 1996 APIC Guidelines Committee. APIC guideline for selection and use of disinfectants. Association for Professionals in Infection Control and Epidemiology, Inc. *Am. J. Infect. Control* 1996;24:313-42.
18. Rutala WA. Disinfection, sterilization and waste disposal. In: Wenzel RP, ed. *Prevention and control of nosocomial infections*. Baltimore: Williams and Wilkins, 1997:539-93.
19. Rutala WA. APIC guideline for selection and use of disinfectants. *Am. J. Infect. Control* 1990;18:99-117.
20. Association of peri-Operative Registered Nurses. Recommended practices for high-level disinfection. *AORN J.* 2005;81:402-12.
21. Garner JS, Favero MS. CDC guidelines for the prevention and control of nosocomial infections. Guideline for handwashing and hospital environmental control, 1985. Supersedes guideline for hospital environmental control published in 1981. *Am. J. Infect. Control* 1986;14:110-29.
22. Centers for Disease Control. Guidelines for prevention of transmission of human immunodeficiency virus

- and hepatitis B virus to health-care and public-safety workers. *MMWR* 1989;38:1-37.
23. Centers for Disease Control. Guidelines for Environmental Infection Control in Health-Care Facilities, 2003. *MMWR* 2003;52 (No. RR-10):1-44.
 24. Bhattachatyya M, Kepnes LJ. The effectiveness of immersion disinfection for flexible fiberoptic laryngoscopes. *Otolaryngol Head Neck* 2004;130:681-5.
 25. Hamasuna R, Nose K, Sueyoshi T, Nagano M, Hasui Y, Osada Y. High-level disinfection of cystoscopic equipment with ortho-phthalaldehyde solution. *J. Hosp. Infect.* 2004;57:346-8.
 26. Foliente RL KB, Aprecio RM, Bains HJ, Kettering JD, Chen YK. Efficacy of high-level disinfectants for reprocessing gastrointestinal endoscopes in simulated-use testing. *Gastrointest. Endosc.* 2001;53:456-62.
 27. Kovacs BJ, Chen YK, Kettering JD, Aprecio RM, Roy I. High-level disinfection of gastrointestinal endoscopes: are current guidelines adequate? *Am. J. Gastroenterol.* 1999;94:1546-50.
 28. Rutala WA, Clontz EP, Weber DJ, Hoffmann KK. Disinfection practices for endoscopes and other semicritical items. *Infect. Control Hosp. Epidemiol.* 1991;12:282-8.
 29. Phillips J, Hulka B, Hulka J, Keith D, Keith L. Laparoscopic procedures: The American Association of Gynecologic Laparoscopists' Membership Survey for 1975. *J. Reprod. Med.* 1977;18:227-32.
 30. Muscarella LF. Current instrument reprocessing practices: Results of a national survey. *Gastrointestinal Nursing* 2001;24:253-60.
 31. Wright EP, Collins CH, Yates MD. *Mycobacterium xenopi* and *Mycobacterium kansasii* in a hospital water supply. *J. Hosp. Infect.* 1985;6:175-8.
 32. Wallace RJ, Jr., Brown BA, Driffith DE. Nosocomial outbreaks/pseudo-outbreaks caused by nontuberculous mycobacteria. *Annu. Rev. Microbiol.* 1998;52:453-90.
 33. Mitchell DH, Hicks LJ, Chiew R, Montanaro JC, Chen SC. Pseudoepidemic of *Legionella pneumophila* serogroup 6 associated with contaminated bronchoscopes. *J. Hosp. Infect.* 1997;37:19-23.
 34. Meenhorst PL, Reingold AL, Groothuis DG, et al. Water-related nosocomial pneumonia caused by *Legionella pneumophila* serogroups 1 and 10. *J. Infect. Dis.* 1985;152:356-64.
 35. Atlas RM. *Legionella*: from environmental habitats to disease pathology, detection and control. *Environ. Microbiol.* 1999;1:283-93.
 36. Rutala WA, Weber DJ. Water as a reservoir of nosocomial pathogens. *Infect. Control Hosp. Epidemiol.* 1997;18:609-16.
 37. Weber DJ, Rutala WA. Environmental issues and nosocomial infections. In: Wenzel RP, ed. *Prevention and control of nosocomial infections*. Baltimore: Williams and Wilkins, 1997:491-514.
 38. Society of Gastroenterology Nurses and Associates. Standards for infection control and reprocessing of flexible gastrointestinal endoscopes. *Gastroenterol. Nurs.* 2000;23:172-9.
 39. Gerding DN, Peterson LR, Vennes JA. Cleaning and disinfection of fiberoptic endoscopes: evaluation of glutaraldehyde exposure time and forced-air drying. *Gastroenterology* 1982;83:613-8.
 40. Society of Gastroenterology Nurses and Associates. Guideline for the use of high-level disinfectants and sterilants in reprocessing of flexible gastrointestinal endoscopes.
 41. Turner AG, Higgins MM, Craddock JG. Disinfection of immersion tanks (Hubbard) in a hospital burn unit. *Arch. Environ. Health* 1974;28:101-4.
 42. Rutala DR, Rutala WA, Weber DJ, Thomann CA. Infection risks associated with spirometry. *Infect. Control Hosp. Epidemiol.* 1991;12:89-92.
 43. Kohn WG, Collins AS, Cleveland JL, Harte JA, Eklund KJ, Malvitz DM. Guidelines for infection control in dental health-care settings-2003. *MMWR* 2003;52 (no. RR-17):1-67.
 44. Sehulster L, Chinn RYW, Healthcare Infection Control Practices Advisory Committee. Guidelines for environmental infection control in health-care facilities. *MMWR* 2003;52:1-44.
 45. Rutala WA, White MS, Gergen MF, Weber DJ. Bacterial contamination of keyboards: Efficacy and functional impact of disinfectants. *Infect Control Hosp Epidemiol* 2006;27:372-7.
 46. Sattar SA, Lloyd-Evans N, Springthorpe VS, Nair RC. Institutional outbreaks of rotavirus diarrhoea: potential role of fomites and environmental surfaces as vehicles for virus transmission. *J. Hyg. (Lond)*. 1986;96:277-89.
 47. Weber DJ, Rutala WA. Role of environmental contamination in the transmission of vancomycin-resistant enterococci. *Infect. Control Hosp. Epidemiol.* 1997;18:306-9.
 48. Ward RL, Bernstein DI, Knowlton DR, et al. Prevention of surface-to-human transmission of rotaviruses by treatment with disinfectant spray. *J. Clin. Microbiol.* 1991;29:1991-6.

49. Tyler R, Ayliffe GA, Bradley C. Virucidal activity of disinfectants: studies with the poliovirus. *J. Hosp. Infect.* 1990;15:339-45.
50. Gwaltney JM, Jr., Hendley JO. Transmission of experimental rhinovirus infection by contaminated surfaces. *Am. J. Epidemiol.* 1982;116:828-33.
51. Sattar SA, Jacobsen H, Springthorpe VS, Cusack TM, Rubino JR. Chemical disinfection to interrupt transfer of rhinovirus type 14 from environmental surfaces to hands. *Appl. Environ. Microbiol.* 1993;59:1579-85.
52. Sattar SA, Jacobsen H, Rahman H, Cusack TM, Rubino JR. Interruption of rotavirus spread through chemical disinfection. *Infect. Control Hosp. Epidemiol.* 1994;15:751-6.
53. Rutala WA, Barbee SL, Aguiar NC, Sobsey MD, Weber DJ. Antimicrobial activity of home disinfectants and natural products against potential human pathogens. *Infect. Control Hosp. Epidemiol.* 2000;21:33-8.
54. Silverman J, Vazquez JA, Sobel JD, Zervos MJ. Comparative in vitro activity of antiseptics and disinfectants versus clinical isolates of *Candida* species. *Infect. Control Hosp. Epidemiol.* 1999;20:676-84.
55. Best M, Sattar SA, Springthorpe VS, Kennedy ME. Efficacies of selected disinfectants against *Mycobacterium tuberculosis*. *J. Clin. Microbiol.* 1990;28:2234-9.
56. Best M, Kennedy ME, Coates F. Efficacy of a variety of disinfectants against *Listeria* spp. *Appl. Environ. Microbiol.* 1990;56:377-80.
57. Best M, Springthorpe VS, Sattar SA. Feasibility of a combined carrier test for disinfectants: studies with a mixture of five types of microorganisms. *Am. J. Infect. Control* 1994;22:152-62.
58. Mbithi JN, Springthorpe VS, Sattar SA. Chemical disinfection of hepatitis A virus on environmental surfaces. *Appl. Environ. Microbiol.* 1990;56:3601-4.
59. Springthorpe VS, Grenier JL, Lloyd-Evans N, Sattar SA. Chemical disinfection of human rotaviruses: efficacy of commercially-available products in suspension tests. *J. Hyg. (Lond).* 1986;97:139-61.
60. Akamatsu T, Tabata K, Hironga M, Kawakami H, Uyeda M. Transmission of *Helicobacter pylori* infection via flexible fiberoptic endoscopy. *Am. J. Infect. Control* 1996;24:396-401.
61. Sattar SA, Springthorpe VS. Survival and disinfectant inactivation of the human immunodeficiency virus: a critical review. *Rev. Infect. Dis.* 1991;13:430-47.
62. Resnick L, Veren K, Salahuddin SZ, Tondreau S, Markham PD. Stability and inactivation of HTLV-III/LAV under clinical and laboratory environments. *JAMA* 1986;255:1887-91.
63. Weber DJ, Barbee SL, Sobsey MD, Rutala WA. The effect of blood on the antiviral activity of sodium hypochlorite, a phenolic, and a quaternary ammonium compound. *Infect. Control Hosp. Epidemiol.* 1999;20:821-7.
64. Rice EW, Clark RM, Johnson CH. Chlorine inactivation of *Escherichia coli* O157:H7. *Emerg. Infect. Dis.* 1999;5:461-3.
65. Pentella MA, Fisher T, Chandler S, Britt-Ohrmund T, Kwa BH, Yangco BG. Are disinfectants accurately prepared for use in hospital patient care areas? *Infect. Control Hosp. Epidemiol.* 2000;21:103.
66. Bhalla A, Pultz NJ, Gries DM, et al. Acquisition of nosocomial pathogens on hands after contact with environmental surfaces near hospitalized patients. *Infect. Control Hosp. Epidemiol.* 2004;25:164-7.
67. Ray AJ, Hoyen CK, Taub TF, Eckstein EC, Donskey CJ. Nosocomial transmission of vancomycin-resistant enterococci from surfaces. *JAMA* 2002;287:1400-1.
68. Westwood JC, Mitchell MA, Legace S. Hospital sanitation: the massive bacterial contamination of the wet mop. *Appl. Microbiol.* 1971;21:693-7.
69. Rutala WA, Weber DJ. Disinfection of endoscopes: review of new chemical sterilants used for high-level disinfection. *Infect. Control Hosp. Epidemiol.* 1999;20:69-76.
70. Russell AD. Bacterial spores and chemical sporicidal agents. *Clin. Microbiol. Rev.* 1990;3:99-119.
71. Terleckyj B, Axler DA. Quantitative neutralization assay of fungicidal activity of disinfectants. *Antimicrob. Agents Chemother.* 1987;31:794-8.
72. Klein M, DeForest A. The inactivation of viruses by germicides. *Chem. Specialists Manuf. Assoc. Proc.* 1963;49:116-8.
73. Rutala WA, Cole EC, Wannamaker NS, Weber DJ. Inactivation of *Mycobacterium tuberculosis* and *Mycobacterium bovis* by 14 hospital disinfectants. *Am. J. Med.* 1991;91:267S-271S.
74. Robison RA, Bodily HL, Robinson DF, Christensen RP. A suspension method to determine reuse life of chemical disinfectants during clinical use. *Appl. Environ. Microbiol.* 1988;54:158-64.
75. Isenberg HD, Giugliano ER, France K, Alperstein P. Evaluation of three disinfectants after in-use stress. *J.*

- Hosp. Infect. 1988;11:278-85.
76. Cole EC, Rutala WA, Nessen L, Wannamaker NS, Weber DJ. Effect of methodology, dilution, and exposure time on the tuberculocidal activity of glutaraldehyde-based disinfectants. *Appl. Environ. Microbiol.* 1990;56:1813-7.
 77. Power EG, Russell AD. Sporicidal action of alkaline glutaraldehyde: factors influencing activity and a comparison with other aldehydes. *J. Appl. Bacteriol.* 1990;69:261-8.
 78. Rutala WA, Gergen MF, Weber DJ. Sporicidal activity of chemical sterilants used in hospitals. *Infect. Control Hosp. Epidemiol.* 1993;14:713-8.
 79. Rutala WA, Gergen MF, Weber DJ. Inactivation of *Clostridium difficile* spores by disinfectants. *Infect. Control Hosp. Epidemiol.* 1993;14:36-9.
 80. Ascenzi JM, Ezzell RJ, Wendt TM. A more accurate method for measurement of tuberculocidal activity of disinfectants. *Appl. Environ. Microbiol.* 1987;53:2189-92.
 81. Collins FM. Use of membrane filters for measurement of mycobactericidal activity of alkaline glutaraldehyde solution. *Appl. Environ. Microbiol.* 1987;53:737-9.
 82. Rubbo SD, Gardner JF, Webb RL. Biocidal activities of glutaraldehyde and related compounds. *J. Appl. Bacteriol.* 1967;30:78-87.
 83. Rutala WA, Weber DJ. FDA labeling requirements for disinfection of endoscopes: a counterpoint. *Infect. Control Hosp. Epidemiol.* 1995;16:231-5.
 84. Collins FM. Kinetics of the tuberculocidal response by alkaline glutaraldehyde in solution and on an inert surface. *J. Appl. Bacteriol.* 1986;61:87-93.
 85. Food and Drug Administration. 2005. FDA-cleared sterilants and high-level disinfectants with general claims for processing reusable medical and dental devices, May 13, 2005. www.fda.gov/cdrh/ode/germlab.html.
 86. Crow S, Metcalf RW, Beck WC, Birnbaum D. Disinfection or sterilization? Four views on arthroscopes. *AORN J.* 1983;37:854-9, 862-8.
 87. Loffer FD. Disinfection vs. sterilization of gynecologic laparoscopy equipment. The experience of the Phoenix Surgicenter. *J. Reprod. Med.* 1980;25:263-6.
 88. Johnson LL, Shneider DA, Austin MD, Goodman FG, Bullock JM, DeBruin JA. Two per cent glutaraldehyde: a disinfectant in arthroscopy and arthroscopic surgery. *J. Bone Joint Surg.* 1982;64:237-9.
 89. Burns S, Edwards M, Jennings J, et al. Impact of variation in reprocessing invasive fiberoptic scopes on patient outcomes. *Infect. Control Hosp. Epidemiol.* 1996;17(suppl):P42.
 90. Fuselier HA, Jr., Mason C. Liquid sterilization versus high level disinfection in the urologic office. *Urology* 1997;50:337-40.
 91. Muscarella LF. High-level disinfection or "sterilization" of endoscopes? *Infect. Control Hosp. Epidemiol.* 1996;17:183-7.
 92. Miles RS. What standards should we use for the disinfection of large equipment? *J. Hosp. Infect.* 1991;18:264-73.
 93. Lee RM, Kozarek RA, Sumida SE, Raltz SL. Risk of contamination of sterile biopsy forceps in disinfected endoscopes. *Gastrointest. Endosc.* 1998;47:377-81.
 94. Kinney TP, Kozarek RA, Raltz S, Attia F. Contamination of single-use biopsy forceps: A prospective in vitro analysis. *Gastrointest. Endosc.* 2002;56:209-12.
 95. Centers for Disease Control. Recommendations for preventing possible transmission of human T-lymphotropic virus type III/lymphadenopathy-associated virus from tears. *MMWR* 1985;34:533-4.
 96. Lettau LA, Bond WW, McDougal JS. Hepatitis and diaphragm fitting. *JAMA* 1985;254:752.
 97. Schembre DB. Infectious complications associated with gastrointestinal endoscopy. *Gastrointest. Endosc. Clin. N. Am.* 2000;10:215-32.
 98. Nelson DB. Infectious disease complications of GI endoscopy: Part II, exogenous infections. *Gastrointest. Endosc.* 2003;57:695-711.
 99. Chu NS, Favero M. The microbial flora of the gastrointestinal tract and the cleaning of flexible endoscopes. *Gastrointest. Endosc. Clin. N. Am.* 2000;10:233-44.
 100. Alfa MJ, Sitter DL. In-hospital evaluation of orthophthalaldehyde as a high level disinfectant for flexible endoscopes. *J. Hosp. Infect.* 1994;26:15-26.
 101. Vesley D, Melson J, Stanley P. Microbial bioburden in endoscope reprocessing and an in-use evaluation of the high-level disinfection capabilities of Cidex PA. *Gastroenterol. Nurs.* 1999;22:63-8.

102. Chu NS, McAlister D, Antonoplos PA. Natural bioburden levels detected on flexible gastrointestinal endoscopes after clinical use and manual cleaning. *Gastrointest. Endosc.* 1998;48:137-42.
103. Rutala WA, Weber DJ. Reprocessing endoscopes: United States perspective. *J. Hosp. Infect.* 2004;56:S27-S39.
104. Hanson PJ, Gor D, Clarke JR, et al. Contamination of endoscopes used in AIDS patients. *Lancet* 1989;2:86-8.
105. Hanson PJ, Gor D, Clarke JR, et al. Recovery of the human immunodeficiency virus from fiberoptic bronchoscopes. *Thorax* 1991;46:410-2.
106. Chaufour X, Deva AK, Vickery K, et al. Evaluation of disinfection and sterilization of reusable angioscopes with the duck hepatitis B model. *J. Vasc. Surg.* 1999;30:277-82.
107. Cheung RJ, Ortiz D, DiMarino AJ, Jr. GI endoscopic reprocessing practices in the United States. *Gastrointest. Endosc.* 1999;50:362-8.
108. American Society for Gastrointestinal Endoscopy. Position statement: reprocessing of flexible gastrointestinal endoscopes. *Gastrointest. Endosc.* 1996;43:541-6.
109. Food and Drug Administration. Content and format of premarket notification [510(k)] submissions for liquid chemical sterilants/high level disinfectants. www.fda.gov/cdrh/ode/397 2000.
110. Urayama S, Kozarek RA, Sumida S, Raltz S, Merriam L, Pethigal P. Mycobacteria and glutaraldehyde: is high-level disinfection of endoscopes possible? *Gastrointest. Endosc.* 1996;43:451-6.
111. Jackson J, Leggett JE, Wilson DA, Gilbert DN. *Mycobacterium gordonae* in fiberoptic bronchoscopes. *Am. J. Infect. Control* 1996;24:19-23.
112. Martiny H, Floss H, Zuhlsdorf B. The importance of cleaning for the overall results of processing endoscopes. *J. Hosp. Infect.* 2004;56:S16-S22.
113. Alvarado CJ, Reichelderfer M. APIC guideline for infection prevention and control in flexible endoscopy. Association for Professionals in Infection Control. *Am. J. Infect. Control* 2000;28:138-55.
114. Society of Gastroenterology Nurses and Associates. Guideline for the use of high-level disinfectants and sterilants for reprocessing of flexible gastrointestinal endoscopes. *Gastroenterol. Nurs.* 2000;23:180-7.
115. Society of Gastroenterology Nurses and Associates. Standards of infection control in reprocessing of flexible gastrointestinal endoscopes. *Gastroenterol. Nurs.* 2006;29:142-8.
116. Nelson DB, Jarvis WR, Rutala WA, et al. Multi-society guideline for reprocessing flexible gastrointestinal endoscopes. *Infect Control Hosp Epidemiol* 2003;24:532-537.
117. Martin MA, Reichelderfer M, 1991, and 1993 APIC Guidelines Committee. APIC guidelines for infection prevention and control in flexible endoscopy. *Am. J. Infect. Control* 1994;22:19-38.
118. Rey JF, Halfon P, Feryn JM, Khiri H, Masseyeff MF, Ouzan D. Risk of transmission of hepatitis C virus by digestive endoscopy. *Gastroenterol. Clin. Biol.* 1995;19:346-9.
119. Cronmiller JR, Nelson DK, Jackson DK, Kim CH. Efficacy of conventional endoscopic disinfection and sterilization methods against *Helicobacter pylori* contamination. *Helicobacter* 1999;4:198-203.
120. Sartor C, Charrel RN, de Lamballerie X, Sambuc R, De Micco P, Boubli L. Evaluation of a disinfection procedure for hysteroscopes contaminated by hepatitis C virus. *Infect. Control Hosp. Epidemiol.* 1999;20:434-6.
121. Hanson PJ, Chadwick MV, Gaya H, Collins JV. A study of glutaraldehyde disinfection of fiberoptic bronchoscopes experimentally contaminated with *Mycobacterium tuberculosis*. *J. Hosp. Infect.* 1992;22:137-42.
122. Merighi A, Contato E, Scagliarini R, et al. Quality improvement in gastrointestinal endoscopy: microbiologic surveillance of disinfection. *Gastrointest. Endosc.* 1996;43:457-62.
123. Bond WW. Endoscope reprocessing: Problems and solutions. In: Rutala WA, ed. *Disinfection, sterilization, and antisepsis in healthcare*. Champlain, New York: Polyscience Publications, 1998:151-163.
124. Deva AK, Vickery K, Zou J, West RH, Harris JP, Cossart YE. Establishment of an in-use testing method for evaluating disinfection of surgical instruments using the duck hepatitis B model. *J. Hosp. Infect.* 1996;33:119-30.
125. Hanson PJ, Gor D, Jeffries DJ, Collins JV. Elimination of high titre HIV from fiberoptic endoscopes. *Gut* 1990;31:657-9.
126. Wu MS, Wang JT, Yang JC, et al. Effective reduction of *Helicobacter pylori* infection after upper gastrointestinal endoscopy by mechanical washing of the endoscope. *Hepatogastroenterology.* 1996;43:1660-4.

127. Kirschke DL, Jones TF, Craig AS, et al. *Pseudomonas aeruginosa* and *Serratia marcescens* contamination associated with a manufacturing defect in bronchoscopes. *N. Engl. J. Med.* 2003;348:214-20.
128. Srinivasan A, Wolfenden LL, Song X, et al. An outbreak of *Pseudomonas aeruginosa* infections associated with flexible bronchoscopes. *N. Engl. J. Med.* 2003;348:221-7.
129. Kaczmarek RG, Moore RM, Jr., McCrohan J, et al. Multi-state investigation of the actual disinfection/sterilization of endoscopes in health care facilities. *Am. J. Med.* 1992;92:257-61.
130. Bradley CR, Babb JR. Endoscope decontamination: automated vs. manual. *J. Hosp. Infect.* 1995;30:537-42.
131. Muscarella LF. Advantages and limitations of automatic flexible endoscope reprocessors. *Am. J. Infect. Control* 1996;24:304-9.
132. Muscarella LF. Automatic flexible endoscope reprocessors. *Gastrointest. Endosc. Clin. N. Am.* 2000;10:245-57.
133. Alvarado CJ, Stolz SM, Maki DG. Nosocomial infections from contaminated endoscopes: a flawed automated endoscope washer. An investigation using molecular epidemiology. *Am. J. Med.* 1991;91:272S-280S.
134. Fraser VJ, Jones M, Murray PR, Medoff G, Zhang Y, Wallace RJ, Jr. Contamination of flexible fiberoptic bronchoscopes with *Mycobacterium chelonae* linked to an automated bronchoscope disinfection machine. *Am. Rev. Respir. Dis.* 1992;145:853-5.
135. Cooke RP, Whyman-Morris A, Umasankar RS, Goddard SV. Bacteria-free water for automatic washer-disinfectors: an impossible dream? *J. Hosp. Infect.* 1998;39:63-5.
136. Muscarella LF. Deja Vu...All over again? The importance of instrument drying. *Infect. Control Hosp. Epidemiol.* 2000;21:628-9.
137. Rutala WA, Weber DJ. Importance of lumen flow in liquid chemical sterilization. *Am. J. Infect. Control* 1999;20:458-9.
138. Dwyer DM, Klein EG, Istre GR, Robinson MG, Neumann DA, McCoy GA. *Salmonella newport* infections transmitted by fiberoptic colonoscopy. *Gastrointest. Endosc.* 1987;33:84-7.
139. Wheeler PW, Lancaster D, Kaiser AB. Bronchopulmonary cross-colonization and infection related to mycobacterial contamination of suction valves of bronchoscopes. *J. Infect. Dis.* 1989;159:954-8.
140. Bond WW. Virus transmission via fiberoptic endoscope: recommended disinfection. *JAMA* 1987;257:843-4.
141. Lynch DA, Porter C, Murphy L, Axon AT. Evaluation of four commercial automatic endoscope washing machines. *Endoscopy* 1992;24:766-70.
142. Bond WW. Disinfection and endoscopy: microbial considerations. *J. Gastroenterol. Hepatol.* 1991;6:31-6.
143. Nelson D. Newer technologies for endoscope disinfection: electrolyzed acid water and disposable-component endoscope systems. *Gastrointest. Endosc. Clin. N. Am.* 2000;10:319-28.
144. Silberman HD. Non-inflatable sterile sheath for introduction of the flexible nasopharyngolaryngoscope. *Ann Otol, Rhinol, Laryngol* 2001;110:385-7.
145. Kruse A, Rey JF. Guidelines on cleaning and disinfection in GI endoscopy. Update 1999. The European Society of Gastrointestinal Endoscopy. *Endoscopy* 2000;32:77-80.
146. British Thoracic Society. British Thoracic Society guidelines on diagnostic flexible bronchoscopy. *Thorax* 2001;56:1-21.
147. Association of Operating Room Nurses. Recommended practices for use and care of endoscopes. 2000 standards, recommended practices, and guidelines. Denver, CO: AORN, 2000:243-7.
148. British Society of Gastroenterology. Cleaning and disinfection of equipment for gastrointestinal endoscopy. Report of a working party of the British Society of Gastroenterology Endoscope Committee. *Gut* 1998;42:585-93.
149. Jackson FW, Ball MD. Correction of deficiencies in flexible fiberoptic sigmoidoscope cleaning and disinfection technique in family practice and internal medicine offices. *Arch. Fam. Med.* 1997;6:578-82.
150. Orsi GB, Filocamo A, Di Stefano L, Tittobello A. Italian National Survey of Digestive Endoscopy Disinfection Procedures. *Endoscopy* 1997;29:732-8; quiz 739-40.
151. Honeybourne D, Neumann CS. An audit of bronchoscopy practice in the United Kingdom: a survey of adherence to national guidelines. *Thorax* 1997;52:709-13.
152. Michele TM, Cronin WA, Graham NM, et al. Transmission of *Mycobacterium tuberculosis* by a fiberoptic bronchoscope. Identification by DNA fingerprinting. *JAMA* 1997;278:1093-5.

153. Bronowicki JP, Venard V, Botte C, et al. Patient-to-patient transmission of hepatitis C virus during colonoscopy. *N. Engl. J. Med.* 1997;337:237-40.
154. Agerton T, Valway S, Gore B, et al. Transmission of a highly drug-resistant strain (strain W1) of *Mycobacterium tuberculosis*. Community outbreak and nosocomial transmission via a contaminated bronchoscope. *JAMA* 1997;278:1073-7.
155. Food and Drug Administration, Centers for Disease Control and Prevention. FDA and CDC public health advisory: Infections from endoscopes inadequately reprocessed by an automated endoscope reprocessing system, Food and Drug Administration, Rockville, MD. 1999.
156. Nelson DB, Muscarella LF. Current issues in endoscope reprocessing and infection control during gastrointestinal endoscopy. *World J Gastroenterol* 2006;12:3953-64.
157. Riley R, Beanland C, Bos H. Establishing the shelf life of flexible colonoscopes. *Gastroenterol. Nurs.* 2002;25:114-9.
158. Rejchrt S, Cermak P, Pavlatova L, Mickova E, Bures J. Bacteriologic testing of endoscopes after high-level disinfection. *Gastrointest. Endosc.* 2004;60:76-8.
159. Willis C. Bacteria-free endoscopy rinse water - a realistic aim? *Epidemiol. Infect.* 2005;134:279-84.
160. Humphreys H, McGrath H, McCormick PA, Walsh C. Quality of final rinse water used in washer-disinfectors for endoscopes. *J. Hosp. Infect.* 2002;51:151-3.
161. Pang J, Perry P, Ross A, Forbes GM. Bacteria-free rinse water for endoscope disinfection. *Gastrointest. Endosc.* 2002;56:402-6.
162. Leung J, Vallero R, Wilson R. Surveillance cultures to monitor quality of gastrointestinal endoscope reprocessing. *Am. J. Gastroenterol.* 2003;98.
163. Moses FM, Lee J. Surveillance cultures to monitor quality of gastrointestinal endoscope reprocessing. *Am. J. Gastroenterol.* 2003;98:77-81.
164. Tunuguntla A, Sullivan MJ. Monitoring quality of flexible endoscopic disinfection by microbiologic surveillance cultures. *Tennessee Med* 2004;October:453-6.
165. Muscarella LF. Application of environmental sampling to flexible endoscope reprocessing: The importance of monitoring the rinse water. *Infect. Control Hosp. Epidemiol.* 2002;23:285-9.
166. Fraser TG, Reiner S, Malcznski M, Yarnold PR, Warren J, Noskin GA. Multidrug-resistant *Pseudomonas aeruginosa* cholangiopancreatography: Failure of routine endoscope cultures to prevent an outbreak. *Infect Control Hosp Epidemiol* 2004;25:856-9.
167. Bond WW, Hedrick ER. Microbiological culturing of environmental and medical-device surfaces. In: Isenberg HD, and M.J.R. Gilchrist, ed. *Clinical Microbiology Procedures Handbook*, Section 11, Epidemiologic and Infection Control Microbiology. Washington, DC: American Society for Microbiology, 1992:11.10.1-11.10.9.
168. Murray PR, Baron EJ, Pfaller MA, Jorgensen JH, Tenover FC, Tenover FC, eds. *Manual of Clinical Microbiology*. In: Murray PR, Baron EJ, Pfaller MA, Jorgensen JH, Tenover FC, Tenover FC, eds. Washington, D.C.: American Society for Microbiology Press, 2003.
169. Blob R, Kampf G. Test models to determine cleaning efficacy with different types of bioburden and its clinical correlation. *J. Hosp. Infect.* 2004;56 (suppl):S44-S48.
170. Obee PC, Griffith CJ, Cooper RA, Cooke RP, Bennion NE, Lewis M. Real-time monitoring in managing the decontamination of flexible gastrointestinal endoscopes. *Am. J. Infect. Control* 2005;33:202-6.
171. Sciortino CV, Xia EL, Mozee A. Assessment of a novel approach to evaluate the outcome of endoscope reprocessing. *Infect Control Hosp Epidemiol* 2004;25:284-90.
172. Murphy C. Inactivated glutaraldehyde: Lessons for infection control. *Am. J. Infect. Control* 1998;26:159-60.
173. Carsaw H, Debacker N. Recall of patients after use of inactive batch of Cidex disinfection solution in Belgian hospitals, Fifth International Conference of the Hospital Infection Society, Edinburgh, September 15-18, 2002. Hospital Infections Society.
174. Ad hoc Committee on Infection Control in the Handling of Endoscopic Equipment. Guidelines for preparation of laparoscopic instrumentation. *AORN J.* 1980;32:65-6, 70, 74, 76.
175. Taylor EW, Mehtar S, Cowan RE, Feneley RC. Endoscopy: disinfectants and health. Report of a meeting held at the Royal College of Surgeons of England, February 1993. *J. Hosp. Infect.* 1994;28:5-14.
176. Hulka JF, Wisler MG, Bruch C. A discussion: laparoscopic instrument sterilization. *Med. Instrum.* 1977;11:122-3.

177. Corson SL, Block S, Mintz C, Dole M, Wainwright A. Sterilization of laparoscopes. Is soaking sufficient? *J. Reprod. Med.* 1979;23:49-56.
178. Corson SL, Dole M, Kraus R, Richards L, Logan B. Studies in sterilization of the laparoscope: II. *J. Reprod. Med.* 1979;23:57-9.
179. Chan-Myers H, McAlister D, Antonoplos P. Natural bioburden levels detected on rigid lumened medical devices before and after cleaning. *Am. J. Infect. Control* 1997;25:471-6.
180. Rodrigues C, Mehta AC, Jha U, Bharucha M, Dastur FD, Udwardia TE. Nosocomial *Mycobacterium chelonae* infection in laparoscopic surgery. *Infect. Control Hosp. Epidemiol.* 2001;22:474-5.
181. Marshburn PB, Rutala WA, Wannamaker NS, Hulka JF. Gas and steam sterilization of assembled versus disassembled laparoscopic equipment. *Microbiologic studies. J. Reprod. Med.* 1991;36:483-7.
182. Bernhang AM. Clostridium pyoarthrosis following arthroscopy. *Arthroscopy* 1987;3:56-8.
183. D'Angelo GL, Ogilvie-Harris DJ. Septic arthritis following arthroscopy, with cost/benefit analysis of antibiotic prophylaxis. *Arthroscopy* 1988;4:10-4.
184. Weber DJ, Rutala WA. Nosocomial ocular infections. In: Mayhall CG, ed. *Infect. Control and Hosp. Epidemiol.* Philadelphia: Lippincott Williams & Wilkins, 1999:287-99.
185. Rutala WA, Peacock JE, Gergen MF, Sobsey MD, Weber DJ. Efficacy of hospital germicides against adenovirus 8, a common cause of epidemic keratoconjunctivitis in health care facilities. *Antimicrob. Agents Chemother.* 2006;50:1419-24.
186. Sattar SA, Springthorpe VS, Karim Y, Loro P. Chemical disinfection of non-porous inanimate surfaces experimentally contaminated with four human pathogenic viruses. *Epidemiol. Infect.* 1989;102:493-505.
187. Chronister CL. Structural damage to Schiottz tonometers after disinfection with solutions. *Optom. Vis. Sci.* 1997;74:164-6.
188. Nagington J, Sutehall GM, Whipp P. Tonometer disinfection and viruses. *Br. J. Ophthalmol.* 1983;67:674-6.
189. Craven ER, Butler SL, McCulley JP, Luby JP. Applanation tonometer tip sterilization for adenovirus type 8. *Ophthalmology* 1987;94:1538-40.
190. American Academy of Ophthalmology. Updated recommendations for ophthalmic practice in relation to the human immunodeficiency virus. American Academy of Ophthalmology, San Francisco, CA, 1988.
191. Pepose JS, Linette G, Lee SF, MacRae S. Disinfection of Goldmann tonometers against human immunodeficiency virus type 1. *Arch. Ophthalmol.* 1989;107:983-5.
192. Ventura LM, Dix RD. Viability of herpes simplex virus type 1 on the applanation tonometer. *Am. J. Ophthalmol.* 1987;103:48-52.
193. Koo D, Bouvier B, Wesley M, Courtright P, Reingold A. Epidemic keratoconjunctivitis in a university medical center ophthalmology clinic; need for re-evaluation of the design and disinfection of instruments. *Infect. Control Hosp. Epidemiol.* 1989;10:547-52.
194. Jernigan JA, Lowry BS, Hayden FG, et al. Adenovirus type 8 epidemic keratoconjunctivitis in an eye clinic: risk factors and control. *J. Infect. Dis.* 1993;167:1307-13.
195. Fritz S, Hust MH, Ochs C, Gratwohl I, Staiger M, Braun B. Use of a latex cover sheath for transesophageal echocardiography (TEE) instead of regular disinfection of the echoscope? *Clin. Cardiol.* 1993;16:737-40.
196. Lawrentschuk N, Chamberlain M. Sterile disposable sheath system for flexible cystoscopes. *Urology* 2005;66:1310-3.
197. Milki AA, Fisch JD. Vaginal ultrasound probe cover leakage: implications for patient care. *Fertil. Steril.* 1998;69:409-11.
198. Storment JM, Monga M, Blanco JD. Ineffectiveness of latex condoms in preventing contamination of the transvaginal ultrasound transducer head. *South. Med. J.* 1997;90:206-8.
199. Hignett M, Claman P. High rates of perforation are found in endovaginal ultrasound probe covers before and after oocyte retrieval for *in vitro* fertilization-embryo transfer. *J. Assist. Reprod. Genet.* 1995;12:606-9.
200. Amis S, Ruddy M, Kibbler CC, Economides DL, MacLean AB. Assessment of condoms as probe covers for transvaginal sonography. *J. Clin. Ultrasound* 2000;28:295-8.
201. Rooks VJ, Yancey MK, Elg SA, Brueske L. Comparison of probe sheaths for endovaginal sonography. *Obstet. Gynecol.* 1996;87:27-9.
202. Odwin CS, Fleischer AC, Kepple DM, Chiang DT. Probe covers and disinfectants for transvaginal transducers. *J. Diagnostic Med. Sonography* 1990;6:130-5.
203. Benson WG. Exposure to glutaraldehyde. *J. Soc. Occup. Med.* 1984;34:63-4.

204. Garland SM, de Crespigny L. Prevention of infection in obstetrical and gynaecological ultrasound practice. *Aust. N. Z. J. Obstet Gynaecol.* 1996;36:392-5.
205. Fowler C, McCracken D. US probes: risk of cross infection and ways to reduce it--comparison of cleaning methods. *Radiology* 1999;213:299-300.
206. Muradali D, Gold WL, Phillips A, Wilson S. Can ultrasound probes and coupling gel be a source of nosocomial infection in patients undergoing sonography? An in vivo and in vitro study. *AJR. Am. J. Roentgenol.* 1995;164:1521-4.
207. Lewis DL, Arens M, Appleton SS, et al. Cross-contamination potential with dental equipment. *Lancet* 1992;340:1252-4.
208. Lewis DL, Boe RK. Cross-infection risks associated with current procedures for using high-speed dental handpieces. *J. Clin. Microbiol.* 1992;30:401-6.
209. American Dental Association. Infection control recommendations for the dental office and the dental laboratory. *JADA* 1996;127:672-80.
210. Centers for Disease Control. Recommended Infection-Control Practices for Dentistry, 1993. *MMWR* 1993;41:1-12.
211. Department of Health and Human Services. Food and Drug Administration. Dental handpiece sterilization, Food and Drug Administration, Rockville, MD, 1992.
212. Silverstone SE, Hill DE. Evaluation of sterilization of dental handpieces by heating in synthetic compressor lubricant. *Gen. Dent.* 1999;47:158-60.
213. Goodman HS, Carpenter RD, Cox MR. Sterilization of dental instruments and devices: an update. *Am. J. Infect. Control* 1994;22:90-4.
214. Occupational Safety and Health Administration. Occupational exposure to bloodborne pathogens; final rule. *Fed. Regist.* 1991;56:64003-182.
215. Occupational Safety and Health Administration. OSHA Memorandum from Stephen Mallinger. EPA-registered disinfectants for HIV/HBV. Washington, DC, 1997.
216. Gurevich I, Dubin R, Cunha BA. Dental instrument and device sterilization and disinfection practices. *J. Hosp. Infect.* 1996;32:295-304.
217. Smith A, Dickson M, Aitken J, Bagg J. Contaminated dental instruments. *J. Hosp. Infect.* 2002;51:233-5.
218. Hastreiter RJ, Molinari JA, Falken MC, Roesch MH, Gleason MJ, Merchant VA. Effectiveness of dental office instrument sterilization procedures. *J. Am. Dent. Assoc.* 1991;122:51-6.
219. Andres MT, Tejerina JM, Fierro JF. Reliability of biologic indicators in a mail-return sterilization-monitoring service: a review of 3 years. *Quintessence Int.* 1995;26:865-70.
220. Miller CH, Sheldrake MA. The ability of biological indicators to detect sterilization failures. *Am. J. Dent.* 1994;7:95-7.
221. Sarin PS, Scheer DI, Kross RD. Inactivation of human T-cell lymphotropic retrovirus (HTLV-III) by LD. *N. Engl. J. Med.* 1985;313:1416.
222. Sarin PS, Scheer DI, Kross RD. Inactivation of human T-cell lymphotropic retrovirus. *Environ Microbiol* 1990;56:1423-8.
223. Ascenzi JM. Standardization of tuberculocidal testing of disinfectants. *J. Hosp. Infect.* 1991;18:256-63.
224. Bond WW, Favero MS, Petersen NJ, Ebert JW. Inactivation of hepatitis B virus by intermediate-to-high-level disinfectant chemicals. *J. Clin. Microbiol.* 1983;18:535-8.
225. Kobayashi H, Tsuzuki M. The effect of disinfectants and heat on hepatitis B virus. *J. Hosp. Infect.* 1984;5:93-4.
226. Spire B, Barre-Sinoussi F, Montagnier L, Chermann JC. Inactivation of lymphadenopathy associated virus by chemical disinfectants. *Lancet* 1984;2:899-901.
227. Martin LS, McDougal JS, Loskoski SL. Disinfection and inactivation of the human T lymphotropic virus type III/Lymphadenopathy-associated virus. *J. Infect. Dis.* 1985;152:400-3.
228. Centers for Disease Control. Recommendations for prevention of HIV transmission in health-care settings. *MMWR* 1987;36:S3-S18.
229. Prince DL, Prince HN, Thraenhart O, Muchmore E, Bonder E, Pugh J. Methodological approaches to disinfection of human hepatitis B virus. *J. Clin. Microbiol.* 1993;31:3296-304.
230. Prince DL, Prince RN, Prince HN. Inactivation of human immunodeficiency virus type 1 and herpes simplex virus type 2 by commercial hospital disinfectants. *Chemical Times and Trends* 1990;13:13-16.
231. Sattar SA, Springthorpe VS, Conway B, Xu Y. Inactivation of the human immunodeficiency virus: an

- update. Rev. Med. Microbiol. 1994;5:139-150.
232. Kaplan JC, Crawford DC, Durno AG, Schooley RT. Inactivation of human immunodeficiency virus by Betadine. Infect. Control 1987;8:412-4.
 233. Hanson PJ, Gor D, Jeffries DJ, Collins JV. Chemical inactivation of HIV on surfaces. Br. Med. J. 1989;298:862-4.
 234. Hanson PJ, Jeffries DJ, Collins JV. Viral transmission and fiberoptic endoscopy. J. Hosp. Infect. 1991;18:136-40.
 235. Payan C, Cottin J, Lemarie C, Ramont C. Inactivation of hepatitis B virus in plasma by hospital in-use chemical disinfectants assessed by a modified HepG2 cell culture. J. Hosp. Infect. 2001;47:282-87.
 236. Chanzy B, Duc-Bin DL, Rousset B, et al. Effectiveness of a manual disinfection procedure in eliminating hepatitis C virus from experimentally contaminated endoscopes. Gastrointest. Endosc. 1999;50:147-51.
 237. Druce JD, Russell JS, Birch CJ, Yates LA, Harper RW, Smolich JJ. A decontamination and sterilization protocol employed during reuse of cardiac electrophysiology catheters inactivates human immunodeficiency virus. Infect. Control Hosp. Epidemiol. 2003;24:184-90.
 238. Payan C, Pivert A, Kampf G, Ramont C, Cottin J, Lemarie C. Assessment of new chemical disinfectants for HBV virucidal activity in a cell culture model. J. Hosp. Infect. 2004;56 (suppl):S58-S63.
 239. Reynolds CD, Rhinehart E, Dreyer P, Goldmann DA. Variability in reprocessing policies and procedures for flexible fiberoptic endoscopes in Massachusetts hospitals. Am. J. Infect. Control 1992;20:283-90.
 240. Handsfield HH, Cummings MJ, Swenson PD. Prevalence of antibody to human immunodeficiency virus and hepatitis B surface antigen in blood samples submitted to a hospital laboratory. Implications for handling specimens. JAMA 1987;258:3395-7.
 241. Baker JL, Kelen GD, Sivertson KT, Quinn TC. Unsuspected human immunodeficiency virus in critically ill emergency patients. JAMA 1987;257:2609-11.
 242. Kelen GD, Fritz S, Qaqish B, et al. Unrecognized human immunodeficiency virus infection in emergency department patients. N. Engl. J. Med. 1988;318:1645-50.
 243. Ishino Y, Ido K, Sugano K. Contamination with hepatitis B virus DNA in gastrointestinal endoscope channels: Risk of infection on reuse after on-site cleaning. Endoscopy 2005;37:548-51.
 244. Agolini G, Russo A, Clementi M. Effect of phenolic and chlorine disinfectants on hepatitis C virus binding and infectivity. Am. J. Infect. Control 1999;27:236-9.
 245. Alter MJ, Tokars JI, Arduino MJ, Favero MS. Nosocomial infections with hemodialysis. In: Mayhall CG, ed. Infect. Control and Hosp. Epidemiol. Philadelphia: Lippincott Williams & Wilkins, 2004:1139-60.
 246. Centers for Disease Control. Recommendations for preventing transmission of infections among chronic hemodialysis patients. MMWR. 2001;50:1-43.
 247. Velandia M, Fridkin SK, Cardenas V, et al. Transmission of HIV in dialysis centre. Lancet 1995;345:1417-22.
 248. Guinto CH, Bottone EJ, Raffalli JT, Montecalvo MA, Wormser GP. Evaluation of dedicated stethoscopes as a potential source of nosocomial pathogens. Am. J. Infect. Control 2002;30:499-502.
 249. Tokars JI, Miller ER, Alter MJ, Arduino MJ. National surveillance of dialysis-associated diseases in the United States, 1997. Semin. Dialysis 2000;13:75-85.
 250. Amato RL, Curtis JM. The practical application of ozone in dialysis. Nephrol. News Issues 2002;September 27-9.
 251. Smeets E, Koonman J, van der Sande F, et al. Prevention of biofilm formation in dialysis water treatment systems. Kidney Int. 2003;63:1574-6.
 252. Finelli L, Miller JT, Tokars JI, Alter MJ, Arduino MJ. National surveillance of dialysis-associated diseases in the United States, 2002. Semin Dialysis 2005;18:52-61.
 253. Association for the Advancement of Medical Instrumentation. Reuse of hemodialyzers: Association for the Advancement of Medical Instrumentation, Arlington VA, 2002/2003:ANSI/AAMI RD47:2002 & RD47:2002/A1:2003; 1-32.
 254. Kim KH, Fekety R, Batts DH, et al. Isolation of *Clostridium difficile* from the environment and contacts of patients with antibiotic-associated colitis. J. Infect. Dis. 1981;143:42-50.
 255. Skoutelis AT, Westenfelder GO, Beckerdite M, Phair JP. Hospital carpeting and epidemiology of *Clostridium difficile*. Am. J. Infect. Control 1994;22:212-7.
 256. Wilcox MH, Fawley WN. Hospital disinfectants and spore formation by *Clostridium difficile*. Lancet 2000;356:1324.

257. Kaatz GW, Gitlin SD, Schaberg DR, et al. Acquisition of *Clostridium difficile* from the hospital environment. *Am. J. Epidemiol.* 1988;127:1289-94.
258. Wilcox MH, Fawley WN, Wigglesworth N, Parnell P, Verity P, Freeman J. Comparison of the effect of detergent versus hypochlorite cleaning on environmental contamination and incidence of *Clostridium difficile* infection. *J. Hosp. Infect.* 2003;54:109-14.
259. Mayfield JL, Leet T, Miller J, Mundy LM. Environmental control to reduce transmission of *Clostridium difficile*. *Clin. Infect. Dis.* 2000;31:995-1000.
260. Marler LM, Siders JA, Wolters LC, Pettigrew Y, Skitt BL, Allen SD. Comparison of five cultural procedures for isolation of *Clostridium difficile* from stools. *J. Clin. Microbiol.* 1992;30:514-6.
261. Brazier JG. The diagnosis of *Clostridium difficile*-associated disease. *J. Antimicrob. Chemother.* 1998;41 (suppl):29-40.
262. Perez J, Springthorpe S, Sattar SA. Activity of selected oxidizing microbicides against spores of *Clostridium difficile*: Relevance to environmental control. *Am. J. Infect. Control* 2005;33:320-5.
263. McFarland LV, Mulligan ME, Kwok RY, Stamm WE. Nosocomial acquisition of *Clostridium difficile* infection. *N. Engl. J. Med.* 1989;320:204-10.
264. Jernigan JA, Siegman-Igra Y, Guerrant RC, Farr BM. A randomized crossover study of disposable thermometers for prevention of *Clostridium difficile* and other nosocomial infections. *Infect Control Hosp Epidemiol* 1998;19:494-9.
265. Hughes CE, Gebhard RL, Peterson LR, Gerding DN. Efficacy of routine fiberoptic endoscope cleaning and disinfection for killing *Clostridium difficile*. *Gastrointest. Endosc.* 1986;32:7-9.
266. Dyas A, Das BC. The activity of glutaraldehyde against *Clostridium difficile*. *J. Hosp. Infect.* 1985;6:41-5.
267. Wullt M, Odenholt I, Walder M. Activity of three disinfectants and acidified nitrite against *Clostridium difficile* spores. *Infect Control Hosp Epidemiol* 2003;24:765-8.
268. Block C. The effect of Perasafe and sodium dichloroisocyanurate (NaDCC) against spores of *Clostridium difficile* and *Bacillus atrophaeus* on stainless steel and polyvinyl chloride surfaces. *J. Hosp. Infect.* 2004;57:144-8.
269. Occupational Safety and Health Administration. OSHA instruction CPL 2-2.44C. Office of Health Compliance Assistance. Washington, DC, 1992.
270. Rutala WA, Weber DJ. Infection control: the role of disinfection and sterilization. *J. Hosp. Infect.* 1999;43:S43-55.
271. Barbee SL, Weber DJ, Sobsey MD, Rutala WA. Inactivation of *Cryptosporidium parvum* oocyst infectivity by disinfection and sterilization processes. *Gastrointest. Endosc.* 1999;49:605-11.
272. Wilson JA, Margolin AB. The efficacy of three common hospital liquid germicides to inactivate *Cryptosporidium parvum* oocysts. *J. Hosp. Infect.* 1999;42:231-7.
273. Fayer R, Graczyk TK, Cranfield MR, Trout JM. Gaseous disinfection of *Cryptosporidium parvum* oocysts. *Appl. Environ. Microbiol.* 1996;62:3908-9.
274. Venkitanarayanan KS, Ezeike GO, Hung YC, Doyle MP. Inactivation of *Escherichia coli* O157:H7 and *Listeria monocytogenes* on plastic kitchen cutting boards by electrolyzed oxidizing water. *J. Food Prot.* 1999;62:857-60.
275. Taormina PJ, Beuchat LR. Behavior of enterohemorrhagic *Escherichia coli* O157:H7 on alfalfa sprouts during the sprouting process as influenced by treatments with various chemicals. *J. Food Prot.* 1999;62:850-6.
276. Taormina PJ, Beuchat LR. Comparison of chemical treatments to eliminate enterohemorrhagic *Escherichia coli* O157:H7 on alfalfa seeds. *J. Food Prot.* 1999;62:318-24.
277. Castillo A, Lucia LM, Kemp GK, Acuff GR. Reduction of *Escherichia coli* O157:H7 and *Salmonella typhimurium* on beef carcass surfaces using acidified sodium chlorite. *J. Food Prot.* 1999;62:580-4.
278. Graham DY, Osato MS. Disinfection of biopsy forceps and culture of *Helicobacter pylori* from gastric mucosal biopsies. *Am. J. Gastroenterol.* 1999;94:1422-3.
279. Kaneko H, Mitsuma T, Kotera H, Uchida K, Furusawa A, Morise K. Are routine cleaning methods sufficient to remove *Helicobacter pylori* from endoscopic equipment? *Endoscopy* 1993;25:435.
280. Langenberg W, Rauws EA, Oudbier JH, Tytgat GN. Patient-to-patient transmission of *Campylobacter pylori* infection by fiberoptic gastroduodenoscopy and biopsy. *J. Infect. Dis.* 1990;161:507-11.
281. Miyaji H, Kohli Y, Azuma T, et al. Endoscopic cross-infection with *Helicobacter pylori*. *Lancet* 1995;345:464.

282. Fantry GT, Zheng QX, James SP. Conventional cleaning and disinfection techniques eliminate the risk of endoscopic transmission of *Helicobacter pylori*. *Am. J. Gastroenterol.* 1995;90:227-32.
283. Shimada T, Terano A, Ota S, Takikawa H, Sumino S. Risk of iatrogenic transmission of *Helicobacter pylori* by gastroscopes. *Lancet* 1996;347:1342-3.
284. Roosendaal R, Kuipers EJ, van den Brule AJ, et al. Detection of *Helicobacter pylori* DNA by PCR in gastrointestinal equipment. *Lancet* 1993;341:900.
285. Johnson CH, Rice EW, Reasoner DJ. Inactivation of *Helicobacter pylori* by chlorination. *Appl. Environ. Microbiol.* 1997;63:4969-70.
286. Chapin M, Yatabe J, Cherry JD. An outbreak of rotavirus gastroenteritis on a pediatric unit. *Am. J. Infect. Control* 1983;11:88-91.
287. Keswick BH, Pickering LK, DuPont HL, Woodward WE. Survival and detection of rotaviruses on environmental surfaces in day care centers. *Appl. Environ. Microbiol.* 1983;46:813-16.
288. Ansari SA, Spingthorpe S, Sattar SA. Survival and vehicular spread of human rotaviruses: Possible relation to seasonality of outbreaks. *Rev. Infect. Dis.* 1991;13:448-61.
289. Ansari SA, Sattar SA, Springthorpe VS, Wells GA, Tostowaryk W. Rotavirus survival on human hands and transfer of infectious virus to animate and nonporous inanimate surfaces. *J. Clin. Microbiol.* 1988;26:1513-8.
290. Sattar SA, Raphael RA, Lochnan H, Springthorpe VS. Rotavirus inactivation by chemical disinfectants and antiseptics used in hospitals. *Can. J. Microbiol.* 1983;29:1464-9.
291. Lloyd-Evans N, Springthorpe VS, Sattar SA. Chemical disinfection of human rotavirus-contaminated inanimate surfaces. *J. Hyg. (Lond).* 1986;97:163-73.
292. Tan JA, Schnagl RD. Inactivation of a rotavirus by disinfectants. *Med. J. Aust.* 1981;1:19-23.
293. Sattar SA, Springthorpe VS, Karim Y, Loro P. Chemical disinfection of non-porous inanimate surfaces experimentally contaminated with four human pathogenic viruses. *Epidemiol Infect* 1989;102:493-505.
294. Green J, Wright PA, Gallimore CI, Mitchell O, Morgan-Capner P, Brown DWG. The role of environmental contamination with small round structured viruses in a hospital outbreak investigated by reverse-transcriptase polymerase chain reaction assay. *J. Hosp. Infect.* 1998;39:39-45.
295. Evans MR, Meldrum R, Lane W, et al. An outbreak of viral gastroenteritis following environmental contamination at a concert hall. *Epidemiol & Infect* 2002;129:355-360.
296. Marks PJ, Vipond IB, Regan FM, Wedgwood K, Fey RE, Caul EO. A school outbreak of Norwalk-like virus: Evidence for airborne transmission. *Epidemiol Infect* 2003;131:727-36.
297. Doultree JC, Druce JD, Birch CJ, Bowden DS, Marshall JA. Inactivation of feline calicivirus, a Norwalk virus surrogate. *J. Hosp. Infect.* 1999;41:51-7.
298. Sattar SA. Microbicides and the environmental control of nosocomial viral infections. *J. Hosp. Infect.* 2004;56 (suppl):S64-S69.
299. Jimenez L, Chiang M. Virucidal activity of a quaternary ammonium compound disinfectant against feline calicivirus: A surrogate for norovirus. *Am. J. Infect. Control* 2006;34:269-73.
300. Gehrke C, Steinmann J, Goroncy-Bermes P. Inactivation of feline calicivirus, a surrogate of norovirus (formerly Norwalk-like viruses), by different types of alcohol in vitro and in vivo. *J. Hosp. Infect.* 2004;56:49-55.
301. Centers for Disease Control and Prevention. Update: Severe acute respiratory syndrome - United States, May 14, 2003. *MMWR* 2003;52:436-8.
302. Saknimit M, Inatsuki I, Sugiyama Y, Yagami K. Virucidal efficacy of physico-chemical treatments against coronaviruses and parvoviruses of laboratory animals. *Jikken Dobutsu.* 1988;37:341-5.
303. Sizun J, Yu MW, Talbot PJ. Survival of human coronaviruses 229E and OC43 in suspension and after drying on surfaces: a possible source of hospital-acquired infections. *J. Hosp. Infect.* 2000;46:55-60.
304. Kariwa H, Fujii N, Takashima I. Inactivation of SARS coronavirus by means of povidone-iodine, physical conditions, and chemical reagents. *Jpn. J. Vet. Res.* 2004;52:105-12.
305. Greub G, Raoult D. Biocides currently used for bronchoscope decontamination are poorly effective against free-living amoebae. *Infect Control Hosp Epidemiol* 2003;24:784-6.
306. Leggiadro RJ. The threat of biological terrorism: A public health and infection control reality. *Infect. Control Hosp. Epidemiol.* 2000;21:53-6.
307. Henderson DA. The looming threat of bioterrorism. *Science* 1999;283:1279-82.
308. Centers for Disease Control. Biological and chemical terrorism: strategic plan for preparedness and

- response. MMWR 2000;49 (no. RR-4):1-14.
309. Weber DJ, Rutala WA. Disinfection and sterilization of potential bioterrorism agents. In: Rutala WA, ed. Disinfection, sterilization and antisepsis: Principles, practices and new research. Washington DC: Association for Professionals in Infection Control and Epidemiology, 2004:86-103.
 310. Ferrier A, Garin D, Crance JM. Rapid inactivation of vaccinia virus in suspension and dried on surfaces. J. Hosp. Infect. 2004;57:73-9.
 311. Butcher W, Ulaeto D. Contact inactivation of orthopoxviruses by household disinfectants. J. Appl. Microbiol. 2005;99:279-84.
 312. Brazis AR, Leslie JE, PW K, RL W. The inactivation of spores of *Bacillus globigii* and *Bacillus anthracis* by free available chlorine. Appl. Microbiol. 1958;6:338-342.
 313. Sattar SA, Springthorpe VS, Adegbonrin O. Is *Bacillus subtilis* (ATCC 19659) a suitable surrogate for evaluating microbicides against *Bacillus anthracis*., Association for Official Analytical Chemists International Annual Meeting, St. Louis, Missouri, 2004.
 314. Whitney EAS, Beatty ME, Taylor TH Jr, et al. Inactivation of *Bacillus anthracis* spores. Emerg. Infect. Dis. 2003;9:623-7.
 315. Weber DJ, Rutala WA. Risks and prevention of nosocomial transmission of rare zoonotic diseases. Clin. Infect. Dis. 2001;32:446-456.
 316. Chataigner D, Garnier R, Sans S, Efthymiou ML. [Acute accidental poisoning with hospital disinfectant. 45 cases of which 13 with fatal outcome]. Presse Med. 1991;20:741-3.
 317. Hess JA, Molinari JA, Gleason MJ, Radecki C. Epidermal toxicity of disinfectants. Am. J. Dent. 1991;4:51-6.
 318. Weber DJ, Rutala WA. Occupational risks associated with the use of selected disinfectants and sterilants. In: Rutala WA, ed. Disinfection, sterilization, and antisepsis in healthcare. Champlain, New York: Polyscience Publications, 1998:211-26.
 319. Cokendolpher JC, Haukos JF. The Practical Application of Disinfection and Sterilization in Health Care Facilities. Chicago: American Hospital Association, 1996.
 320. Rideout K, Teschke K, Dimich-Ward H, Kennedy SM. Considering risks to healthcare workers from glutaraldehyde alternatives in high-level disinfection. J. Hosp. Infect. 2005;59:4-11.
 321. Oie S, Kamiya A. Assessment of and intervention for the misuse of aldehyde disinfectants in Japan. Infect. Control Hosp. Epidemiol. 2002;23:98-9.
 322. American Conference of Governmental Industrial Hygienists (ACGIH). Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Cincinnati: ACGIH, 2001.
 323. Jordan SLP, Russo MR, Blessing RL, Grab LA. Glutaraldehyde safety: inactivation and disposal. Abstract. Am. J. Infect. Control 1997;25:154-55.
 324. Jordan SL. The correct use of glutaraldehyde in the healthcare environment. Gastroenterol. Nurs. 1995;18:143-5.
 325. Cheung HY, Brown MR. Evaluation of glycine as an inactivator of glutaraldehyde. J Pharmacy Pharmacol 1982;34:211-4.
 326. Daschner F. The hospital and pollution: Role of the hospital epidemiologist in protecting the environment. In: Wenzel RP, ed. Prevention and control of nosocomial infections. Baltimore: Williams and Wilkins, 1997:595-605.
 327. Rutala WA, Cole EC, Thomann CA, Weber DJ. Stability and bactericidal activity of chlorine solutions. Infect. Control Hosp. Epidemiol. 1998;19:323-7.
 328. Rutala WA, Weber DJ. Uses of inorganic hypochlorite (bleach) in health-care facilities. Clin. Microbiol. Rev. 1997;10:597-610.
 329. Dychdala GR. Chlorine and chlorine compounds. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lippincott Williams & Wilkins, 2001:135-157.
 330. Rutala WA, Weber DJ. Principles of disinfecting patient-care items. In: Rutala WA, ed. Disinfection, sterilization, and antisepsis in healthcare. Champlain, New York: Polyscience Publications, 1998:133-49.
 331. Luebbert P. Home care. In: Pfeiffer JA, ed. APIC text of infection control and epidemiology. Vol. 1. Washington: Association for Professionals in Infection control and epidemiology, 2000:44-7.
 332. Parnes CA. Efficacy of sodium hypochlorite bleach and "alternative" products in preventing transfer of bacteria to and from inanimate surfaces. Environ. Health 1997;59:14-20.
 333. Karapinar M, Gonul SA. Effects of sodium bicarbonate, vinegar, acetic and citric acids on growth and

- survival of *Yersinia enterocolitica*. Int. J. Food Microbiol. 1992;16:343-7.
334. McMurry LM, Oethinger M, Levy SB. Triclosan targets lipid synthesis. Nature 1998;394:531-2.
335. Moken MC, McMurry LM, Levy SB. Selection of multiple-antibiotic-resistant (*mar*) mutants of *Escherichia coli* by using the disinfectant pine oil: roles of the *mar* and *acrAB* loci. Antimicrob. Agents Chemother. 1997;41:2770-2.
336. Scott E, Bloomfield SF, Barlow CG. An investigation of microbial contamination in the home. J. Hyg. (Lond). 1982;89:279-93.
337. Rusin P, Orosz-Coughlin P, Gerba C. Reduction of faecal coliform, coliform and heterotrophic plate count bacteria in the household kitchen and bathroom by disinfection with hypochlorite cleaners. J. Appl. Microbiol. 1998;85:819-28.
338. Gilbert P, McBain AJ. Potential impact of increased use of biocides in consumer products on prevalence of antibiotic resistance. Clin Microbiol Reviews 2003;16:189-208.
339. Bueumer R, Bloomfield SF, Exner M, Fara G, Scott EA. The need for a home hygiene policy and guidelines on home hygiene. Ann. Ig. 1999;11:11-26.
340. International Scientific Forum on Home Hygiene. www.ifh-homehygiene.org.
341. Russell AD, Russell NJ. Biocides: activity, action and resistance. In: Hunter PA, Darby GK, Russell NJ, eds. Fifty years of antimicrobials: past perspectives and future trends. England: Cambridge University Press, 1995:327-65.
342. Russell AD. Bacterial resistance to disinfectants: Present knowledge and future problems. J. Hosp. Infect. 1998;43:S57-S68.
343. Russell AD. Plasmids and bacterial resistance to biocides. J. Appl. Microbiol. 1997;83:155-65.
344. Russell AD. Bacterial resistance to disinfectants: present knowledge and future problems. J. Hosp. Infect. 1998;43:S57-68.
345. Russell AD. Principles of antimicrobial activity and resistance. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lippincott Williams & Wilkins, 2001:31-55.
346. McDonnell G, Russell AD. Antiseptics and disinfectants: activity, action, and resistance. Clin. Microbiol. Rev. 1999;12:147-79.
347. Gerba CP, Rusin P. Relationship between the use of antiseptics/disinfectants and the development of antimicrobial resistance. In: Rutala WA, ed. Disinfection, sterilization and antisepsis: principles and practices in healthcare facilities. Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:187-94.
348. Townsend DE, Ashdown N, Greed LC, Grubb WB. Transposition of gentamicin resistance to staphylococcal plasmids encoding resistance to cationic agents. J. Antimicrob. Chemother. 1984;14:115-24.
349. Brumfitt W, Dixon S, Hamilton-Miller JM. Resistance to antiseptics in methicillin and gentamicin resistant *Staphylococcus aureus*. Lancet 1985;1:1442-3.
350. Al-Masaudi SB, Day MJ, Russell AD. Sensitivity of methicillin-resistant *Staphylococcus aureus* strains to some antibiotics, antiseptics and disinfectants. J. Appl. Bacteriol. 1988;65:329-37.
351. Tennent JM, Lyon BR, Midgley M, Jones IG, Purewal AS, Skurray RA. Physical and biochemical characterization of the *qacA* gene encoding antiseptic and disinfectant resistance in *Staphylococcus aureus*. J. Gen. Microbiol. 1989;135:1-10.
352. Kaulfers PM, Laufs R. [Transmissible formaldehyde resistance in *Serratia marcescens*]. Zentralblatt für Bakteriologie, Mikrobiologie und Hygiene - 1 - Abt - Originale B, Hygiene 1985;181:309-19.
353. Tennent JM, Lyon BR, Gillespie MT, May JW, Skurray RA. Cloning and expression of *Staphylococcus aureus* plasmid-mediated quaternary ammonium resistance in *Escherichia coli*. Antimicrob. Agents Chemother. 1985;27:79-83.
354. Rutala WA, Stiegel MM, Sarubbi FA, Weber DJ. Susceptibility of antibiotic-susceptible and antibiotic-resistant hospital bacteria to disinfectants. Infect. Control Hosp. Epidemiol. 1997;18:417-21.
355. Anderson RL, Carr JH, Bond WW, Favero MS. Susceptibility of vancomycin-resistant enterococci to environmental disinfectants. Infect. Control Hosp. Epidemiol. 1997;18:195-9.
356. Sakagami Y, Kajimura K. Bactericidal activities of disinfectants against vancomycin-resistant enterococci. J. Hosp. Infect. 2002;50:140-4.
357. Sehulster LM, Anderson RL. Susceptibility of glycopeptide-intermediate resistant *Staphylococcus aureus* (GISA) to surface disinfectants, hand washing chemicals, and a skin antiseptic. Abstract Y-3. 98th General

- Meeting of American Society for Microbiology, May, 1998:547.
358. Rutala WA, Weber DJ, Gergen MF. Studies on the disinfection of VRE-contaminated surfaces. *Infect. Control Hosp. Epidemiol.* 2000;21:548.
359. Byers KE, Durbin LJ, Simonton BM, Anglim AM, Adal KA, Farr BM. Disinfection of hospital rooms contaminated with vancomycin-resistant *Enterococcus faecium*. *Infect. Control Hosp. Epidemiol.* 1998;19:261-4.
360. Carling PC, Briggs JL, Perkins J, Highlander D. Improved cleaning of patient rooms using a new targeting method. *Clin Inf Dis* 2006;42:385-8.
361. Russell AD, Suller MT, Maillard JY. Do antiseptics and disinfectants select for antibiotic resistance? *J. Med. Microbiol.* 1999;48:613-5.
362. Russell AD. Bacterial adaptation and resistance to antiseptics, disinfectants and preservatives is not a new phenomenon. *J. Hosp. Infect.* 2004;57:97-104.
363. Levy SB. The challenge of antibiotic resistance. *Scientific Am.* 1998;278:46-53.
364. Jones RD, Jampani HB, Newman JL, Lee AS. Triclosan: a review of effectiveness and safety in health care settings. *Am. J. Infect. Control* 2000;28:184-96.
365. Russell AD, McDonnell G. Concentration: a major factor in studying biocidal action. *J. Hosp. Infect.* 2000;44:1-3.
366. Russell AD, Maillard JY. Reaction and response-relationship between antibiotic resistance and resistance to antiseptics and disinfectants. *Am. J. Infect. Control* 2000;28:204-6.
367. Murtough SM, Hiom SJ, Palmer M, Russell AD. Biocide rotation in the healthcare setting: is there a case for policy implementation? *J. Hosp. Infect.* 2001;48:1-6.
368. Murtough SM, Hiom SJ, Palmer M, Russell AD. A survey of rotational use of biocides in hospital pharmacy aseptic units. *J. Hosp. Infect.* 2002;50:228-31.
369. Gebel J, Sonntag H-G, Werner H-P, Vavata V, Exner M, Kistemann T. The higher disinfectant resistance of nosocomial isolates of *Klebsiella oxytoca*: How reliable are indicator organisms in disinfectant testing? *J. Hosp. Infect.* 2002;50:309-11.
370. Ruden H, Daschner F. Should we routinely disinfect floors? *J. Hosp. Infect.* 2002;51:309.
371. Rutala WA, DJ W. Should we routinely disinfect floors? Reply to Professor F. Daschner. *J. Hosp. Infect.* 2002;51:309-11.
372. Rutala WA, Weber DJ. The benefits of surface disinfection. *Am. J. Infect. Control* 2004;32:226-31.
373. Dettenkofer M, Wenzler S, Amthor S, Antes G, Motschall E, Daschner FD. Does disinfection of environmental surfaces influence nosocomial infection rates? A systematic review. *Am. J. Infect. Control* 2004;32:84-9.
374. Daschner F, Schuster A. Disinfection and the prevention of infectious disease-no adverse effects? *Am. J. Infect. Control* 2004;32:224-5.
375. Cozad A, Jones RD. Disinfection and the prevention of infectious disease. *Am. J. Infect. Control* 2003;31:243-54.
376. Favero MS, Bond WW. Chemical disinfection of medical and surgical materials. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lea & Febiger, 1991:617-41.
377. Rheinbaben FV, Schunemann S, Grob T, Wolff MH. Transmission of viruses via contact in a household setting: experiments using bacteriophage OX174 as a model virus. *J. Hosp. Infect.* 2000;46:61-66.
378. Rutala WA, Weber DJ. Surface disinfection: should we do it? *J. Hosp. Infect.* 2001;48 (supplement A):S64-S68.
379. Ayliffe GAJ, Collins DM, Lowbury EJJ. Cleaning and disinfection of hospital floors. *Brit. Med. J.* 1966;2:442-5.
380. Ayliffe GA, Collins BJ, Lowbury EJ, Babb JR, Lilly HA. Ward floors and other surfaces as reservoirs of hospital infection. *J. Hyg. (Lond)*. 1967;65:515-36.
381. Exner M, Vacata V, Hornei B, Dietlein E, Gebel J. Household cleaning and surface disinfection: New insights and strategies. *J. Hosp. Infect.* 2004;56 (suppl):S70-S75.
382. Dharan S, Mourouga P, Copin P, Bessmer G, Tschanz B, Pittet D. Routine disinfection of patients' environmental surfaces. Myth or reality? *J. Hosp. Infect.* 1999;42:113-7.
383. Engelhart S KL, Glasmacher A, Fischnaller E, Marklein G, Exner M. *Pseudomonas aeruginosa* outbreak in a haematology-oncology unit associated with contaminated surface cleaning equipment. *J. Hosp. Infect.* 2002;52:93-98.

384. Denton M, Wilcox MH, Parnell P, et al. Role of environmental cleaning in controlling an outbreak of *Acinetobacter baumannii* on a neurosurgical intensive care unit. *J. Hosp. Infect.* 2004;56:106-10.
385. Barker J, Vipond IB, Bloomfield SF. Effects of cleaning and disinfection in reducing the spread of Norovirus contamination via environmental surfaces. *J. Hosp. Infect.* 2004;58:42-9.
386. Garner JS. Guideline for isolation precautions in hospitals. The Hospital Infection Control Practices Advisory Committee. *Infect. Control Hosp. Epidemiol.* 1996;17:53-80.
387. Maki DG, Alvarado CJ, Hassemer CA, Zilz MA. Relation of the inanimate hospital environment to endemic nosocomial infection. *N. Engl. J. Med.* 1982;307:1562-6.
388. Daschner F, Rabbenstein G, Langmaack H. [Surface decontamination in the control of hospital infections: comparison of different methods (author's transl)]. *Dtsch. Med. Wochenschr.* 1980;105:325-9.
389. Danforth D, Nicolle LE, Hume K, Alfieri N, Sims H. Nosocomial infections on nursing units with floors cleaned with a disinfectant compared with detergent. *J. Hosp. Infect.* 1987;10:229-35.
390. Smith TL, Iwen PC, Olson SB, Rupp ME. Environmental contamination with vancomycin-resistant enterococci in an outpatient setting. *Infect. Control Hosp. Epidemiol.* 1998;19:515-8.
391. Boyce JM, Potter-Bynoe G, Chenevert C, King T. Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. *Infect. Control Hosp. Epidemiol.* 1997;18:622-7.
392. Bonten MJM, Hayden MJ, Nathan C, et al. Epidemiology of colonisation of patients and environment with vancomycin-resistant enterococci. *Lancet* 1996;348:1615-9.
393. Hardy KJ, Oppenheim BA, Gossain S, Gao F, Hawkey PM. A study of the relationship between environmental contamination with methicillin-resistant *Staphylococcus aureus* (MRSA) and patients' acquisition of MRSA. *Infect Control Hosp Epidemiol* 2006;27:127-32.
394. Hota B. Contamination, disinfection, and cross-contamination: Are hospital surfaces reservoirs for nosocomial infection? *Clin. Infect. Dis.* 2004;39:1182-9.
395. Neely AN, Maley MP. Survival of enterococci and staphylococci on hospital fabrics and plastic. *J. Clin. Microbiol.* 2000;38:724-6.
396. Wendt C, Wiensenthal B, Dietz E, Ruden H. Survival of enterococci on dry surfaces. *J. Clin. Microbiol.* 1998;36:3734-6.
397. Neely AN, Maley MP. The 1999 Lindberg award. 3% hydrogen peroxide for the gram-positive disinfection of fabrics. *J. Burn Care Rehabil.* 1999;20:471-7.
398. Griffith CJ, Cooper RA, Gilmore J, Davies C, Lewis M. An evaluation of hospital cleaning regimes and standards. *J. Hosp. Infect.* 2000;45:19-28.
399. Tiller JC, Liao CJ, Lewis K, Klivanov AM. Designing surfaces that kill bacteria on contact. *Proc. Natl. Acad. Sci.* 2001;98:5981-5.
400. Rutala WA, Weber DJ. New disinfection and sterilization methods. *Emerg. Inf. Dis.* 2001;7:348-53.
401. Whitby JL, Rampling A. *Pseudomonas aeruginosa* contamination in domestic and hospital environments. *Lancet* 1972;1:15-7.
402. Scott E, Bloomfield SF. The survival and transfer of microbial contamination via cloths, hand and utensils. *J. Appl. Bacteriol.* 1990;68:271-8.
403. Scott E, Bloomfield SF. Investigations of the effectiveness of detergent washing, drying and chemical disinfection on contamination of cleaning cloths. *J. Appl. Bacteriol.* 1990;68:279-83.
404. Rutala WA, Cole EC. Antiseptics and disinfectants--safe and effective? *Infect. Control* 1984;5:215-8.
405. Oie S, Huang Y, Kamiya A, Konishi H, Nakazawa T. Efficacy of disinfectants against biofilm cells of methicillin-resistant *Staphylococcus aureus*. *Microbios* 1996;85:223-30.
406. Sartor C, Jacomo V, Duvivier C, Tissot-Dupont H, Sambuc R, Drancourt M. Nosocomial *Serratia marcescens* infections associated with extrinsic contamination of a liquid nonmedicated soap. *Infect. Control Hosp. Epidemiol.* 2000;21:196-9.
407. Reiss I, Borkhardt A, Fussle R, Sziegoleit A, Gortner L. Disinfectant contaminated with *Klebsiella oxytoca* as a source of sepsis in babies. *Lancet* 2000;356:310.
408. O'Rourke E, Runyan D, O'Leary J, Stern J. Contaminated iodophor in the operating room. *Am. J. Infect. Control* 2003;31:255-6.
409. Chuanchuen R, Karkhoff-Schweizer RR, Schweizer HP. High-level triclosan resistance in *Pseudomonas aeruginosa* is solely a result of efflux. *Am. J. Infect. Control* 2003;31:124-7.
410. Newman KA, Tenney JH, Oken HA, Moody MR, Wharton R, Schimpff SC. Persistent isolation of an

- unusual *Pseudomonas* species from a phenolic disinfectant system. *Infect. Control* 1984;5:219-22.
411. Bean HS. Types and characteristics of disinfectants. *J. Appl. Bacteriol.* 1967;30:6-16.
412. Russell AD, Hugo WB, Ayliffe GAJ. *Principles and Practice of Disinfection, Preservation and Sterilization*. Oxford, England: Blackwell Scientific Publications, 1999.
413. Russell AD. Factors influencing the efficacy of germicides. In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research*. Washington DC: Association for Professionals in Infection Control and Epidemiology, 2004:162-70.
414. Gillis RJ, Schmidt WC. Scanning electron microscopy of spores on inoculated product surfaces. *MD* 1983;46-9.
415. Favero MS, Petersen NJ, Carson LA, Bond WW, Hindman SH. Gram-negative water bacteria in hemodialysis systems. *Health Lab. Sci.* 1975;12:321-34.
416. Rutala WA, Cole EC. Ineffectiveness of hospital disinfectants against bacteria: a collaborative study. *Infect. Control* 1987;8:501-6.
417. Favero MS. Naturally occurring microorganisms and their resistance to physical and chemical agents. In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research*. Washington DC: Association for Professionals in Infection Control and Epidemiology, 2004:1-14.
418. Lee DH, Miles RJ, Perry BF. The mycoplasmacidal properties of sodium hypochlorite. *J. Hyg. (Lond)*. 1985;95:243-53.
419. Scott GH, Williams JC. Susceptibility of *Coxiella burnetii* to chemical disinfectants. *Ann. N. Y. Acad. Sci.* 1990;590:291-6.
420. Russell AD. Factors influencing the efficacy of antimicrobial agents. In: Russell AD, Hugo WB, Ayliffe GAJ, eds. *Principles and practice of disinfection, preservation and sterilization*. Oxford: Blackwell Science, 1999:95-123.
421. Rutala WA. Selection and use of disinfectants in healthcare. In: Mayhall CG, ed. *Infect. Control and Hosp. Epidemiol.* Philadelphia: Lippincott Williams & Wilkins, 1999:1161-87.
422. Lewis DL, Arens M. Resistance of microorganisms to disinfection in dental and medical devices. *Nat. Med.* 1995;1:956-8.
423. Muscarella LF. Sterilizing dental equipment. *Nat. Med.* 1995;1:1223-5.
424. Abbott CF, Cockton J, Jones W. Resistance of crystalline substances to gas sterilization. *J. Pharm. Pharmacol.* 1956;8:709-20.
425. Doyle JE, Ernst RR. Resistance of *Bacillus subtilis* var. *niger* spores occluded in water-insoluble crystals to three sterilization agents. *Appl. Microbiol.* 1967;15:726-30.
426. Jacobs P. Cleaning: Principles, methods and benefits. In: Rutala WA, ed. *Disinfection, sterilization, and antisepsis in healthcare*. Champlain, New York: Polyscience Publications, 1998:165-81.
427. Gorham RA, Jacobs P, Roberts CG. Laboratory artifacts due to protein and salt crystals on the inactivation of *Bacillus stearothermophilus*. *J. Hosp. Infect.* 1998;40:abstract P.9.2.2.
428. Cole EC, Rutala WA, Carson JL, Alfano EM. *Pseudomonas* pellicle in disinfectant testing: electron microscopy, pellicle removal, and effect on test results. *Appl. Environ. Microbiol.* 1989;55:511-3.
429. Anderson RL, Holland BW, Carr JK, Bond WW, Favero MS. Effect of disinfectants on pseudomonads colonized on the interior surface of PVC pipes. *Am. J. Public Health* 1990;80:17-21.
430. Anderson RL, Vess RW, Carr JH, Bond WW, Panlilio AL, Favero MS. Investigations of intrinsic *Pseudomonas cepacia* contamination in commercially manufactured povidone-iodine. *Infect. Control Hosp. Epidemiol.* 1991;12:297-302.
431. LeChevallier MW, Cawthon CD, Lee RG. Inactivation of biofilm bacteria. *Appl. Environ. Microbiol.* 1988;54:2492-9.
432. LeChevallier MW, Cawthon CD, Lee RG. Factors promoting survival of bacteria in chlorinated water supplies. *Appl. Environ. Microbiol.* 1988;54:649-54.
433. Costerton JS, Stewart PS, Greenberg EP. Bacterial biofilms: a common cause of persistent infections. *Science* 1999;284:1318-22.
434. Donlan RM, Costerton JW. Biofilms: Survival mechanisms of clinically relevant microorganisms. *Clin. Microbiol. Rev.* 2002;15:167-93.
435. Dunne WM. Bacterial adhesion: Seen any good biofilms lately? *Clin. Microbiol. Rev.* 2002;15:155-66.
436. Vickery K, Pajkos A, Cossart Y. Removal of biofilm from endoscopes: Evaluation of detergent efficiency.

- Am. J. Infect. Control 2004;32:170-6.
437. Marion K, Freney J, James G, Bergeron E, Renaud FNR, Costerton JW. Using an efficient biofilm detaching agent: An essential step for the improvement of endoscope reprocessing protocols. *J. Hosp. Infect.* 2006;In press.
 438. Marion-Ferey K, Pasmore M, Stoodley P, Wilson S, Husson GP, Costerton JW. Biofilm removal from silicone tubing: an assessment of the efficacy of dialysis machine decontamination procedures using an in vitro model. *J. Hosp. Infect.* 2003;53:64-71.
 439. Brown ML, Aldrich HC, Gauthier JJ. Relationship between glycocalyx and povidone-iodine resistance in *Pseudomonas aeruginosa* (ATCC 27853) biofilms. *Appl. Environ. Microbiol.* 1995;61:187-93.
 440. Price D, Ahearn DG. Incidence and persistence of *Pseudomonas aeruginosa* in whirlpools. *J. Clin. Microbiol.* 1988;26:1650-4.
 441. Anonymous. Dental Unit Waterlines: Approaching the Year 2000. ADA Council on Scientific Affairs. *JADA* 1999;130:1653-64.
 442. Donlan RM. Biofilms: a source of infection? In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: principles and practices in healthcare facilities*. Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:219-26.
 443. Loukili NH, Zink E, Grandadam S, Bientz M, Meunier O. Effectiveness of detergent-disinfecting agents on *Escherichia coli* 54127 biofilm. *J. Hosp. Infect.* 2004;57:175-8.
 444. Johansen C, Falholt P, Gram L. Enzymatic removal and disinfection of bacterial biofilms. *Appl. Environ. Microbiol.* 1997;63:3724-8.
 445. Reichert M. Preparation of supplies for terminal sterilization. In: Reichert M, Young JH, eds. *Sterilization technology for the health care facility*. Gaithersburg, MD: Aspen Publication, 1997:36-50.
 446. Miller CH, Riggen SD, Sheldrake MA, Neeb JM. Presence of microorganisms in used ultrasonic cleaning solutions. *Am. J. Dent.* 1993;6:27-31.
 447. Jatzwauk L, Schone H, Pietsch H. How to improve instrument disinfection by ultrasound. *J. Hosp. Infect.* 2001;48 (Supple):S80-S83.
 448. Richburg FA, Reidy JJ, Apple DJ, Olson RJ. Sterile hypopyon secondary to ultrasonic cleaning solution. *J. Cataract Refract. Surg.* 1986;12:248-51.
 449. Schultz JK. Decontamination alternative. *Infect. Control Hosp. Epidemiol.* 1990;11:8-9.
 450. Rutala WA, Shafer KM. General information on cleaning, disinfection, and sterilization. In: Pfeiffer JA, ed. *APIC infection control and applied epidemiology: principles and practice*,. St. Louis: Mosby, 1996:15.1-15.17.
 451. Leonard DL, Mills SE. Comparison of automated instrument cleaning: preliminary results. *Infect. Control Steril. Technol.* 1997;20-23, 26-28.
 452. Ransjo U, Engstrom L, Hakansson P, et al. A test for cleaning and disinfection processes in a washer-disinfector. *APMIS* 2001;109:299-304.
 453. American Society for Hospital Central Service Personnel. *Training manual for central service technicians*. Chicago: American Hospital Association, 2001:1-271.
 454. Ninemeier JD. *Central service technical manual*. Chicago: International Association of Healthcare Central Service Materiel Management, 1998.
 455. Reichert M, Young JH. *Sterilization technology for the health care facility*. Gaithersburg: Aspen Publication, 1997:307.
 456. Vesley D, Norlien KG, Nelson B, Ott B, Streifel AJ. Significant factors in the disinfection and sterilization of flexible endoscopes. *Am. J. Infect. Control* 1992;20:291-300.
 457. Roberts CG. Studies on the bioburden on medical devices and the importance of cleaning. In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: principles and practices in healthcare facilities*. Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:63-9.
 458. Baxter RL, Baxter HC, Campbell GA, et al. Quantitative analysis of residual protein contamination on reprocessed surgical instruments. *J. Hosp. Infect.* 2006;63:439-44.
 459. Murdoch H, Taylor D, Dickinson J, et al. Surface decontamination of surgical instruments: An ongoing dilemma. *J. Hosp. Infect.* 2006;63:432-8.
 460. Alfa MJ, Nemes R. Manual versus automated methods for cleaning reusable accessory devices used for minimally invasive surgical procedures. *J. Hosp. Infect.* 2004;58:50-8.
 461. Alfa MJ, Nemes R, Olson N, Mulaire A. Manual methods are suboptimal compared with automated

- methods for cleaning of single-use biopsy forceps. *Infect Control Hosp Epidemiol* 2006;27:841-6.
462. Lee CH, Cheng SM, Humar A, et al. Acute febrile reactions with hypotension temporally associated with the introduction of a concentrated bioenzyme preparation in the cleaning and sterilization process of endomyocardial biopones. *Infect. Control Hosp. Epidemiol.* 2000;21:102.
463. Hutchisson B, LeBlanc C. The truth and consequences of enzymatic detergents. *Gastroenterol. Nurs.* 2005;28:372-6.
464. Zuhlsdorf B EM, Floss H, Martiny H,. Cleaning efficacy of nine different cleaners in a washer-disinfector designed for flexible endoscopes. *J. Hosp. Infect.* 2002;52:206-11.
465. Merritt K, Hitchins VM, Brown SA. Safety and cleaning of medical materials and devices. *J. Biomed. Mater. Res.* 2000;53:131-6.
466. Babb JR, Bradley CR. Endoscope decontamination: where do we go from here? *J. Hosp. Infect.* 1995;30:543-51.
467. Zuhlsdorf B, Floss H, Martiny H. Efficacy of 10 different cleaning processes in a washer-disinfector for flexible endoscopes. *J. Hosp. Infect.* 2004;56:305-11.
468. Alfa MJ, Jackson M. A new hydrogen peroxide-based medical-device detergent with germicidal properties: Comparison with enzymatic cleaners. *Am. J. Infect. Control* 2001;29:168-77.
469. Alfa MJ, DeGagne P, Olson N, Puchalski T. Comparison of ion plasma, vaporized hydrogen peroxide and 100% ethylene oxide sterilizers to the 12/88 ethylene oxide gas sterilizer. *Infect. Control Hosp. Epidemiol.* 1996;17:92-100.
470. Alfa MJ. Flexible endoscope reprocessing. *Infect. Control Steril. Technol.* 1997;3:26-36.
471. Alfa MJ, Degagne P, Olson N. Worst-case soiling levels for patient-used flexible endoscopes before and after cleaning. *Am. J. Infect. Control* 1999;27:392-401.
472. Rutala WA, Weber DJ. Low-temperature sterilization technology: Do we need to redefine sterilization? *Infect. Control Hosp. Epidemiol.* 1996;17:89-91.
473. Dancer SJ. How do we assess hospital cleaning? A proposal for microbiological standards for surface hygiene in hospitals. *J. Hosp. Infect.* 2004;56:10-5.
474. Pfeifer M. Standardised test soil blood 1:Composition, preparation, application. *Zentr. Steril.* 1998;6:381-5.
475. Pfeifer M. Blood as a soil on surgical instruments: Chemical profile, cleaning, detection. *Zentr. Steril.* 1998;6:304-10.
476. Fengier TW, Pahike H, Bisson S, Michels W. Are processed surgical instruments free of protein? *Zentr. Steril.* 2001;9:20-32.
477. Takashina M. Application of a bioluminescent method for checking cleaning results. *Zentr. Steril.* 2001;9:248-58.
478. Lipscomb IP, Sihota AK, Botham M, Harris KL, Keevil CW. Rapid method for the sensitive detection of protein contamination on surgical instruments. *J. Hosp. Infect.* 2006;62:141-8.
479. Malik RE, Cooper RA, Griffith CJ. Use of audit tools to evaluate the efficacy of cleaning systems in hospitals. *Am. J. Infect. Control* 2003;31:181-7.
480. Hansen KS. Occupational dermatoses in hospital cleaning women. *Contact Dermatitis* 1983;9:343-51.
481. Melli MC, Giorgini S, Sertoli A. Sensitization from contact with ethyl alcohol. *Contact Dermatitis* 1986;14:315.
482. Spaulding EH. Alcohol as a surgical disinfectant. *AORN J.* 1964;2:67-71.
483. Morton HE. The relationship of concentration and germicidal efficiency of ethyl alcohol. *Ann N.Y. Acad. Sci.* 1950;53:191-96.
484. Ali Y, Dolan MJ, Fendler EJ, Larson EL. Alcohols. In: Block SS, ed. *Disinfection, sterilization, and preservation.* Philadelphia: Lippincott Williams & Wilkins, 2001:229-54.
485. Morton HE. Alcohols. In: Block SS, ed. *Disinfection, sterilization, and preservation.* Philadelphia: Lea & Febiger, 1983:225-239.
486. Sykes G. The influence of germicides on the dehydrogenases of *Bact. coli*. Part I. The succinic acid dehydrogenase of *Bact. coli*. *J. Hyg. (Camb)* 1939;39:463-69.
487. Dagley S, Dawes EA, Morrison GA. Inhibition of growth of *Aerobacter aerogenes*: the mode of action of phenols, alcohols, acetone and ethyl acetate. *J. Bacteriol.* 1950;60:369-78.
488. Tilley FW, Schaffer JM. Relation between the chemical constitution and germicidal activity of the monohydric alcohols and phenols. *J. Bacteriol.* 1926;12:303-9.
489. Coulthard CE, Sykes G. The germicidal effect of alcohol with special reference to its action on bacterial

- spores. *Pharmaceutical J.* 1936;137:79-81.
490. Tyler R, Ayliffe GA. A surface test for virucidal activity of disinfectants: preliminary study with herpes virus. *J. Hosp. Infect.* 1987;9:22-9.
491. Kurtz JB, Lee TW, Parsons AJ. The action of alcohols on rotavirus, astrovirus and enterovirus. *J. Hosp. Infect.* 1980;1:321-5.
492. Smith CR. Alcohol as a disinfectant against the tubercle bacillus. *Public Health Rep.* 1947;62:1285-95.
493. Kruse RH, Green TD, Chambers RC, Jones MW. Disinfection of aerosolized pathogenic fungi on laboratory surfaces. I. Tissue phase. *Appl. Microbiol.* 1963;11:436-45.
494. Kruse RH, Green TD, Chambers RC, Jones MW. Disinfection of aerosolized pathogenic fungi on laboratory surfaces. II Culture phase. *Appl. Microbiol.* 1964;12:155-60.
495. Connor CG, Hopkins SL, Salisbury RD. Effectivity of contact lens disinfection systems against *Acanthamoeba culbertsoni*. *Optom. Vis. Sci.* 1991;68:138-41.
496. Turner NA, Russell AD, Furr JR, Lloyd D. *Acanthamoeba* spp., antimicrobial agents and contact lenses. *Sci. Prog.* 1999;82:1-8.
497. Nye RN, Mallory TB. A note on the fallacy of using alcohol for the sterilization of surgical instruments. *Boston Med. Surg. J.* 1923;189:561-3.
498. Frobisher M, Sommermeyer L, Blackwell MJ. Studies on disinfection of clinical thermometers. I. Oral thermometers. *Appl. Microbiol.* 1973;1:187-94.
499. Sommermeyer L, Frobisher M. Laboratory studies on disinfection of rectal thermometers. *Nurs. Res.* 1973;2:85-9.
500. Singh D, Kaur H, Gardner WG, Treen LB. Bacterial contamination of hospital pagers. *Infect. Control Hosp. Epidemiol.* 2002;23:274-6.
501. Embil JM, Zhanel GG, Plourde J, Hoban D. Scissors: A potential source of nosocomial infection. *Infect. Control Hosp. Epidemiol.* 2002;23:147-51.
502. Zachary KC, Bayne PS, Morrison VJ, Ford DS, Silver LC, Hooper DC. Contamination of gowns, gloves, and stethoscopes with vancomycin-resistant enterococci. *Infect. Control Hosp. Epidemiol.* 2001;22:560-4.
503. Babb JR, Bradley CR, Deverill CE, Ayliffe GA, Melikian V. Recent advances in the cleaning and disinfection of fibrescopes. *J. Hosp. Infect.* 1981;2:329-40.
504. Garcia de Cabo A, Martinez Larriba PL, Checa Pinilla J, Guerra Sanz F. A new method of disinfection of the flexible fibrobronchoscope. *Thorax* 1978;33:270-2.
505. Elson CO, Hattori K, Blackstone MO. Polymicrobial sepsis following endoscopic retrograde cholangiopancreatography. *Gastroenterology* 1975;69:507-10.
506. Weber DJ, Wilson MB, Rutala WA, Thomann CA. Manual ventilation bags as a source for bacterial colonization of intubated patients. *Am. Rev. Respir. Dis.* 1990;142:892-4.
507. Cavagnolo RZ. Inactivation of herpesvirus on CPR manikins utilizing a currently recommended disinfecting procedure. *Infect. Control* 1985;6:456-8.
508. Ohara T, Itoh Y, Itoh K. Ultrasound instruments as possible vectors of staphylococcal infection. *J. Hosp. Infect.* 1998;40:73-7.
509. Talbot GH, Skros M, Provencher M. 70% alcohol disinfection of transducer heads: experimental trials. *Infect. Control* 1985;6:237-9.
510. Platt R, Lehr JL, Marino S, Munoz A, Nash B, Raemer DB. Safe and cost-effective cleaning of pressure-monitoring transducers. *Infect. Control Hosp. Epidemiol.* 1988;9:409-16.
511. Beck-Sague CM, Jarvis WR. Epidemic bloodstream infections associated with pressure transducers: a persistent problem. *Infect. Control Hosp. Epidemiol.* 1989;10:54-9.
512. Chronister CL, Russo P. Effects of disinfecting solutions on tonometer tips. *Optom. Vis. Sci.* 1990;67:818-21.
513. Lingel NJ, Coffey B. Effects of disinfecting solutions recommended by the Centers for Disease Control on Goldmann tonometer biprisms. *J. Am. Optom. Assoc.* 1992;63:43-8.
514. Soukiasian SH, Asdourian GK, Weiss JS, Kachadoorian HA. A complication from alcohol-swabbed tonometer tips. *Am. J. Ophthalmol.* 1988;105:424-5.
515. Jakobsson SW, Rajs J, Jonsson JA, Persson H. Poisoning with sodium hypochlorite solution. Report of a fatal case, supplemented with an experimental and clinico-epidemiological study. *Am. J. Forensic Med. Pathol.* 1991;12:320-7.
516. Heidemann SM, Goetting MG. Treatment of acute hypoxemic respiratory failure caused by chlorine

- exposure. *Pediatr. Emerg. Care* 1991;7:87-8.
517. Hoy RH. Accidental systemic exposure to sodium hypochlorite (Clorox) during hemodialysis. *Am. J. Hosp. Pharm.* 1981;38:1512-4.
 518. Landau GD, Saunders WH. The effect of chlorine bleach on the esophagus. *Arch. Otolaryngol.* 1964;80:174-6.
 519. French RJ, Tabb HG, Rutledge LJ. Esophageal stenosis produced by ingestion of bleach: report of two cases. *South. Med. J.* 1970;63:1140-4.
 520. Ward MJ, Routledge PA. Hypernatraemia and hyperchloraemic acidosis after bleach ingestion. *Hum. Toxicol.* 1988;7:37-8.
 521. Ingram TA. Response of the human eye to accidental exposure to sodium hypochlorite. *J Endodontics* 1990;16:235-8.
 522. Haag JR, Gieser RG. Effects of swimming pool water on the cornea. *JAMA* 1983;249:2507-8.
 523. Mrvos R, Dean BS, Krenzelok EP. Home exposures to chlorine/chloramine gas: review of 216 cases. *South. Med. J.* 1993;86:654-7.
 524. Reisz GR, Gammon RS. Toxic pneumonitis from mixing household cleaners. *Chest* 1986;89:49-52.
 525. Gapany-Gapanavicius M, Yellin A, Almog S, Tirosh M. Pneumomediastinum. A complication of chlorine exposure from mixing household cleaning agents. *JAMA* 1982;248:349-50.
 526. Hoffman PN, Death JE, Coates D. The stability of sodium hypochlorite solutions. In: Collins CH, Allwood MC, Bloomfield SF, Fox A, eds. *Disinfectants: their use and evaluation of effectiveness.* London: Academic Press, 1981:77-83.
 527. Gamble MR. Hazard: formaldehyde and hypochlorites. *Lab. Anim.* 1977;11:61.
 528. Helms C, Massanari R, Wenzel R, et al. Control of epidemic nosocomial legionellosis: a 5 year progress report on continuous hyperchlorination of a water distribution system. *Abstracts of 27th Interscience Conference of Antimicrobial Agents and Chemotherapy*, 1987:349, p.158.
 529. Environmental Protection Agency. R.E.D. Facts sodium and calcium hypochlorite salts. 1991.
 530. Coates D. Comparison of sodium hypochlorite and sodium dichloroisocyanurate disinfectants: neutralization by serum. *J. Hosp. Infect.* 1988;11:60-7.
 531. Coates D. A comparison of sodium hypochlorite and sodium dichloroisocyanurate products. *J. Hosp. Infect.* 1985;6:31-40.
 532. Coates D, Wilson M. Use of sodium dichloroisocyanurate granules for spills of body fluids. *J. Hosp. Infect.* 1989;13:241-51.
 533. Bloomfield SF, Uso EE. The antibacterial properties of sodium hypochlorite and sodium dichloroisocyanurate as hospital disinfectants. *J. Hosp. Infect.* 1985;6:20-30.
 534. Coates D. An evaluation of the use of chlorine dioxide (Tristel One-Shot) in an automated washer/disinfector (Medivator) fitted with a chlorine dioxide generator for decontamination of flexible endoscopes. *J. Hosp. Infect.* 2001;48:55-65.
 535. Isomoto H, Urata M, Kawazoe K, et al. Endoscope disinfection using chlorine dioxide in an automated washer-disinfector. *J. Hosp. Infect.* 2006;63:298-305.
 536. Sampson MN MA. Not all super-oxidized waters are the same. *J. Hosp. Infect.* 2002;52:227-8.
 537. Selkon JB, Babb JR, Morris R. Evaluation of the antimicrobial activity of a new super-oxidized water, Sterilox®, for the disinfection of endoscopes. *J. Hosp. Infect.* 1999;41:59-70.
 538. Fraise AP. Choosing disinfectants. *J. Hosp. Infect.* 1999;43:255-64.
 539. Tanaka H, Hirakata Y, Kaku M, et al. Antimicrobial activity of superoxidized water. *J. Hosp. Infect.* 1996;34:43-9.
 540. Tanaka N, Fujisawa T, Daimon T, Fujiwara K, Yamamoto M, Abe T. The use of electrolyzed solutions for the cleaning and disinfecting of dialyzers. *Artif. Organs* 2000;24:921-8.
 541. Williams ND, Russell AD. The effects of some halogen-containing compounds on *Bacillus subtilis* endospores. *J. Appl. Bacteriol.* 1991;70:427-36.
 542. Babb JR, Bradley CR, Ayliffe GAJ. Sporicidal activity of glutaraldehydes and hypochlorites and other factors influencing their selection for the treatment of medical equipment. *J. Hosp. Infect.* 1980;1:63-75.
 543. Brown DG, Skyllis TP, Fekety FR. Comparison of chemical sterilant/disinfectant solutions against spores of *Clostridium difficile*. *Abstracts of the American Society for Microbiology*, 1983:Q39,267.
 544. Grant D, Venneman M, Burns RM. Mycobactericidal activity of Alcide an experimental liquid sterilant. *Abstracts of the Annual Meeting of the American Society of Microbiology*, 1982: Q101,226.

545. Korich DG, Mead JR, Madore MS, Sinclair NA, Sterling CR. Effects of ozone, chlorine dioxide, chlorine, and monochloramine on *Cryptosporidium parvum* oocyst viability. *Appl. Environ. Microbiol.* 1990;56:1423-8.
546. Griffiths PA, Babb JR, Fraise AP. Mycobactericidal activity of selected disinfectants using a quantitative suspension test. *J. Hosp. Infect.* 1999;41:111-21.
547. Centers for Disease Control. Bacteremia associated with reuse of disposable hollow-fiber hemodialyzers. *MMWR* 1986;35:417-8.
548. Bloomfield SF, Miller EA. A comparison of hypochlorite and phenolic disinfectants for disinfection of clean and soiled surfaces and blood spillages. *J. Hosp. Infect.* 1989;13:231-9.
549. Shetty N, Srinivasan S, Holton J, Ridgway GL. Evaluation of microbicidal activity of a new disinfectant: Sterilox® 2500 against *Clostridium difficile* spores, *Helicobacter pylori*, vancomycin resistant *Enterococcus* species, *Candida albicans* and several *Mycobacterium* species. *J. Hosp. Infect.* 1999;41:101-5.
550. Urata M, Isomot H, Murase K, et al. Comparison of the microbicidal activities of superoxidized and ozonated water in the disinfection of endoscopes. *J Intern Med Res* 2003;31:299-306.
551. Tsuji S, Kawano S, Oshita M, et al. Endoscope disinfection using acidic electrolytic water. *Endoscopy* 1999;31:528-35.
552. Lee JH, Rhee PL, Kim JH, et al. Efficacy of electrolyzed acid water in reprocessing patient-used flexible upper endoscopes: Comparison with 2% alkaline glutaraldehyde. *J. Gastroenterol. Hepatol.* 2004;19:897-904.
553. Centers for Disease Control. Acquired immune deficiency syndrome (AIDS): precautions for clinical and laboratory staffs. *MMWR* 1982;31:577-80.
554. Garner JS, Simmons BP. Guideline for isolation precautions in hospitals. *Infect. Control* 1983;4:245-325.
555. Van Bueren J, Simpson RA, Salman H, Farrelly HD, Cookson BD. Inactivation of HIV-1 by chemical disinfectants: sodium hypochlorite. *Epidemiol. Infect.* 1995;115:567-79.
556. Coates D. Disinfection of spills of body fluids: how effective is a level of 10,000 ppm available chlorine? *J. Hosp. Infect.* 1991;18:319-22.
557. Chitnis V, Chitnis S, Patil S, Chitnis D. Practical limitations of disinfection of body fluid spills with 10,000 ppm sodium hypochlorite (NaOCl). *Am. J. Infect. Control* 2004;32:306-8.
558. Anonymous. Recommendations for decontaminating manikins used in cardiopulmonary resuscitation training, 1983 update. *Infect. Control* 1984;5:399-401.
559. Centers for Disease Control. Use of bleach for disinfection of drug injection equipment. *MMWR* 1993;42:418-9.
560. Shapshak P, McCoy CB, Rivers JE, et al. Inactivation of human immunodeficiency virus-1 at short time intervals using undiluted bleach. *J. Acquir. Immune Defic. Syndr.* 1993;6:218-9.
561. Shapshak P, McCoy CB, Shah SM, et al. Preliminary laboratory studies of inactivation of HIV-1 in needles and syringes containing infected blood using undiluted household bleach. *J. Acquir. Immune Defic. Syndr.* 1994;7:754-9.
562. Brystrom A, Sundqvist G. Bacteriologic evaluation of the effect of 0.5 percent sodium hypochlorite in endodontic therapy. *Oral Surg. Oral Med. Oral Pathol.* 1983;55:307-12.
563. Favero MJ, Tokars JI, Arduino MJ, Alter MJ. Nosocomial infections associated with hemodialysis. In: Mayhall CG, ed. *Infect. Control and Hosp. Epidemiol.* Philadelphia: Lippincott Williams & Wilkins, 1999:897-917.
564. Helms CM, Massanari RM, Zeitler R, et al. Legionnaires' disease associated with a hospital water system: a cluster of 24 nosocomial cases. *Ann. Intern. Med.* 1983;99:172-8.
565. Heffelfinger JD, Kool JL, Fridkin S, et al. Risk of hospital-acquired legionnaires' disease in cities using monochloramine versus other water disinfectants. *Infect Control Hosp Epidemiol* 2003;24:569-74.
566. Moore MR, Pryor M, Fields B, Lucas C, Phelan M, Besser RE. Introduction of monochloramine into a municipal water system: Impact on colonization of buildings by *Legionella* spp. *Appl. Environ. Microbiol.* 2006;72:378-83.
567. Srinivasan A, Bova G, Ross T, et al. A 17-month evaluation of a chlorine dioxide water treatment system to control *Legionella* species in a hospital water supply. *Infect Control Hosp Epidemiol* 2003;24:575-9.
568. Steve L, Goodhart P, Alexander J. Hydrotherapy burn treatment: use of chloramine-T against resistant microorganisms. *Arch. Phys. Med. Rehabil.* 1979;60:301-3.

569. Coates D, Wilson M. Powders, composed of chlorine-releasing agent acrylic resin mixtures or based on peroxygen compounds, for spills of body fluids. *J. Hosp. Infect.* 1992;21:241-52.
570. Tulis JJ. Formaldehyde as a gas. In: Phillips GB, Miller WS, eds. *Industrial Sterilization*. Durham: Duke University Press, 1972:209-38.
571. Emmons CW. Fungicidal action of some common disinfectants on two dermatophytes. *Arch. Dermatol. Syphil.* 1933;28:15-21.
572. McCulloch EC, Costigan S. A comparison of the efficiency of phenol, liquor cresolis, formaldehyde, sodium hypochlorite and sodium hydroxide against *Eberthella typhi* at various temperatures. *J. Infect. Dis.* 1936;59:281-4.
573. Sagripanti JL, Eklund CA, Trost PA, et al. Comparative sensitivity of 13 species of pathogenic bacteria to seven chemical germicides. *Am. J. Infect. Control* 1997;25:335-9.
574. NIOSH. Formaldehyde: evidence of carcinogenicity. NIOSH Current Intelligence Bulletin 34. DHEW (NIOSH) Publication No. 81-111. 1981.
575. Occupational Safety and Health Administration. OSHA amends formaldehyde standard. *Occupational Safety and Health News* 1991:1.
576. Occupational Health and Safety Administration. OSHA Fact Sheet: Formaldehyde: Occupational Safety and Health Administration, U.S. Department of Labor, 2002.
577. Occupational Safety and Health Administration. Air Contaminants Final Rule. *Fed. Regist.* 1993;58:35338-51.
578. Occupational Safety and Health Administration. Formaldehyde: OSHA Fact Sheet: Occupational Safety and Health Administration, 2002.
579. Centers for Disease Control. Occupational exposures to formaldehyde in dialysis units. *MMWR* 1986;35:399-01.
580. Centers for Disease Control. Formaldehyde exposures in a gross anatomy laboratory - Colorado. *MMWR* 1983;52:698-700.
581. Tokars JI, Miller ER, Alter MJ, Arduino MJ. National surveillance of dialysis associated diseases in the United States, 1995. *ASAIO J.* 1998;44:98-107.
582. Favero MS, Alter MJ, Tokars JI, Bland LA. Dialysis-associated disease and their control. In: Bennett JV, Brachman PS, eds. *Hospital Infections*. Boston: Little, Brown and Company, 1998:357-80.
583. Bland LA, Favero MS. Microbial contamination control strategies for hemodialysis system. *Plant, Technology & Safety Management Series: infection control issues in PTSM 1990*. Oakbrook Terrace, Illinois.
584. Boucher RM. Potentiated acid 1,5 pentanedial solution--a new chemical sterilizing and disinfecting agent. *Am. J. Hosp. Pharm.* 1974;31:546-57.
585. Miner NA, McDowell JW, Willcockson GW, Bruckner NI, Stark RL, Whitmore EJ. Antimicrobial and other properties of a new stabilized alkaline glutaraldehyde disinfectant/sterilizer. *Am. J. Hosp. Pharm.* 1977;34:376-82.
586. Pepper RE. Comparison of the activities and stabilities of alkaline glutaraldehyde sterilizing solutions. *Infect. Control* 1980;1:90-2.
587. Leach ED. A new synergized glutaraldehyde-phenate sterilizing solution and concentrated disinfectant. *Infect. Control* 1981;2:26-30.
588. Miner NA, Ross C. Clinical evaluation of ColdSpor, a glutaraldehyde-phenolic disinfectant. *Respir. Care* 1991;36:104-9.
589. Collins FM, Montalbino V. Mycobactericidal activity of glutaraldehyde solutions. *J. Clin. Microbiol.* 1976;4:408-12.
590. Masferrer R, Marquez R. Comparison of two activated glutaraldehyde solutions: Cidex Solution and Sonacide. *Respir. Care* 1977;22:257-62.
591. Jette LP, Ringuette L, Ishak M, Miller M, Saint-Antoine P. Evaluation of three glutaraldehyde-based disinfectants used in endoscopy. *J. Hosp. Infect.* 1995;30:295-303.
592. Scott EM, Gorman SP. Glutaraldehyde. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001:361-81.
593. Scott EM, Gorman SP. Glutaraldehyde. In: Block SS, ed. *Disinfection, Sterilization, and Preservation*. Philadelphia: Lea & Febiger, 1991:596-616.
594. Stonehill AA, Krop S, Borick PM. Buffered glutaraldehyde - a new chemical sterilizing solution. *Am. J.*

- Hosp. Pharm. 1963;20:458-65.
595. Borick PM, Dondershine FH, Chandler VL. Alkalinized glutaraldehyde, a new antimicrobial agent. J. Pharm. Sci. 1964;53:1273-5.
596. Russell AD. Glutaraldehyde: current status and uses. Infect. Control Hosp. Epidemiol. 1994;15:724-33.
597. Hanson PJ, Bennett J, Jeffries DJ, Collins JV. Enteroviruses, endoscopy and infection control: an applied study. J. Hosp. Infect. 1994;27:61-7.
598. van Klingeren B, Pullen W. Glutaraldehyde resistant mycobacteria from endoscope washers. J. Hosp. Infect. 1993;25:147-9.
599. Griffiths PA, Babb JR, Bradley CR, Fraise AP. Glutaraldehyde-resistant *Mycobacterium chelonae* from endoscope washer disinfectors. J. Appl. Microbiol. 1997;82:519-26.
600. Dauendorffer JN, Laurain C, Weber M, Dailloux M. Evaluation of the bactericidal efficiency of a 2% alkaline glutaraldehyde solution on *Mycobacterium xenopi*. J. Hosp. Infect. 2000;46:73-6.
601. Nomura K, Ogawa M, Miyamoto H, Muratani T, Taniguchi H. Antibiotic susceptibility of glutaraldehyde-tolerant *Mycobacterium chelonae* from bronchoscope washing machines. J. Hosp. Infect. 2004;32:185-8.
602. Webster E, Ribner B, Streed LL, Hutton N. Microbial contamination of activated 2% glutaraldehyde used in high-level disinfection of endoscopes (abstract). Am. J. Infect. Control 1996;24:153.
603. Casemore DP, Blewett DA, Wright SE. Cleaning and disinfection of equipment for gastrointestinal flexible endoscopy: interim recommendations of a Working Party of the British Society of Gastroenterology. Gut 1989;30:1156-7.
604. Laskowski LF, Marr JJ, Spernoga JF, et al. Fastidious mycobacteria grown from porcine prosthetic-heart-valve cultures. N. Engl. J. Med. 1977;297:101-2.
605. Collins FM. Bactericidal activity of alkaline glutaraldehyde solution against a number of atypical mycobacterial species. J. Appl. Bacteriol. 1986;61:247-51.
606. Food and Drug Administration. Sterilants and high level disinfectants cleared by FDA in a 510(k) as of January 30, 2002 with general claims for processing reusable medical and dental devices, <http://www.fda.gov/cdrh/ode/germlab.html>, 2001.
607. Hernandez A, Martro E, Pizo C, et al. In-use evaluation of Perasafe compared with Cidex in fiberoptic bronchoscope disinfection. J. Hosp. Infect. 2003;54:46-52.
608. Leong D, Dorsey G, Klapp M. Dilution of glutaraldehyde by automatic endoscope machine washers: the need for a quality control program. Abstracts of the 14th Annual Educational Conference of Association for Practitioners in Infection Control, 1987:108, p.130.
609. Mbithi JN, Springthorpe VS, Sattar SA, Pacquette M. Bactericidal, virucidal, and mycobactericidal activities of reused alkaline glutaraldehyde in an endoscopy unit. J. Clin. Microbiol. 1993;31:2988-95.
610. Kleier DJ, Averbach RE. Glutaraldehyde nonbiologic monitors. Infect. Control Hosp. Epidemiol. 1990;11:439-41.
611. Kleier DJ, Tucker JE, Averbach RE. Clinical evaluation of glutaraldehyde nonbiologic monitors. Quintessence Int. 1989;20:271-7.
612. Overton D, Burgess JO, Beck B, Matis B. Glutaraldehyde test kits: evaluation for accuracy and range. Gen. Dent. 1989;37:126, 128.
613. Cooke RPD, Goddard SV, Chatterley R, Whyman-Morris A, Cheale J. Monitoring glutaraldehyde dilution in automated washer/disinfectors. J. Hosp. Infect. 2001;48:242-6.
614. Ayliffe GA, Babb JR, Bradley CR. Disinfection of endoscopes. J. Hosp. Infect. 1986;7:296-9.
615. Centers for Disease Control. Federal regulatory action against sporicidin cold sterilizing solution. MMWR 1991;40:880-1.
616. Husni L, Kale E, Climer C, Bostwick B, Parker TF, 3rd. Evaluation of a new disinfectant for dialyzer reuse. Am. J. Kidney Dis. 1989;14:110-8.
617. Townsend TR, Wee SB, Koblin B. An efficacy evaluation of a synergized glutaraldehyde-phenate solution in disinfecting respiratory therapy equipment contaminated during patient use. Infect. Control 1982;3:240-4.
618. Petersen NJ, Carson LA, Doto IL, Aguerro SM, Favero MS. Microbiologic evaluation of a new glutaraldehyde-based disinfectant for hemodialysis systems. Trans. Am. Soc. Artif. Intern. Organs 1982;28:287-90.
619. Gundogdu H, Ocal K, Caglikulekci M, Karabiber N, Bayramoglu E, Karahan M. High-level disinfection with 2% alkalinized glutaraldehyde solution for reuse of laparoscopic disposable plastic trocars. J.

- Laparoendosc. Adv. Surg. Techniques. Part A 1998;8:47-52.
620. Castelli M, Qizilbash A, Seaton T. Post-colonoscopy proctitis. *Am. J. Gastroenterol.* 1986;81:887.
621. Jonas G, Mahoney A, Murray J, Gertler S. Chemical colitis due to endoscope cleaning solutions: a mimic of pseudomembranous colitis. *Gastroenterology* 1988;95:1403-8.
622. Levine DS. Proctitis following colonoscopy. *Gastrointest. Endosc.* 1988;34:269-72.
623. Riney S, Grimes M, Khalife K, Warbasse L, Massanari M. Diarrhea associated with disinfection of sigmoidoscopes. [abstract]. *Am J Infect Control* 1991;19:109.
624. Durante L, Zulty JC, Israel E, et al. Investigation of an outbreak of bloody diarrhea: association with endoscopic cleaning solution and demonstration of lesions in an animal model. *Am. J. Med.* 1992;92:476-80.
625. Burtin P, Ruget O, Petit R, Boyer J. Glutaraldehyde-induced proctitis after endorectal ultrasound examination: a higher risk of incidence than expected? *Gastrointest. Endosc.* 1993;39:859-60.
626. Babb RR, Paaso BT. Glutaraldehyde proctitis. *West. J. Med.* 1995;163:477-8.
627. Ryan CK, Potter GD. Disinfectant colitis. Rinse as well as you wash. *J. Clin. Gastroenterol.* 1995;21:6-9.
628. Rozen P, Somjen GJ, Baratz M, Kimel R, Arber N, Gilat T. Endoscope-induced colitis: description, probable cause by glutaraldehyde, and prevention. *Gastrointest. Endosc.* 1994;40:547-53.
629. West AB, Kuan SF, Bennick M, Lagarde S. Glutaraldehyde colitis following endoscopy: clinical and pathological features and investigation of an outbreak. *Gastroenterology* 1995;108:1250-5.
630. Dolce P, Gourdeau M, April N, Bernard PM. Outbreak of glutaraldehyde-induced proctocolitis. *Am. J. Infect. Control* 1995;23:34-9.
631. Farina A, Fievet MH, Plassart F, Menet MC, Thuillier A. Residual glutaraldehyde levels in fiberoptic endoscopes: measurement and implications for patient toxicity. *J. Hosp. Infect.* 1999;43:293-7.
632. Dailey JR, Parnes RE, Aminlari A. Glutaraldehyde keratopathy. *Am. J. Ophthalmol.* 1993;115:256-8.
633. Courtright P, Lewallen S, Holland SP, Wendt TM. Corneal decompensation after cataract surgery. An outbreak investigation in Asia. *Ophthalmology* 1995;102:1461-5.
634. Leinster P, Baum JM, Baxter PJ. An assessment of exposure to glutaraldehyde in hospitals: typical exposure levels and recommended control measures. *Br. J. Ind. Med.* 1993;50:107-11.
635. Beauchamp RO, St Clair MB, Fennell TR, Clarke DO, Morgan KT. A critical review of the toxicology of glutaraldehyde. *Crit. Rev. Toxicol.* 1992;22:143-74.
636. Corrado OJ, Osman J, Davies RJ. Asthma and rhinitis after exposure to glutaraldehyde in endoscopy units. *Hum. Toxicol.* 1986;5:325-8.
637. Norback D. Skin and respiratory symptoms from exposure to alkaline glutaraldehyde in medical services. *Scand. J. Work, Environ. Health* 1988;14:366-71.
638. Mwaniki DL, Guthua SW. Occupational exposure to glutaraldehyde in tropical climates. *Lancet* 1992;340:1476-7.
639. Centers for Disease Control. Symptoms of irritation associated with exposure to glutaraldehyde. *MMWR* 1987;36:190-1.
640. Wiggins P, McCurdy SA, Zeidenberg W. Epistaxis due to glutaraldehyde exposure. *J. Occup. Med.* 1989;31:854-6.
641. Di Prima T, De Pasquale R, Nigro M. Contact dermatitis from glutaraldehyde. *Contact Dermatitis* 1988;19:219-20.
642. Fowler JF, Jr. Allergic contact dermatitis from glutaraldehyde exposure. *J. Occup. Med.* 1989;31:852-3.
643. Fisher AA. Allergic contact dermatitis of the hands from Sporicidin (glutaraldehyde-phenate) used to disinfect endoscopes. *Cutis* 1990;45:227-8.
644. Nethercott JR, Holness DL, Page E. Occupational contact dermatitis due to glutaraldehyde in health care workers. *Contact Dermatitis* 1988;18:193-6.
645. Gannon PF, Bright P, Campbell M, O'Hickey SP, Burge PS. Occupational asthma due to glutaraldehyde and formaldehyde in endoscopy and x ray departments. *Thorax* 1995;50:156-9.
646. Chan-Yeung M, McMurren T, Catonio-Begley F, Lam S. Occupational asthma in a technologist exposed to glutaraldehyde. *J. Allergy Clin. Immunol.* 1993;91:974-8.
647. Schnuch A, Uter W, Geier J, Frosch PJ, Rustemeyer T. Contact allergies in healthcare workers. Results from the IVDK. *Acta Derm. Venereol.* 1998;78:358-63.
648. Wellons SL, Trawick EG, Stowers MF, Jordan SL, Wass TL. Laboratory and hospital evaluation of four personal monitoring methods for glutaraldehyde in ambient air. *Am. Ind. Hyg. Assoc. J.* 1998;59:96-103.

649. Newman MA, Kachuba JB. Glutaraldehyde: a potential health risk to nurses. *Gastroenterol. Nurs.* 1992;14:296-300, discussion 300-1.
650. Association for the Advancement of Medical Instrumentation. Safe use and handling of glutaraldehyde-based products in healthcare facilities. Arlington, VA: AAMI, 1995.
651. Anonymous. Glutaraldehyde. New York: Occupational Health Services, Inc., 1992.
652. Rutala WA, Hamory BH. Expanding role of hospital epidemiology: employee health--chemical exposure in the health care setting. *Infect. Control Hosp. Epidemiol.* 1989;10:261-6.
653. Turner FJ. Hydrogen peroxide and other oxidant disinfectants. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lea & Febiger, 1983:240-50.
654. Block SS. Peroxygen compounds. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001:185-204.
655. Sattar SA, Springthorpe VS, Rochon M. A product based on accelerated and stabilized hydrogen peroxide: Evidence for broad-spectrum germicidal activity. *Canadian J Infect Control* 1998 (Winter):123-30.
656. Omidbakhsh N, Sattar SA. Broad-spectrum microbicidal activity, toxicologic assessment, and materials compatibility of a new generation of accelerated hydrogen peroxide-based environmental surface disinfectant. *Am. J. Infect. Control* 2006;34:251-7.
657. Schaeffer AJ, Jones JM, Amundsen SK. Bacterial effect of hydrogen peroxide on urinary tract pathogens. *Appl. Environ. Microbiol.* 1980;40:337-40.
658. Wardle MD, Renninger GM. Bactericidal effect of hydrogen peroxide on spacecraft isolates. *Appl. Microbiol.* 1975;30:710-1.
659. Sagripanti JL, Bonifacino A. Comparative sporicidal effect of liquid chemical germicides on three medical devices contaminated with spores of *Bacillus subtilis*. *Am. J. Infect. Control* 1996;24:364-71.
660. Sagripanti JL, Bonifacino A. Effects of salt and serum on the sporicidal activity of liquid disinfectants. *J. AOAC Int.* 1997;80:1198-207.
661. Saurina G, Landman D, Quale JM. Activity of disinfectants against vancomycin-resistant *Enterococcus faecium*. *Infect. Control Hosp. Epidemiol.* 1997;18:345-7.
662. Kilvington S. Moist-heat disinfection of *Acanthamoeba* cysts. *Rev. Infect. Dis.* 1991;13:S418.
663. Sattar SA, Adegbonrin O, Ramirez J. Combined application of simulated reuse and quantitative carrier test to assess high-level disinfection: Experiments with an accelerated hydrogen peroxide-based formulation. *Am. J. Infect. Control* 2002;30:449-57.
664. Leaper S. Influence of temperature on the synergistic sporicidal effect of peracetic acid plus hydrogen peroxide in *Bacillus subtilis* SA22(NCA 72-52). *Food Microbiol.* 1984;1:199-203.
665. Mentel R, Schmidt J. Investigations on rhinovirus inactivation by hydrogen peroxide. *Acta Virol.* 1973;17:351-4.
666. Sattar SA. Effect of liquid chemical germicides on mycobacteria including multi-drug resistant isolates of *Mycobacteria tuberculosis*. Abstracts of the 37th Interscience Conference on Antimicrobial Agents of Chemotherapy; September 28-October 1, 1997; Toronto, Ontario, Canada; E166., 1997.
667. Reckitt & Colman. Sporox sterilant and high-level disinfectant technical report. Montvale, NJ: Reckitt & Colman, 1997:1-12.
668. Sattar SA, Taylor YE, Paquette M, Rubino J. In-hospital evaluation of 7.5% hydrogen peroxide as a disinfectant for flexible endoscopes. *Can. J. Infect. Control* 1996;11:51-4.
669. Hobson DW, Seal LA. Evaluation of a novel, rapid-acting, sterilizing solution at room temperature. *Am. J. Infect. Control* 2000;28:370-5.
670. Anonymous. Hydrogen peroxide, ACS reagent. Vol. 2001: Sigma Product Information Sheet, <http://www.sigma-sial.com/sigma/proddata/h0904.htm>.
671. Silvany RE, Dougherty JM, McCulley JP, Wood TS, Bowman RW, Moore MB. The effect of currently available contact lens disinfection systems on *Acanthamoeba castellanii* and *Acanthamoeba polyphaga*. *Ophthalmology* 1990;97:286-90.
672. Moore MB. *Acanthamoeba keratitis* and contact lens wear: the patient is at fault. *Cornea* 1990;9:S33-5; discussion S39-40.
673. Judd PA, Tomlin PJ, Whitby JL, Inglis TC, Robinson JS. Disinfection of ventilators by ultrasonic nebulisation. *Lancet* 1968;2:1019-20.
674. Levenson JE. Corneal damage from improperly cleaned tonometer tips. *Arch. Ophthalmol.* 1989;107:1117.
675. Thompson RL, Haley CE, Searcy MA, et al. Catheter-associated bacteriuria. Failure to reduce attack rates

- using periodic instillations of a disinfectant into urinary drainage systems. JAMA 1984;251:747-51.
676. Bilotta JJ, Wayne JD. Hydrogen peroxide enteritis: the "snow white" sign. Gastrointest. Endosc. 1989;35:428-30.
677. Gottardi W. Iodine and iodine compounds. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lea & Febiger, 1991:152-66.
678. Gottardi W. Iodine and iodine compounds. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lippincott Williams & Wilkins, 2001:159-84.
679. Craven DE, Moody B, Connolly MG, Kollisch NR, Stottmeier KD, McCabe WR. Pseudobacteremia caused by povidone-iodine solution contaminated with *Pseudomonas cepacia*. N. Engl. J. Med. 1981;305:621-3.
680. Berkelman RL, Lewin S, Allen JR, et al. Pseudobacteremia attributed to contamination of povidone-iodine with *Pseudomonas cepacia*. Ann. Intern. Med. 1981;95:32-6.
681. Parrott PL, Terry PM, Whitworth EN, et al. *Pseudomonas aeruginosa* peritonitis associated with contaminated poloxamer-iodine solution. Lancet 1982;2:683-5.
682. Favero MS. Iodine--champagne in a tin cup. Infect. Control 1982;3:30-2.
683. Berkelman RL, Holland BW, Anderson RL. Increased bactericidal activity of dilute preparations of povidone-iodine solutions. J. Clin. Microbiol. 1982;15:635-9.
684. Chang SL. Modern concept of disinfection. J. Sanit. Eng. Div. Proc. Am. Soc. Civ. Eng. 1971:689-705.
685. Wallbank AM, Drulak M, Poffenroth L, Barnes C, Kay C, Lebttag I. Wescodyne: lack of activity against poliovirus in the presence of organic matter. Health Lab. Sci. 1978;15:133-7.
686. Carson JA, Favero MS. Comparative resistance of nontuberculous mycobacteria to iodophor germicides. Abstracts of the Annual Meeting of the American Society for Microbiology, 1984:Q101, p.221.
687. Medcom. Medcom Frequently Asked Questions. www.medcompnet.com/faq/faq/html, 2000.
688. Simons C, Walsh SE, Maillard JY, Russell AD. A note: ortho-phthalaldehyde: proposed mechanism of action of a new antimicrobial agent. Lett. Appl. Microbiol. 2000;31:299-302.
689. Walsh SE, Maillard JY, Simons C, Russell AD. Studies on the mechanisms of the antibacterial action of ortho-phthalaldehyde. J. Appl. Microbiol. 1999;87:702-10.
690. Fraud S, Hann AC, Maillard J-Y, Russell AD. Effects of ortho-phthalaldehyde, glutaraldehyde and chlorhexidine diacetate on *Mycobacterium chelonae* and *Mycobacterium abscessus* strains with modified permeability. J. Antimicrob. Chemother. 2003;51:575-84.
691. Cabrera-Martinez RM, Setlow B, Setlow P. Studies on the mechanisms of the sporicidal action of ortho-phthalaldehyde. J. Appl. Microbiol. 2002;92:675-80.
692. Gordon MD, Ezzell RJ, Bruckner NI, Ascenzi JM. Enhancement of mycobactericidal activity of glutaraldehyde with α,β -unsaturated and aromatic aldehydes. J. Indust. Microbiol. 1994;13:77-82.
693. Gregory AW, Schaalje GB, Smart JD, Robison RA. The mycobactericidal efficacy of ortho-phthalaldehyde and the comparative resistances of *Mycobacterium bovis*, *Mycobacterium terrae*, and *Mycobacterium chelonae*. Infect. Control Hosp. Epidemiol. 1999;20:324-30.
694. Walsh SE, Maillard JY, Russell AD. Ortho-phthalaldehyde: a possible alternative to glutaraldehyde for high level disinfection. J. Appl. Microbiol. 1999;86:1039-46.
695. Roberts CG, Chan Myers H. Mycobactericidal activity of dilute ortho-phthalaldehyde solutions. In: Abstracts in Environmental and General Applied Microbiology, Q-265, ASM 98th General Meeting, Atlanta, Georgia, USA, 1998:464-5.
696. Chan-Myers H. Sporicidal activity of ortho-phthalaldehyde as a function of temperature (abstract). Infect. Control Hosp. Epidemiol. 2000;21:101.
697. Chan-Myers H, Roberts C. Effect of temperature and organic soil concentration on biocidal activity of ortho-phthalaldehyde solution (abstract). 2000 Education Meeting of the Association for Professional in Infection Control and Epidemiology, Minneapolis, MN, 2000:31.
698. Bruckner NI, Gordon MD, Howell RG. Odorless aromatic dialdehyde disinfecting and sterilizing composition. US Patent 4,851,449. July, 1989.
699. McDonnell G, Pretzer D. New and developing chemical antimicrobials. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lippincott Williams & Wilkins, 2001:431-43.
700. Fraud S, Maillard J-Y, Russell AD. Comparison of the mycobactericidal activity of ortho-phthalaldehyde, glutaraldehyde, and other dialdehydes by a quantitative suspension test. J. Hosp. Infect. 2001;48:214-21.

701. Sattar SA, Springthorpe VS. New methods for efficacy testing of disinfectants and antiseptics. In: Rutala WA, ed. Disinfection, sterilization and antisepsis: principles and practices in healthcare facilities. Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:174-86.
702. Walsh SE, Maillard J-Y, Russell AD, Hann AC. Possible mechanisms for the relative efficiencies of ortho-phthalaldehyde and glutaraldehyde against glutaraldehyde-resistant *Mycobacterium chelonae*. J. Appl. Microbiol. 2001;91:80-92.
703. Herruzo-Cabrera R, Vizcaino-Alcaide MJ, Rodriguez J. Comparison of the microbicidal efficacy on germ carriers of several tertiary amine compounds with ortho-phthalaldehyde and Perasafe. J. Hosp. Infect. 2006;63:73-8.
704. Herruzo-Cabrera R, Vizcaino-Alcaide MJ, Fernandez-Acenero MJ. The influence of laboratory adaptation on test strains, such as *Pseudomonas aeruginosa*, in the evaluation of the antimicrobial efficacy of ortho-phthalaldehyde. J. Hosp. Infect. 2004;57:217-22.
705. Favero MS. Naturally occurring microorganisms and their resistance to physical and chemical agents. In: Rutala WA, ed. Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research. Washington, DC: Association for Professionals in Infection Control and Epidemiology, 2004:1-15.
706. Cooke RPD, Goddard SV, Whyman-Morris A, Sherwood J, Chatterly R. An evaluation of Cidex OPA (0.55% ortho-phthalaldehyde) as an alternative to 2% glutaraldehyde for high-level disinfection of endoscopes. J. Hosp. Infect. 2003;54:226-31.
707. Streckenbach SC, Alston TA. Perioral stains after ortho-phthalaldehyde disinfection of echo probes. Anesthesiol 2003;99:1032.
708. Wardle E, Jones D. Determination of rinsing volumes following manual endoscope disinfection with ortho-phthalaldehyde (OPA). J Gastroenterol Nurses College Australia 2003;January:7-9.
709. Sokol WN. Nine episodes of anaphylaxis following cystoscopy caused by Cidex OPA (ortho-phthalaldehyde) high-level disinfectant in 4 patients after cystoscopy. J. Allergy Clin. Immunol. 2004;114:392-7.
710. Hession SM. Endoscopic disinfection by ortho-phthalaldehyde in a clinical setting: An evaluation of reprocessing time and costs compared with glutaraldehyde. Gastroenterol. Nurs. 2003;26:110-4.
711. Tucker RC, Lestini BJ, Marchant RE. Surface analysis of clinically used expanded PTFE endoscopic tubing treated by the STERIS PROCESS. ASAIO J. 1996;42:306-13.
712. Hernandez A, Martro E, Matas L, Ausina V. In-vitro evaluation of Pearsafe compared with 2% alkaline glutaraldehyde against *Mycobacterium* spp. J. Hosp. Infect. 2003;54:52-6.
713. Vizcaino-Alcaide MJ, Herruzo-Cabrera R, Fernandez-Acenero MJ. Comparison of the disinfectant efficacy of Persafe and 2% glutaraldehyde in in vitro tests. J. Hosp. Infect. 2003;53:124-8.
714. Lensing HH, Oei HL. Investigations on the sporicidal and fungicidal activity of disinfectants. Zentralblatt für Bakteriologie, Mikrobiologie und Hygiene - 1 - Abt - Originale B, Hygiene 1985;181:487-95.
715. Sagripanti JL, Bonifacino A. Comparative sporicidal effects of liquid chemical agents. Appl. Environ. Microbiol. 1996;62:545-51.
716. Crow S. Peracetic acid sterilization: a timely development for a busy healthcare industry. Infect. Control Hosp. Epidemiol. 1992;13:111-3.
717. Malchesky PS. Medical applications of peracetic acid. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lippincott Williams & Wilkins, 2001:979-96.
718. Mannion PT. The use of peracetic acid for the reprocessing of flexible endoscopes and rigid cystoscopes and laparoscopes. J. Hosp. Infect. 1995;29:313-5.
719. Bradley CR, Babb JR, Ayliffe GA. Evaluation of the Steris System 1 Peracetic Acid Endoscope Processor. J. Hosp. Infect. 1995;29:143-51.
720. Duc DL, Ribiollet A, Dode X, Duceil G, Marchetti B, Calop J. Evaluation of the microbicidal efficacy of Steris System I for digestive endoscopes using GERMANDE and ASTM validation protocols. J. Hosp. Infect. 2001;48:135-41.
721. Alfa MJ, Olson N, Degagne P, Hizon R. New low temperature sterilization technologies: microbicidal activity and clinical efficacy. In: Rutala WA, ed. Disinfection, sterilization, and antisepsis in healthcare. Champlain, New York: Polyscience Publications, 1998:67-78.
722. Alfa MJ, DeGagne P, Olson N, Hizon R. Comparison of liquid chemical sterilization with peracetic acid and ethylene oxide sterilization for long narrow lumens. Am. J. Infect. Control 1998;26:469-77.

723. Seballos RJ, Walsh AL, Mehta AC. Clinical evaluation of a liquid chemical sterilization system for flexible bronchoscopes. *J. Bronch.* 1995;2:192-99.
724. Wallace CG, Agee PM, Demicco DD. Liquid chemical sterilization using peracetic acid. An alternative approach to endoscope processing. *ASAIO J.* 1995;41:151-4.
725. Centers for Disease Control and Prevention. Bronchoscopy-related infections and pseudoinfections - New York, 1996 and 1998. *MMWR* 1999;48:557-60.
726. Middleton AM, Chadwick MV, Gaya H. Disinfection of bronchoscopes, contaminated in vitro with *Mycobacterium tuberculosis*, *Mycobacterium avium-intracellulare* and *Mycobacterium chelonae* in sputum, using stabilized, buffered peracetic acid solution ('Nu-Cidex'). *J. Hosp. Infect.* 1997;37:137-43.
727. Holton J, Shetty N. In-use stability of Nu-Cidex. *J. Hosp. Infect.* 1997;35:245-8.
728. Alasri A, Roques C, Michel G, Cabassud C, Aptel P. Bactericidal properties of peracetic acid and hydrogen peroxide, alone and in combination, and chlorine and formaldehyde against bacterial water strains. *Can. J. Microbiol.* 1992;38:635-42.
729. Stanley P. Destruction of a glutaraldehyde-resistant mycobacterium by a per-oxygen disinfectant. (Abstract). *Am. J. Infect. Control* 1998;26:185.
730. Fleming SJ, Foreman K, Shanley K, Mihrshahi R, Siskind V. Dialyser reprocessing with Renalin. *Am. J. Nephrol.* 1991;11:27-31.
731. Kahn G. Depigmentation caused by phenolic detergent germicides. *Arch. Dermatol.* 1970;102:177-87.
732. Prindle RF. Phenolic compounds. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lea & Febiger, 1983:197-224.
733. Hegna IK. A comparative investigation of the bactericidal and fungicidal effects of three phenolic disinfectants. *J. Appl. Bacteriol.* 1977;43:177-81.
734. Hegna IK. An examination of the effect of three phenolic disinfectants on *Mycobacterium tuberculosis*. *J. Appl. Bacteriol.* 1977;43:183-7.
735. Bergan T, Lystad A. Antitubercular action of disinfectants. *J. Appl. Bacteriol.* 1971;34:751-6.
736. Narang HK, Codd AA. Action of commonly used disinfectants against enteroviruses. *J. Hosp. Infect.* 1983;4:209-12.
737. Cole EC, Rutala WA, Samsa GP. Disinfectant testing using a modified use-dilution method: collaborative study. *J. Assoc. Off. Anal. Chem.* 1988;71:1187-94.
738. Goddard PA, McCue KA. Phenolic compounds. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001:255-81.
739. Wysowski DK, Flynt JW, Jr., Goldfield M, Altman R, Davis AT. Epidemic neonatal hyperbilirubinemia and use of a phenolic disinfectant detergent. *Pediatrics* 1978;61:165-70.
740. Doan HM, Keith L, Shennan AT. Phenol and neonatal jaundice. *Pediatrics* 1979;64:324-5.
741. Shickman MD, Guze LB, Pearce ML. Bacteremia following cardiac catheterization. *N. Engl. J. Med.* 1959;260:1164-6.
742. Ehrenkranz NJ, Bolyard EA, Wiener M, Cleary TJ. Antibiotic-sensitive *Serratia marcescens* infections complicating cardiopulmonary operations: contaminated disinfectant as a reservoir. *Lancet* 1980;2:1289-92.
743. Shere L. Some comparisons of the disinfecting properties of hypochlorites and quaternary ammonium compounds. *Milk Plant Monthly* March 1948:66-9.
744. MacDougall KD, Morris C. Optimizing disinfectant application in healthcare facilities. *Infect Control Today* 2006;June:62-7.
745. Sykes G. *Disinfection and sterilization*. London: E & FN Spon Ltd, 1965.
746. Merianos JJ. Surface-active agents. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001:283-320.
747. Purohit A, Kopferschmitt-Kubler MC, Moreau C, Popin E, Blaumeiser M, Pauli G. Quaternary ammonium compounds and occupational asthma. *International Archives of Occupational & Environmental Health* 2000;73:423-7.
748. Petrocci AN. Surface active agents: quaternary ammonium compounds. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lea & Febiger, 1983:309-29.
749. Smith CR, Nishihara H, Golden F, Hoyt A, Guss CO, Kloetzel MC. The bactericidal effect of surface-active agents on tubercle bacilli. *Public Health Rep.* 1950;48:1588-1600.
750. Broadley SJ, Furr JR, Jenkins PA, Russell AD. Antimycobacterial activity of 'Virkon'. *J. Hosp. Infect.*

- 1993;23:189-97.
751. Angelillo IF, Bianco A, Nobile CG, Pavia M. Evaluation of the efficacy of glutaraldehyde and peroxygen for disinfection of dental instruments. *Lett. Appl. Microbiol.* 1998;27:292-6.
 752. Coates D. Disinfectants and spills of body fluids. *Nurs. RSA* 1992;7:25-7.
 753. Hamouda T, Hayes MM, Cao ZH, et al. A novel surfactant nanoemulsion with broad-spectrum sporicidal activity against *Bacillus* species. *J. Infect. Dis.* 1999;180:1939-49.
 754. Hamouda T, Myc A, Donovan B, Shih AY, Reuter JD, Baker JR. A novel surfactant nanoemulsion with a unique non-irritant topical antimicrobial activity against bacteria, enveloped viruses and fungi. *Microbiol. Res.* 2001;156:1-7.
 755. Hamouda T, Baker JR, Jr. Antimicrobial mechanism of action of surfactant lipid preparations in enteric Gram-negative bacilli. *J. Appl. Microbiol.* 2000;89:397-403.
 756. Widmer AF, Frei R. Antimicrobial activity of glucoprotamin: A clinical study of a new disinfectant for instruments. *Infect Control Hosp Epidemiol* 2003;24:762-4.
 757. Wilson M. Light-activated antimicrobial coating for the continuous disinfection of surfaces. *Infect Control Hosp Epidemiol* 2003;24:782-4.
 758. Schneider PM. New technologies for disinfection and sterilization. In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research.* Washington DC: Association for Professionals in Infection Control and Epidemiology, 2004:127-39.
 759. Rotter ML. Handwashing, hand disinfection, and skin disinfection. In: Wenzel RP, ed. *Prevention and control of nosocomial infections.* Baltimore: Williams and Wilkins, 1997:691-709.
 760. Mandel GL, Bennett JE, Dolin R. *Principles and practices of infectious diseases.* New York: Livingstone, 2000.
 761. Weber DJ, Rutala WA. Use of metals and microbicides in the prevention of nosocomial infections. In: Rutala W, ed. *Disinfection, sterilization, and antisepsis in healthcare.* Champlain, New York: Polyscience Publications, 1995:271-85.
 762. Weber DJ, Rutala WA. Use of metals as microbicides in preventing infections in healthcare. In: Block SS, ed. *Disinfection, sterilization, and preservation.* Philadelphia: Lippincott Williams & Wilkins, 2001:415-30.
 763. Brady MJ, Lisay CM, Yurkovetskiy AV, Sawan SP. Persistent silver disinfectant for the environmental control of pathogenic bacteria. *Am. J. Infect. Control* 2003;31:208-214.
 764. Rusin P, Bright K, Gerba C. Rapid reduction of *Legionella pneumophila* on stainless steel with zeolite coatings containing silver and zinc ions. *Lett. Appl. Microbiol.* 2003;36:69-72.
 765. Bright KR, Gerba CP, Rusin PA. Rapid reduction of *Staphylococcus aureus* populations on stainless steel surfaces by zeolite ceramic coatings containing silver and zinc ions. *J. Hosp. Infect.* 2002;52:307-9.
 766. Landeen LK, Yahya MT, Gerba CP. Efficacy of copper and silver ions and reduced levels of free chlorine in inactivation of *Legionella pneumophila*. *Appl. Environ. Microbiol.* 1989;55:3045-50.
 767. Pyle BH, Broadaway SC, McFeters GA. Efficacy of copper and silver ions with iodine in the inactivation of *Pseudomonas cepacia*. *J. Appl. Bacteriol.* 1992;72:71-9.
 768. Yahya MT, Landeen LK, Messina MC, Kutz SM, Schulze R, Gerba CP. Disinfection of bacteria in water systems by using electrolytically generated copper:silver and reduced levels of free chlorine. *Can. J. Microbiol.* 1990;36:109-16.
 769. Liu Z, Stout JE, Tedesco L, et al. Controlled evaluation of copper-silver ionization in eradicating *Legionella pneumophila* from a hospital water distribution system. *J. Infect. Dis.* 1994;169:919-22.
 770. Noyce JO, Michels H, Keevil CW. Potential use of copper surfaces to reduce survival of epidemic methicillin-resistant *Staphylococcus aureus* in the healthcare environment. *J. Hosp. Infect.* 2006;63:289-97.
 771. Goetz A, Yu VL. Copper-silver ionization: cautious optimism for *Legionella* disinfection and implications for environmental culturing. *Am. J. Infect. Control* 1997;25:449-51.
 772. Miuetzner S, Schwille RC, Farley A, et al. Efficacy of thermal treatment and copper-silver ionization for controlling *Legionella pneumophila* in high-volume hot water plumbing systems in hospitals. *Am. J. Infect. Control* 1997;25:452-7.
 773. Stout JE, Lin YS, Goetz AM, Muder RR. Controlling *Legionella* in hospital water systems: experience with the superheat-and-flush method and copper-silver ionization. *Infect. Control Hosp. Epidemiol.* 1998;19:911-4.

774. Stout JE, Yu VL. Experience of the first 16 hospitals using copper-silver ionization for *Legionella* control: Implications for the evaluation of other disinfection modalities. *Infect Control Hosp Epidemiol* 2003;24:563-8.
775. Russell AD. Ultraviolet radiation. In: Russell AD, Hugo WB, Ayliffe GAJ, eds. Principles and practices of disinfection, preservation and sterilization. Oxford: Blackwell Science, 1999:688-702.
776. Hall KK, Giannetta ET, Getchell-White SI, Durbin LJ, Farr BM. Ultraviolet light disinfection of hospital water for preventing nosocomial *Legionella* infection: A 13-year follow-up. *Infect Control Hosp Epidemiol* 2003;24:580-3.
777. Singh S, Schaaf NG. Dynamic sterilization of titanium implants with ultraviolet light. *Internat. J. Oral Maxillofac. Implants* 1989;4:139-46.
778. Dolman PJ, Dobrogowski MJ. Contact lens disinfection by ultraviolet light. *Am. J. Ophthalmol.* 1989;108:665-9.
779. Shechmeister IL. Sterilization by ultraviolet irradiation. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lea & Febiger, 1991:553-65.
780. National Research Council. Postoperative wound infections - the influence of ultraviolet irradiation of the operating room and of various other factors. *Ann. Surg.* 1964;160:1-125.
781. Sensakovic JW, Smith LG. Nosocomial ultraviolet keratoconjunctivitis. *Infect. Control* 1982;3:475-6.
782. Cefai C, Richards J, Gould FK, McPeake P. An outbreak of respiratory tract infection resulting from incomplete disinfection of ventilatory equipment. *J. Hosp. Infect.* 1990;15:177-82.
783. Gurevich I, Tafuro P, Ristuccia P, Herrmann J, Young AR, Cunha BA. Disinfection of respirator tubing: a comparison of chemical versus hot water machine-assisted processing. *J. Hosp. Infect.* 1983;4:199-208.
784. Rutala WA, Weber DJ, Gergen MF, Gratta AR. Efficacy of a washer-pasteurizer for disinfection of respiratory-care equipment. *Infect. Control Hosp. Epidemiol.* 2000;21:333-6.
785. Jette LP, Lambert NG. Evaluation of two hot water washer disinfectors for medical instruments. *Infect. Control Hosp. Epidemiol.* 1988;9:194-9.
786. Wang C-Y, Wu H-D, Lee L-N, et al. Pasteurization is effective against multidrug-resistant bacteria. *Am. J. Infect. Control* 2006;34:320-2.
787. Dempsey KM, Chiew RF, McKenzie JA, Mitchell DH. Evaluation of the cleaning and disinfection efficacy of the DEKO-190; award-based automated washer/disinfectant. *J. Hosp. Infect.* 2000;46:50-4.
788. Kearns AM, Freeman R, Lightfoot NF. Nosocomial enterococci: resistance to heat and sodium hypochlorite. *J. Hosp. Infect.* 1995;30:193-9.
789. Bradley CR, Fraise AP. Heat and chemical resistance of enterococci. *J. Hosp. Infect.* 1996;34:191-6.
790. Chadwick PR, Oppenheim BA. Vancomycin-resistant enterococci and bedpan washer machines. *Lancet* 1994;344:685.
791. Nystrom B. New technology for sterilization and disinfection. *Am. J. Med.* 1991;91:264S-266S.
792. Sanders FT, Morrow MS. The EPA's role in the regulation of antimicrobial pesticides in the United States. In: Rutala WA, ed. Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research. Washington, DC: Association for Professionals in Infection Control and Epidemiology, 2004:29-41.
793. Anonymous. Memorandum of understanding between the Food and Drug Administration, Public Health Service, and the Environmental Protection Agency, 1993.
794. Ulatowski TA. Current activities concerning the premarket evaluation of infection control devices at the Food and Drug Administration. In: Rutala WA, ed. Disinfection, sterilization, and antisepsis in healthcare. Champlain, New York: Polyscience Publications, 1998:1-7.
795. Cole EC, Rutala WA. Bacterial numbers on penicylinders used in disinfectant testing: use of 24 hour adjusted broth cultures. *J. Assoc. Off. Anal. Chem.* 1988;71:9-11.
796. Cole EC, Rutala WA, Alfano EM. Comparison of stainless steel penicylinders used in disinfectant testing. *J. Assoc. Off. Anal. Chem.* 1988;71:288-9.
797. Cole EC, Rutala WA, Carson JL. Evaluation of penicylinders used in disinfectant testing: bacterial attachment and surface texture. *J. Assoc. Off. Anal. Chem.* 1987;70:903-6.
798. Cole EC, Rutala WA, Samsa GP. Standardization of bacterial numbers of penicylinders used in disinfectant testing: interlaboratory study. *J. Assoc. Off. Anal. Chem.* 1987;70:635-7.
799. Alfano EM, Cole EC, Rutala WA. Quantitative evaluation of bacteria washed from stainless steel penicylinders during AOAC use-dilution method. *J. Assoc. Off. Anal. Chem.* 1988;71:868-71.

800. Favero MS, Groschel DHM. Chemical germicides in the health care field: current status and evaluation of efficacy and research needs. Washington, DC: American Society for Microbiology, 1987.
801. Sattar SA. Microbicidal testing of germicides: an update. In: Rutala WA, ed. Disinfection, sterilization, and antisepsis in healthcare. Champlain, New York: Polyscience Publications, 1998:227-40.
802. Best M. Development of a combined carrier test for disinfectant efficacy. Ottawa, Canada: University of Ottawa, 1994.
803. Sattar SA, Springthorpe VS. Recent developments in methods for testing the germicidal activity of disinfectants and antiseptics. In: Rutala WA, ed. Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research. Washington, DC: Association for Professionals in Infection Control and Epidemiology, 2004:180-8.
804. Sanders FT. Environmental protection agency's role in the regulation of antimicrobial pesticides in the United States. In: Rutala WA, ed. Disinfection, Sterilization and Antisepsis: principles and practices in healthcare facilities. Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:28-40.
805. Groschel DHM. Caveat emptor: do your disinfectants work? Infect. Control 1983;4:144.
806. United States General Accounting Office. Disinfectants: EPA lacks assurance they work., 1990.
807. Johnston MD, Lambert RJW, Hanlon GW, Denyer SP. A rapid method for assessing the suitability of quenching agents for individual biocides as well as combinations. J. Appl. Microbiol. 2002;92:784-9.
808. Russell AD. Neutralization procedures in the evaluation of bactericidal activity. In: Collins CH, Allwood MC, Bloomfield SF, Fox A, eds. Disinfectants: their use and evaluation of effectiveness. London: Academic Press, 1981:45-59.
809. Russell AD, Ahonkhai I, Rogers DT. Microbiological applications of the inactivation of antibiotics and other antimicrobial agents. J. Appl. Bacteriol. 1979;46:207-45.
810. Engley FB, Jr, Dey BP. A universal neutralizing medium for antimicrobial chemicals. Chem. Specialists Manuf. Assoc. Proc. 1970:100-6.
811. Association for the Advancement of Medical Instrumentation. Good hospital practice: Steam sterilization and sterility assurance. AAMI. Arlington, VA, 1993.
812. Association for the Advancement of Medical Instrumentation. Flash sterilization: Steam sterilization of patient care items for immediate use. AAMI. Arlington, VA, 1996.
813. Association for the Advancement of Medical Instrumentation. Steam sterilization and sterility assurance in health care facilities. ANSI/AAMI ST46. Arlington, VA, 2002:ANSI/AAMI ST46:2002.
814. Association for the Advancement of Medical Instrumentation. Ethylene oxide sterilization in health care facilities: Safety and effectiveness. AAMI. Arlington, VA, 1999.
815. Association of Operating Room Nurses. Recommended practices for sterilization in perioperative practice settings. 2000 Standards, Recommended Practices, and Guidelines. Denver, CO: AORN, 2000:347-58.
816. Association for peri-Operative Registered Nurses. Recommended practices for cleaning and caring for surgical instruments and powered equipment. AORN J. 2002;75:727-41.
817. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for prevention of surgical site infection, 1999. Hospital Infection Control Practices Advisory Committee. Infect. Control Hosp. Epidemiol. 1999;20:250-78.
818. Education Design. Best practices for the prevention of surgical site infection. Denver Colorado: Education Design, 1998.
819. Association for the Advancement of Medical Instrumentation. Comprehensive guide to steam sterilization and sterility assurance in health care facilities, ANSI/AAMI ST79. 2006.
820. Association for peri-Operative Registered Nurses. Recommended practice for sterilization in the perioperative practice setting. AORN J. 2006;83:700-22.
821. Singh J, Bhatia R, Gandhi JC, et al. Outbreak of viral hepatitis B in a rural community in India linked to inadequately sterilized needles and syringes. Bull. World Health Organ. 1998;76:93-8.
822. Eickhoff TC. An outbreak of surgical wound infections due to *Clostridium perfringens*. Surg. Gynecol. Obstet. 1962;114:102-8.
823. Favero MS. Sterility assurance: Concepts for patient safety. In: Rutala WA, ed. Disinfection, sterilization and antisepsis: principles and practices in healthcare facilities. Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:110-9.
824. Oxborrow GS, Berube R. Sterility testing-validation of sterilization processes, and sporicide testing. In:

- Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lea & Febiger, 1991:1047-57.
825. Rutala WA, Weber DJ. Clinical effectiveness of low-temperature sterilization technologies. *Infect. Control Hosp. Epidemiol.* 1998;19:798-804.
826. Adler S, Scherrer M, Daschner FD. Costs of low-temperature plasma sterilization compared with other sterilization methods. *J. Hosp. Infect.* 1998;40:125-34.
827. Joslyn L. Sterilization by heat. In: Block SS, ed. Disinfection, sterilization, and preservation. Philadelphia: Lippincott Williams & Wilkins, 2001:695-728.
828. Bucx MJ, Veldman DJ, Beenhakker MM, Koster R. The effect of steam sterilization at 134 degrees C on light intensity provided by fibrelight Macintosh laryngoscopes. *Anaesthesia* 2000;55:185-6.
829. Gilbert JA, Phillips HO. The effect of steam sterilization on plaster casting material. *Clinical Orthopaed Rel Res* 1984:241-4.
830. Agaloco JP, Akers JE, Madsen RE. Moist heat sterilization--myths and realities. *PDA J. Pharmaceutical Sci. Technol.* 1998;52:346-50.
831. Rutala WA, Stiegel MM, Sarubbi FA, Jr. Decontamination of laboratory microbiological waste by steam sterilization. *Appl. Environ. Microbiol.* 1982;43:1311-6.
832. Lauer JL, Battles DR, Vesley D. Decontaminating infectious laboratory waste by autoclaving. *Appl. Environ. Microbiol.* 1982;44:690-4.
833. Rhodes P, Zelner L, Laufman H. A new disposable bowie-Dick-type test pack for prevacuum high-temperature sterilizers. *Med. Instrum.* 1982;16:117-20.
834. Association for the Advancement of Medical Instrumentation. Technical Information Report on process challenge devices/test packs for use in health care facilities, 2003.
835. Young M. Sterilization process monitoring. *Managing Infect Control* 2004;August:70-6.
836. American Society for Healthcare Central Service Professionals. Training Manual for Health Care Central Service Technicians. In: Association AH, ed. Chicago: The Jossey-Bass/American Hospital Association Press Series, 2001:1-271.
837. Crow S. Steam sterilizers: an evolution in design. *Infect. Control Hosp. Epidemiol.* 1993;14:488-90.
838. Gurevich I, Jacobsen E, Cunha BA. Pseudoautoclave failure caused by differences in spore test steam sensitivities. *Am. J. Infect. Control* 1996;24:402-4.
839. Bryce EA, Roberts FJ, Clements B, MacLean S. When the biological indicator is positive: investigating autoclave failures. *Infect. Control Hosp. Epidemiol.* 1997;18:654-6.
840. Barone MA, Faisal AJ, Andrews L, Ahmed J, Rashida B, Kristensen D. Adaptation and validation of a portable steam sterilizer for processing intrauterine device insertion instruments and supplies in low-resource settings. *Am. J. Infect. Control* 1997;25:350-6.
841. Young JH. Sterilization with steam under pressure. In: Morrissey RF, Phillips GB, eds. Sterilization technology: a practical guide for manufacturers and users of health care product. New York: Van Nostrand Reinhold, 1993:81-119.
842. Palenik CJ, Cumberlander ND. Effects of steam sterilization on the contents of sharps containers. *Am. J. Infect. Control* 1993;21:28-33.
843. Rutala WA. Disinfection and flash sterilization in the operating room. *J. Ophthal. Nurs. Technol.* 1991;10:106-15.
844. Maki DG, Hassemer CA. Flash sterilization: carefully measured haste. *Infect. Control* 1987;8:307-10.
845. Barrett T. Flash sterilization: What are the risks? In: Rutala WA, ed. Disinfection, sterilization and antisepsis: principles and practices in healthcare facilities. Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:70-6.
846. Vesley D, Langholz AC, Rohlfing SR, Foltz WE. Fluorimetric detection of a *Bacillus stearothermophilus* spore-bound enzyme, α -D-glucosidase, for rapid identification of flash sterilization failure. *Appl. Environ. Microbiol.* 1992;58:717-9.
847. Rutala WA, Gergen MF, Weber DJ. Evaluation of a rapid readout biological indicator for flash sterilization with three biological indicators and three chemical indicators. *Infect. Control Hosp. Epidemiol.* 1993;14:390-4.
848. Strzelecki LR, Nelson JH. Evaluation of closed container flash sterilization system. *Orthoped. Nurs.* 1989;8:21-4.
849. Hood E, Stout N, Catto B. Flash sterilization and neurosurgical site infections: Guilt by association. *Am. J.*

- Infect. Control 1997;25:156.
850. Rutala WA, Weber DJ, Chappell KJ. Patient injury from flash-sterilized instruments. *Infect. Control Hosp. Epidemiol.* 1999;20:458.
 851. Schneider PM. Low-temperature sterilization alternatives in the 1990s. *Tappi J.* 1994;77:115-9.
 852. Environmental Protection Agency. Protection of stratospheric ozone; Proposed Rule. 40 CFR Part 82. *Fed. Regist.* 1993.
 853. Schneider PM. Emerging low temperature sterilization technologies (non-FDA approved). In: Rutala WA, ed. *Disinfection, sterilization, and antisepsis in healthcare.* Champlain, New York: Polyscience Publications, 1998:79-92.
 854. Gross D. Ethylene oxide sterilization and alternative methods. *Surg. Serv. Management* 1995;1:16-7.
 855. Holler C, Martiny H, Christiansen B, Ruden H, Gundermann KO. The efficacy of low temperature plasma (LTP) sterilization, a new sterilization technique. *Zentralbl. Hyg. Umweltmed.* 1993;194:380-91.
 856. Rutala WA, Gergen MF, Weber DJ. Comparative evaluation of the sporicidal activity of new low-temperature sterilization technologies: ethylene oxide, 2 plasma sterilization systems, and liquid peracetic acid. *Am. J. Infect. Control* 1998;26:393-8.
 857. Ernst RR, Doyle JE. Sterilization with gaseous ethylene oxide: a review of chemical and physical factors. *Biotech. Bioeng.* 1968;10.
 858. Joslyn L. Gaseous chemical sterilization. In: Block SS, ed. *Disinfection, sterilization, and preservation.* Philadelphia: Lippincott Williams & Wilkins, 2001:337-60.
 859. Fisher AA. Ethylene oxide dermatitis. *Cutis* 1984;34:20, 22, 24.
 860. Jay WM, Swift TR, Hull DS. Possible relationship of ethylene oxide exposure to cataract formation. *Am. J. Ophthalmol.* 1982;93:727-32.
 861. Salinas E, Sasich L, Hall DH, Kennedy RM, Morriss H. Acute ethylene oxide intoxication. *Drug Intell. Clin. Pharm.* 1981;15:384-6.
 862. Marchand M, Delesvonx R, Claeys C. The toxicity of ethylene oxide and a report on three fatal cases of poisoning. *Am. Arch. Indust. Health* 1958;18:60.
 863. Finelli PF, Morgan TF, Yaar I, Granger CV. Ethylene oxide-induced polyneuropathy. A clinical and electrophysiologic study. *Arch. Neurol.* 1983;40:419-21.
 864. Estrin WJ, Becker CE. Evidence of neurologic dysfunction related to long-term ethylene oxide exposure. *Arch. Neurol.* 1987;44:1283-6.
 865. Estrin WJ, Bowler RM, Lash A, Becker CE. Neurotoxicological evaluation of hospital sterilizer workers exposed to ethylene oxide. *J. Toxicol. Clin. Toxicol.* 1990;28:1-20.
 866. Crystal HA, Schaumburg HH, Grober E, Fuld PA, Lipton RB. Cognitive impairment and sensory loss associated with chronic low-level ethylene oxide exposure. *Neurology* 1988;38:567-9.
 867. Shaham J, Levi Z, Gurvich R, Shain R, Ribak J. Hematological changes in hospital workers due to chronic exposure to low levels of ethylene oxide. *J. Occup. Environ. Med.* 2000;42:843-50.
 868. Lindbohm ML, Hemminki K, Bonhomme MG, et al. Effects of paternal occupational exposure on spontaneous abortions. *Am. J. Public Health* 1991;81:1029-33.
 869. Hemminki K, Mutanen P, Saloniemi I, Niemi M-L, Vainio H. Spontaneous abortions in hospital staff engaged in sterilising instruments with chemical agents. *Br. Med. J.* 1982;285:1461-3.
 870. Rowland AS, Baird DD, Shore DL, Darden B, Wilcox AJ. Ethylene oxide exposure may increase the risk of spontaneous abortion, preterm birth, and postterm birth. *Epidemiology* 1996;7:363-8.
 871. National Toxicology Program. <http://ntp-server.niehs.nih.gov/>.
 872. Anonymous. Ethylene oxide sterilization: How hospitals can adapt to the changes. *Health Devices* 1994;23:485-92.
 873. Occupational Safety and Health Administration. Ethylene Oxide: OSHA Fact Sheet: Occupational Safety and Health Administration, 2002.
 874. Cardenas-Camarena L. Ethylene oxide burns from improperly sterilized mammary implants. *Ann. Plast. Surg.* 1998;41:361-9.
 875. Windebank AJ, Blehrud MD. Residual ethylene oxide in hollow fiber hemodialysis units is neurotoxic in vitro. *Ann. Neurol.* 1989;26:63-8.
 876. Occupational Health and Safety Administration. Chemical sampling information-Ethylene chlorohydrin: Occupational Safety and Health Administration, 2002.
 877. Parisi AN, Young WE. Sterilization with ethylene oxide and other gases. In: Block SS, ed. *Disinfection,*

- sterilization, and preservation. Philadelphia: Lea & Febiger, 1991:580-95.
878. Ries MD, Weaver K, Beals N. Safety and efficacy of ethylene oxide sterilized polyethylene in total knee arthroplasty. *Clin. Orthop.* 1996;159-63.
879. Alfa MJ, DeGagne P, Olson N. Bacterial killing ability of 10% ethylene oxide plus 90% hydrochlorofluorocarbon sterilizing gas. *Infect. Control Hosp. Epidemiol.* 1997;18:641-5.
880. Parker HH, Johnson RB. Effectiveness of ethylene oxide for sterilization of dental handpieces. *J. Dent.* 1995;23:113-5.
881. Jacobs PT, Lin SM. Sterilization processes utilizing low-temperature plasma. In: Block SS, ed. *Disinfection, sterilization, and preservation.* Philadelphia: Lippincott Williams & Wilkins, 2001:747-63.
882. Rutala WA, Gergen MF, Weber DJ. Sporicidal activity of a new low-temperature sterilization technology: the Sterrad 50 sterilizer. *Infect. Control Hosp. Epidemiol.* 1999;20:514-6.
883. Kyi MS, Holton J, Ridgway GL. Assessment of the efficacy of a low temperature hydrogen peroxide gas plasma sterilization system. *J. Hosp. Infect.* 1995;31:275-84.
884. Jacobs PT, Smith D. The new Sterrad 100S sterilization system: Features and advantages. *Zentr. Steril.* 1998;6:86-94.
885. Rudolph H, Hilbert M. Practical testing of the new plasma sterilizer "Sterrad 100S" in the Diakonkrankenhaus Rotenburg. *Zentr. Steril.* 1997;5:207-15.
886. Bar W, Marquez de Bar G, Naumann A, Rusch-Gerdes S. Contamination of bronchoscopes with *Mycobacterium tuberculosis* and successful sterilization by low-temperature hydrogen peroxide plasma sterilization. *Am. J. Infect. Control* 2001;29:306-11.
887. Centers for Disease Control and Prevention. Corneal decompensation after intraocular ophthalmic surgery—Missouri, 1998. *MMWR* 1998;47:306-9.
888. Duffy RE, Brown SE, Caldwell KL, et al. An epidemic of corneal destruction caused by plasma gas sterilization. *Arch. Ophthalmol.* 2000;118:1167-76.
889. Jarvis WR. Hospital Infections Program, Centers for Disease Control and Prevention: On-site outbreak investigations, 1990-1999: How often are germicides or sterilants the source? In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: principles and practices in healthcare facilities.* Washington, DC: Association for Professional in Infection Control and Epidemiology, 2001:41-8.
890. Borneff M, Ruppert J, Okpara J, et al. Efficacy testing of low-temperature plasma sterilization (LTP) with test object models simulating practice conditions. *Zentr. Steril.* 1995;3:361-71.
891. Borneff-Lipp M, Okpara J, Bodendorf M, Sonntag HG. Validation of low-temperature-plasma (LPT) sterilization systems: Comparison of two technical versions, the Sterrad 100, 1.8 and the 100S. *Hygiene und Mikrobiologie* 1997;3:21-8.
892. Roberts C, Antonoplos P. Inactivation of human immunodeficiency virus type 1, hepatitis A virus, respiratory syncytial virus, vaccinia virus, herpes simplex virus type 1, and poliovirus type 2 by hydrogen peroxide gas plasma sterilization. *Am. J. Infect. Control* 1998;26:94-101.
893. Okpara-Hofmann J, Knoll M, Durr M, Schmitt B, Borneff-Lipp M. Comparison of low-temperature hydrogen peroxide gas plasma sterilization for endoscopes using various Sterrad models. *J. Hosp. Infect.* 2005;59:280-5.
894. Timm D, Gonzales D. Effect of sterilization on microstructure and function of microsurgical scissors. *Surg. Serv. Management* 1997;3:47-9.
895. Feldman LA, Hui HK. Compatibility of medical devices and materials with low-temperature hydrogen peroxide gas plasma. *Med. Dev. Diag. Indust.* 1997;19:57-62.
896. Muscarella LF. Leading a horse to water: Are crucial lessons in endoscopy and outbreak investigations being learned? *Infect. Control Hosp. Epidemiol.* 2002;23:358-60.
897. Gurevich I, Qadri SMH, Cunha BA. False-positive results of spore tests from improper clip use with the Steris chemical sterilant system. *Infect. Control Hosp. Epidemiol.* 1992;21:42-3.
898. Kralovic RC. Use of biological indicators designed for steam or ethylene oxide to monitor a liquid chemical sterilization process. *Infect. Control Hosp. Epidemiol.* 1993;14:313-9.
899. Bond WW. Biological indicators for a liquid chemical sterilizer. *Infect. Control Hosp. Epidemiol.* 1993;14:565.
900. Bond WW. Biological indicators for a liquid chemical sterilizer: a solution to the instrument reprocessing problem? *Infect. Control Hosp. Epidemiol.* 1993;14:309-12.
901. Malchesky PS. Biological indicators for a liquid chemical sterilizer. *Infect. Control Hosp. Epidemiol.*

- 1993;14:563-6.
902. Daschner F. STERIS SYSTEM 1 in Germany. *Infect. Control Hosp. Epidemiol.* 1994;15:294, 296.
 903. Sorin M, Segal-Maurer S, Urban C, Combest A, Rahal JJ. Nosocomial transmission of imipenem-resistant *Pseudomonas aeruginosa* following bronchoscopy associated with improper connection to the STERIS System 1 Processor. *Infect. Control Hosp. Epidemiol.* 2001;22:409-13.
 904. Food and Drug Administration, Division of General and Restorative Devices. Guidance on Premarket Notification [510(k)] Submissions for Sterilizers Intended for Use in Health Care Facilities. Rockville, MD. 1993.
 905. Vickery K, Deva AK, Zou J, Kumaradeva P, Bissett L, Cossart YE. Inactivation of duck hepatitis B virus by a hydrogen peroxide gas plasma sterilization system: laboratory and 'in use' testing. *J. Hosp. Infect.* 1999;41:317-22.
 906. Vassal S, Favennec L, Ballet JJ, Brasseur P. Hydrogen peroxide gas plasma sterilization is effective against *Cryptosporidium parvum* oocysts. *Am. J. Infect. Control* 1998;26:136-8.
 907. Penna TC, Ferraz CA, Cassola MA. The presterilization microbial load on used medical devices and the effectiveness of hydrogen peroxide gas plasma against *Bacillus subtilis* spores. *Infect. Control Hosp. Epidemiol.* 1999;20:465-72.
 908. Bryce EA, Chia E, Logelin G, Smith JA. An evaluation of the AbTox Plazlyte Sterilization System. *Infect. Control Hosp. Epidemiol.* 1997;18:646-53.
 909. Graham GS, Riley R. Sterilization manufacturers: Interactions with regulatory agencies. In: Rutala WA, ed. *Disinfection, sterilization, and antisepsis in healthcare.* Champlain, New York: Polyscience Publications, 1998:41-8.
 910. Royce A, Bowler C. Ethylene oxide sterilisation-some experiences and some practical limitations. *J. Pharm. Pharmacol.* 1961;13:87t-94t.
 911. Nystrom B. Disinfection of surgical instruments. *J. Hosp. Infect.* 1981;2:363-8.
 912. Rutala WA, Gergen MF, Jones JF, Weber DJ. Levels of microbial contamination on surgical instruments. *Am. J. Infect. Control* 1998;26:143-5.
 913. Alfa MJ, Nemes R. Inadequacy of manual cleaning for reprocessing single-use, triple-lumen sphinctertomes: Simulated-use testing comparing manual with automated cleaning methods. *Am. J. Infect. Control* 2003;31:193-207.
 914. Alfa MJ, Nemes R. Reprocessing of lumened instruments. In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research.* Washington DC: Association for Professionals in Infection Control and Epidemiology, 2004:189-99.
 915. Bargmann LS, Bargmann BC, Collier JP, Currier BH, Mayor MB. Current sterilization and packaging methods for polyethylene. *Clin. Orthop.* 1999:49-58.
 916. Williams IR, Mayor MB, Collier JP. The impact of sterilization method on wear in knee arthroplasty. *Clin. Orthop.* 1998:170-80.
 917. Russell AD. Ionizing radiation. In: Russell AD, Hugo WB, Ayliffe GAJ, eds. *Principles and practices of disinfection, preservation and sterilization.* Oxford: Blackwell Science, 1999:675-87.
 918. Hansen JM, Shaffer HL. Sterilization and preservation by radiation sterilization. In: Block SS, ed. *Disinfection, sterilization, and preservation.* Philadelphia: Lippincott Williams & Wilkins, 2001:729-46.
 919. Lagergren ER. Recent advances in sterilization. *J Infect Control (Asia)* 1998;1:11-3.
 920. Perkins JJ. *Principles and methods of sterilization in health sciences.* Springfield, IL: Charles C Thomas, 1969.
 921. Favero MS, Bond WW. The use of liquid chemical germicides. In: Morrissey RF, Phillips GB, eds. *Sterilization technology: A practical guide for manufacturers and users of health care products.* New York: Van Nostrand Reinhold, 1993:309-334.
 922. Muscarella LF. Are all sterilization processes alike? *AORN J.* 1998;67:966-70, 973-6.
 923. Levy RV. Sterile filtration of liquids and gases. In: Block SS, ed. *Disinfection, sterilization, and preservation.* Philadelphia: Lippincott Williams & Wilkins, 2001:795-822.
 924. Wallhausser KH. Is the removal of microorganisms by filtration really a sterilization method? *J. Parenter. Drug Assoc.* 1979;33:156-70.
 925. Webb BC, Thomas CJ, Harty DW, Willcox MD. Effectiveness of two methods of denture sterilization. *J. Oral Rehabil.* 1998;25:416-23.
 926. Rohrer MD, Bulard RA. Microwave sterilization. *J. Am. Dent. Assoc.* 1985;110:194-8.

927. Rohrer MD, Terry MA, Bulard RA, Graves DC, Taylor EM. Microwave sterilization of hydrophilic contact lenses. *Am. J. Ophthalmol.* 1986;101:49-57.
928. Douglas C, Burke B, Kessler DL, Cicmanec JF, Bracken RB. Microwave: practical cost-effective method for sterilizing urinary catheters in the home. *Urology* 1990;35:219-22.
929. Kindle G, Busse A, Kampa D, Meyer-Konig U, Daschner FD. Killing activity of microwaves in milk. *J. Hosp. Infect.* 1996;33:273-8.
930. Harris MG, Rechberger J, Grant T, Holden BA. In-office microwave disinfection of soft contact lenses. *Optom. Vis. Sci.* 1990;67:129-32.
931. Mervine J, Temple R. Using a microwave oven to disinfect intermittent-use catheters. *Rehabil. Nurs.* 1997;22:318-20.
932. Najdovski L, Dragas AZ, Kotnik V. The killing activity of microwaves on some non-sporogenic and sporogenic medically important bacterial strains. *J. Hosp. Infect.* 1991;19:239-47.
933. Rosaspina S, Salvatorelli G, Anzanel D, Bovolenta R. Effect of microwave radiation on *Candida albicans*. *Microbios* 1994;78:55-9.
934. Welt BA, Tong CH, Rossen JL, Lund DB. Effect of microwave radiation on inactivation of *Clostridium sporogenes* (PA 3679) spores. *Appl. Environ. Microbiol.* 1994;60:482-8.
935. Latimer JM, Matsen JM. Microwave oven irradiation as a method for bacterial decontamination in a clinical microbiology laboratory. *J. Clin. Microbiol.* 1977;6:340-2.
936. Sanborn MR, Wan SK, Bulard R. Microwave sterilization of plastic tissue culture vessels for reuse. *Appl. Environ. Microbiol.* 1982;44:960-4.
937. Rosaspina S, Salvatorelli G, Anzanel D. The bactericidal effect of microwaves on *Mycobacterium bovis* dried on scalpel blades. *J. Hosp. Infect.* 1994;26:45-50.
938. Engelhardt JP, Grun L, Dahl HJ. Factors affecting sterilization in glass bead sterilizers. *J. Endod.* 1984;10:465-70.
939. Smith GE. Glass bead sterilization of orthodontic bands. *Am. J. Orthod. Dentofacial Orthop.* 1986;90:243-9.
940. Sisco V, Winters LL, Zange LL, Brennan PC. Efficacy of various methods of sterilization of acupuncture needles. *J. Manip. Physiol. Therap.* 1988;11:94-7.
941. Klapes NA, Vesley D. Vapor-phase hydrogen peroxide as a surface decontaminant and sterilant. *Appl. Environ. Microbiol.* 1990;56:503-6.
942. French GL, Otter JA, Shannon KP, Adams NMT, Watling D, Parks MJ. Tackling contamination of the hospital environment by methicillin-resistant *Staphylococcus aureus* (MRSA): A comparison between conventional terminal cleaning and hydrogen peroxide vapour decontamination. *J. Hosp. Infect.* 2004;57:31-7.
943. Jeanes A, Rao G, Osman M, Merrick P. Eradication of persistent environmental MRSA. *J. Hosp. Infect.* 2005;61:85-6.
944. Bates CJ, Pearse R. Use of hydrogen peroxide vapour for environmental control during a *Serratia* outbreak in a neonatal intensive care unit. *J. Hosp. Infect.* 2005;61:364-6.
945. Boyce JM, Havill NL, Otter JA, et al. Impact of hydrogen peroxide vapor room bio-decontamination on environmental contamination and nosocomial transmission of *Clostridium difficile*. *The Society of Healthcare Epidemiology of America*, 2006;Abstract 155:109.
946. Berrington AW, Pedler SJ. Investigation of gaseous ozone for MRSA decontamination of hospital side-rooms. *J. Hosp. Infect.* 1998;40:61-5.
947. Gaspar MC, Pelaez B, Fernandez C, Fereres J. Microbiological efficacy of Sterrad 100S and LTSF sterilisation systems compared to ethylene oxide. *Zentr. Steril.* 2002;10:91-9.
948. Kanemitsu K, Kunishima H, Imasaka T, et al. Evaluation of a low-temperature steam and formaldehyde sterilizer. *J. Hosp. Infect.* 2003;55:47-52.
949. Kanemitsu K, Imasaka T, Ishikawa S, et al. A comparative study of ethylene oxide gas, hydrogen peroxide gas plasma, and low-temperature steam formaldehyde sterilization. *Infect Control Hosp Epidemiol* 2005;26:486-9.
950. Roncoroni AJ, Casewell MW, Phillips I. The disinfection of clinically contaminated Matburn suction pumps and baby incubators in an 'Aseptor' formalin cabinet. *J. Hosp. Infect.* 1980;1:251-9.
951. Cumberland NS, Botting FG. Formaldehyde vapour cabinets. *J. Hosp. Infect.* 1991;19:67-70.
952. Jeng DK, Woodworth AG. Chlorine dioxide gas sterilization of oxygenators in an industrial scale

- sterilizer: a successful model. *Artif. Organs* 1990;14:361-8.
953. Knapp JE, Battisti DL. Chloride dioxide. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001:215-27.
954. Kowalski JB. Sterilization of medical devices, pharmaceutical components, and barrier isolation systems with gaseous chlorine dioxide. In: Morrissey RF, Kowalski JB, eds. *Sterilization of medical products*. Champlain, NY: Polyscience Publications, 1998:313-23.
955. Portner DM, Hoffman RK. Sporicidal effect of peracetic acid vapor. *Appl. Microbiol.* 1968;16:1782-5.
956. Mata-Portuguez VH, Perez LS, Acosta-Gio E. Sterilization of heat-resistant instruments with infrared radiation. *Infect. Control Hosp. Epidemiol.* 2002;23.
957. Frey R. The structural and functional prerequisites for a central sterile supply department (CSSD). *Zentr. Steril.* 2000;8:128-40.
958. Reich RR, Fleming W, Burgess DJ. Sterilization validation: it's not just for industry. *Infect. Control Steril. Technol.* 1996;2.
959. American Institute of Architects. *Guidelines for design and construction of hospital and health care facilities*. Washington, DC: The American Institute of Architects Press, 2001.
960. DesCoteaux JG, Poulin EC, Julien M, Guidoin R. Residual organic debris on processed surgical instruments. *AORN J.* 1995;62:23-30.
961. Rutala WA, Weber DJ. A review of the use of gowns and drapes (single use and reusable) in healthcare. *Infect. Control Hosp. Epidemiol.* 2001;22:248-57.
962. Association of peri-Operative Registered Nurses. Recommended practices for sterilization in the perioperative practice setting. *AORN J.* 2006;83:700-22.
963. Taurasi R. Comfortable PPE? Maximum tray weight? *Healthcare Purchasing News* 2004;July:48.
964. Chobin N, Furr D, Nuyttens A. Wet packs and plastic accessory cases. *Infect Control Today* 2004;August:24, 28-30.
965. Dunkelberg H, Fleitmann-Glende F. Measurement of the microbial barrier effectiveness of sterilization containers in terms of the log reduction value for prevention of nosocomial infections. *Am. J. Infect. Control* 2006;34:285-9.
966. Rutala WA, Weber DJ. Choosing a sterilization wrap. *Infect. Control Today* 2000;4:64,70.
967. Maloney JM, Kohut RD. Infection control, barrier protection and the treatment environment. *Dent. Hyg. (Chic.)* 1987;61:310-3.
968. Mayworm D. Sterile shelf life and expiration dating. *J. Hosp. Supply, Process. Distri.* 1984;2:32-5.
969. Cardo DM, Sehulster LM. Central sterile supply. In: Mayhall CG, ed. *Infect. Control and Hosp. Epidemiol.* Philadelphia: Lippincott Williams & Wilkins, 1999:1023-30.
970. Klapes NA, Greene VW, Langholz AC. Microbial contamination associated with routine aseptic practice. *J. Hosp. Infect.* 1987;10:299-304.
971. Butt WE, Bradley DV, Jr., Mayhew RB, Schwartz RS. Evaluation of the shelf life of sterile instrument packs. *Oral Surg. Oral Med. Oral Pathol.* 1991;72:650-4.
972. Webster J, Lloyd W, Ho P, Burrige C, George N. Rethinking sterilization practices: Evidence for event-related outdated. *Infect. Control Hosp. Epidemiol.* 2003;24:622-4.
973. Widmer AF, Houston A, Bollinger E, Wenzel RP. A new standard for sterility testing for autoclaved surgical trays. *J. Hosp. Infect.* 1992;21:253-60.
974. Schneider PM, Reich RR, Kirckof SS, Foltz WG. Performance of various steam sterilization indicators under optimum and sub-optimum exposure conditions. In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research*. Washington DC: Association for Professionals in Infection Control and Epidemiology, 2004:200-23.
975. Greene VW. Control of sterilization process. In: Russell AD, Hugo WB, Ayliffe GAJ, eds. *Principles and practice of disinfection, preservation and sterilization*. Oxford, England: Blackwell Scientific Publications, 1992:605-24.
976. Vesley D, Nellis MA, Allwood PB. Evaluation of a rapid readout biological indicator for 121°C gravity and 132°C vacuum-assisted steam sterilization cycles. *Infect. Control Hosp. Epidemiol.* 1995;16:281-6.
977. Rutala WA, Jones SM, Weber DJ. Comparison of a rapid readout biological indicator for steam sterilization with four conventional biological indicators and five chemical indicators. *Infect. Control Hosp. Epidemiol.* 1996;17:423-8.
978. Alfa MJ, Olson N, DeGagne P, Jackson M. Evaluation of rapid readout biological indicators for 132°C

- gravity and 132°C vacuum-assisted steam sterilization cycles using a new automated fluorescent reader. *Infect. Control Hosp. Epidemiol.* 2002;23:388-92.
979. Koncur P, Janes JE, Ortiz PA. 20 second sterilization indicator tests equivalent to BIs. *Infect. Control Steril. Technol.* 1998;26-8, 30, 32-4.
980. Perkins RE, Bodman HA, Kundsins RB, Walter CW. Monitoring steam sterilization of surgical instruments: a dilemma. *Appl. Environ. Microbiol.* 1981;42:383-7.
981. Kotilainen HR, Gantz NM. An evaluation of three biological indicator systems in flash sterilization. *Infect. Control* 1987;8:311-6.
982. Kleinegger CL, Yeager DL, Huling JK, Drake DR. The effects of contamination on biological monitoring. *Infect. Control Hosp. Epidemiol.* 2001;22:391-2.
983. Centers for Disease Control. False-positive results of spore tests in ethylene oxide sterilizers - Wisconsin. *MMWR* 1981;30:238-40.
984. Association of Operating Room Nurses. AORN standards and recommended practices for perioperative nursing. 1987:Section III:14.1-III:14.11, AORN, Denver, CO.
985. Gurevich I, Holmes JE, Cunha BA. Presumed autoclave failure due to false-positive spore strip tests. *Infect. Control* 1982;3:388-92.
986. Epstein BJ, Lattimer JM, Matsen JM, Garibaldi RA. False positive spore strip sterility tests with steam sterilization. *Am. J. Infect. Control* 1983;11:71-3.
987. Association for the Advancement of Medical Instrumentation. Good hospital practice: steam sterilization and sterility assurance. Arlington, VA: AAMI, 1988.
988. Baird RM. Sterility assurance: Concepts, methods and problems. In: Russell AD, Hugo WB, Ayliffe GAJ, eds. *Principles and practice of disinfection, preservation and sterilization*. Oxford, England: Blackwell Scientific Publications, 1999:787-99.
989. Coulter WA, Chew-Graham CA, Cheung SW, Burke FJT. Autoclave performance and operator knowledge of autoclave use in primary care: a survey of UK practices. *J. Hosp. Infect.* 2001;48:180-5.
990. Greene VW. Reuse of disposable devices. In: Mayhall CG, ed. *Infect. Control and Hosp. Epidemiol.* Philadelphia: Lippincott Williams & Wilkins, 1999:1201-8.
991. Avital B, Khan M, Krum D, Jazayeri M, Hare J. Repeated use of ablation catheters: a prospective study. *J. Am. Coll. Cardiol.* 1993;22:1367-72.
992. Dunnigan A, Roberts C, McNamara M, Benson DW, Jr., Benditt DG. Success of re-use of cardiac electrode catheters. *Am. J. Cardiol.* 1987;60:807-10.
993. Aton EA, Murray P, Fraser V, Conaway L, Cain ME. Safety of reusing cardiac electrophysiology catheters. *Am. J. Cardiol.* 1994;74:1173-5.
994. Brown SA, Merritt K, Woods TO, McNamee SG, Hitchins VM. Effects of different disinfection and sterilization methods on tensile strength of materials used for single-use devices. *Biomed. Instrum. Technol.* 2002;January/February:23-7.
995. Food and Drug Administration. Enforcement Priorities for Single-Use Devices Reprocessed by Third Parties and Hospitals, Rockville, MD., 2000.
996. Ulatowski TA. FDA: Reuse of Single-Use Devices. In: Rutala WA, ed. *Disinfection, sterilization and antisepsis: Principles, practices, challenges, and new research*. Washington, DC: Association for Professionals in Infection Control and Epidemiology, 2004:15-23.
997. Occupational Health and Safety Administration. Hazard Communication Standard. 29 CFR 1910.1200, OSHA, Washington, DC.
998. Edens AL. Occupational Safety and Health Administration: Regulations affecting healthcare facilities. In: Rutala WA, ed. *Disinfection, Sterilization and Antisepsis: Principles and practices in healthcare facilities*. Washington, D.C.: Association for Professionals in Infection Control and Epidemiology, Inc., 2001:49-58.
999. Schultz JK. Decontamination: recommended practices. In: Reichert M, Young JH, eds. *Sterilization technology for the health care facility*. Gaithersburg, MD: Aspen Publication, 1997:10-20.
1000. Occupational Health and Safety Administration. Ethylene Oxide Standard. Vol. 29 CFR 1910.1047, OSHA, Washington, DC.
1001. Occupational Safety and Health Administration. Formaldehyde Standard. Vol. 29 CFR 1910.1048, Washington, DC.
1002. Buxton AE, Anderson RL, Werdegar D, Atlas E. Nosocomial respiratory tract infection and colonization with *Acinetobacter calcoaceticus*. Epidemiologic characteristics. *Am. J. Med.* 1978;65:507-13.

1003. Snyderman DR. Hepatitis B infection from medical personnel. *JAMA* 1976;236:1009.
1004. Martiny H, Floss H. Residuals on medical devices following reprocessing. *J. Hosp. Infect.* 2001;48 (Supplement):S88-S92.
1005. Taylor DM. Inactivation of prions by physical and chemical means. *J. Hosp. Infect.* 1999;43 (supplement):S69-S76.
1006. Best M, Sattar SA, Springthorpe VS, Kennedy ME. Comparative mycobactericidal efficacy of chemical disinfectants in suspension and carrier tests. *Appl. Environ. Microbiol.* 1988;54:2856-8.
1007. Weber DJ, Rutala WA. Environmental issues and nosocomial infections. In: Wenzel RP, ed. *Prevention and control of nosocomial infections*. Baltimore: Williams and Wilkins, 1991:1042-64.
1008. Palmer PH, Yeoman DM. A study to assess the value of disinfectants when washing ward floors. *Med. J. Aust.* 1972;2:1237-9.
1009. Centers for Disease Control and Prevention. Preventing the spread of vancomycin resistance - report from the Hospital Infection Control Practices Advisory Committee. *Fed. Regist.* 1994;25758-63.
1010. Ayliffe GA, Collins BJ, Lowbury EJ. Cleaning and disinfection of hospital floors. *BMJ* 1966;5511:442-5.
1011. Neely AN. A survey of gram-negative bacteria survival on hospital fabrics and plastics. *J. Burn Care Rehabil.* 2000;21:523-7.
1012. Rutala WA. Disinfection and sterilization of patient-care items. *Infect. Control Hosp. Epidemiol.* 1996;17:377-84.
1013. Environmental Protection Agency. Pesticides: Regulating Pesticides. <http://www.epa.gov/oppad001/chemregindex.htm>, 2003.
1014. Hoffman PN, Layzell SK. Household bleach as disinfectant for use by injecting drug users. *Lancet* 1993;342:743.
1015. Chu NS, Chan-Myers H, Ghazanfari N, Antonoplos P. Levels of naturally occurring microorganisms on surgical instruments after clinical use and after washing. *Am. J. Infect. Control* 1999;27:315-9.
1016. Hoffmann KK, Weber DJ, Rutala WA. Pseudoepidemic of *Rhodotorula rubra* in patients undergoing fiberoptic bronchoscopy. *Infect. Control Hosp. Epidemiol.* 1989;10:511-4.
1017. Lowry PW, Jarvis WR. Use of tap water and disinfection practices in outpatient settings. A survey of otolaryngologists. *Arch. Otolaryngol. Head Neck Surg.* 1991;117:886-8.
1018. Fahey BJ, Koziol DE, Banks SM, Henderson DK. Frequency of nonparenteral occupational exposures to blood and body fluids before and after universal precautions training. *Am. J. Med.* 1991;90:145-53.
1019. Beekmann SE, Vlahov D, Koziol DE, McShalley ED, Schmitt JM, Henderson DK. Temporal association between implementation of universal precautions and a sustained, progressive decrease in percutaneous exposures to blood. *Clin. Infect. Dis.* 1994;18:562-9.
1020. Gerberding JL, Littell C, Tarkington A, Brown A, Schechter WP. Risk of exposure of surgical personnel to patients' blood during surgery at San Francisco General Hospital. *N. Engl. J. Med.* 1991;324:1788-93.
1021. Mast ST, Woolwine JD, Gerberding JL. Efficacy of gloves in reducing blood volumes transferred during simulated needlestick injury. *J. Infect. Dis.* 1993;168:1589-92.
1022. Wendt C, Herwaldt LA. Epidemics: Identification and Management. In: Wenzel RP, ed. *Prevention and Control of Nosocomial Infections*. Baltimore: Williams & Wilkins, 1997:175-214.
1023. Feigal DW, Gardner SN, McClellan M. Ensuring safe and effective medical devices. *N. Engl. J. Med.* 2003;348:191-2.
1024. Oie S, Kamiya A. Microbial contamination of antiseptics and disinfectants. *Am. J. Infect. Control* 1996;24:389-95.
1025. Strzelecki LR, Nelson JH. Evaluation of closed container flash sterilization system. *Orthop. Nurs.* 1989;8:21-4.
1026. Burgess DJ, Reich RR. Industrial ethylene oxide sterilization. In: Morrissey RF, Phillips GB, eds. *Sterilization technology: a practical guide for manufacturers and users of health care product*. New York: Van Nostrand Reinhold, 1993:120-51.
1027. Conviser CA, C W. Ethylene oxide sterilization: sterilant alternatives. In: Reichert M, Young JH, eds. *Sterilization technology for the health care facility*. Gaithersburg, MD: Aspen Publication, 1997:189-99.
1028. Young JH. Steam sterilization: scientific principles. In: Reichert M, Young JH, eds. *Sterilization technology for the health care facility*. Gaithersburg, MD: Aspen Publication, 1997:123-144.
1029. Alfa MJ. Importance of lumen flow in liquid chemical sterilization. *Am. J. Infect. Control* 1999;27:373-5.
1030. Mallison GF, Standard PG. Safe storage times for sterile packs. *Hospitals* 1974;48:77-8, 80.

1031. Klapes NA, Greene VW, Langholz AC, Hunstiger C. Effect of long-term storage on sterile status of devices in surgical packs. *Infect. Control* 1987;8:289-93.
1032. Japp NF. Packaging: Shelf life. In: Reichert M, Young JH, eds. *Sterilization Technology*. Gaithersburg, Maryland: Aspen, 1997:99-102.
1033. Joint Commission for the Accreditation of Healthcare Organizations. *Comprehensive accreditation manual for hospitals*, JCAHO, Chicago, IL. 2003.
1034. Block SS. Definition of terms. In: Block SS, ed. *Disinfection, sterilization, and preservation*. Philadelphia: Lippincott Williams & Wilkins, 2001:19-28.
1035. Molinari JA, Gleason MJ, Cottone JA, Barrett ED. Comparison of dental surface disinfectants. *Gen. Dent.* 1987;35:171-5.

The recommendations in this guideline for Ebola Virus Disease has been superseded by [CDC's Infection Prevention and Control Recommendations for Hospitalized Patients with Known or Suspected Ebola Virus Disease in U.S. Hospitals](#) and by [CDC's Interim Guidance for Environmental Infection Control in Hospitals for Ebola Virus](#) issued on August 1, 2014.

This information is on pages [12](#), [13](#), [113](#) and [124](#).

[Click here](#) for current information on how Ebola virus is transmitted.

Guidelines for Environmental Infection Control in Health-Care Facilities

**Recommendations of CDC and the Healthcare Infection Control
Practices Advisory Committee (HICPAC)**

**U.S. Department of Health and Human Services
Centers for Disease Control and Prevention (CDC)
Atlanta, GA 30333**

2003

Suggested Citations:

Available from the CDC Internet Site:

The full-text version of the guidelines appears as a web-based document at the CDC's Division of Healthcare Quality Promotion's Internet site at:
www.cdc.gov/ncidod/hip/enviro/guide.htm

The full-text version of the guidelines should be cited when reference is made primarily to material in Parts I and IV. The print version of the guidelines appears as:

Sehulster LM, Chinn RYW, Arduino MJ, Carpenter J, Donlan R, Ashford D, Besser R, Fields B, McNeil MM, Whitney C, Wong S, Juranek D, Cleveland J. Guidelines for environmental infection control in health-care facilities. Recommendations from CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). Chicago IL; American Society for Healthcare Engineering/American Hospital Association; 2004.

Part II of these guidelines appeared in the CDC's "Morbidity and Mortality Weekly Report:"

Centers for Disease Control and Prevention. Guidelines for environmental infection control in health-care facilities: recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). MMWR 2003; 52 (No. RR-10): 1–48.

Updates to the Part II recommendations also appeared in the MMWR in 2003 as "Errata: Vol. 52 (No. RR-10)" (MMWR Vol. 52 [42]: 1025–6) on October 24, 2003 and as a "Notice to Readers" scheduled to appear in February 2004. The full-text version of these guidelines (this document) incorporates these updates.

Centers for Disease Control and Prevention Healthcare Infection Control Practices Advisory Committee (HICPAC)

Guidelines for Environmental Infection Control in Health-Care Facilities

Abstract

Background:

Although the environment serves as a reservoir for a variety of microorganisms, it is rarely implicated in disease transmission except in the immunocompromised population. Inadvertent exposures to environmental opportunistic pathogens (e.g., *Aspergillus* spp. and *Legionella* spp.) or airborne pathogens (e.g., *Mycobacterium tuberculosis* and varicella-zoster virus) may result in infections with significant morbidity and/or mortality. Lack of adherence to established standards and guidance (e.g., water quality in dialysis, proper ventilation for specialized care areas such as operating rooms, and proper use of disinfectants) can result in adverse patient outcomes in health-care facilities.

Objective:

The objective is to develop an environmental infection-control guideline that reviews and reaffirms strategies for the prevention of environmentally-mediated infections, particularly among health-care workers and immunocompromised patients. The recommendations are evidence-based whenever possible.

Search Strategies:

The contributors to this guideline reviewed predominantly English-language articles identified from MEDLINE literature searches, bibliographies from published articles, and infection-control textbooks.

Criteria for Selecting Citations and Studies for This Review:

Articles dealing with outbreaks of infection due to environmental opportunistic microorganisms and epidemiological- or laboratory experimental studies were reviewed. Current editions of guidelines and standards from organizations (i.e., American Institute of Architects [AIA], Association for the Advancement of Medical Instrumentation [AAMI], and American Society of Heating, Refrigeration, and Air-Conditioning Engineers [ASHRAE]) were consulted. Relevant regulations from federal agencies (i.e., U.S. Food and Drug Administration [FDA]; U.S. Department of Labor, Occupational Safety and Health Administration [OSHA]; U.S. Environmental Protection Agency [EPA]; and U.S. Department of Justice) were reviewed. Some topics did not have well-designed, prospective studies nor reports of outbreak investigations. Expert opinions and experience were consulted in these instances.

Types of Studies:

Reports of outbreak investigations, epidemiological assessment of outbreak investigations with control strategies, and *in vitro* environmental studies were assessed. Many of the recommendations are derived

from empiric engineering concepts and reflect industry standards. A few of the infection-control measures proposed cannot be rigorously studied for ethical or logistical reasons.

Outcome Measures:

Infections caused by the microorganisms described in this guideline are rare events, and the effect of these recommendations on infection rates in a facility may not be readily measurable. Therefore, the following steps to measure performance are suggested to evaluate these recommendations:

1. Document whether infection-control personnel are actively involved in all phases of a health-care facility's demolition, construction, and renovation. Activities should include performing a risk assessment of the necessary types of construction barriers, and daily monitoring and documenting of the presence of negative airflow within the construction zone or renovation area.
2. Monitor and document daily the negative airflow in airborne infection isolation rooms (AII) and positive airflow in protective environment rooms (PE), especially when patients are in these rooms.
3. Perform assays at least once a month by using standard quantitative methods for endotoxin in water used to reprocess hemodialyzers, and for heterotrophic, mesophilic bacteria in water used to prepare dialysate and for hemodialyzer reprocessing.
4. Evaluate possible environmental sources (e.g., water, laboratory solutions, or reagents) of specimen contamination when nontuberculous mycobacteria (NTM) of unlikely clinical importance are isolated from clinical cultures. If environmental contamination is found, eliminate the probable mechanisms.
5. Document policies to identify and respond to water damage. Such policies should result in either repair and drying of wet structural materials within 72 hours, or removal of the wet material if drying is unlikely within 72 hours.

Main Results:

Infection-control strategies and engineering controls, when consistently implemented, are effective in preventing opportunistic, environmentally-related infections in immunocompromised populations. Adherence to proper use of disinfectants, proper maintenance of medical equipment that uses water (e.g., automated endoscope reprocessors and hydrotherapy equipment), water-quality standards for hemodialysis, and proper ventilation standards for specialized care environments (i.e., airborne infection isolation [AII], protective environment [PE], and operating rooms [ORs]), and prompt management of water intrusion into facility structural elements will minimize health-care-associated infection risks and reduce the frequency of pseudo-outbreaks. Routine environmental sampling is not advised except in the few situations where sampling is directed by epidemiologic principles and results can be applied directly to infection control decisions, and for water quality determinations in hemodialysis.

Reviewers' Conclusions:

Continued compliance with existing environmental infection control measures will decrease the risk of health-care-associated infections among patients, especially the immunocompromised, and health-care workers.

**Centers for Disease Control and Prevention
Healthcare Infection Control Practices Advisory Committee (HICPAC)**

***Guidelines for Environmental Infection Control in
Health-Care Facilities***

Table of Contents

Executive Summary	1
Part I. Background Information: Environmental Infection Control in Health-Care Facilities	3
A. Introduction	3
B. Key Terms Used in this Guideline	5
C. Air	6
1. Modes of Transmission of Airborne Diseases	6
2. Airborne Infectious Diseases in Health-Care Facilities	7
3. Heating, Ventilation, and Air Conditioning Systems in Health-Care Facilities	13
4. Construction, Renovation, Remediation, Repair, and Demolition	21
5. Environmental Infection-Control Measures for Special Health-Care Settings.....	34
6. Other Aerosol Hazards in Health-Care Facilities	40
D. Water	40
1. Modes of Transmission of Waterborne Diseases	40
2. Waterborne Infectious Diseases in Health-Care Facilities	41
3. Water Systems in Health-Care Facilities	46
4. Strategies for Controlling Waterborne Microbial Contamination	53
5. Cooling Towers and Evaporative Condensers.....	57
6. Dialysis Water Quality and Dialysate.....	59
7. Ice Machines and Ice	65
8. Hydrotherapy Tanks and Pools.....	67
9. Miscellaneous Medical/Dental Equipment Connected to Main Water Systems	69
E. Environmental Services	71
1. Principles of Cleaning and Disinfecting Environmental Surfaces.....	71
2. General Cleaning Strategies for Patient-Care Areas.....	74
3. Cleaning Strategies for Spills of Blood and Body Substances	77
4. Carpeting and Cloth Furnishings	78
5. Flowers and Plants in Patient-Care Areas	80
6. Pest Control	81
7. Special Pathogen Concerns.....	82
F. Environmental Sampling	88
1. General Principles: Microbiologic Sampling of the Environment	88
2. Air Sampling.....	89
3. Water Sampling	94
4. Environmental Surface Sampling.....	95
G. Laundry and Bedding	98
1. General Information	98
2. Epidemiology and General Aspects of Infection Control.....	98
3. Collecting, Transporting, and Sorting Contaminated Textiles and Fabrics.....	99

4. Parameters of the Laundry Process	100
5. Special Laundry Situations.....	102
6. Surgical Gowns, Drapes, and Disposable Fabrics.....	103
7. Antimicrobial-Impregnated Articles and Consumer Items Bearing Antimicrobial Labeling ..	103
8. Standard Mattresses, Pillows, and Air-Fluidized Beds	104
H. Animals in Health-Care Facilities	105
1. General Information	105
2. Animal-Assisted Activities, Animal-Assisted Therapy, and Resident Animals	106
3. Service Animals	108
4. Animals as Patients in Human Health-Care Facilities	110
5. Research Animals in Health-Care Facilities	111
I. Regulated Medical Waste	112
1. Epidemiology	112
2. Categories of Medical Waste	112
3. Management of Regulated Medical Waste in Health-Care Facilities	113
4. Treatment of Regulated Medical Waste.....	113
5. Discharging Blood, Fluids to Sanitary Sewers or Septic Tanks.....	116
6. Medical Waste and CJD	116
Part II. Recommendations for Environmental Infection Control in Health-Care Facilities	117
A. Rationale for Recommendations	117
B. Rating Categories.....	117
C. Recommendations—Air	118
D. Recommendations—Water	125
E. Recommendations—Environmental Services	133
F. Recommendations—Environmental Sampling	138
G. Recommendations—Laundry and Bedding	138
H. Recommendations—Animals in Health-Care Facilities.....	141
I. Recommendations—Regulated Medical Waste.....	143
Part III. References.....	145
Part IV. Appendices.....	201
Appendix A. Glossary of Terms	201
Appendix B. Air	210
1. Airborne Contaminant Removal	210
2. Air Sampling for Aerosols Containing Legionellae.....	210
3. Calculation of Air Sampling Results.....	211
4. Ventilation Specifications for Health-Care Facilities.....	212
Appendix C. Water	220
1. Biofilms	220
2. Water and Dialysate Sampling Strategies in Dialysis.....	222
3. Water Sampling Strategies and Culture Techniques for Detecting Legionellae	223
4. Procedure for Cleaning Cooling Towers and Related Equipment	225
5. Maintenance Procedures Used to Decrease Survival and Multiplications of <i>Legionella</i> spp. in Potable-Water Distribution Systems	227
Appendix D. Insects and Microorganisms.....	228
Appendix E. Information Resources.....	229
Appendix F. Areas of Future Research	230
Index—Parts I and IV	231

List of Figures, Boxes, and Tables

Figures

Figure 1. Diagram of a ventilation system	14
Figure 2. Example of positive-pressure room control for protection from airborne environmental microbes (PE)	35
Figure 3. Example of negative-pressure room control for airborne infection isolation (AII)	36
Figure 4. Example of airborne infection isolation (AII) room with anteroom and neutral anteroom	37
Figure 5. Diagram of a typical air conditioning (induced draft) cooling tower	58
Figure 6. Dialysis water treatment system	60

Boxes

Box 1. Environmental infection control: performance measures	2
Box 2. Eight criteria for evaluating the strength of evidence for environmental sources of infection	4
Box 3. Chain of infection components	4
Box 4. Suggested members and functions of a multi-disciplinary coordination team for construction, renovation, repair, and demolition projects	24
Box 5. Construction design and function considerations for environmental infection control	25
Box 6. Unresolved issues associated with microbiologic air sampling	28
Box 7. Construction/repair projects that require barrier structures	32
Box 8. Strategy for managing TB patients and preventing airborne transmission in operating rooms	39
Box 9. Recovery and remediation measures for water-related emergencies	52
Box 10. Contingency planning for flooding	53
Box 11. Steps in an epidemiologic investigation for legionellosis	56
Box 12. General steps for cleaning and maintaining ice machines, dispensers, and storage chests	66
Box 13. Preliminary concerns for conducting air sampling	90
Box 14. Selecting an air sampling device	93
Box 15. Undertaking environmental-surface sampling	95
Box C.1. Potential sampling sites for <i>Legionella</i> spp. in health-care facilities	224
Box C.2. Procedures for collecting and processing environmental specimens for <i>Legionella</i> spp.	225

Tables

Table 1. Clinical and epidemiologic characteristics of aspergillosis	7
Table 2. Environmental fungal pathogens: entry into and contamination of the health-care facility	8
Table 3. Clinical and epidemiologic characteristics of tuberculosis (TB)	10
Table 4. Microorganisms associated with airborne transmission	13
Table 5. Filtration methods	15
Table 6. Engineered specifications for positive- and negative pressure rooms	19
Table 7. Ventilation hazards in health-care facilities that may be associated with increased potential of airborne disease transmission	22
Table 8. Strategies to reduce dust and moisture intrusion during external demolition and construction	30

Table 9. Infection-control measures for internal construction and repair projects	32
Table 10. Summary of ventilation specifications in selected areas of health-care facilities.....	39
Table 11. Clinical and epidemiologic characteristics of legionellosis/Legionnaires disease.....	41
Table 12. <i>Pseudomonas aeruginosa</i> infections in health-care facilities	42
Table 13. Other gram-negative bacteria associated with water and moist environments	43
Table 14. Nontuberculous mycobacteria—environmental vehicles	45
Table 15. Water and point-of-use fixtures as sources and reservoirs of waterborne pathogens	47
Table 16. Water demand in health-care facilities during water disruption emergencies	50
Table 17. Additional infection-control measures to prevent exposure of high-risk patients to waterborne pathogens.....	57
Table 18. Microbiologic limits for hemodialysis fluids.....	62
Table 19. Factors influencing microbial contamination in hemodialysis systems.....	63
Table 20. Microorganisms and their sources in ice and ice machines	65
Table 21. Infections associated with use of hydrotherapy equipment	67
Table 22. Levels of disinfection by type of microorganism	72
Table 23. Air sampling methods and examples of equipment	91
Table 24. Examples of eluents and diluents for environmental-surface sampling.....	96
Table 25. Methods of environmental-surface sampling.....	97
Table 26. Examples of diseases associated with zoonotic transmission.....	105
Table 27. Microorganisms and biologicals identified as select agents	115
Table B.1. Air changes/hour (ACH) and time required for airborne-contaminant removal efficiencies of 99% and 99.9%	210
Table B.2. Ventilation requirements for areas affecting patient care in hospitals and outpatient facilities	212
Table B.3. Pressure relationships and ventilation of certain areas of nursing facilities.....	217
Table B.4. Filter efficiencies for central ventilation and air conditioning systems in general hospitals	219
Table B.5. Filter efficiencies for central ventilation and air conditioning systems in outpatient facilities	219
Table B.6. Filter efficiencies for central ventilation and air conditioning systems in nursing facilities	220
Table B.7. Filter efficiencies for central ventilation and air conditioning systems in psychiatric hospitals	220
Table D.1. Microorganisms isolated from arthropods in health-care settings	228

List of Abbreviations Used in This Publication

AAA	animal-assisted activity
AAMI	Association for the Advancement of Medical Instrumentation
AAT	animal-assisted therapy
ACGIH	American Council of Governmental Industrial Hygienists
ACH	air changes per hour
ADA	Americans with Disabilities Act
AER	automated endoscope reprocessor
AFB	acid-fast bacilli
AHA	American Hospital Association
AHJ	authorities having jurisdiction
AIA	American Institute of Architects
AII	airborne infection isolation
AmB	amphotericin B
ANC	absolute neutrophil count
ANSI	American National Standards Institute
AORN	Association of periOperative Registered Nurses
ASHE	American Society for Healthcare Engineering
ASHRAE	American Society of Heating, Refrigeration, and Air-Conditioning Engineers
BCG	Bacille Calmette-Guérin
BCYE	buffered charcoal yeast extract medium
BHI	brain-heart infusion
BMBL	CDC/NIH publication “Biosafety in Microbiological and Biomedical Laboratories”
BOD	biological oxygen demand
BSE	bovine spongiform encephalopathy
BSL	biosafety level
C	Centigrade
CAPD	continuous ambulatory peritoneal dialysis
CCPD	continual cycling peritoneal dialysis
CMAD	count median aerodynamic diameter
CDC	U.S. Centers for Disease Control and Prevention
CFR	Code of Federal Regulations
CFU	colony-forming unit
CJD	Creutzfeldt-Jakob disease
cm	centimeter
CMS	U.S. Centers for Medicare and Medicaid Services
CPL	compliance document (OSHA)
CT/EC	cooling tower/evaporative condenser
DFA	direct fluorescence assay; direct fluorescent antibody
DHHS	U.S. Department of Health and Human Services
DHBV	duck hepatitis B virus
DNA	deoxyribonucleic acid
DOP	dioctylphthalate
DOT	U.S. Department of Transportation
EC	environment of care (JCAHO)
ELISA	enzyme-linked immunosorbent assay
EPA	U.S. Environmental Protection Agency
ESRD	end-stage renal disease

EU	endotoxin unit
F	Fahrenheit
FDA	U.S. Food and Drug Administration
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FRC	free residual chlorine
ft	foot (feet)
FTC	U.S. Federal Trade Commission
GISA	glycopeptide intermediate resistant <i>Staphylococcus aureus</i>
HBV	hepatitis B virus
HCV	hepatitis C virus
HEPA	high efficiency particulate air
HICPAC	Healthcare Infection Control Practices Advisory Committee
HIV	human immunodeficiency virus
HPV	human papilloma virus
HSCT	hematopoietic stem cell transplant
HVAC	heating, ventilation, air conditioning
ICRA	infection control risk assessment
ICU	intensive care unit
ID₅₀	50% median infectious dose
IPD	intermittent peritoneal dialysis
JCAHO	Joint Commission on Accreditation of Healthcare Organizations
kg	kilogram
L	liter
MAC	<i>Mycobacterium avium</i> complex; also used to denote MacConkey agar
MDRO	multiple-drug resistant organism
MIC	minimum inhibitory concentration
µm	micrometer; micron
mL	milliliter
min	minute
mg	milligram
MMAD	mass median aerodynamic diameter
MMWR	“Morbidity and Mortality Weekly Report”
MRSA	methicillin-resistant <i>Staphylococcus aureus</i>
MSDS	material safety data sheet
N	Normal
NaCl	sodium chloride
NaOH	sodium hydroxide
NCID	National Center for Infectious Diseases
NCCDPHP	National Center for Chronic Disease Prevention and Health Promotion
NCCLS	National Committee for Clinical Laboratory Standards
ng	nanogram
NICU	neonatal intensive care unit
NIH	U.S. National Institutes of Health
NIOSH	National Institute for Occupational Safety and Health
nm	nanometer
NNIS	National Nosocomial Infection Surveillance
NTM	nontuberculous mycobacteria
OPL	on-premises laundry
OSHA	U.S. Occupational Safety and Health Administration
Pa	Pascal
PCP	<i>Pneumocystis carinii</i> pneumonia

PCR	polymerase chain reaction
PD	peritoneal dialysis
PE	protective environment
PEL	permissible exposure limit
PPE	personal protective equipment
ppm	parts per million
PVC	polyvinylchloride
RAPD	randomly amplified polymorphic DNA
RODAC	replicate organism direct agar contact
RSV	respiratory syncytial virus
RO	reverse osmosis
SARS	severe acute respiratory syndrome
SARS-CoV	SARS coronavirus
sec	second
spp	species
SSI	surgical site infection
TB	tuberculosis
TLV®-TWA	threshold limit value-time weighted average
TSA	tryptic soy agar
TSB	tryptic soy broth
TSE	transmissible spongiform encephalopathy
U.S.	United States
USC	United States Code
USDA	U.S. Department of Agriculture
USPS	U.S. Postal Service
UV	ultraviolet
UVGI	ultraviolet germicidal irradiation
VAV	variable air ventilation
vCJD	variant Creutzfeldt-Jakob disease
VISA	vancomycin intermediate resistant <i>Staphylococcus aureus</i>
VRE	vancomycin-resistant <i>Enterococcus</i>
VRSA	vancomycin-resistant <i>Staphylococcus aureus</i>
v/v	volume/volume
VZV	varicella-zoster virus

Note: Use of trade names and commercial sources is for identification only and does not imply endorsement by the U.S. Department of Health and Human Services. References to non-CDC sites on the Internet are provided as a service to the reader and does not constitute or imply endorsement of these organization s or their programs by CDC or the U.S. Department of Health and Human Services. CDC is not responsible for the content of pages found at these sites.

The following CDC staff member and HICPAC member prepared this report:

Lynne Schulster, PhD
Division of Healthcare Quality Promotion
National Center for Infectious Diseases

Raymond Y.W. Chinn, MD
HICPAC Advisor
Sharp Memorial Hospital
San Diego, California

Disclosure of Relationship: Raymond Y.W. Chinn is a private-practice physician and salaried employee of Sharp Memorial Hospital in San Diego, California. Dr. Chinn received no research funds from commercial sources either directly, or indirectly through awards made to the hospital, before or during the development of these guidelines.

Contributions were made by the following CDC staff members:

Matthew Arduino, DrPH
Joe Carpenter, PE
Rodney Donlan, PhD
Lynne Schulster, PhD
Division of Healthcare Quality Promotion
National Center for Infectious Diseases

David Ashford, DVM, Dsc, MPH
Richard Besser, MD
Barry Fields, PhD
Michael M. McNeil, MBBS, MPH
Cynthia Whitney, MD, MPH
Stephanie Wong, DMV, MPH
Division of Bacterial and Mycotic Diseases
National Center for Infectious Diseases

Dennis Juranek, DVM, MSC
Division of Parasitic Diseases
National Center for Infectious Diseases

Jennifer Cleveland, DDS, MPH
Division of Oral Health
National Center for Chronic Disease Prevention and Health Promotion

***In collaboration with the Healthcare Infection Control Practices Advisory Committee
(HICPAC)***

HICPAC Members, February 2002

Robert A. Weinstein, MD
Chair
Cook County Hospital
Chicago, IL

Jane D. Siegel, MD
Co-Chair
University of Texas Southwestern Medical Center
Dallas, TX

Michele L. Pearson, MD
Executive Secretary
Centers for Disease Control and Prevention
Atlanta, GA

Raymond Y.W. Chinn, MD
Sharp Memorial Hospital
San Diego, CA

Alfred DeMaria, Jr., MD
Massachusetts Department of Public Health
Jamaica Plain, MA

Elaine L. Larson, RN, PhD
Columbia University School of Nursing
New York, NY

James T. Lee, MD, PhD
University of Minnesota
VA Medical Center
St. Paul, MN

Ramon E. Moncada, MD
Coronado Physician's Medical Center
Coronado, CA

William A. Rutala, PhD, MPH, CIC
University of North Carolina School of
Medicine
Chapel Hill, NC

William E. Scheckler, MD
University of Wisconsin Medical School
Madison, WI

Beth H. Stover, RN, CIC
Kosair Children's Hospital
Louisville, KY

Marjorie A. Underwood, RN, BSN, CIC
Mt. Diablo Medical Center
Concord, CA

Liaison Members

Loretta L. Fauerbach, MS, CIC
Association for Professionals in Infection
Control and Epidemiology (APIC)
Washington, DC

Sandra L. Fitzler, RN
American Health Care Association
Washington, DC

Dorothy M. Fogg, RN, BSN, MA
Association of periOperative Registered
Nurses (AORN)
Denver, CO

Stephen F. Jencks, MD, MPH
U.S. Centers for Medicare and Medicaid
Services
Baltimore, MD

Chiu S. Lin, PhD
U.S. Food and Drug Administration
Rockville, MD

James P. Steinberg, MD
Society for Healthcare Epidemiology of America,
Inc. (SHEA)
Atlanta, GA

Liaison Members (continued)

Michael L. Tapper, MD
Advisory Committee for the Elimination of
Tuberculosis (ACET)
New York, NY

Expert Reviewers

Trisha Barrett, RN, MBA, CIC
Alta Bates Medical Center
Berkeley, CA

Michael Berry
University of North Carolina
Chapel Hill, NC

Walter W. Bond, MS
RCSA, Inc.
Lawrenceville, GA

Douglas Erickson, FASHE
American Society for Healthcare
Engineering (ASHE)
Park Ridge, IL

Richard Miller, MD
University of Louisville School of Medicine
Louisville, KY

Gina Pugliese, RN, MS
Premier Safety Institute
Oak Brook, IL

James D. Scott, PE
Michigan Department of Consumer and
Industry Services
Lansing, MI

Dale Woodin
American Society for Healthcare Engineering
(ASHE)
Chicago, IL

Judene Bartley, MS, MPH, CIC
Epidemiology Consulting Services, Inc.
Beverly Hills, MI

Col. Nancy Bjerke, BSN, MA, MEd, MPH, CIC
(USAF, Retired)
Infection Control Associates (ICA)
San Antonio, TX

Cheryl Carter, RN
University of Iowa Health Center
Iowa City, IA

Martin S. Favero, PhD
Advanced Sterilization Products, Johnson and
Johnson
Irvine, CA

Col. Shannon E. Mills, DDS
HQ USAF / Surgeon General Detail
Bolin AFB, DC

Craig E. Rubens, MD, PhD
Children's Hospital and Medical Center
Seattle, WA

Andrew J. Streifel, MPH, REHS
University of Minnesota
Minneapolis, MN

Executive Summary

The *Guidelines for Environmental Infection Control in Health-Care Facilities* is a compilation of recommendations for the prevention and control of infectious diseases that are associated with health-care environments. This document a) revises multiple sections from previous editions of the Centers for Disease Control and Prevention [CDC] document titled *Guideline for Handwashing and Hospital Environmental Control*;^{1,2} b) incorporates discussions of air and water environmental concerns from CDC's *Guideline for the Prevention of Nosocomial Pneumonia*;³ c) consolidates relevant environmental infection-control measures from other CDC guidelines;⁴⁻⁹ and d) includes two topics not addressed in previous CDC guidelines — infection-control concerns related to animals in health-care facilities and water quality in hemodialysis settings.

Part I of this report, *Background Information: Environmental Infection Control in Health-Care Facilities*, provides a comprehensive review of the scientific literature. Attention is given to engineering and infection-control concerns during construction, demolition, renovation, and repairs of health-care facilities. Use of an infection-control risk assessment is strongly supported before the start of these or any other activities expected to generate dust or water aerosols. Also reviewed in Part I are infection-control measures used to recover from catastrophic events (e.g., flooding, sewage spills, loss of electricity and ventilation, and disruption of the water supply) and the limited effects of environmental surfaces, laundry, plants, animals, medical wastes, cloth furnishings, and carpeting on disease transmission in healthcare facilities.

Part II of this guideline, *Recommendations for Environmental Infection Control in Health-Care Facilities*, outlines environmental infection control in health-care facilities, describing measures for preventing infections associated with air, water, and other elements of the environment. These recommendations represent the views of different divisions within CDC's National Center for Infectious Diseases (NCID) (e.g., the Division of Healthcare Quality Promotion [DHQP] and the Division of Bacterial and Mycotic Diseases [DBMD]) and the consensus of the Healthcare Infection Control Practices Advisory Committee (HICPAC), a 12-member group that advises CDC on concerns related to the surveillance, prevention, and control of health-care-associated infections, primarily in U.S. health-care facilities.¹⁰ In 1999, HICPAC's infection-control focus was expanded from acute-care hospitals to all venues where health care is provided (e.g., outpatient surgical centers, urgent care centers, clinics, outpatient dialysis centers, physicians' offices, and skilled nursing facilities). The topics addressed in this guideline are applicable to the majority of health-care venues in the United States. This document is intended for use primarily by infection-control professionals (ICPs), epidemiologists, employee health and safety personnel, information system specialists, administrators, engineers, facility managers, environmental service professionals, and architects for health-care facilities.

Key recommendations include a) infection-control impact of ventilation system and water system performance; b) establishment of a multidisciplinary team to conduct infection-control risk assessment; c) use of dust-control procedures and barriers during construction, repair, renovation, or demolition; d) environmental infection-control measures for special care areas with patients at high risk; e) use of airborne particle sampling to monitor the effectiveness of air filtration and dust-control measures; f) procedures to prevent airborne contamination in operating rooms when infectious tuberculosis [TB] patients require surgery; g) guidance regarding appropriate indications for routine culturing of water as part of a comprehensive control program for legionellae; h) guidance for recovering from water system disruptions, water leaks, and natural disasters [e.g., flooding]; i) infection-control concepts for equipment that uses water from main lines [e.g., water systems for hemodialysis, ice machines, hydrotherapy equipment, dental unit water lines, and automated endoscope reprocessors]; j) environmental surface cleaning and disinfection strategies with respect to antibiotic-resistant

microorganisms; k) infection-control procedures for health-care laundry; l) use of animals in health care for activities and therapy; m) managing the presence of service animals in health-care facilities; n) infection-control strategies for when animals receive treatment in human health-care facilities; and o) a call to reinstate the practice of inactivating amplified cultures and stocks of microorganisms on-site during medical waste treatment.

Whenever possible, the recommendations in Part II are based on data from well-designed scientific studies. However, certain of these studies were conducted by using narrowly defined patient populations or for specific health-care settings (e.g., hospitals versus long-term care facilities), making generalization of findings potentially problematic. Construction standards for hospitals or other health-care facilities may not apply to residential home-care units. Similarly, infection-control measures indicated for immunosuppressed patient care are usually not necessary in those facilities where such patients are not present. Other recommendations were derived from knowledge gained during infectious disease investigations in health-care facilities, where successful termination of the outbreak was often the result of multiple interventions, the majority of which cannot be independently and rigorously evaluated. This is especially true for construction situations involving air or water.

Other recommendations are derived from empiric engineering concepts and may reflect an industry standard rather than an evidence-based conclusion. Where recommendations refer to guidance from the American Institute of Architects (AIA), the statements reflect standards intended for new construction or renovation. Existing structures and engineered systems are expected to be in continued compliance with the standards in effect at the time of construction or renovation. Also, in the absence of scientific confirmation, certain infection-control recommendations that cannot be rigorously evaluated are based on a strong theoretical rationale and suggestive evidence. Finally, certain recommendations are derived from existing federal regulations. The references and the appendices comprise Parts III and IV of this document, respectively.

Infections caused by the microorganisms described in these guidelines are rare events, and the effect of these recommendations on infection rates in a facility may not be readily measurable. Therefore, the following steps to measure performance are suggested to evaluate these recommendations (Box 1):

Box 1. Environmental infection control: performance measures

-
- 1. Document whether infection-control personnel are actively involved in all phases of a health-care facility's demolition, construction, and renovation. Activities should include performing a risk assessment of the necessary types of construction barriers, and daily monitoring and documenting of the presence of negative airflow within the construction zone or renovation area.**
 - 2. Monitor and document daily the negative airflow in airborne infection isolation (AII) rooms and positive airflow in protective environment (PE) rooms, especially when patients are in these rooms.**
 - 3. Perform assays at least once a month by using standard quantitative methods for endotoxin in water used to reprocess hemodialyzers, and for heterotrophic and mesophilic bacteria in water used to prepare dialysate and for hemodialyzer reprocessing.**
 - 4. Evaluate possible environmental sources (e.g., water, laboratory solutions, or reagents) of specimen contamination when nontuberculous mycobacteria (NTM) of unlikely clinical importance are isolated from clinical cultures. If environmental contamination is found, eliminate the probable mechanisms.**
 - 5. Document policies to identify and respond to water damage. Such policies should result in either repair and drying of wet structural or porous materials within 72 hours, or removal of the wet material if drying is unlikely with 72 hours.**
-

Topics outside the scope of this document include a) noninfectious adverse events (e.g., sick building syndrome); b) environmental concerns in the home; c) home health care; d) bioterrorism; and e) health-care–associated foodborne illness. This document includes only limited discussion of a) handwashing/hand hygiene; b) standard precautions; and c) infection-control measures used to prevent instrument or equipment contamination during patient care (e.g., preventing waterborne contamination of nebulizers or ventilator humidifiers). These topics are mentioned only if they are important in minimizing the transfer of pathogens to and from persons or equipment and the environment. Although the document discusses principles of cleaning and disinfection as they are applied to maintenance of environmental surfaces, the full discussion of sterilization and disinfection of medical instruments and direct patient-care devices is deferred for inclusion in the *Guideline for Disinfection and Sterilization in Health-Care Facilities*, a document currently under development. Similarly, the full discussion of hand hygiene is available as the *Guideline for Hand Hygiene in Health-Care Settings: Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force*. Where applicable, the *Guidelines for Environmental Infection Control in Health-Care Facilities* are consistent in content to the drafts available as of October 2002 of both the revised *Guideline for Prevention of Health-Care–Associated Pneumonia* and *Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health-Care Facilities*.

This guideline was prepared by CDC staff members from NCID and the National Center for Chronic Disease Prevention and Health Promotion (NCCDPHP) and the designated HICPAC advisor. Contributors to this document reviewed predominantly English-language manuscripts identified from reference searches using the National Library of Medicine’s MEDLINE, bibliographies of published articles, and infection-control textbooks. Working drafts of the guideline were reviewed by CDC scientists, HICPAC committee members, and experts in infection control, engineering, internal medicine, infectious diseases, epidemiology, and microbiology. All recommendations in this guideline may not reflect the opinions of all reviewers.

Part I. Background Information: Environmental Infection Control in Health-Care Facilities

A. Introduction

The health-care environment contains a diverse population of microorganisms, but only a few are significant pathogens for susceptible humans. Microorganisms are present in great numbers in moist, organic environments, but some also can persist under dry conditions. Although pathogenic microorganisms can be detected in air and water and on fomites, assessing their role in causing infection and disease is difficult.¹¹ Only a few reports clearly delineate a “cause and effect” with respect to the environment and in particular, housekeeping surfaces.

Eight criteria are used to evaluate the strength of evidence for an environmental source or means of transmission of infectious agents (Box 2).^{11,12} Applying these criteria to disease investigations allows scientists to assess the contribution of the environment to disease transmission. An example of this application is the identification of a pathogen (e.g., vancomycin-resistant enterococci [VRE]) on an environmental surface during an outbreak. The presence of the pathogen does not establish its causal role; its transmission from source to host could be through indirect means (e.g., via hand transferral).¹¹ The surface, therefore, would be considered one of a number of potential reservoirs for the pathogen, but not the “de facto” source of exposure. An understanding of how infection occurs after exposure,

based on the principles of the “chain of infection,” is also important in evaluating the contribution of the environment to health-care–associated disease.¹³ All of the components of the “chain” must be operational for infection to occur (Box 3).

Box 2. Eight criteria for evaluating the strength of evidence for environmental sources of infection* +

1. **The organism can survive after inoculation onto the fomite.**
 2. **The organism can be cultured from in-use fomites.**
 3. **The organism can proliferate in or on the fomite.**
 4. **Some measure of acquisition of infection cannot be explained by other recognized modes of transmission.**
 5. **Retrospective case-control studies show an association between exposure to the fomite and infection.**
 6. **Prospective case-control studies may be possible when more than one similar type of fomite is in use.**
 7. **Prospective studies allocating exposure to the fomite to a subset of patients show an association between exposure and infection.**
 8. **Decontamination of the fomite results in the elimination of infection transmission.**
-

* These criteria are listed in order of strength of evidence.

+ Adapted from references 11 and 12.

Box 3. Chain of infection components*

1. **Adequate number of pathogenic organisms (dose)**
 2. **Pathogenic organisms of sufficient virulence**
 3. **A susceptible host**
 4. **An appropriate mode of transmission or transferal of the organism in sufficient number from source to host**
 5. **The correct portal of entry into the host**
-

* Adapted from reference 13.

The presence of the susceptible host is one of these components that underscores the importance of the health-care environment and opportunistic pathogens on fomites and in air and water. As a result of advances in medical technology and therapies (e.g., cytotoxic chemotherapy and transplantation medicine), more patients are becoming immunocompromised in the course of treatment and are therefore at increased risk for acquiring health-care–associated opportunistic infections. Trends in health-care delivery (e.g., early discharge of patients from acute care facilities) also are changing the distribution of patient populations and increasing the number of immunocompromised persons in non-acute-care hospitals. According to the American Hospital Association (AHA), in 1998, the number of hospitals in the United States totaled 6,021; these hospitals had a total of 1,013,000 beds,¹⁴ representing a 5.5% decrease in the number of acute-care facilities and a 10.2% decrease in the number of beds over the 5-year period 1994–1998.¹⁴ In addition, the total average daily number of patients receiving care in U.S. acute-care hospitals in 1998 was 662,000 (65.4%) – 36.5% less than the 1978 average of 1,042,000.¹⁴ As the number of acute-care hospitals declines, the length of stay in these facilities is concurrently decreasing, particularly for immunocompetent patients. Those patients remaining in acute-care facilities are likely to be those requiring extensive medical interventions who therefore are at high risk for opportunistic infection.

The growing population of severely immunocompromised patients is at odds with demands on the health-care industry to remain viable in the marketplace; to incorporate modern equipment, new diagnostic procedures, and new treatments; and to construct new facilities. Increasing numbers of health-care facilities are likely to be faced with construction in the near future as hospitals consolidate to reduce costs, defer care to ambulatory centers and satellite clinics, and try to create more “home-like” acute-care settings. In 1998, approximately 75% of health-care–associated construction projects focused on renovation of existing outpatient facilities or the building of such facilities;¹⁵ the number of projects associated with outpatient health care rose by 17% from 1998 through 1999.¹⁶ An aging population is also creating increasing demand for assisted-living facilities and skilled nursing centers. Construction of assisted-living facilities in 1998 increased 49% from the previous year, with 138 projects completed at a cost of \$703 million.¹⁶ Overall, from 1998 to 1999, health-care–associated construction costs increased by 28.5%, from \$11.56 billion to \$14.86 billion.¹⁶

Environmental disturbances associated with construction activities near health-care facilities pose airborne and waterborne disease threats risks for the substantial number of patients who are at risk for health-care–associated opportunistic infections. The increasing age of hospitals and other health-care facilities is also generating ongoing need for repair and remediation work (e.g., installing wiring for new information systems, removing old sinks, and repairing elevator shafts) that can introduce or increase contamination of the air and water in patient-care environments. Aging equipment, deferred maintenance, and natural disasters provide additional mechanisms for the entry of environmental pathogens into high-risk patient-care areas.

Architects, engineers, construction contractors, environmental health scientists, and industrial hygienists historically have directed the design and function of hospitals’ physical plants. Increasingly, however, because of the growth in the number of susceptible patients and the increase in construction projects, the involvement of hospital epidemiologists and infection-control professionals is required. These experts help make plans for building, maintaining, and renovating health-care facilities to ensure that the adverse impact of the environment on the incidence of health-care–associated infections is minimal. The following are examples of adverse outcomes that could have been prevented had such experts been involved in the planning process: a) transmission of infections caused by *Mycobacterium tuberculosis*, varicella-zoster virus (VZV), and measles (i.e., rubeola) facilitated by inappropriate air-handling systems in health-care facilities;⁶ b) disease outbreaks caused by *Aspergillus* spp.,^{17–19} *Mucoraceae*,²⁰ and *Penicillium* spp. associated with the absence of environmental controls during periods of health-care facility-associated construction;²¹ c) infections and/or colonizations of patients and staff with vancomycin-resistant *Enterococcus faecium* [VRE] and *Clostridium difficile* acquired indirectly from contact with organisms present on environmental surfaces in health-care facilities,^{22–25} and d) outbreaks and pseudoepidemics of legionellae,^{26, 27} *Pseudomonas aeruginosa*,^{28–30} and the nontuberculous mycobacteria (NTM)^{31, 32} linked to water and aqueous solutions used in health-care facilities. The purpose of this guideline is to provide useful information for both health-care professionals and engineers in efforts to provide a safe environment in which quality health care may be provided to patients. The recommendations herein provide guidance to minimize the risk for and prevent transmission of pathogens in the indoor environment.

B. Key Terms Used in this Guideline

Although Appendix A provides definitions for terms discussed in Part I, several terms that pertain to specific patient-care areas and patients who are at risk for health-care–associated opportunistic infections are presented here. Specific engineering parameters for these care areas are discussed more

fully in the text. **Airborne Infection Isolation (AII)** refers to the isolation of patients infected with organisms spread via airborne droplet nuclei $<5\ \mu\text{m}$ in diameter. This isolation area receives numerous air changes per hour (ACH) (≥ 12 ACH for new construction as of 2001; ≥ 6 ACH for construction before 2001), and is under negative pressure, such that the direction of the airflow is from the outside adjacent space (e.g., corridor) into the room. The air in an AII room is preferably exhausted to the outside, but may be recirculated provided that the return air is filtered through a high efficiency particulate air (HEPA) filter. The use of personal respiratory protection is also indicated for persons entering these rooms.

Protective Environment (PE) is a specialized patient-care area, usually in a hospital, with a positive airflow relative to the corridor (i.e., air flows from the room to the outside adjacent space). The combination of HEPA filtration, high numbers of air changes per hour (≥ 12 ACH), and minimal leakage of air into the room creates an environment that can safely accommodate patients who have undergone allogeneic hematopoietic stem cell transplant (HSCT).

Immunocompromised patients are those patients whose immune mechanisms are deficient because of immunologic disorders (e.g., human immunodeficiency virus [HIV] infection, congenital immune deficiency syndrome, chronic diseases [such as diabetes, cancer, emphysema, and cardiac failure]) or immunosuppressive therapy (e.g., radiation, cytotoxic chemotherapy, anti-rejection medication, and steroids). Immunocompromised patients who are identified as **high-risk patients** have the greatest risk of infection caused by airborne or waterborne microorganisms. Patients in this subset include those who are severely neutropenic for prolonged periods of time (i.e., an absolute neutrophil count [ANC] of ≤ 500 cells/mL), allogeneic HSCT patients, and those who have received intensive chemotherapy (e.g., childhood acute myelogenous leukemia patients).

C. Air

1. Modes of Transmission of Airborne Diseases

A variety of airborne infections in susceptible hosts can result from exposures to clinically significant microorganisms released into the air when environmental reservoirs (i.e., soil, water, dust, and decaying organic matter) are disturbed. Once these materials are brought indoors into a health-care facility by any of a number of vehicles (e.g., people, air currents, water, construction materials, and equipment), the attendant microorganisms can proliferate in various indoor ecological niches and, if subsequently disbursed into the air, serve as a source for airborne health-care-associated infections.

Respiratory infections can be acquired from exposure to pathogens contained either in droplets or droplet nuclei. Exposure to microorganisms in droplets (e.g., through aerosolized oral and nasal secretions from infected patients³³) constitutes a form of direct contact transmission. When droplets are produced during a sneeze or cough, a cloud of infectious particles $>5\ \mu\text{m}$ in size is expelled, resulting in the potential exposure of susceptible persons within 3 feet of the source person.⁶ Examples of pathogens spread in this manner are influenza virus, rhinoviruses, adenoviruses, and respiratory syncytial virus (RSV). Because these agents primarily are transmitted directly and because the droplets tend to fall out of the air quickly, measures to control air flow in a health-care facility (e.g., use of negative pressure rooms) generally are not indicated for preventing the spread of diseases caused by these agents. Strategies to control the spread of these diseases are outlined in another guideline.³

The spread of airborne infectious diseases via droplet nuclei is a form of indirect transmission.³⁴ Droplet nuclei are the residuals of droplets that, when suspended in air, subsequently dry and produce

particles ranging in size from 1–5 μm . These particles can a) contain potentially viable microorganisms, b) be protected by a coat of dry secretions, c) remain suspended indefinitely in air, and d) be transported over long distances. The microorganisms in droplet nuclei persist in favorable conditions (e.g., a dry, cool atmosphere with little or no direct exposure to sunlight or other sources of radiation). Pathogenic microorganisms that can be spread via droplet nuclei include *Mycobacterium tuberculosis*, VZV, measles virus (i.e., rubeola), and smallpox virus (i.e., variola major).⁶ Several environmental pathogens have life-cycle forms that are similar in size to droplet nuclei and may exhibit similar behavior in the air. The spores of *Aspergillus fumigatus* have a diameter of 2–3.5 μm , with a settling velocity estimated at 0.03 cm/second (or about 1 meter/hour) in still air. With this enhanced buoyancy, the spores, which resist desiccation, can remain airborne indefinitely in air currents and travel far from their source.³⁵

2. Airborne Infectious Diseases in Health-Care Facilities

a. Aspergillosis and Other Fungal Diseases

Aspergillosis is caused by molds belonging to the genus *Aspergillus*. *Aspergillus* spp. are prototype health-care-acquired pathogens associated with dusty or moist environmental conditions. Clinical and epidemiologic aspects of aspergillosis (Table 1) are discussed extensively in another guideline.³

Table 1. Clinical and epidemiologic characteristics of aspergillosis

		References
Causative agents	<i>Aspergillus fumigatus</i> (90%–95% of <i>Aspergillus</i> infections among hematopoietic stem cell transplant (HSCT) patients; <i>A. flavus</i> , <i>A. niger</i> , <i>A. terreus</i> , <i>A. nidulans</i>)	36–43
Modes of transmission	Airborne transmission of fungal spores; direct inhalation; direct inoculation from environmental sources (rare)	37
Activities associated with infection	Construction, renovation, remodeling, repairs, building demolition; rare episodes associated with fomites	44–51
Clinical syndromes and diseases	Acute invasive: pneumonia; ulcerative tracheobronchitis; osteomyelitis; abscesses (aspergillomas) of the lungs, brain, liver, spleen, and kidneys; thrombosis of deep blood vessels; necrotizing skin ulcers; endophthalmitis; and sinusitis Chronic invasive: chronic pneumonitis Hypersensitivity: allergic bronchopulmonary aspergillosis Cutaneous: primary skin and burn-wound infections	44, 45, 52–58
Patient populations at greatest risk	Hematopoietic stem cell transplant patients (HSCT): immunocompromised patients (i.e., those with underlying disease), patients undergoing chemotherapy, organ transplant recipients, preterm neonates, hemodialysis patients, patients with identifiable immune system deficiencies who receive care in general intensive care units (ICUs), and cystic fibrosis patients (may be colonized, occasionally become infected)	36, 59–78
Factors affecting severity and outcomes	The immune status of the patient and the duration of severe neutropenia	79, 80
Occurrence	Rare and sporadic, but increasing as proportion of immunocompromised patients increases; 5% of HSCT patients infected, <5% of solid organ transplant recipients infected	36, 37, 81–88
Mortality rate	Rate can be as high as 100% if severe neutropenia persists; 13%–80% mortality among leukemia patients	58, 83, 89, 90

Aspergillus spp. are ubiquitous, aerobic fungi that occur in soil, water, and decaying vegetation; the organism also survives well in air, dust, and moisture present in health-care facilities.^{91–93} The presence of aspergilli in the health-care facility environment is a substantial extrinsic risk factor for opportunistic invasive aspergillosis (invasive aspergillosis being the most serious form of the disease).^{69, 94} Site renovation and construction can disturb *Aspergillus*-contaminated dust and produce bursts of airborne

fungal spores. Increased levels of atmospheric dust and fungal spores have been associated with clusters of health-care–acquired infections in immunocompromised patients.^{17, 20, 44, 47, 49, 50, 95–98}

Absorbent building materials (e.g., wallboard) serve as an ideal substrate for the proliferation of this organism if they become and remain wet, thereby increasing the numbers of fungal spores in the area. Patient-care items, devices, and equipment can become contaminated with *Aspergillus* spp. spores and serve as sources of infection if stored in such areas.⁵⁷

Most cases of aspergillosis are caused by *Aspergillus fumigatus*, a thermotolerant/thermophilic fungus capable of growing over a temperature range from 53.6°F–127.4°F (12°C–53°C); optimal growth occurs at approximately 104°F (40°C), a temperature inhibitory to most other saprophytic fungi.⁹⁹ It can use cellulose or sugars as carbon sources; because its respiratory process requires an ample supply of carbon, decomposing organic matter is an ideal substrate.

Other opportunistic fungi that have been occasionally linked with health-care–associated infections are members of the order *Mucorales* (e.g., *Rhizopus* spp.) and miscellaneous moniliaceous molds (e.g., *Fusarium* spp. and *Penicillium* spp.) (Table 2). Many of these fungi can proliferate in moist environments (e.g., water-damaged wood and building materials). Some fungi (e.g., *Fusarium* spp. and *Pseudoallescheria* spp.) also can be airborne pathogens.¹⁰⁰ As with aspergillosis, a major risk factor for disease caused by any of these pathogens is the host’s severe immunosuppression from either underlying disease or immunosuppressive therapy.^{101, 102}

Table 2. Environmental fungal pathogens: entry into and contamination of the health-care facility

Implicated environmental vehicle	References
<i>Aspergillus</i> spp.	
Improperly functioning ventilation systems	20, 46, 47, 97, 98, 103, 104
Air filters ^{*,†}	17, 18, 105–107
Air filter frames	17, 18
Window air conditioners	96
Backflow of contaminated air	107
Air exhaust contamination [†]	104
False ceilings	48, 57, 97, 108
Fibrous insulation and perforated metal ceilings	66
Acoustic ceiling tiles, plasterboard	18, 109
Fireproofing material	48, 49
Damp wood building materials	49
Opening doors to construction site	110
Construction	69
Open windows	20, 108, 111
Disposal conduit door	68
Hospital vacuum cleaner	68
Elevator	112
Arm boards	57
Walls	113
Unit kitchen	114
Food	21
Ornamental plants	21
<i>Mucorales</i> / <i>Rhizopus</i> spp.	
Air filter	20, 115
False ceilings	97
Heliport	115
<i>Scedosporium</i> spp.	
Construction	116

(Table 2. continued)

Implicated environmental vehicles	References
<i>Penicillium</i> spp.	
Rotting cabinet wood, pipe leak	21
Ventilation duct fiberglass insulation	112
Air filters	105
Topical anesthetic	117
<i>Acromonium</i> spp.	
Air filters	105
<i>Cladosporium</i> spp.	
Air filters	105
<i>Sporothrix</i>	
Construction (pseudoe epidemic)	118

- *. Pigeons, their droppings and roosts are associated with spread of *Aspergillus*, *Cryptococcus*, and *Histoplasma* spp. There have been at least three outbreaks linked to contamination of the filtering systems from bird droppings^{98, 103, 104} Pigeon mites may gain access into a health-care facility through the ventilation system.¹¹⁹
- +. The American Institute of Architects (AIA) standards stipulate that for new or renovated construction a) exhaust outlets are to be placed >25 feet from air intake systems, b) the bottom of outdoor air intakes for HVAC systems should be 6 feet above ground or 3 feet above roof level, and c) exhaust outlets from contaminated areas are situated above the roof level and arranged to minimize the recirculation of exhausted air back into the building.¹²⁰

Infections due *Cryptococcus neoformans*, *Histoplasma capsulatum*, or *Coccidioides immitis* can occur in health-care settings if nearby ground is disturbed and a malfunction of the facility's air-intake components allows these pathogens to enter the ventilation system. *C. neoformans* is a yeast usually 4–8 μm in size. However, viable particles of <2 μm diameter (and thus permissive to alveolar deposition) have been found in soil contaminated with bird droppings, particularly from pigeons.^{98, 103, 104, 121} *H. capsulatum*, with the infectious microconidia ranging in size from 2–5 μm , is endemic in the soil of the central river valleys of the United States. Substantial numbers of these infectious particles have been associated with chicken coops and the roosts of blackbirds.^{98, 103, 104, 122} Several outbreaks of histoplasmosis have been associated with disruption of the environment; construction activities in an endemic area may be a potential risk factor for health-care-acquired airborne infection.^{123, 124} *C. immitis*, with arthrospores of 3–5 μm diameter, has similar potential, especially in the endemic southwestern United States and during seasons of drought followed by heavy rainfall. After the 1994 earthquake centered near Northridge, California, the incidence of coccidioidomycosis in the surrounding area exceeded the historical norm.¹²⁵

Emerging evidence suggests that *Pneumocystis carinii*, now classified as a fungus, may be spread via airborne, person-to-person transmission.¹²⁶ Controlled studies in animals first demonstrated that *P. carinii* could be spread through the air.¹²⁷ More recent studies in health-care settings have detected nucleic acids of *P. carinii* in air samples from areas frequented or occupied by *P. carinii*-infected patients but not in control areas that are not occupied by these patients.^{128, 129} Clusters of cases have been identified among immunocompromised patients who had contact with a source patient and with each other. Recent studies have examined the presence of *P. carinii* DNA in oropharyngeal washings and the nares of infected patients, their direct contacts, and persons with no direct contact.^{130, 131} Molecular analysis of the DNA by polymerase chain reaction (PCR) provides evidence for airborne transmission of *P. carinii* from infected patients to direct contacts, but immunocompetent contacts tend to become transiently colonized rather than infected.¹³¹ The role of colonized persons in the spread of *P. carinii* pneumonia (PCP) remains to be determined. At present, specific modifications to ventilation systems to control spread of PCP in a health-care facility are not indicated. Current recommendations

outline isolation procedures to minimize or eliminate contact of immunocompromised patients not on PCP prophylaxis with PCP-infected patients.^{6, 132}

b. Tuberculosis and Other Bacterial Diseases

The bacterium most commonly associated with airborne transmission is *Mycobacterium tuberculosis*. A comprehensive review of the microbiology and epidemiology of *M. tuberculosis* and guidelines for tuberculosis (TB) infection control have been published.^{4, 133, 134} A summary of the clinical and epidemiologic information from these materials is provided in this guideline (Table 3).

Table 3. Clinical and epidemiologic characteristics of tuberculosis (TB)*

Causative agents	<i>Mycobacterium tuberculosis</i> , <i>M. bovis</i> , <i>M. africanum</i>
Mode of transmission	Airborne transmission via droplet nuclei 1–5 µm in diameter
Patient factors associated with infectivity and transmission	<ul style="list-style-type: none"> ▪ Disease of the lungs, airways, or larynx; presence of cough or other forceful expiratory measures ▪ Presence of acid-fast bacilli (AFB) in the sputum ▪ Failure of the patient to cover the mouth and nose when coughing or sneezing ▪ Presence of cavitation on chest radiograph ▪ Inappropriate or shortened duration of chemotherapy
Activities associated with infections	<ul style="list-style-type: none"> ▪ Exposures in relatively small, enclosed spaces ▪ Inadequate ventilation resulting in insufficient removal of droplet nuclei ▪ Cough-producing procedures done in areas without proper environmental controls ▪ Recirculation of air containing infectious droplet nuclei ▪ Failure to use respiratory protection when managing open lesions for patients with suspected extrapulmonary TB¹³⁵
Clinical syndromes and disease	Pulmonary TB ; extrapulmonary TB can affect any organ system or tissue; laryngeal TB is highly contagious
Populations at greatest risk	<ul style="list-style-type: none"> ▪ Immunocompromised persons (e.g., HIV-infected persons) ▪ Medically underserved persons, urban poor, homeless persons, elderly persons, migrant farm workers, close contacts of known patients ▪ Substance abusers, present and former prison inmates ▪ Foreign-born persons from areas with high prevalence of TB ▪ Health-care workers
Factors affecting severity and outcomes	<ul style="list-style-type: none"> ▪ Concentration of droplet nuclei in air, duration of exposure ▪ Age at infection ▪ Immunosuppression due to therapy or disease, underlying chronic medical conditions, history of malignancies or lesions of the lungs
Occurrence	Worldwide; incidence in the United States is 5.6 cases/100,000 population (2001) ¹³⁶
Mortality	930 deaths in the United States (1999) ¹³⁶
Chemoprophylaxis / treatment	Treatment of latent infection includes isoniazid (INH) or rifampin (RIF). ^{4, 134, 137–139} Directly observed therapy (DOT) for active cases as indicated: INH, RIF, pyrazinamide (PZA), ethambutol (EMB), streptomycin (SM) in various combinations determined by prevalent levels of specific resistance. ^{4, 134, 137–139} Consult therapy guidelines for specific treatment indications. ¹³⁹

* Material in this table is compiled from references 4, 133–141.

M. tuberculosis is carried by droplet nuclei generated when persons (primarily adults and adolescents) who have pulmonary or laryngeal TB sneeze, cough, speak, or sing;¹³⁹ normal air currents can keep these particles airborne for prolonged periods and spread them throughout a room or building.¹⁴² However, transmission of TB has occurred from mycobacteria aerosolized during provision of care (e.g., wound/lesion care or during handling of infectious peritoneal dialysis fluid) for extrapulmonary TB patients.^{135, 140}

Gram-positive cocci (i.e., *Staphylococcus aureus*, group A beta-hemolytic streptococci), also important health-care-associated pathogens, are resistant to inactivation by drying and can persist in the

environment and on environmental surfaces for extended periods. These organisms can be shed from heavily colonized persons and discharged into the air. Airborne dispersal of *S. aureus* is directly associated with the concentration of the bacterium in the anterior nares.¹⁴³ Approximately 10% of healthy carriers will disseminate *S. aureus* into the air, and some persons become more effective disseminators of *S. aureus* than others.^{144–148} The dispersal of *S. aureus* into air can be exacerbated by concurrent viral upper respiratory infection, thereby turning a carrier into a “cloud shedder.”¹⁴⁹ Outbreaks of surgical site infections (SSIs) caused by group A beta-hemolytic streptococci have been traced to airborne transmission from colonized operating-room personnel to patients.^{150–153} In these situations, the strain causing the outbreak was recovered from the air in the operating room^{150, 151, 154} or on settle plates in a room in which the carrier exercised.^{151–153} *S. aureus* and group A streptococci have not been linked to airborne transmission outside of operating rooms, burn units, and neonatal nurseries.^{155, 156} Transmission of these agents occurs primarily via contact and droplets.

Other gram-positive bacteria linked to airborne transmission include *Bacillus* spp. which are capable of sporulation as environmental conditions become less favorable to support their growth. Outbreaks and pseudo-outbreaks have been attributed to *Bacillus cereus* in maternity, pediatric, intensive care, and bronchoscopy units; many of these episodes were secondary to environmental contamination.^{157–160}

Gram-negative bacteria rarely are associated with episodes of airborne transmission because they generally require moist environments for persistence and growth. The main exception is *Acinetobacter* spp., which can withstand the inactivating effects of drying. In one epidemiologic investigation of bloodstream infections among pediatric patients, identical *Acinetobacter* spp. were cultured from the patients, air, and room air conditioners in a nursery.¹⁶¹

Aerosols generated from showers and faucets may potentially contain legionellae and other gram-negative waterborne bacteria (e.g., *Pseudomonas aeruginosa*). Exposure to these organisms is through direct inhalation. However, because water is the source of the organisms and exposure occurs in the vicinity of the aerosol, the discussion of the diseases associated with such aerosols and the prevention measures used to curtail their spread is discussed in another section of the Guideline (see Part I: Water).

c. Airborne Viral Diseases

Some human viruses are transmitted from person to person via droplet aerosols, but very few viruses are consistently airborne in transmission (i.e., are routinely suspended in an infective state in air and capable of spreading great distances), and health-care–associated outbreaks of airborne viral disease are limited to a few agents. Consequently, infection-control measures used to prevent spread of these viral diseases in health-care facilities primarily involve patient isolation, vaccination of susceptible persons, and antiviral therapy as appropriate rather than measures to control air flow or quality.⁶ Infections caused by VZV frequently are described in health-care facilities. Health-care–associated airborne outbreaks of VZV infections from patients with primary infection and disseminated zoster have been documented; patients with localized zoster have, on rare occasions, also served as source patients for outbreaks in health-care facilities.^{162–166} VZV infection can be prevented by vaccination, although patients who develop a rash within 6 weeks of receiving varicella vaccine or who develop breakthrough varicella following exposure should be considered contagious.¹⁶⁷

Viruses whose major mode of transmission is via droplet contact rarely have caused clusters of infections in group settings through airborne routes. The factors facilitating airborne distribution of these viruses in an infective state are unknown, but a presumed requirement is a source patient in the early stage of infection who is shedding large numbers of viral particles into the air. Airborne transmission of measles has been documented in health-care facilities.^{168–171} In addition, institutional outbreaks of influenza virus infections have occurred predominantly in nursing homes,^{172–176} and less frequently in medical and neonatal intensive care units, chronic-care areas, HSCT units, and pediatric

12 [Click here for current information on how Ebola virus is transmitted.](#)

wards.^{177–180} Some evidence supports airborne transmission of influenza viruses by droplet nuclei,^{181, 182} and case clusters in pediatric wards suggest that droplet nuclei may play a role in transmitting certain respiratory pathogens (e.g., adenoviruses and respiratory syncytial virus [RSV]).^{177, 183, 184} Some evidence also supports airborne transmission of enteric viruses. An outbreak of a Norwalk-like virus infection involving more than 600 staff personnel over a 3-week period was investigated in a Toronto, Ontario hospital in 1985; common sources (e.g., food and water) were ruled out during the investigation, leaving airborne spread as the most likely mode of transmission.¹⁸⁵

Smallpox virus, a potential agent of bioterrorism, is spread predominantly via direct contact with infectious droplets, but it also can be associated with airborne transmission.^{186, 187} A German hospital study from 1970 documented the ability of this virus to spread over considerable distances and cause infection at low doses in a well-vaccinated population; factors potentially facilitating transmission in this situation included a patient with cough and an extensive rash, indoor air with low relative humidity, and faulty ventilation patterns resulting from hospital design (e.g., open windows).¹⁸⁸ Smallpox patients with extensive rash are more likely to have lesions present on mucous membranes and therefore have greater potential to disseminate virus into the air.¹⁸⁸ In addition to the smallpox transmission in Germany, two cases of laboratory-acquired smallpox virus infection in the United Kingdom in 1978 also were thought to be caused by airborne transmission.¹⁸⁹

Airborne transmission may play a role in the natural spread of hantaviruses and certain hemorrhagic fever viruses (e.g., Ebola, Marburg, and Lassa), but evidence for airborne spread of these agents in health-care facilities is inconclusive.¹⁹⁰ Although hantaviruses can be transmitted when aerosolized from rodent excreta,^{191, 192} person-to-person spread of hantavirus infection from source patients has not occurred in health-care facilities.^{193–195} Nevertheless, health-care workers are advised to contain potentially infectious aerosols and wear National Institute of Occupational Safety and Health (NIOSH) approved respiratory protection when working with this agent in laboratories or autopsy suites.¹⁹⁶ Lassa virus transmission via aerosols has been demonstrated in the laboratory and incriminated in health-care-associated infections in Africa,^{197–199} but airborne spread of this agent in hospitals in developed nations likely is inefficient.^{200, 201} Yellow fever is considered to be a viral hemorrhagic fever agent with high aerosol infectivity potential, but health-care-associated transmission of this virus has not been described.²⁰² Viral hemorrhagic fever diseases primarily occur after direct exposure to infected blood and body fluids, and the use of standard and droplet precautions prevents transmission early in the course of these illnesses.^{203, 204} However, whether these viruses can persist in droplet nuclei that might remain after droplet production from coughs or vomiting in the latter stages of illness is unknown.²⁰⁵ Although the use of a negative-pressure room is not required during the early stages of illness, its use might be prudent at the time of hospitalization to avoid the need for subsequent patient transfer. Current CDC guidelines recommend negative-pressure rooms with anterooms for patients with hemorrhagic fever and use of HEPA respirators by persons entering these rooms when the patient has prominent cough, vomiting, diarrhea, or hemorrhage.^{6, 203} Face shields or goggles will help to prevent mucous-membrane exposure to potentially-aerosolized infectious material in these situations. If an anteroom is not available, portable, industrial-grade high efficiency particulate air (HEPA) filter units can be used to provide the equivalent of additional air changes per hour (ACH).

Table 4. Microorganisms associated with airborne transmission*

	Fungi	Bacteria	Viruses
Numerous reports in health-care facilities	<i>Aspergillus</i> spp.+ <i>Mucorales (Rhizopus</i> spp.) ^{97, 115}	<i>Mycobacterium tuberculosis</i> +	Measles (rubeola) virus ¹⁶⁸⁻¹⁷⁰ Varicella-zoster virus ¹⁶²⁻¹⁶⁶
Atypical, occasional reports	<i>Acremonium</i> spp. ^{105, 206} <i>Fusarium</i> spp. ¹⁰² <i>Pseudoallescheria boydii</i> ¹⁰⁰ <i>Scedosporium</i> spp. ¹¹⁶ <i>Sporothrix cyanescens</i> ¶ ¹¹⁸	<i>Acinetobacter</i> spp. ¹⁶¹ <i>Bacillus</i> spp.¶ ^{160, 207} <i>Brucella</i> spp.** ²⁰⁸⁻²¹¹ <i>Staphylococcus aureus</i> ^{148, 156} Group A <i>Streptococcus</i> ¹⁵¹	Smallpox virus (variola)§ ^{188, 189} Influenza viruses ^{181, 182} Respiratory syncytial virus ¹⁸³ Adenoviruses ¹⁸⁴ Norwalk-like virus ¹⁸⁵
Airborne in nature; airborne transmission in health care settings not described	<i>Coccidioides immitis</i> ¹²⁵ <i>Cryptococcus</i> spp. ¹²¹ <i>Histoplasma capsulatum</i> ¹²⁴	<i>Coxiella burnetii</i> (Q fever) ²¹²	Hantaviruses ^{193, 195} Lassa virus ²⁰⁵ Marburg virus ²⁰⁵ Ebola virus ²⁰⁵ Crimean-Congo virus ²⁰⁵
Under investigation	<i>Pneumocystis carinii</i> ¹³¹	—	—

* This list excludes microorganisms transmitted from aerosols derived from water.

+ Refer to the text for references for these disease agents.

§ Airborne transmission of smallpox is infrequent. Potential for airborne transmission increases with patients who are effective disseminators present in facilities with low relative humidity in the air and faulty ventilation.

¶ Documentation of pseudoepidemic during construction.

** Airborne transmission documented in the laboratory but not in patient-care areas

3. Heating, Ventilation, and Air Conditioning Systems in Health-Care Facilities

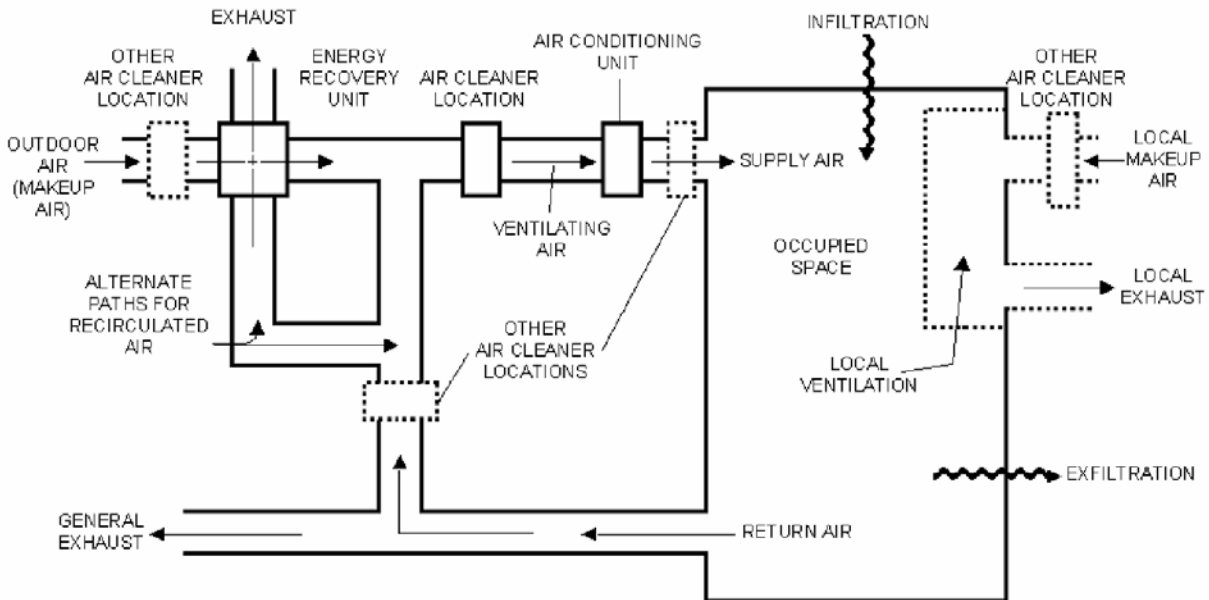
a. Basic Components and Operations

Heating, ventilation, and air conditioning (HVAC) systems in health-care facilities are designed to a) maintain the indoor air temperature and humidity at comfortable levels for staff, patients, and visitors; b) control odors; c) remove contaminated air; d) facilitate air-handling requirements to protect susceptible staff and patients from airborne health-care-associated pathogens; and e) minimize the risk for transmission of airborne pathogens from infected patients.^{35, 120} An HVAC system includes an outside air inlet or intake; filters; humidity modification mechanisms (i.e., humidity control in summer, humidification in winter); heating and cooling equipment; fans; ductwork; air exhaust or out-takes; and registers, diffusers, or grilles for proper distribution of the air (Figure 1).^{213, 214} Decreased performance of healthcare facility HVAC systems, filter inefficiencies, improper installation, and poor maintenance can contribute to the spread of health-care-associated airborne infections.

The American Institute of Architects (AIA) has published guidelines for the design and construction of new health-care facilities and for renovation of existing facilities. These AIA guidelines address indoor air-quality standards (e.g., ventilation rates, temperature levels, humidity levels, pressure relationships, and minimum air changes per hour [ACH]) specific to each zone or area in health-care facilities (e.g., operating rooms, laboratories, diagnostic areas, patient-care areas, and support departments).¹²⁰ These guidelines represent a consensus document among authorities having jurisdiction (AHJ), governmental regulatory agencies (i.e., Department of Health and Human Services [DHHS]; Department of Labor, Occupational Safety and Health Administration [OSHA]), health-care professionals, professional organizations (e.g., American Society of Heating, Refrigeration, and Air-Conditioning Engineers [ASHRAE], American Society for Healthcare Engineering [ASHE]), and accrediting organizations (i.e., Joint Commission on Accreditation of Healthcare Organizations [JCAHO]). More than 40 state agencies that license health-care facilities have either incorporated or adopted by reference these

guidelines into their state standards. JCAHO, through its surveys, ensures that facilities are in compliance with the ventilation guidelines of this standard for new construction and renovation.

Figure 1. Diagram of a ventilation system*



Outdoor air and recirculated air pass through air cleaners (e.g., filter banks) designed to reduce the concentration of airborne contaminants. Air is conditioned for temperature and humidity before it enters the occupied space as supply air. Infiltration is air leakage inward through cracks and interstitial spaces of walls, floors, and ceilings. Exfiltration is air leakage outward through these same cracks and spaces. Return air is largely exhausted from the system, but a portion is recirculated with fresh, incoming air.

* Used with permission of the publisher of reference 214 (ASHRAE)

Engineering controls to contain or prevent the spread of airborne contaminants center on a) local exhaust ventilation [i.e., source control], b) general ventilation, and c) air cleaning.⁴ General ventilation encompasses a) dilution and removal of contaminants via well-mixed air distribution of filtered air, b) directing contaminants toward exhaust registers and grilles via uniform, non-mixed airflow patterns, c) pressurization of individual spaces relative to all other spaces, and d) pressurization of buildings relative to the outdoors and other attached buildings.

A centralized HVAC system operates as follows. Outdoor air enters the system, where low-efficiency or “roughing” filters remove large particulate matter and many microorganisms. The air enters the distribution system for conditioning to appropriate temperature and humidity levels, passes through an additional bank of filters for further cleaning, and is delivered to each zone of the building. After the conditioned air is distributed to the designated space, it is withdrawn through a return duct system and delivered back to the HVAC unit. A portion of this “return air” is exhausted to the outside while the remainder is mixed with outdoor air for dilution and filtered for removal of contaminants.²¹⁵ Air from toilet rooms or other soiled areas is usually exhausted directly to the atmosphere through a separate duct exhaust system. Air from rooms housing tuberculosis patients is exhausted to the outside if possible, or passed through a HEPA filter before recirculation. Ultraviolet germicidal irradiation (UVGI) can be used as an adjunct air-cleaning measure, but it cannot replace HEPA filtration.

b. Filtration

i. Filter Types and Methods of Filtration

Filtration, the physical removal of particulates from air, is the first step in achieving acceptable indoor air quality. Filtration is the primary means of cleaning the air. Five methods of filtration can be used (Table 5). During filtration, outdoor air passes through two filter beds or banks (with efficiencies of 20%–40% and $\geq 90\%$, respectively) for effective removal of particles 1–5 μm in diameter.^{35, 120} The low-to-medium efficiency filters in the first bank have low resistance to airflow, but this feature allows some small particulates to pass onto heating and air conditioning coils and into the indoor environment.³⁵ Incoming air is mixed with recirculated air and reconditioned for temperature and humidity before being filtered by the second bank of filters. The performance of filters with $\leq 90\%$ efficiency is measured using either the dust-spot test or the weight-arrestance test.^{35, 216}

Table 5. Filtration methods*

Basic method	Principle of performance	Filtering efficiency
Straining	Particles in the air are larger than the openings between the filter fibers, resulting in gross removal of large particles.	Low
Impingement	Particles collide with filter fibers and remain attached to the filter. Fibers may be coated with adhesive.	Low
Interception	Particles enter into the filter and become entrapped and attached to the filter fibers.	Medium
Diffusion	Small particles, moving in erratic motion, collide with filter fibers and remain attached.	High
Electrostatic	Particles bearing negative electrostatic charge are attracted to the filter with positively charged fibers.	High

* Material in this table was compiled from information in reference 217.

The second filter bank usually consists of high-efficiency filters. This filtration system is adequate for most patient-care areas in ambulatory-care facilities and hospitals, including the operating room environment and areas providing central services.¹²⁰ Nursing facilities use 90% dust-spot efficient filters as the second bank of filters,¹²⁰ whereas a HEPA filter bank may be indicated for special-care areas of hospitals. HEPA filters are at least 99.97% efficient for removing particles $\geq 0.3 \mu\text{m}$ in diameter. (As a reference, *Aspergillus* spores are 2.5–3.0 μm in diameter.) Examples of care areas where HEPA filters are used include PE rooms and those operating rooms designated for orthopedic implant procedures.³⁵

Maintenance costs associated with HEPA filters are high compared with other types of filters, but use of in-line disposable prefilters can increase the life of a HEPA filter by approximately 25%. Alternatively, if a disposable prefilter is followed by a filter that is 90% efficient, the life of the HEPA filter can be extended ninefold. This concept, called progressive filtration, allows HEPA filters in special care areas to be used for 10 years.²¹³ Although progressive filtering will extend the mechanical ability of the HEPA filter, these filters may absorb chemicals in the environment and later desorb those chemicals, thereby necessitating a more frequent replacement program. HEPA filter efficiency is monitored with the dioctylphthalate (DOP) particle test using particles that are 0.3 μm in diameter.²¹⁸

HEPA filters are usually framed with metal, although some older versions have wood frames. A metal frame has no advantage over a properly fitted wood frame with respect to performance, but wood can compromise the air quality if it becomes and remains wet, allowing the growth of fungi and bacteria. Hospitals are therefore advised to phase out water-damaged or spent wood-framed filter units and replace them with metal-framed HEPA filters.

HEPA filters are usually fixed into the HVAC system; however, portable, industrial grade HEPA units are available that can filter air at the rate of 300–800 ft³/min. Portable HEPA filters are used to a) temporarily recirculate air in rooms with no general ventilation, b) augment systems that cannot provide adequate airflow, and c) provide increased effectiveness in airflow.⁴ Portable HEPA units are useful engineering controls that help clean the air when the central HVAC system is undergoing repairs,²¹⁹ but these units do not satisfy fresh-air requirements.²¹⁴ The effectiveness of the portable unit for particle removal is dependent on a) the configuration of the room, b) the furniture and persons in the room, c) the placement of the units relative to the contents and layout of the room, and d) the location of the supply and exhaust registers or grilles. If portable, industrial-grade units are used, they should be capable of recirculating all or nearly all of the room air through the HEPA filter, and the unit should be designed to achieve the equivalent of ≥ 12 ACH.⁴ (An average room has approximately 1,600 ft³ of airspace.) The hospital engineering department should be contacted to provide ACH information in the event that a portable HEPA filter unit is necessary to augment the existing fixed HVAC system for air cleaning.

ii. Filter Maintenance

Efficiency of the filtration system is dependent on the density of the filters, which can create a drop in pressure unless compensated by stronger and more efficient fans, thus maintaining air flow. For optimal performance, filters require monitoring and replacement in accordance with the manufacturer's recommendations and standard preventive maintenance practices.²²⁰ Upon removal, spent filters can be bagged and discarded with the routine solid waste, regardless of their patient-care area location.²²¹ Excess accumulation of dust and particulates increases filter efficiency, requiring more pressure to push the air through. The pressure differential across filters is measured by use of manometers or other gauges. A pressure reading that exceeds specifications indicates the need to change the filter. Filters also require regular inspection for other potential causes of decreased performance. Gaps in and around filter banks and heavy soil and debris upstream of poorly maintained filters have been implicated in health-care-associated outbreaks of aspergillosis, especially when accompanied by construction activities at the facility.^{17, 18, 106, 222}

c. Ultraviolet Germicidal Irradiation (UVGI)

As a supplemental air-cleaning measure, UVGI is effective in reducing the transmission of airborne bacterial and viral infections in hospitals, military housing, and classrooms, but it has only a minimal inactivating effect on fungal spores.^{223–228} UVGI is also used in air handling units to prevent or limit the growth of vegetative bacteria and fungi. Most commercially available UV lamps used for germicidal purposes are low-pressure mercury vapor lamps that emit radiant energy predominantly at a wave-length of 253.7 nm.^{229, 230} Two systems of UVGI have been used in health-care settings – duct irradiation and upper-room air irradiation. In duct irradiation systems, UV lamps are placed inside ducts that remove air from rooms to disinfect the air before it is recirculated. When properly designed, installed, and maintained, high levels of UVGI can be attained in the ducts with little or no exposure of persons in the rooms.^{231, 232} In upper-room air irradiation, UV lamps are either suspended from the ceiling or mounted on the wall.⁴ Upper air UVGI units have two basic designs: a) a “pan” fixture with UVGI unshielded above the unit to direct the irradiation upward and b) a fixture with a series of parallel plates to columnize the irradiation outward while preventing the light from getting to the eyes of the room's occupants. The germicidal effect is dependent on air mixing via convection between the room's irradiated upper zone and the lower patient-care zones.^{233, 234}

Bacterial inactivation studies using BCG mycobacteria and *Serratia marcescens* have estimated the effect of UVGI as equivalent to 10 ACH–39 ACH.^{235, 236} Another study, however, suggests that UVGI may result in fewer equivalent ACH in the patient-care zone, especially if the mixing of air between zones is insufficient.²³⁴ The use of fans or HVAC systems to generate air movement may increase the effectiveness of UVGI if airborne microorganisms are exposed to the light energy for a sufficient length

of time.^{233, 235, 237–239} The optimal relationship between ventilation and UVGI is not known.

Because the clinical effectiveness of UV systems may vary, UVGI is not recommended for air management prior to air recirculation from airborne isolation rooms. It is also not recommended as a substitute for HEPA filtration, local exhaust of air to the outside, or negative pressure.⁴ The use of UV lamps and HEPA filtration in a single unit offers only minimal infection-control benefits over those provided by the use of a HEPA filter alone.²⁴⁰ Duct systems with UVGI are not recommended as a substitute for HEPA filters if the air from isolation rooms must be recirculated to other areas of the facility.⁴ Regular maintenance of UVGI systems is crucial and usually consists of keeping the bulbs free of dust and replacing old bulbs as necessary. Safety issues associated with the use of UVGI systems are described in other guidelines.⁴

d. Conditioned Air in Occupied Spaces

Temperature and humidity are two essential components of conditioned air. After outside air passes through a low- or medium-efficiency filter, the air undergoes conditioning for temperature and humidity control before it passes through high-efficiency or HEPA filtration.

i. Temperature

HVAC systems in health-care facilities are often single-duct or dual-duct systems.^{35, 241} A single-duct system distributes cooled air (55°F [12.8°C]) throughout the building and uses thermostatically controlled reheat boxes located in the terminal ductwork to warm the air for individual or multiple rooms. The dual-duct system consists of parallel ducts, one with a cold air stream and the other with a hot air stream. A mixing box in each room or group of rooms mixes the two air streams to achieve the desired temperature. Temperature standards are given as either a single temperature or a range, depending on the specific health-care zone. Cool temperature standards (68°F–73°F [20°C–23°C]) usually are associated with operating rooms, clean workrooms, and endoscopy suites.¹²⁰ A warmer temperature (75°F [24°C]) is needed in areas requiring greater degrees of patient comfort. Most other zones use a temperature range of 70°F–75°F (21°C–24°C).¹²⁰ Temperatures outside of these ranges may be needed occasionally in limited areas depending on individual circumstances during patient care (e.g., cooler temperatures in operating rooms during specialized operations).

ii. Humidity

Four measures of humidity are used to quantify different physical properties of the mixture of water vapor and air. The most common of these is relative humidity, which is the ratio of the amount of water vapor in the air to the amount of water vapor air can hold at that temperature.²⁴² The other measures of humidity are specific humidity, dew point, and vapor pressure.²⁴²

Relative humidity measures the percentage of saturation. At 100% relative humidity, the air is saturated. For most areas within health-care facilities, the designated comfort range is 30%–60% relative humidity.^{120, 214} Relative humidity levels >60%, in addition to being perceived as uncomfortable, promote fungal growth.²⁴³ Humidity levels can be manipulated by either of two mechanisms.²⁴⁴ In a water-wash unit, water is sprayed and drops are taken up by the filtered air; additional heating or cooling of this air sets the humidity levels. The second mechanism is by means of water vapor created from steam and added to filtered air in humidifying boxes. Reservoir-type humidifiers are not allowed in health-care facilities as per AIA guidelines and many state codes.¹²⁰ Cool-mist humidifiers should be avoided, because they can disseminate aerosols containing allergens and microorganisms.²⁴⁵ Additionally, the small, personal-use versions of this equipment can be difficult to clean.

iii. Ventilation

The control of air pollutants (e.g., microorganisms, dust, chemicals, and smoke) at the source is the most effective way to maintain clean air. The second most effective means of controlling indoor air pollution is through ventilation. Ventilation rates are voluntary unless a state or local government specifies a standard in health-care licensing or health department requirements. These standards typically apply to only the design of a facility, rather than its operation.^{220, 246} Health-care facilities without specific ventilation standards should follow the AIA guideline specific to the year in which the building was built or the ANSI/ASHRAE Standard 62, *Ventilation for Acceptable Indoor Air Quality*.^{120, 214, 241}

Ventilation guidelines are defined in terms of air volume per minute per occupant and are based on the assumption that occupants and their activities are responsible for most of the contaminants in the conditioned space.²¹⁵ Most ventilation rates for health-care facilities are expressed as room ACH. Peak efficiency for particle removal in the air space occurs between 12 ACH–15 ACH.^{35, 247, 248} Ventilation rates vary among the different patient-care areas of a health-care facility (Appendix B).¹²⁰

Health-care facilities generally use recirculated air.^{35, 120, 241, 249, 250} Fans create sufficient positive pressure to force air through the building duct work and adequate negative pressure to evacuate air from the conditioned space into the return duct work and/or exhaust, thereby completing the circuit in a sealed system (Figure 1). However, because gaseous contaminants tend to accumulate as the air recirculates, a percentage of the recirculated air is exhausted to the outside and replaced by fresh outdoor air. In hospitals, the delivery of filtered air to an occupied space is an engineered system design issue, the full discussion of which is beyond the scope of this document.

Hospitals with areas not served by central HVAC systems often use through-the-wall or fan coil air conditioning units as the sole source of room ventilation. AIA guidelines for newly installed systems stipulate that through-the-wall fan-coil units be equipped with permanent (i.e., cleanable) or replaceable filters with a minimum efficiency of 68% weight arrestance.¹²⁰ These units may be used only as recirculating units; all outdoor air requirements must be met by a separate central air handling system with proper filtration, with a minimum of two outside air changes in general patient rooms (D. Erickson, ASHE, 2000).¹²⁰ If a patient room is equipped with an individual through-the-wall fan coil unit, the room should not be used as either AII or as PE.¹²⁰ These requirements, although directed to new HVAC installations also are appropriate for existing settings. Non-central air-handling systems are prone to problems associated with excess condensation accumulating in drip pans and improper filter maintenance; health-care facilities should clean or replace the filters in these units on a regular basis while the patient is out of the room.

Laminar airflow ventilation systems are designed to move air in a single pass, usually through a bank of HEPA filters either along a wall or in the ceiling, in a one-way direction through a clean zone with parallel streamlines. Laminar airflow can be directed vertically or horizontally; the unidirectional system optimizes airflow and minimizes air turbulence.^{63, 241} Delivery of air at a rate of 0.5 meters per second (90 ± 20 ft/min) helps to minimize opportunities for microorganism proliferation.^{63, 251, 252} Laminar airflow systems have been used in PE to help reduce the risk for health-care-associated airborne infections (e.g., aspergillosis) in high-risk patients.^{63, 93, 253, 254} However, data that demonstrate a survival benefit for patients in PE with laminar airflow are lacking. Given the high cost of installation and apparent lack of benefit, the value of laminar airflow in this setting is questionable.^{9, 37} Few data support the use of laminar airflow systems elsewhere in a hospital.²⁵⁵

iv. Pressurization

Positive and negative pressures refer to a pressure differential between two adjacent air spaces (e.g., rooms and hallways). Air flows away from areas or rooms with positive pressure (pressurized), while

air flows into areas with negative pressure (depressurized). All rooms are set at negative pressure to prevent airborne microorganisms in the room from entering hallways and corridors. PE rooms housing severely neutropenic patients are set at positive pressure to keep airborne pathogens in adjacent spaces or corridors from coming into and contaminating the airspace occupied by such high-risk patients. Self-closing doors are mandatory for both of these areas to help maintain the correct pressure differential.^{4,6,120} Older health-care facilities may have variable pressure rooms (i.e., rooms in which the ventilation can be manually switched between positive and negative pressure). These rooms are no longer permitted in the construction of new facilities or in renovated areas of the facility,¹²⁰ and their use in existing facilities has been discouraged because of difficulties in assuring the proper pressure differential, especially for the negative pressure setting, and because of the potential for error associated with switching the pressure differentials for the room. Continued use of existing variable pressure rooms depends on a partnership between engineering and infection control. Both positive- and negative-pressure rooms should be maintained according to specific engineering specifications (Table 6).

Table 6. Engineered specifications for positive- and negative pressure rooms*

	Positive pressure areas (e.g., protective environments [PE])	Negative pressure areas (e.g., airborne infection isolation [AII])
Pressure differentials	> +2.5 Pa§ (0.01" water gauge)	> -2.5 Pa (0.01" water gauge)
Air changes per hour (ACH)	>12	≥12 (for renovation or new construction)
Filtration efficiency	Supply: 99.97% @ 0.3 µm DOP¶ Return: none required**	Supply: 90% (dust spot test) Return: 99.97% @ 0.3 µm DOP¶ †
Room airflow direction	Out to the adjacent area	In to the room
Clean-to-dirty airflow in room	Away from the patient (high-risk patient, immunosuppressed patient)	Towards the patient (airborne disease patient)
Ideal pressure differential	> + 8 Pa	> - 2.5 Pa

* Material in this table was compiled from references 35 and 120. Table adapted from and used with permission of the publisher of reference 35 (Lippincott Williams and Wilkins).

§ Pa is the abbreviation for Pascal, a metric unit of measurement for pressure based on air velocity; 250 Pa equals 1.0 inch water gauge.

¶ DOP is the abbreviation for dioctylphthalate particles of 0.3 µm diameter.

** If the patient requires both PE and AII, return air should be HEPA-filtered or otherwise exhausted to the outside.

† HEPA filtration of exhaust air from AII rooms should not be required, providing that the exhaust is properly located to prevent re-entry into the building.

Health-care professionals (e.g., infection control, hospital epidemiologists) must perform a risk assessment to determine the appropriate number of AII rooms (negative pressure) and/or PE rooms (positive pressure) to serve the patient population. The AIA guidelines require a certain number of AII rooms as a minimum, and it is important to refer to the edition under which the building was built for appropriate guidance.¹²⁰

In large health-care facilities with central HVAC systems, sealed windows help to ensure the efficient operation of the system, especially with respect to creating and maintaining pressure differentials. Sealing the windows in PE areas helps minimize the risk of airborne contamination from the outside. One outbreak of aspergillosis among immunosuppressed patients in a hospital was attributed in part to an open window in the unit during a time when both construction and a fire happened nearby; sealing the window prevented further entry of fungal spores into the unit from the outside air.¹¹¹ Additionally, all emergency exits (e.g., fire escapes and emergency doors) in PE wards should be kept closed (except during emergencies) and equipped with alarms.

e. Infection Control Impact of HVAC System Maintenance and Repair

A failure or malfunction of any component of the HVAC system may subject patients and staff to discomfort and exposure to airborne contaminants. Only limited information is available from formal

studies on the infection-control implications of a complete air-handling system failure or shutdown for maintenance. Most experience has been derived from infectious disease outbreaks and adverse outcomes among high-risk patients when HVAC systems are poorly maintained. (See Table 7 for potential ventilation hazards, consequences, and correction measures.)

AIA guidelines prohibit U.S. hospitals and surgical centers from shutting down their HVAC systems for purposes other than required maintenance, filter changes, and construction.¹²⁰ Airflow can be reduced; however, sufficient supply, return, and exhaust must be provided to maintain required pressure relationships when the space is not occupied. Maintaining these relationships can be accomplished with special drives on the air-handling units (i.e., a variable air ventilation [VAV] system).

Microorganisms proliferate in environments wherever air, dust, and water are present, and air-handling systems can be ideal environments for microbial growth.³⁵ Properly engineered HVAC systems require routine maintenance and monitoring to provide acceptable indoor air quality efficiently and to minimize conditions that favor the proliferation of health-care-associated pathogens.^{35, 249} Performance monitoring of the system includes determining pressure differentials across filters, regular inspection of system filters, DOP testing of HEPA filters, testing of low- or medium efficiency filters, and manometer tests for positive- and negative-pressure areas in accordance with nationally recognized standards, guidelines, and manufacturers' recommendations. The use of hand-held, calibrated equipment that can provide a numerical reading on a daily basis is preferred for engineering purposes (A. Streifel, University of Minnesota, 2000).²⁵⁶ Several methods that provide a visual, qualitative measure of pressure differentials (i.e., airflow direction) include smoke-tube tests or placing flutter strips, ping-pong balls, or tissue in the air stream.

Preventive filter and duct maintenance (e.g., cleaning ductwork vents, replacing filters as needed, and properly disposing spent filters into plastic bags immediately upon removal) is important to prevent potential exposures of patients and staff during HVAC system shut-down. The frequency of filter inspection and the parameters of this inspection are established by each facility to meet their unique needs. Ductwork in older health-care facilities may have insulation on the interior surfaces that can trap contaminants. This insulation material tends to break down over time to be discharged from the HVAC system. Additionally, a malfunction of the air-intake system can overburden the filtering system and permit aerosolization of fungal pathogens. Keeping the intakes free from bird droppings, especially those from pigeons, helps to minimize the concentration of fungal spores entering from the outside.⁹⁸

Accumulation of dust and moisture within HVAC systems increases the risk for spread of health-care-associated environmental fungi and bacteria. Clusters of infections caused by *Aspergillus* spp., *P. aeruginosa*, *S. aureus*, and *Acinetobacter* spp. have been linked to poorly maintained and/or malfunctioning air conditioning systems.^{68, 161, 257, 258} Efforts to limit excess humidity and moisture in the infrastructure and on air-stream surfaces in the HVAC system can minimize the proliferation and dispersion of fungal spores and waterborne bacteria throughout indoor air.²⁵⁹⁻²⁶² Within the HVAC system, water is present in water-wash units, humidifying boxes, or cooling units. The dual-duct system may also create conditions of high humidity and excess moisture that favor fungal growth in drain pans as well as in fibrous insulation material that becomes damp as a result of the humid air passing over the hot stream and condensing.

If moisture is present in the HVAC system, periods of stagnation should be avoided. Bursts of organisms can be released upon system start-up, increasing the risk of airborne infection.²⁰⁶ Proper engineering of the HVAC system is critical to preventing dispersal of airborne organisms. In one hospital, endophthalmitis caused by *Acremonium kiliense* infection following cataract extraction in an ambulatory surgical center was traced to aerosols derived from the humidifier water in the ventilation system.²⁰⁶ The organism proliferated because the ventilation system was turned off routinely when the

center was not in operation; the air was filtered before humidification, but not afterwards.

Most health-care facilities have contingency plans in case of disruption of HVAC services. These plans include back-up power generators that maintain the ventilation system in high-risk areas (e.g., operating rooms, intensive-care units, negative- and positive-pressure rooms, transplantation units, and oncology units). Alternative generators are required to engage within 10 seconds of a loss of main power. If the ventilation system is out of service, rendering indoor air stagnant, sufficient time must be allowed to clean the air and re-establish the appropriate number of ACH once the HVAC system begins to function again. Air filters may also need to be changed, because reactivation of the system can dislodge substantial amounts of dust and create a transient burst of fungal spores.

Duct cleaning in health-care facilities has benefits in terms of system performance, but its usefulness for infection control has not been conclusively determined. Duct cleaning typically involves using specialized tools to dislodge dirt and a high-powered vacuum cleaner to clean out debris.²⁶³ Some duct-cleaning services also apply chemical biocides or sealants to the inside surfaces of ducts to minimize fungal growth and prevent the release of particulate matter. The U.S. Environmental Protection Agency (EPA), however, has concerns with the use of sanitizers and/or disinfectants to treat the surfaces of ductwork, because the label indications for most of these products may not specifically include the use of the product in HVAC systems.²⁶⁴ Further, EPA has not evaluated the potency of disinfectants in such applications, nor has the agency examined the potential attendant health and safety risks. The EPA recommends that companies use only those chemical biocides that are registered for use in HVAC systems.²⁶⁴ Although infrequent cleaning of the exhaust ducts in AII areas has been documented as a cause of diminishing negative pressure and a decrease in the air exchange rates,²¹⁴ no data indicate that duct cleaning, beyond what is recommended for optimal performance, improves indoor air quality or reduces the risk of infection. Exhaust return systems should be cleaned as part of routine system maintenance. Duct cleaning has not been shown to prevent any health problems,²⁶⁵ and EPA studies indicate that airborne particulate levels do not increase as a result of dirty air ducts, nor do they diminish after cleaning, presumably because much of the dirt inside air ducts adheres to duct surfaces and does not enter the conditioned space.²⁶⁵ Additional research is needed to determine if air-duct contamination can significantly increase the airborne infection risk in general areas of health-care facilities.

4. Construction, Renovation, Remediation, Repair, and Demolition

a. General Information

Environmental disturbances caused by construction and/or renovation and repair activities (e.g., disruption of the above-ceiling area, running cables through the ceiling, and structural repairs) in and near health-care facilities markedly increase the airborne *Aspergillus* spp. spore counts in the indoor air of such facilities, thereby increasing the risk for health-care-associated aspergillosis among high-risk patients. Although one case of health-care-associated aspergillosis is often difficult to link to a specific environmental exposure, the occurrence of temporarily clustered cases increase the likelihood that an environmental source within the facility may be identified and corrected.

Table 7. Ventilation hazards in health-care facilities that may be associated with increased potential of airborne disease transmission*

Problem§	Consequences	Possible solutions
Water-damaged building materials (18, 266)	Water leaks can soak wood, wall board, insulation, wall coverings, ceiling tiles, and carpeting. All of these materials can provide microbial habitat when wet. This is especially true for fungi growing on gypsum board.	<ol style="list-style-type: none"> 1. Replace water-damaged materials. 2. Incorporate fungistatic compounds into building materials in areas at risk for moisture problems. 3. Test for all moisture and dry in less than 72 hours. Replace if the material cannot dry within 72 hours.
Filter bypasses (17)	Rigorous air filtration requires air flow resistance. Air stream will elude filtration if openings are present because of filter damage or poor fit.	<ol style="list-style-type: none"> 1. Use pressure gauges to ensure that filters are performing at proper static pressure. 2. Make ease of installation and maintenance criteria for filter selection. 3. Properly train maintenance personnel in HVAC concerns. 4. Design system with filters downstream from fans. 5. Avoid water on filters or insulation.
Improper fan setting (267)	Air must be delivered at design volume to maintain pressure balances. Air flow in special vent rooms reverses.	<ol style="list-style-type: none"> 1. Routinely monitor air flow and pressure balances throughout critical parts of HVAC system. 2. Minimize or avoid using rooms that switch between positive and negative pressure.
Ductwork disconnections (268)	Dislodged or leaky supply duct runs can spill into and leaky returns may draw from hidden areas. Pressure balance will be interrupted, and infectious material may be disturbed and entrained into hospital air supply.	<ol style="list-style-type: none"> 1. Design a ductwork system that is easy to access, maintain, and repair. 2. Train maintenance personnel to regularly monitor air flow volumes and pressure balances throughout the system. 3. Test critical areas for appropriate air flow
Air flow impedance (213)	Debris, structural failure, or improperly adjusted dampers can block duct work and prevent designed air flow.	<ol style="list-style-type: none"> 1. Design and budget for a duct system that is easy to inspect, maintain, and repair. 2. Alert contractors to use caution when working around HVAC systems during the construction phase. 3. Regularly clean exhaust grilles. 4. Provide monitoring for special ventilation areas.
Open windows (96, 247)	Open windows can alter fan-induced pressure balance and allow dirty-to-clean air flow.	<ol style="list-style-type: none"> 1. Use sealed windows. 2. Design HVAC systems to deliver sufficient outdoor dilution ventilation. 3. Ensure that OSHA indoor air quality standards are met.
Dirty window air conditioners (96, 269)	Dirt, moisture, and bird droppings can contaminate window air conditioners, which can then introduce infectious material into hospital rooms.	<ol style="list-style-type: none"> 1. Eliminate such devices in plans for new construction. 2. Where they must be used, make sure that they are routinely cleaned and inspected.

Problem§	Consequences	Possible solutions
Inadequate filtration (270)	Infectious particles may pass through filters into vulnerable patient areas.	<ol style="list-style-type: none"> 1. Specify appropriate filters during new construction design phase. 2. Make sure that HVAC fans are sized to overcome pressure demands of filter system. 3. Inspect and test filters for proper installation.
Maintenance disruptions (271)	Fan shut-offs, dislodged filter cake material contaminates downstream air supply and drain pans. This may compromise air flow in special ventilation areas.	<ol style="list-style-type: none"> 1. Budget for a rigorous maintenance schedule when designing a facility. 2. Design system for easy maintenance. 3. Ensure communication between engineering and maintenance personnel. 4. Institute an ongoing training program for all involved staff members.
Excessive moisture in the HVAC system (120)	Chronically damp internal lining of the HVAC system, excessive condensate, and drip pans with stagnant water may result from this problem.	<ol style="list-style-type: none"> 1. Locate duct humidifiers upstream of the final filters. 2. Identify a means to remove water from the system. 3. Monitor humidity; all duct take-offs should be downstream of the humidifiers so that moisture is absorbed completely. 4. Use steam humidifiers in the HVAC system.
Duct contamination (18, 272)	Debris is released during maintenance or cleaning.	<ol style="list-style-type: none"> 1. Provide point-of-use filtration in the critical areas. 2. Design air-handling systems with insulation of the exterior of the ducts. 3. Do not use fibrous sound attenuators. 4. Decontaminate or encapsulate contamination.

* Reprinted with permission of the publisher of reference 35 (Lippincott Williams and Wilkins).

§ Numbers in parentheses are reference citations.

Construction, renovation, repair, and demolition activities in health-care facilities require substantial planning and coordination to minimize the risk for airborne infection both during projects and after their completion. Several organizations and experts have endorsed a multi-disciplinary team approach (Box 4) to coordinate the various stages of construction activities (e.g., project inception, project implementation, final walk-through, and completion).^{120, 249, 250, 273–276} Environmental services, employee health, engineering, and infection control must be represented in construction planning and design meetings should be convened with architects and design engineers. The number of members and disciplines represented is a function of the complexity of a project. Smaller, less complex projects and maintenance may require a minimal number of members beyond the core representation from engineering, infection control, environmental services, and the directors of the specialized departments.

Box 4. Suggested members and functions of a multi-disciplinary coordination team for construction, renovation, repair, and demolition projects

Members

Infection-control personnel, including hospital epidemiologists
Laboratory personnel
Facility administrators or their designated representatives, facility managers
Director of engineering
Risk-management personnel
Directors of specialized programs (e.g., transplantation, oncology and ICU* programs)
Employee safety personnel, industrial hygienists, and regulatory affairs personnel
Environmental services personnel
Information systems personnel
Construction administrators or their designated representatives
Architects, design engineers, project managers, and contractors

Functions and responsibilities

Coordinate members' input in developing a comprehensive project management plan.
Conduct a risk assessment of the project to determine potential hazards to susceptible patients.
Prevent unnecessary exposures of patients, visitors, and staff to infectious agents.
Oversee all infection-control aspects of construction activities.
Establish site-specific infection-control protocols for specialized areas.
Provide education about the infection-control impact of construction to staff and construction workers.
Ensure compliance with technical standards, contract provisions, and regulations.
Establish a mechanism to address and correct problems quickly.
Develop contingency plans for emergency response to power failures, water supply disruptions, and fires.
Provide a water-damage management plan (including drying protocols) for handling water intrusion from floods, leaks, and condensation.
Develop a plan for structural maintenance.

* ICU is intensive care unit.

Education of maintenance and construction workers, health-care staff caring for high-risk patients, and persons responsible for controlling indoor air quality heightens awareness that minimizing dust and moisture intrusion from construction sites into high-risk patient-care areas helps to maintain a safe environment.^{120, 250, 271, 275–278} Visual and printed educational materials should be provided in the language spoken by the workers. Staff and construction workers also need to be aware of the potentially catastrophic consequences of dust and moisture intrusion when an HVAC system or water system fails during construction or repair; action plans to deal quickly with these emergencies should be developed in advance and kept on file. Incorporation of specific standards into construction contracts may help to prevent departures from recommended practices as projects progress. Establishing specific lines of communication is important to address problems (e.g., dust control, indoor air quality, noise levels, and vibrations), resolve complaints, and keep projects moving toward completion. Health-care facility staff should develop a mechanism to monitor worker adherence to infection-control guidelines on a daily basis in and around the construction site for the duration of the project.

b. Preliminary Considerations

The three major topics to consider before initiating any construction or repair activity are as follows: a) design and function of the new structure or area, b) assessment of environmental risks for airborne disease and opportunities for prevention, and c) measures to contain dust and moisture during construction or repairs. A checklist of design and function considerations can help to ensure that a planned structure or area can be easily serviced and maintained for environmental infection control (Box 5).^{17, 250, 273, 275–277} Specifications for the construction, renovation, remodeling, and maintenance of health-care facilities are outlined in the AIA document, *Guidelines for Design and Construction of Hospitals and Health Care Facilities*.^{120, 275}

Box 5. Construction design and function considerations for environmental infection control

Location of sinks and dispensers for handwashing products and hand hygiene products
Types of faucets (e.g., aerated vs. non-aerated)
Air-handling systems engineered for optimal performance, easy maintenance, and repair
ACH and pressure differentials to accommodate special patient-care areas
Location of fixed sharps containers
Types of surface finishes (e.g., porous vs. non-porous)
Well-caulked walls with minimal seams
Location of adequate storage and supply areas
Appropriate location of medicine preparations areas (e.g., ≥ 3 ft. from a sink)
Appropriate location and type of ice machines (e.g., preferably ice dispensers rather than ice bins)
Appropriate materials for sinks and wall coverings
Appropriate traffic flow (e.g., no “dirty” movement through “clean” areas)
Isolation rooms with anterooms as appropriate
Appropriate flooring (e.g., seamless floors in dialysis units)
Sensible use carpeting (e.g., avoiding use of carpeting in special care areas or areas likely to become wet)*
Convenient location of soiled utility areas
Properly engineered areas for linen services and solid waste management
Location of main generator to minimize the risk of system failure from flooding or other emergency
Installation guidelines for sheetrock

* Use of carpet cleaning methods (e.g., “bonneting”) that disperse microorganisms into the air may increase the risk of airborne infection among at-risk patients, especially if they are in the vicinity of the cleaning activity.¹¹¹

Proactive strategies can help prevent environmentally mediated airborne infections in health-care facilities during demolition, construction, and renovation. The potential presence of dust and moisture and their contribution to health-care-associated infections must be critically evaluated early in the planning of any demolition, construction, renovation, and repairs.^{120, 250, 251, 273, 274, 276–279} Consideration must extend beyond dust generated by major projects to include dust that can become airborne if disturbed during routine maintenance and minor renovation activities (e.g., exposure of ceiling spaces for inspection; installation of conduits, cable, or sprinkler systems; rewiring; and structural repairs or replacement).^{273, 276, 277} Other projects that can compromise indoor air quality include construction and repair jobs that inadvertently allow substantial amounts of raw, unfiltered outdoor air to enter the facility (e.g., repair of elevators and elevator shafts) and activities that dampen any structure, area, or item made of porous materials or characterized by cracks and crevices (e.g., sink cabinets in need of repair, carpets, ceilings, floors, walls, vinyl wall coverings, upholstery, drapes, and countertops).^{18, 273, 277} Molds grow and proliferate on these surfaces when they become and remain wet.^{21, 120, 250, 266, 270, 272, 280} Scrubbable

materials are preferred for use in patient-care areas.

Containment measures for dust and/or moisture control are dictated by the location of the construction site. Outdoor demolition and construction require actions to keep dust and moisture out of the facility (e.g., sealing windows and vents and keeping doors closed or sealed). Containment of dust and moisture generated from construction inside a facility requires barrier structures (either pre-fabricated or constructed of more durable materials as needed) and engineering controls to clean the air in and around the construction or repair site.

c. Infection-Control Risk Assessment

An infection-control risk assessment (ICRA) conducted before initiating repairs, demolition, construction, or renovation activities can identify potential exposures of susceptible patients to dust and moisture and determine the need for dust and moisture containment measures. This assessment centers on the type and extent of the construction or repairs in the work area but may also need to include adjacent patient-care areas, supply storage, and areas on levels above and below the proposed project. An example of designing an ICRA as a matrix, the policy for performing an ICRA and implementing its results, and a sample permit form that streamlines the communication process are available.²⁸¹ Knowledge of the air flow patterns and pressure differentials helps minimize or eliminate the inadvertent dispersion of dust that could contaminate air space, patient-care items, and surfaces.^{57, 282, 283} A recent aspergillosis outbreak among oncology patients was attributed to depressurization of the building housing the HSCT unit while construction was underway in an adjacent building. Pressure readings in the affected building (including 12 of 25 HSCT-patient rooms) ranged from 0.1 Pa–5.8 Pa. Unfiltered outdoor air flowed into the building through doors and windows, exposing patients in the HSCT unit to fungal spores.²⁸³ During long-term projects, providing temporary essential services (e.g., toilet facilities) and conveniences (e.g., vending machines) to construction workers within the site will help to minimize traffic in and out of the area. The type of barrier systems necessary for the scope of the project must be defined.^{12, 120, 250, 279, 284}

Depending on the location and extent of the construction, patients may need to be relocated to other areas in the facility not affected by construction dust.^{51, 285} Such relocation might be especially prudent when construction takes place within units housing immunocompromised patients (e.g., severely neutropenic patients and patients on corticosteroid therapy). Advance assessment of high-risk locations and planning for the possible transport of patients to other departments can minimize delays and waiting time in hallways.⁵¹ Although hospitals have provided immunocompromised patients with some form of respiratory protection for use outside their rooms, the issue is complex and remains unresolved until more research can be done. Previous guidance on this issue has been inconsistent.⁹ Protective respirators (i.e., N95) were well tolerated by patients when used to prevent further cases of construction-related aspergillosis in a recent outbreak.²⁸³ The routine use of the N95 respirator by patients, however, has not been evaluated for preventing exposure to fungal spores during periods of non-construction. Although health-care workers who would be using the N95 respirator for personal respiratory protection must be fit-tested, there is no indication that either patients or visitors should undergo fit-testing.

Surveillance activities should augment preventive strategies during construction projects.^{3, 4, 20, 110, 286, 287} By determining baseline levels of health-care-acquired airborne and waterborne infections, infection-control staff can monitor changes in infection rates and patterns during and immediately after construction, renovations, or repairs.³

d. Air Sampling

Air sampling in health-care facilities may be conducted both during periods of construction and on a periodic basis to determine indoor air quality, efficacy of dust-control measures, or air-handling system performance via parametric monitoring. Parametric monitoring consists of measuring the physical

performance of the HVAC system in accordance with the system manufacturer's specifications. A periodic assessment of the system (e.g., air flow direction and pressure, ACH, and filter efficiency) can give assurance of proper ventilation, especially for special care areas and operating rooms.²⁸⁸

Air sampling is used to detect aerosols (i.e., particles or microorganisms). Particulate sampling (i.e., total numbers and size range of particulates) is a practical method for evaluating the infection-control performance of the HVAC system, with an emphasis on filter efficiency in removing respirable particles (<5 μm in diameter) or larger particles from the air. Particle size is reported in terms of the mass median aerodynamic diameter (MMAD), whereas count median aerodynamic diameter (CMAD) is useful with respect to particle concentrations.

Particle counts in a given air space within the health-care facility should be evaluated against counts obtained in a comparison area. Particle counts indoors are commonly compared with the particulate levels of the outdoor air. This approach determines the "rank order" air quality from "dirty" (i.e., the outdoor air) to "clean" (i.e., air filtered through high-efficiency filters [90%–95% filtration]) to "cleanest" (i.e., HEPA-filtered air).²⁸⁸ Comparisons from one indoor area to another may also provide useful information about the magnitude of an indoor air-quality problem. Making rank-order comparisons between clean, highly-filtered areas and dirty areas and/or outdoors is one way to interpret sampling results in the absence of air quality and action level standards.^{35, 289}

In addition to verifying filter performance, particle counts can help determine if barriers and efforts to control dust dispersion from construction are effective. This type of monitoring is helpful when performed at various times and barrier perimeter locations during the project. Gaps or breaks in the barriers' joints or seals can then be identified and repaired. The American Conference of Governmental Industrial Hygienists (ACGIH) has set a threshold limit value-time weighted average (TLV®-TWA) of 10 mg/m^3 for nuisance dust that contains no asbestos and <1% crystalline silica.²⁹⁰ Alternatively, OSHA has set permissible exposure limits (PELs) for inert or nuisance dust as follows: respirable fraction at 5 mg/m^3 and total dust at 15 mg/m^3 .²⁹¹ Although these standards are not measures of a bioaerosol, they are used for indoor air quality assessment in occupational settings and may be useful criteria in construction areas. Application of ACGIH guidance to health-care settings has not been standardized, but particulate counts in health-care facilities are likely to be well below this threshold value and approaching clean-room standards in certain care areas (e.g., operating rooms).¹⁰⁰

Particle counters and anemometers are used in particulate evaluation. The anemometer measures air flow velocity, which can be used to determine sample volumes. Particulate sampling usually does not require microbiology laboratory services for the reporting of results.

Microbiologic sampling of air in health-care facilities remains controversial because of currently unresolved technical limitations and the need for substantial laboratory support (Box 6). Infection-control professionals, laboratorians, and engineers should determine if microbiologic and/or particle sampling is warranted and assess proposed methods for sampling. The most significant technical limitation of air sampling for airborne fungal agents is the lack of standards linking fungal spore levels with infection rates. Despite this limitation, several health-care institutions have opted to use microbiologic sampling when construction projects are anticipated and/or underway in efforts to assess the safety of the environment for immunocompromised patients.^{35, 289} Microbiologic air sampling should be limited to assays for airborne fungi; of those, the thermotolerant fungi (i.e., those capable of growing at 95°F–98.6°F [35°C–37°C]) are of particular concern because of their pathogenicity in immunocompromised hosts.³⁵ Use of selective media (e.g., Sabouraud dextrose agar and inhibitory mold agar) helps with the initial identification of recovered organisms.

Microbiologic sampling for fungal spores performed as part of various airborne disease outbreak

investigations has also been problematic.^{18, 49, 106, 111, 112, 289} The precise source of a fungus is often difficult to trace with certainty, and sampling conducted after exposure may neither reflect the circumstances that were linked to infection nor distinguish between health-care–acquired and community-acquired infections. Because fungal strains may fluctuate rapidly in the environment, health-care–acquired *Aspergillus* spp. infection cannot be confirmed or excluded if the infecting strain is not found in the health-care setting.²⁸⁷ Sensitive molecular typing methods (e.g., randomly amplified polymorphic DNA (RAPD) techniques and a more recent DNA fingerprinting technique that detects restriction fragment length polymorphisms in fungal genomic DNA) to identify strain differences among *Aspergillus* spp., however, are becoming increasingly used in epidemiologic investigations of health-care–acquired fungal infection (A. Streifel, University of Minnesota, 2000).^{68, 110, 286, 287, 292–296} During case cluster evaluation, microbiologic sampling may provide an isolate from the environment for molecular typing and comparison with patient isolates. Therefore, it may be prudent for the clinical laboratory to save *Aspergillus* spp. isolated from colonizations and invasive disease cases among patients in PE, oncology, and transplant services for these purposes.

Box 6. Unresolved issues associated with microbiologic air sampling*

Lack of standards linking fungal spore levels with infection rates (i.e., no safe level of exposure)
Lack of standard protocols for testing (e.g., sampling intervals, number of samples, sampling locations)
Need for substantial laboratory support
Culture issues (e.g., false negatives, insensitivity, lag time between sampling and recording the results)
New, complex polymerase chain reaction (PCR) analytical methods
Unknown incubation period for *Aspergillus* spp. infection
Variability of sampler readings
Sensitivity of the sampler used (i.e., the volumes of air sampled)
Lack of details in the literature about describing sampling circumstances (e.g., unoccupied rooms vs. ongoing activities in rooms, expected fungal concentrations, and rate of outdoor air penetration)
Lack of correlation between fungal species and strains from the environment and clinical specimens
Confounding variables with high-risk patients (e.g., visitors and time spent outside of protective environment [PE] without respiratory protection)
Need for determination of ideal temperature for incubating fungal cultures (95°F [35°C] is the most commonly used temperature)

* Material in this box is compiled from references 35, 100, 222, 289, and 297.

Sedimentation methods using settle plates and volumetric sampling methods using solid impactors are commonly employed when sampling air for bacteria and fungi. Settle plates have been used by numerous investigators to detect airborne bacteria or to measure air quality during medical procedures (e.g., surgery).^{17, 60, 97, 151, 161, 287} Settle plates, because they rely on gravity during sampling, tend to select for larger particles and lack sensitivity for respirable particles (e.g., individual fungal spores), especially in highly-filtered environments. Therefore, they are considered impractical for general use.^{35, 289, 298–301} Settle plates, however, may detect fungi aerosolized during medical procedures (e.g., during wound dressing changes), as described in a recent outbreak of aspergillosis among liver transplant patients.³⁰²

The use of slit or sieve impactor samplers capable of collecting large volumes of air in short periods of time are needed to detect low numbers of fungal spores in highly filtered areas.^{35, 289} In some

outbreaks, aspergillosis cases have occurred when fungal spore concentrations in PE ambient air ranged as low as 0.9–2.2 colony-forming units per cubic meter (CFU/m³) of air.^{18, 94} On the basis of the expected spore counts in the ambient air and the performance parameters of various types of volumetric air samplers, investigators of a recent aspergillosis outbreak have suggested that an air volume of at least 1000 L (1 m³) should be considered when sampling highly filtered areas.²⁸³ Investigators have also suggested limits of 15 CFU/m³ for gross colony counts of fungal organisms and <0.1 CFU/m³ for *Aspergillus fumigatus* and other potentially opportunistic fungi in heavily filtered areas (≥12 ACH and filtration of ≥99.97% efficiency).¹²⁰ No correlation of these values with the incidence of health-care-associated fungal infection rates has been reported.

Air sampling in health-care facilities, whether used to monitor air quality during construction, to verify filter efficiency, or to commission new space prior to occupancy, requires careful notation of the circumstances of sampling. Most air sampling is performed under undisturbed conditions. However, when the air is sampled during or after human activity (e.g., walking and vacuuming), a higher number of airborne microorganisms likely is detected.²⁹⁷ The contribution of human activity to the significance of air sampling and its impact on health-care-associated infection rates remain to be defined. Comparing microbiologic sampling results from a target area (e.g., an area of construction) to those from an unaffected location in the facility can provide information about distribution and concentration of potential airborne pathogens. A comparison of microbial species densities in outdoor air versus indoor air has been used to help pinpoint fungal spore bursts. Fungal spore densities in outdoor air are variable, although the degree of variation with the seasons appears to be more dramatic in the United States than in Europe.^{92, 287, 303}

Particulate and microbiologic air sampling have been used when commissioning new HVAC system installations; however, such sampling is particularly important for newly constructed or renovated PE or operating rooms. Particulate sampling is used as part of a battery of tests to determine if a new HVAC system is performing to specifications for filtration and the proper number of ACH.^{268, 288, 304} Microbiologic air sampling, however, remains controversial in this application, because no standards for comparison purposes have been determined. If performed, sampling should be limited to determining the density of fungal spores per unit volume of air space. High numbers of spores may indicate contamination of air-handling system components prior to installation or a system deficiency when culture results are compared with known filter efficiencies and rates of air exchange.

e. External Demolition and Construction

External demolition, planned building implosions, and dirt excavation generate considerable dust and debris that can contain airborne microorganisms. In one study, peak concentrations in outdoor air at grade level and HVAC intakes during site excavation averaged 20,000 CFU/m³ for all fungi and 500 CFU/m³ for *Aspergillus fumigatus*, compared with 19 CFU/m³ and 4 CFU/m³, respectively, in the absence of construction.²⁸⁰ Many health-care institutions are located in large, urban areas; building implosions are becoming a more frequent concern. Infection-control risk assessment teams, particularly those in facilities located in urban renewal areas, would benefit by developing risk management strategies for external demolition and construction as a standing policy. In light of the events of 11 September 2001, it may be necessary for the team to identify those dust exclusion measures that can be implemented rapidly in response to emergency situations (Table 8). Issues to be reviewed prior to demolition include a) proximity of the air intake system to the work site, b) adequacy of window seals and door seals, c) proximity of areas frequented by immunocompromised patients, and d) location of the underground utilities (D. Erickson, ASHE, 2000).^{120, 250, 273, 276, 277, 280, 305}

Table 8. Strategies to reduce dust and moisture intrusion during external demolition and construction

<i>Item</i>	<i>Recommendation</i>
Demolition site	<ul style="list-style-type: none"> ● Shroud the site if possible to reduce environmental contamination.
Dust-generating equipment	<ul style="list-style-type: none"> ● Prior to placing dust-generating equipment, evaluate the location to ensure that dust produced by the equipment will not enter the building through open doorways or windows, or through ventilation air intakes.
Construction materials storage	<ul style="list-style-type: none"> ● Locate this storage away from the facility and ventilation air intakes.
Adjacent air intakes HVAC system	<ul style="list-style-type: none"> ● Seal off affected intakes, if possible, or move if funds permit. ● Consult with the facility engineer about pressure differentials and air recirculation options; keep facility air pressure positive to outside air.
Filters	<ul style="list-style-type: none"> ● Ensure that filters are properly installed; change roughing filters frequently to prevent dust build-up on high-efficiency filters.
Windows	<ul style="list-style-type: none"> ● Seal and caulk to prevent entry of airborne fungal spores.
Doors	<ul style="list-style-type: none"> ● Keep closed as much as possible; do not prop open; seal and caulk unused doors (i.e., those that are not designated as emergency exits); use mats with tacky surfaces at outside entrances.
Water utilities	<ul style="list-style-type: none"> ● Note location relative to construction area to prevent intrusion of dust into water systems.*
Medical gas piping	<ul style="list-style-type: none"> ● Ensure that these lines/pipes are insulated during periods of vibration.
Rooftops	<ul style="list-style-type: none"> ● Temporarily close off during active demolition/construction those rooftop areas that are normally open to the public (e.g., rooftop atrium).
Dust generation	<ul style="list-style-type: none"> ● Provide methods (e.g., misting the area with water) to minimize dust.
Immunocompromised patients	<ul style="list-style-type: none"> ● Use walk-ways protected from demolition/construction sites; avoid outside areas close to these sites; avoid rooftops.
Pedestrian traffic	<ul style="list-style-type: none"> ● Close off entry ways as needed to minimize dust intrusion.
Truck traffic	<ul style="list-style-type: none"> ● Reroute if possible, or arrange for frequent street cleaning.
Education and awareness+	<ul style="list-style-type: none"> ● Encourage reporting of hazardous or unsafe incidents associated with construction.

* Contamination of water pipes during demolition activities has been associated with health-care-associated transmission of *Legionella* spp.³⁰⁵

+ When health-care facilities have immunosuppressed patients in their census, telephoning the city building department each month to find out if buildings are scheduled for demolition is prudent.

Minimizing the entry of outside dust into the HVAC system is crucial in reducing the risk for airborne contaminants. Facility engineers should be consulted about the potential impact of shutting down the system or increasing the filtration. Selected air handlers, especially those located close to excavation sites, may have to be shut off temporarily to keep from overloading the system with dust and debris. Care is needed to avoid significant facility-wide reductions in pressure differentials that may cause the building to become negatively pressured relative to the outside. To prevent excessive particulate overload and subsequent reductions in effectiveness of intake air systems that cannot be shut off temporarily, air filters must be inspected frequently for proper installation and function. Excessive dust

penetration can be avoided if recirculated air is maximally utilized while outdoor air intakes are shut down. Scheduling demolition and excavation during the winter, when *Aspergillus* spp. spores may be present in lower numbers, can help, although seasonal variations in spore density differ around the world.^{92, 287, 303} Dust control can be managed by misting the dirt and debris during heavy dust-generating activities. To decrease the amount of aerosols from excavation and demolition projects, nearby windows, especially in areas housing immunocompromised patients, can be sealed and window and door frames caulked or weather-stripped to prevent dust intrusion.^{50, 301, 306} Monitoring for adherence to these control measures throughout demolition or excavation is crucial. Diverting pedestrian traffic away from the construction sites decreases the amount of dust tracked back into the health-care facility and minimizes exposure of high-risk patients to environmental pathogens. Additionally, closing entrances near construction or demolition sites might be beneficial; if this is not practical, creating an air lock (i.e., pressurizing the entry way) is another option.

f. Internal Demolition, Construction, Renovations, and Repairs

The focus of a properly implemented infection-control program during interior construction and repairs is containment of dust and moisture. This objective is achieved by a) educating construction workers about the importance of control measures; b) preparing the site; c) notifying and issuing advisories for staff, patients, and visitors; d) moving staff and patients and relocating patients as needed; e) issuing standards of practice and precautions during activities and maintenance; f) monitoring for adherence to control measures during construction and providing prompt feedback about lapses in control; g) monitoring HVAC performance; h) implementing daily clean-up, terminal cleaning and removal of debris upon completion; and i) ensuring the integrity of the water system during and after construction. These activities should be coordinated with engineering staff and infection-control professionals.

Physical barriers capable of containing smoke and dust will confine dispersed fungal spores to the construction zone.^{279, 284, 307, 308} The specific type of physical barrier required depends on the project's scope and duration and on local fire codes. Short-term projects that result in minimal dust dispersion (e.g., installation of new cables or wiring above ceiling tiles) require only portable plastic enclosures with negative pressure and HEPA filtration of the exhaust air from the enclosed work area. The placement of a portable industrial-grade HEPA filter device capable of filtration rate of 300–800 ft³/min. adjacent to the work area will help to remove fungal spores, but its efficacy is dependent on the supplied ACH and size of the area. If the project is extensive but short-term, dust-abatement, fire-resistant plastic curtains (e.g., Visqueen®) may be adequate. These should be completely airtight and sealed from ceiling to floor with overlapping curtains;^{276, 277, 309} holes, tears, or other perforations should be repaired promptly with tape. A portable, industrial-grade HEPA filter unit on continuous operation is needed within the contained area, with the filtered air exhausted to the outside of the work zone. Patients should not remain in the room when dust-generating activities are performed. Tools to assist the decision-making process regarding selection of barriers based on an ICRA approach are available.²⁸¹

More elaborate barriers are indicated for long-term projects that generate moderate to large amounts of dust. These barrier structures typically consist of rigid, noncombustible walls constructed from sheet rock, drywall, plywood, or plaster board and covered with sheet plastic (e.g., Visqueen®). Barrier requirements to prevent the intrusion of dust into patient-care areas include a) installing a plastic dust abatement curtain before construction of the rigid barrier; b) sealing and taping all joint edges including the top and bottom; c) extending the barrier from floor to floor, which takes into account the space [approximately 2–8 ft.] above the finished, lay-down ceiling; and d) fitting or sealing any temporary doors connecting the construction zone to the adjacent area. (See Box 7 for a list of the various construction and repair activities that require the use of some type of barrier.)

Box 7. Construction/repair projects that require barrier structures*

Demolition of walls, wallboard, plaster, ceramic tiles, ceiling tiles, and ceilings
Removal of flooring and carpeting, windows and doors, and casework
Working with sinks and plumbing that could result in aerosolization of water in high-risk areas
Exposure of ceiling spaces for demolition and for installation or rerouting of utility services (e.g., rewiring, electrical conduction installation, HVAC ductwork, and piping)
Crawling into ceiling spaces for inspection in a manner that may dislodge dust
Demolition, repair, or construction of elevator shafts
Repairing water damage

* Material for this box was compiled from references 120, 250, 273, 276, and 277.

Dust and moisture abatement and control rely primarily on the impermeable barrier containment approach; as construction continues, numerous opportunities can lead to dispersion of dust to other areas of the health-care facility. Infection-control measures that augment the use of barrier containment should be undertaken (Table 9).

Dust-control measures for clinical laboratories are an essential part of the infection-control strategy during hospital construction or renovation. Use of plastic or solid barriers may be needed if the ICRA determines that air flow from construction areas may introduce airborne contaminants into the laboratory space. In one facility, pseudofungemia clusters attributed to *Aspergillus* spp. and *Penicillium* spp. were linked to improper air flow patterns and construction projects adjacent to the laboratory; intrusion of dust and spores into a biological safety cabinet from construction activity immediately next to the cabinet resulted in a cluster of cultures contaminated with *Aspergillus niger*.^{310, 311} Reportedly, no barrier containment was used and the HEPA filtration system was overloaded with dust. In addition, an outbreak of pseudobacteremia caused by *Bacillus* spp. occurred in another hospital during construction above a storage area for blood culture bottles.²⁰⁷ Airborne spread of *Bacillus* spp. spores resulted in contamination of the bottles' plastic lids, which were not disinfected or handled with proper aseptic technique prior to collection of blood samples.

Table 9. Infection-control measures for internal construction and repair projects*+

Infection-control measure	Steps for implementation
Prepare for the project.	<ol style="list-style-type: none"> 1. Use a multi-disciplinary team approach to incorporate infection control into the project. 2. Conduct the risk assessment and a preliminary walk-through with project managers and staff.
Educate staff and construction workers.	<ol style="list-style-type: none"> 1. Educate staff and construction workers about the importance of adhering to infection-control measures during the project. 2. Provide educational materials in the language of the workers. 3. Include language in the construction contract requiring construction workers and subcontractors to participate in infection-control training.
Issue hazard and warning notices.	<ol style="list-style-type: none"> 1. Post signs to identify construction areas and potential hazards. 2. Mark detours requiring pedestrians to avoid the work area.
Relocate high-risk patients as needed, especially if the construction is in or adjacent to a PE area.	<ol style="list-style-type: none"> 1. Identify target patient populations for relocation based on the risk assessment. 2. Arrange for the transfer in advance to avoid delays. 3. At-risk patients should wear protective respiratory equipment (e.g., a high-efficiency mask) when outside their PE rooms.
Establish alternative traffic patterns for staff, patients, visitors, and construction workers.	<ol style="list-style-type: none"> 1. Determine appropriate alternate routes from the risk assessment. 2. Designate areas (e.g., hallways, elevators, and entrances/exits) for construction-worker use. 3. Do not transport patients on the same elevator with construction materials and debris.

Infection-control measure	Steps for implementation
Erect appropriate barrier containment.	<ol style="list-style-type: none"> 1. Use prefabricated plastic units or plastic sheeting for short-term projects that will generate minimal dust. 2. Use durable rigid barriers for ongoing, long-term projects.
Establish proper ventilation.	<ol style="list-style-type: none"> 1. Shut off return air vents in the construction zone, if possible, and seal around grilles. 2. Exhaust air and dust to the outside, if possible. 3. If recirculated air from the construction zone is unavoidable, use a pre-filter and a HEPA filter before the air returns to the HVAC system. 4. When vibration-related work is being done that may dislodge dust in the ventilation system or when modifications are made to ductwork serving occupied spaces, install filters on the supply air grilles temporarily. 5. Set pressure differentials so that the contained work area is under negative pressure. 6. Use air flow monitoring devices to verify the direction of the air pattern. 7. Exhaust air and dust to the outside, if possible. 8. Monitor temperature, air changes per hour (ACH), and humidity levels (humidity levels should be <65%). 9. Use portable, industrial grade HEPA filters in the adjacent area and/or the construction zone for additional ACH. 10. Keep windows closed, if possible.
Control solid debris.	<ol style="list-style-type: none"> 1. When replacing filters, place the old filter in a bag prior to transport and dispose as a routine solid waste. 2. Clean the construction zone daily or more often as needed. 3. Designate a removal route for small quantities of solid debris. 4. Mist debris and cover disposal carts before transport (i.e., leaving the construction zone). 5. Designate an elevator for construction crew use. 6. Use window chutes and negative pressure equipment for removal of larger pieces of debris while maintaining pressure differentials in the construction zone. 7. Schedule debris removal to periods when patient exposures to dust is minimal.
Control water damage.	<ol style="list-style-type: none"> 1. Make provisions for dry storage of building materials. 2. Do not install wet, porous building materials (i.e., sheet rock). 3. Replace water-damaged porous building materials if they cannot be completely dried out within 72 hours.
Control dust in air and on surfaces.	<ol style="list-style-type: none"> 1. Monitor the construction area daily for compliance with the infection-control plan. 2. Protective outer clothing for construction workers should be removed before entering clean areas. 3. Use mats with tacky surfaces within the construction zone at the entry; cover sufficient area so that both feet make contact with the mat while walking through the entry. 4. Construct an anteroom as needed where coveralls can be donned and removed. 5. Clean the construction zone and all areas used by construction workers with a wet mop. 6. If the area is carpeted, vacuum daily with a HEPA-filtered–equipped vacuum. 7. Provide temporary essential services (e.g., toilets) and worker conveniences (e.g., vending machines) in the construction zone as appropriate. 8. Damp-wipe tools if removed from the construction zone or left in the area. 9. Ensure that construction barriers remain well sealed; use particle sampling as needed. 10. Ensure that the clinical laboratory is free from dust contamination.

Infection-control measure	Steps for implementation
Complete the project.	<ol style="list-style-type: none"> 1. Flush the main water system to clear dust-contaminated lines. 2. Terminally clean the construction zone before the construction barriers are removed. 3. Check for visible mold and mildew and eliminate (i.e., decontaminate and remove), if present. 4. Verify appropriate ventilation parameters for the new area as needed. 5. Do not accept ventilation deficiencies, especially in special care areas. 6. Clean or replace HVAC filters using proper dust-containment procedures. 7. Remove the barriers and clean the area of any dust generated during this work. 8. Ensure that the designated air balances in the operating rooms (OR) and protective environments (PE) are achieved before occupancy. 9. Commission the space as indicated, especially in the OR and PE, ensuring that the room's required engineering specifications are met.

* Material in this table includes information from D. Erickson, ASHE, 2000.

+ Material in this table was compiled from references 19, 51, 67, 80, 106, 120, 250, 266, 273, 276–278, 280, 285, and 309, 312–315.

5. Environmental Infection-Control Measures for Special Health-Care Settings

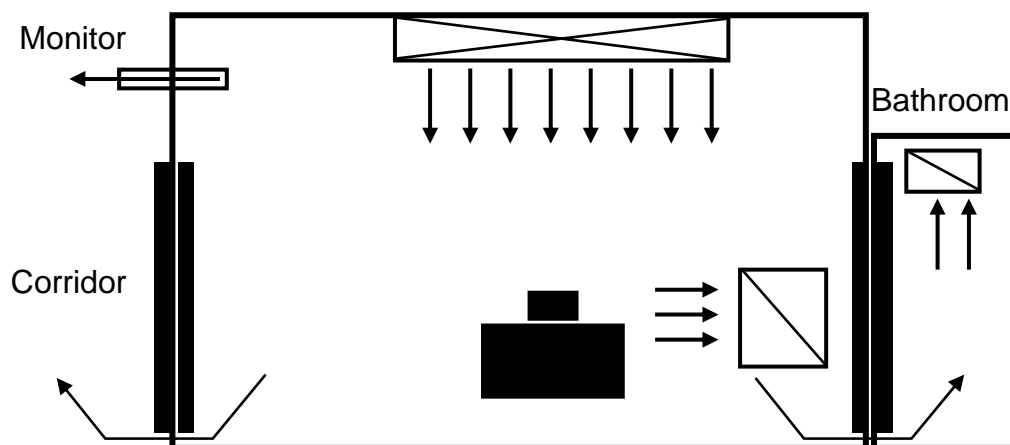
Areas in health-care facilities that require special ventilation include a) operating rooms; b) PE rooms used by high-risk, immunocompromised patients; and c) AII rooms for isolation of patients with airborne infections (e.g., those caused by *M. tuberculosis*, VZV, or measles virus). The number of rooms required for PE and AII are determined by a risk assessment of the health-care facility.⁶ Continuous, visual monitoring of air flow direction is required for new or renovated pressurized rooms.^{120, 256}

a. Protective Environments (PE)

Although the exact configuration and specifications of PEs might differ among hospitals, these care areas for high-risk, immunocompromised patients are designed to minimize fungal spore counts in air by maintaining a) filtration of incoming air by using central or point-of-use HEPA filters; b) directed room air flow [i.e., from supply on one side of the room, across the patient, and out through the exhaust on the opposite side of the room]; c) positive room air pressure of 2.5 Pa [0.01" water gauge] relative to the corridor; d) well-sealed rooms; and e) ≥ 12 ACH.^{44, 120, 251, 254, 316–319} Air flow rates must be adjusted accordingly to ensure sufficient ACH, and these rates vary depending on certain factors (e.g., room air leakage area). For example, to provide ≥ 12 ACH in a typical patient room with 0.5 sq. ft. air leakage, the air flow rate will be minimally 125 cubic feet/min (cfm).^{320, 321} Higher air flow rates may be needed. A general ventilation diagram for a positive-pressure room is given in Figure 2. Directed room air flow in PE rooms is not laminar; parallel air streams are not generated. Studies attempting to demonstrate patient benefit from laminar air flow in a PE setting are equivocal.^{316, 318, 319, 322–327}

Air flow direction at the entrances to these areas should be maintained and verified, preferably on a daily basis, using either a visual means of indication (e.g., smoke tubes and flutter strips) or manometers. Permanent installation of a visual monitoring device is indicated for new PE construction and renovation.¹²⁰ Facility service structures can interfere with the proper unidirectional air flow from the patients' rooms to the adjacent corridor. In one outbreak investigation, *Aspergillus* spp. infections in a critical care unit may have been associated with a pneumatic specimen transport system, a textile disposal duct system, and central vacuum lines for housekeeping, all of which disrupted proper air flow from the patients' rooms to the outside and allowed entry of fungal spores into the unit (M.McNeil, CDC, 2000).

Figure 2. Example of positive-pressure room control for protection from airborne environmental microbes (PE)* + §



* Stacked black boxes represent patient's bed. Long open box with cross-hatch represents supply air. Open boxes with single, diagonal slashes represent air exhaust registers. Arrows indicate directions of air flow.

+ Possible uses include immunocompromised patient rooms (e.g., hematopoietic stem cell transplant or solid organ transplant procedure rooms) and orthopedic operating rooms.

§ Positive-pressure room engineering features include

- positive pressure (greater supply than exhaust air volume);
- pressure differential range of 2.5–8 Pa (0.01–0.03-in. water gauge), ideal at 8 Pa;
- air flow volume differential >125-cfm supply versus exhaust;
- sealed room, approximately 0.5-sq. ft. leakage;
- clean to dirty air flow;
- monitoring;
- ≥ 12 air changes per hour (ACH); and
- return air if refiltered.

¶ This diagram is a generic illustration of air flow in a typical installation. Alternative air flow arrangements are recognized. Adapted and used with permission from A. Streifel and the publisher of reference 328 (Penton Media, Inc.)

The use of surface fungicide treatments is becoming more common, especially for building materials.³²⁹ Copper-based compounds have demonstrated anti-fungal activity and are often applied to wood or paint. Copper-8-quinolinolate was used on environmental surfaces contaminated with *Aspergillus* spp. to control one reported outbreak of aspergillosis.³¹⁰ The compound was also incorporated into the fireproofing material of a newly constructed hospital to help decrease the environmental spore burden.³¹⁶

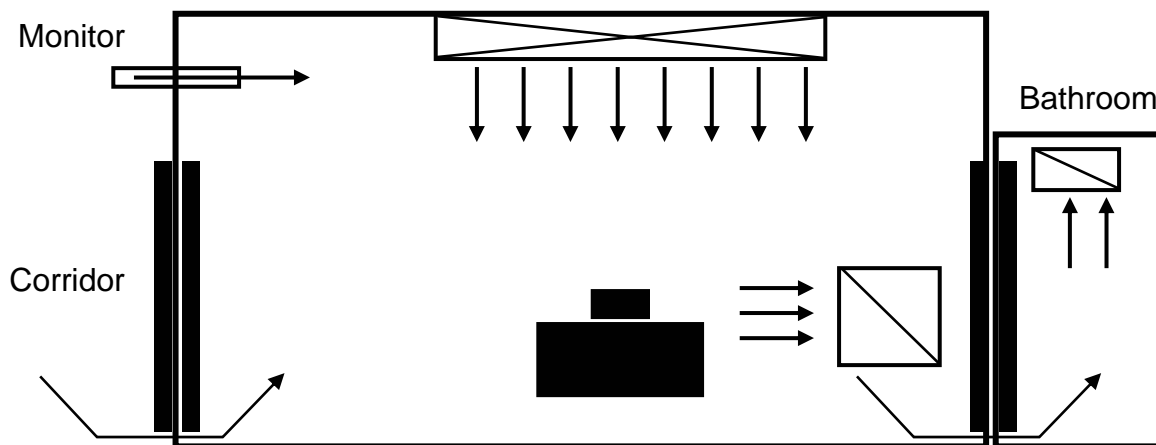
b. Airborne Infection Isolation (AII)

Acute-care inpatient facilities need at least one room equipped to house patients with airborne infectious disease. Every health-care facility, including ambulatory and long-term care facilities, should undertake an ICRA to identify the need for AII areas. Once the need is established, the appropriate ventilation equipment can be identified. Air handling systems for this purpose need not be restricted to central systems. Guidelines for the prevention of health-care-acquired TB have been published in response to multiple reports of health-care-associated transmission of multi-drug resistant strains.^{4, 330} In reports documenting health-care-acquired TB, investigators have noted a failure to comply fully with prevention measures in established guidelines.³³¹⁻³⁴⁵ These gaps highlight the importance of prompt recognition of the disease, isolation of patients, proper treatment, and engineering controls. AII rooms

are also appropriate for the care and management of smallpox patients.⁶ Environmental infection control with respect to smallpox is currently being revisited (see Appendix E).

Salient features of engineering controls for AII areas include a) use of negative pressure rooms with close monitoring of air flow direction using manometers or temporary or installed visual indicators [e.g., smoke tubes and flutter strips] placed in the room with the door closed; b) minimum 6 ACH for existing facilities, ≥ 12 ACH for areas under renovation or for new construction; and c) air from negative pressure rooms and treatment rooms exhausted directly to the outside if possible.^{4, 120, 248} As with PE, airflow rates need to be determined to ensure the proper numbers of ACH.^{320, 321} AII rooms can be constructed either with (Figure 3) or without (Figure 4) an anteroom. When the recirculation of air from AII rooms is unavoidable, HEPA filters should be installed in the exhaust duct leading from the room to the general ventilation system. In addition to UVGI fixtures in the room, UVGI can be placed in the ducts as an adjunct measure to HEPA filtration, but it can not replace the HEPA filter.^{4, 346} A UVGI fixture placed in the upper room, coupled with a minimum of 6 ACH, also provides adequate air cleaning.²⁴⁸

Figure 3. Example of negative-pressure room control for airborne infection isolation (AII)* + §¶



* Stacked black boxes represent patient's bed. Long open box with cross-hatch represents supply air. Open boxes with single, diagonal slashes represent air exhaust registers. Arrows indicate direction of air flow.

+ Possible uses include treatment or procedure rooms, bronchoscopy rooms, and autopsy.

§ Negative-pressure room engineering features include

- negative pressure (greater exhaust than supply air volume);
- pressure differential of 2.5 Pa (0.01-in. water gauge);
- air flow volume differential >125 -cfm exhaust versus supply;
- sealed room, approximately 0.5-sq. ft. leakage;
- clean to dirty air flow;
- monitoring;
- ≥ 12 air changes per hour (ACH) new or renovation, 6 ACH existing; and
- exhaust to outside or HEPA-filtered if recirculated.

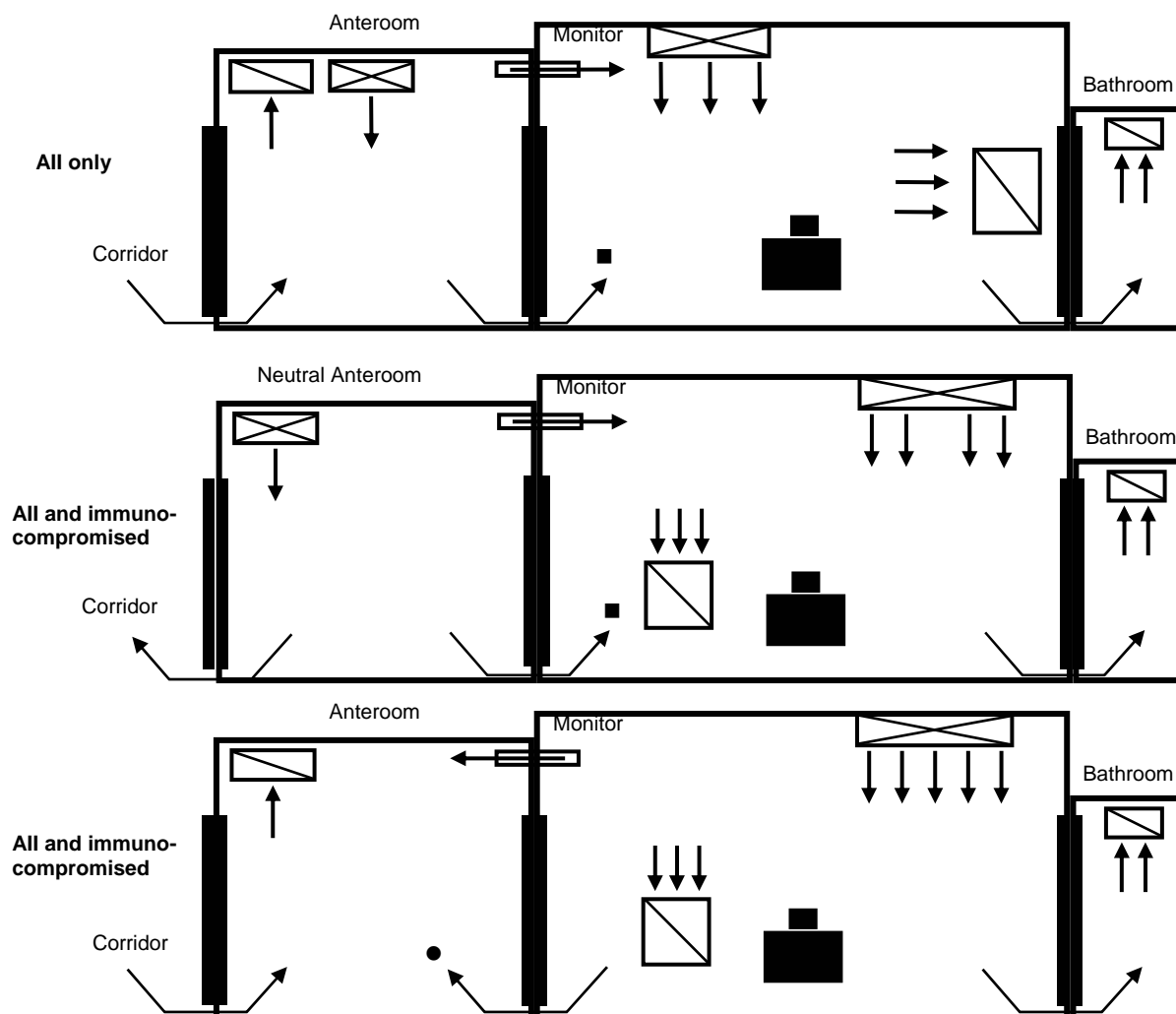
¶ This diagram is a generic illustration of air flow in a typical installation. Alternative air flow arrangements are recognized. Adapted and used with permission from A. Streifel and the publisher of reference 328 (Penton Media, Inc.)

One of the components of airborne infection isolation is respiratory protection for health-care workers and visitors when entering AII rooms.^{4, 6, 347} Recommendations of the type of respiratory protection are dependent on the patient's airborne infection (indicating the need for AII) and the risk of infection to

persons entering the AII room. A more in-depth discussion of respiratory protection in this instance is presented in the current isolation guideline;⁶ a revision of this guideline is in development. Cough-inducing procedures (e.g., endotracheal intubation and suctioning of known or suspected TB patients, diagnostic sputum induction, aerosol treatments, and bronchoscopy) require similar precautions.^{348–350}

Additional engineering measures are necessary for the management of patients requiring PE (i.e., allogeneic HSCT patients) who concurrently have airborne infection. For this type of patient treatment, an anteroom (Figure 4) is required in new construction and renovation as per AIA guidelines.¹²⁰

Figure 4. Example of airborne infection isolation (AII) room with anteroom and neutral anteroom* + §



* The top diagram indicates air flow patterns when patient with only airborne infectious disease occupies room. Middle and bottom diagrams indicate recommended air flow patterns when room is occupied by immunocompromised patient with airborne infectious disease. Stacked black boxes represent patient beds. Long open boxes with cross-hatches represent supply air. Open boxes with single, diagonal slashes represent air exhaust registers. Arrows indicate directions of air flow.

+ AII isolation room with anteroom engineering features include

- pressure differential of 2.5 Pa (0.01-in. water gauge) measured at the door between patient room and anteroom;
- air flow volume differential >125-cfm, depending on anteroom air flow direction (pressurized versus depressurized);

- sealed room with approximately 0.5-sq. ft. leakage;
- clean to dirty air flow
- monitoring;
- ≥ 12 air changes per hour (ACH) new or renovation, 6 ACH existing; and
- anteroom air flow patterns. The small ■ in panels 1 and 2 indicate the anteroom is pressurized (supply versus exhaust), while the small • in panel 3 indicates the anteroom is depressurized (exhaust versus supply).

§ Used with permission of A. Streifel, University of Minnesota

The pressure differential of an anteroom can be positive or negative relative to the patient in the room.¹²⁰ An anteroom can act as an airlock (Figure 4). If the anteroom is positive relative to the air space in the patient's room, staff members do not have to mask prior to entry into the anteroom if air is directly exhausted to the outside and a minimum of 10 ACH (Figure 4, top diagram).¹²⁰ When an anteroom is negative relative to both the AII room and the corridor, health-care workers must mask prior to entering the anteroom (Figure 4, bottom diagram). If an AII room with an anteroom is not available, use of a portable, industrial-grade HEPA filter unit may help to increase the number of ACHs while facilitating the removal of fungal spores; however, a fresh air source must be present to achieve the proper air exchange rate. Incoming ambient air should receive HEPA filtration.

c. Operating Rooms

Operating room air may contain microorganisms, dust, aerosol, lint, skin squamous epithelial cells, and respiratory droplets. The microbial level in operating room air is directly proportional to the number of people moving in the room.³⁵¹ One study documented lower infection rates with coagulase-negative staphylococci among patients when operating room traffic during the surgical procedure was limited.³⁵² Therefore, efforts should be made to minimize personnel traffic during operations. Outbreaks of SSIs caused by group A beta-hemolytic streptococci have been traced to airborne transmission from colonized operating-room personnel to patients.^{150–154} Several potential health-care-associated pathogens (e.g., *Staphylococcus aureus* and *Staphylococcus epidermidis*) and drug-resistant organisms have also been recovered from areas adjacent to the surgical field,³⁵³ but the extent to which the presence of bacteria near the surgical field influences the development of postoperative SSIs is not clear.³⁵⁴

Proper ventilation, humidity (<68%), and temperature control in the operating room is important for the comfort of surgical personnel and patients, but also in preventing environmental conditions that encourage growth and transmission of microorganisms.³⁵⁵ Operating rooms should be maintained at positive pressure with respect to corridors and adjacent areas.³⁵⁶ Operating rooms typically do not have a variable air handling system. Variable air handling systems are permitted for use in operating rooms only if they continue to provide a positive pressure with respect to the corridors and adjacent areas and the proper ACHs are maintained when the room is occupied. Conventional operating-room ventilation systems produce a minimum of about 15 ACH of filtered air for thermal control, three (20%) of which must be fresh air.^{120, 357, 358} Air should be introduced at the ceiling and exhausted near the floor.^{357, 359}

Laminar airflow and UVGI have been suggested as adjunct measures to reduce SSI risk for certain operations. Laminar airflow is designed to move particle-free air over the aseptic operating field at a uniform velocity (0.3–0.5 m/sec), sweeping away particles in its path. This air flow can be directed vertically or horizontally, and recirculated air is passed through a HEPA filter.^{360–363} Neither laminar airflow nor UV light, however, has been conclusively shown to decrease overall SSI risk.^{356, 364–370}

Elective surgery on infectious TB patients should be postponed until such patients have received adequate drug therapy. The use of general anesthesia in TB patients poses infection-control challenges because intubation can induce coughing, and the anesthesia breathing circuit apparatus potentially can become contaminated.³⁷¹ Although operating room suites at 15 ACH exceed the air exchanges required

for TB isolation, the positive air flow relative to the corridor could result in health-care-associated transmission of TB to operating-room personnel. If feasible, intubation and extubation of the TB surgical patient should be performed in AII. AIA currently does not recommend changing pressure from positive to negative or setting it to neutral; most facilities lack the capability to do so.¹²⁰ When emergency surgery is indicated for a suspected/diagnosed infectious TB patient, taking specific infection-control measures is prudent (Box 8).

Box 8. Strategy for managing TB patients and preventing airborne transmission in operating rooms*

1. If emergency surgery is indicated for a patient with active TB, schedule the TB patient as the last surgical case to provide maximum time for adequate ACH.
2. Operating room personnel should use NIOSH-approved N95 respirators without exhalation valves.³⁴⁷
3. Keep the operating room door closed after the patient is intubated, and allow adequate time for sufficient ACH to remove 99% of airborne particles (Appendix B, Table B.1.):
 - a) after the patient is intubated and particularly if intubation produces coughing;
 - b) if the door to the operating suite must be opened, and intubation induces coughing in the patient; or
 - c) after the patient is extubated and suctioned [unless a closed suctioning system is present].
4. Extubate the patient in the operating room or allow the patient to recover in AII rather than in the regular open recovery facilities.
5. Temporary use of a portable, industrial grade HEPA filter may expedite removal of airborne contaminants (fresh-air exchange requirements for proper ventilation must still be met).+
6. Breathing circuit filters with 0.1–0.2 μm pore size can be used as an adjunct infection-control measure.^{373, 374}

* Material in this table was compiled from references 4, 347, and 372–374.

+ The placement of portable HEPA filter units in the operating room must be carefully evaluated for potential disruptions in normal air flow. The portable unit should be turned off while the surgical procedure is underway and turned on following extubation. Portable HEPA filter units previously placed in construction areas may be used in subsequent patient care, provided that all internal and external surfaces are cleaned and the filter's performance is verified with appropriate particle testing and is changed, if needed.

Table 10. Summary of ventilation specifications in selected areas of health-care facilities*

Specifications	AII room+	PE room	Critical care room§	Isolation anteroom	Operating room
Air pressure¶	Negative	Positive	Positive, negative, or neutral	Positive or negative	Positive
Room air changes	≥ 6 ACH (for existing rooms); ≥ 12 ACH (for renovation or new construction)	≥ 12 ACH	≥ 6 ACH	≥ 10 ACH	≥ 15 ACH
Sealed**	Yes	Yes	No	Yes	Yes
Filtration supply	90% (dust-spot ASHRAE 52.1 1992)	99.97%++	$\geq 90\%$	$\geq 90\%$	90%
Recirculation	No§§	Yes	Yes	No	Yes

* Material in this table is compiled from references 35 and 120.

+ Includes bronchoscopy suites.

§ Positive pressure and HEPA filters may be preferred in some rooms in intensive care units (ICUs) caring for large numbers of immunocompromised patients.

¶ Clean-to-dirty: negative to an infectious patient, positive away from an immunocompromised patient.

** Minimized infiltration for ventilation control; pertains to windows, closed doors, and surface joints.

++ Fungal spore filter at point of use (HEPA at 99.97% of 0.3 μm particles).

§§ Recirculated air may be used if the exhaust air is first processed through a HEPA filter.

¶¶ Table used with permission of the publisher of reference 35 (Lippincott Williams and Wilkins).

6. Other Aerosol Hazards in Health-Care Facilities

In addition to infectious bioaerosols, several crucial non-infectious, indoor air-quality issues must be addressed by health-care facilities. The presence of sensitizing and allergenic agents and irritants in the workplace (e.g., ethylene oxide, glutaraldehyde, formaldehyde, hexachlorophene, and latex allergens³⁷⁵) is increasing. Asthma and dermatologic and systemic reactions often result with exposure to these chemicals. Anesthetic gases and aerosolized medications (e.g., ribavirin, pentamidine, and aminoglycosides) represent some of the emerging potentially hazardous exposures to health-care workers. Containment of the aerosol at the source is the first level of engineering control, but personal protective equipment (e.g., masks, respirators, and glove liners) that distances the worker from the hazard also may be needed.

Laser plumes and surgical smoke represent another potential risk for health-care workers.^{376–378} Lasers transfer electromagnetic energy into tissues, resulting in the release of a heated plume that includes particles, gases, tissue debris, and offensive smells. One concern is that aerosolized infectious material in the laser plume might reach the nasal mucosa of surgeons and adjacent personnel. Although some viruses (i.e., varicella-zoster virus, pseudorabies virus, and herpes simplex virus) do not aerosolize efficiently,^{379, 380} other viruses and bacteria (e.g., human papilloma virus [HPV], HIV, coagulase-negative *Staphylococcus*, *Corynebacterium* spp., and *Neisseria* spp.) have been detected in laser plumes.^{381–387} The presence of an infectious agent in a laser plume may not, however, be sufficient to cause disease from airborne exposure, especially if the normal mode of transmission for the agent is not airborne. No evidence indicated that HIV or hepatitis B virus (HBV) has been transmitted via aerosolization and inhalation.³⁸⁸

Although continuing studies are needed to fully evaluate the risk of laser plumes to surgical personnel, the prevention measures in these other guidelines should be followed: a) NIOSH recommendations,³⁷⁸ b) the *Recommended Practices for Laser Safety in Practice Settings* developed by the Association of periOperative Registered Nurses [AORN],³⁸⁹ c) the assessments of ECRI,^{390–392} and d) the ANSI standard.³⁹³ These guidelines recommend the use of a) respirators (N95 or N100) or full face shields and masks,²⁶⁰ b) central wall-suction units with in-line filters to collect particulate matter from minimal plumes, and c) dedicated mechanical smoke exhaust systems with a high-efficiency filter to remove large amounts of laser plume. Although transmission of TB has occurred as a result of abscess management practices that lacked airborne particulate control measures and respiratory protection, use of a smoke evacuator or needle aspirator and a high degree of clinical awareness can help protect health-care workers when excising and draining an extrapulmonary TB abscess.¹³⁷

D. Water

1. Modes of Transmission of Waterborne Diseases

Moist environments and aqueous solutions in health-care settings have the potential to serve as reservoirs for waterborne microorganisms. Under favorable environmental circumstances (e.g., warm temperature and the presence of a source of nutrition), many bacterial and some protozoal microorganisms can either proliferate in active growth or remain for long periods in highly stable, environmentally resistant (yet infectious) forms. Modes of transmission for waterborne infections

include a) direct contact [e.g., that required for hydrotherapy]; b) ingestion of water [e.g., through consuming contaminated ice]; c) indirect-contact transmission [e.g., from an improperly reprocessed medical device];⁶ d) inhalation of aerosols dispersed from water sources;³ and e) aspiration of contaminated water. The first three modes of transmission are commonly associated with infections caused by gram-negative bacteria and nontuberculous mycobacteria (NTM). Aerosols generated from water sources contaminated with *Legionella* spp. often serve as the vehicle for introducing legionellae to the respiratory tract.³⁹⁴

2. Waterborne Infectious Diseases in Health-Care Facilities

a. Legionellosis

Legionellosis is a collective term describing infection produced by *Legionella* spp., whereas Legionnaires disease is a multi-system illness with pneumonia.³⁹⁵ The clinical and epidemiologic aspects of these diseases (Table 11) are discussed extensively in another guideline.³ Although Legionnaires disease is a respiratory infection, infection-control measures intended to prevent health-care-associated cases center on the quality of water—the principal reservoir for *Legionella* spp.

Table 11. Clinical and epidemiologic characteristics of legionellosis/Legionnaires disease

		References
Causative agent	<i>Legionella pneumophila</i> (90% of infections); <i>L. micdadei</i> , <i>L. bozemanii</i> , <i>L. dumoffii</i> , <i>L. longbeachii</i> , (14 additional species can cause infection in humans)	395–399
Mode of transmission	Aspiration of water, direct inhalation or water aerosols	3, 394–398, 400
Source of exposure	Exposure to environmental sources of <i>Legionella</i> spp. (i.e., water or water aerosols)	31, 33, 401–414
Clinical syndromes and diseases	Two distinct illnesses: a) Pontiac fever [a milder, influenza-like illness]; and b) progressive pneumonia that may be accompanied by cardiac, renal, and gastrointestinal involvement	3, 397–399, 415–422
Populations at greatest risk	Immunosuppressed patients (e.g., transplant patients, cancer patients, and patients receiving corticosteroid therapy); immunocompromised patients (e.g., surgical patients, patients with underlying chronic lung disease, and dialysis patients); elderly persons; and patients who smoke	395–397, 423–433
Occurrence	Proportion of community-acquired pneumonia caused by <i>Legionella</i> spp. ranges from 1%–5%; estimated annual incidence among the general population is 8,000–18,000 cases in the United States; the incidence of health-care-associated pneumonia (0%–14%) may be underestimated if appropriate laboratory diagnostic methods are unavailable.	396, 397, 434–444
Mortality rate	Mortality declined markedly during 1980–1998, from 34% to 12% for all cases; the mortality rate is higher among persons with health-care-associated pneumonia compared with the rate among community-acquired pneumonia patients (14% for health-care-associated pneumonia versus 10% for community-acquired pneumonia [1998 data]).	395–397, 445

Legionella spp. are commonly found in various natural and man-made aquatic environments^{446, 447} and can enter health-care facility water systems in low or undetectable numbers.^{448, 449} Cooling towers, evaporative condensers, heated potable water distribution systems, and locally-produced distilled water can provide environments for multiplication of legionellae.^{450–454} In several hospital outbreaks, patients have been infected through exposure to contaminated aerosols generated by cooling towers, showers, faucets, respiratory therapy equipment, and room-air humidifiers.^{401–410, 455} Factors that enhance

colonization and amplification of legionellae in man-made water environments include a) temperatures of 77°F–107.6°F [25°C–42°C],^{456–460} b) stagnation,⁴⁶¹ c) scale and sediment,⁴⁶² and d) presence of certain free-living aquatic amoebae that can support intracellular growth of legionellae.^{462, 463} The bacteria multiply within single-cell protozoa in the environment and within alveolar macrophages in humans.

b. Other Gram-Negative Bacterial Infections

Other gram-negative bacteria present in potable water also can cause health-care–associated infections. Clinically important, opportunistic organisms in tap water include *Pseudomonas aeruginosa*, *Pseudomonas* spp., *Burkholderia cepacia*, *Ralstonia pickettii*, *Stenotrophomonas maltophilia*, and *Sphingomonas* spp. (Tables 12 and 13). Immunocompromised patients are at greatest risk of developing infection. Medical conditions associated with these bacterial agents range from colonization of the respiratory and urinary tracts to deep, disseminated infections that can result in pneumonia and bloodstream bacteremia. Colonization by any of these organisms often precedes the development of infection. The use of tap water in medical care (e.g., in direct patient care, as a diluent for solutions, as a water source for medical instruments and equipment, and during the final stages of instrument disinfection) therefore presents a potential risk for exposure. Colonized patients also can serve as a source of contamination, particularly for moist environments of medical equipment (e.g., ventilators).

In addition to *Legionella* spp., *Pseudomonas aeruginosa* and *Pseudomonas* spp. are among the most clinically relevant, gram-negative, health-care–associated pathogens identified from water. These and other gram-negative, non-fermentative bacteria have minimal nutritional requirements (i.e., these organisms can grow in distilled water) and can tolerate a variety of physical conditions. These attributes are critical to the success of these organisms as health-care–associated pathogens. Measures to prevent the spread of these organisms and other waterborne, gram-negative bacteria include hand hygiene, glove use, barrier precautions, and eliminating potentially contaminated environmental reservoirs.^{464, 465}

Table 12. *Pseudomonas aeruginosa* infections in health-care facilities

		References
Clinical syndromes and diseases	Septicemia, pneumonia (particularly ventilator-associated), chronic respiratory infections among cystic fibrosis patients, urinary tract infections, skin and soft-tissue infections (e.g., tissue necrosis and hemorrhage), burn-wound infections, folliculitis, endocarditis, central nervous system infections (e.g., meningitis and abscess), eye infections, and bone and joint infections	466–503
Modes of transmission	Direct contact with water, aerosols; aspiration of water and inhalation of water aerosols; and indirect transfer from moist environmental surfaces via hands of health-care workers	28, 502–506
Environmental sources of pseudomonads in health-care settings	Potable (tap) water, distilled water, antiseptic solutions contaminated with tap water, sinks, hydrotherapy pools, whirlpools and whirlpool spas, water baths, lithotripsy therapy tanks, dialysis water, eyewash stations, flower vases, and endoscopes with residual moisture in the channels	28, 29, 466, 468, 507–520
Environmental sources of pseudomonads in the community	Fomites (e.g., drug injection equipment stored in contaminated water)	494, 495
Populations at greatest risk	Intensive care unit (ICU) patients (including neonatal ICU), transplant patients (organ and hematopoietic stem cell), neutropenic patients, burn therapy and hydrotherapy patients, patients with malignancies, cystic fibrosis patients, patients with underlying medical conditions, and dialysis patients	28, 466, 467, 472, 477, 493, 506–508, 511, 512, 521–526

Table 13. Other gram-negative bacteria associated with water and moist environments

Implicated contaminated environmental vehicle	References
<i>Burkholderia cepacia</i>	
Distilled water	527
Contaminated solutions and disinfectants	528, 529
Dialysis machines	527
Nebulizers	530–532
Water baths	533
Intrinsically-contaminated mouthwash*	534
Ventilator temperature probes	535
<i>Stenotrophomonas maltophilia, Sphingomonas spp.</i>	
Distilled water	536, 537
Contaminated solutions and disinfectants	529
Dialysis machines	527
Nebulizers	530–532
Water	538
Ventilator temperature probes	539
<i>Ralstonia pickettii</i>	
Fentanyl solutions	540
Chlorhexidine	541
Distilled water	541
Contaminated respiratory therapy solution	541, 542
<i>Serratia marcescens</i>	
Potable water	543
Contaminated antiseptics (i.e., benzalkonium chloride and chlorhexidine)	544–546
Contaminated disinfectants (i.e., quaternary ammonium compounds and glutaraldehyde)	547, 548
<i>Acinetobacter spp.</i>	
Medical equipment that collects moisture (e.g., mechanical ventilators, cool mist humidifiers, vaporizers, and mist tents)	549–556
Room humidifiers	553, 555
Environmental surfaces	557–564
<i>Enterobacter spp.</i>	
Humidifier water	565
Intravenous fluids	566–578
Unsterilized cotton swabs	573
Ventilators	565, 569
Rubber piping on a suctioning machine	565, 569
Blood gas analyzers	570

* This report describes intrinsic contamination (i.e., occurring during manufacture) prior to use by the health-care facility staff. All other entries reflect extrinsic sources of contamination.

Two additional gram-negative bacterial pathogens that can proliferate in moist environments are *Acinetobacter spp.* and *Enterobacter spp.*^{571, 572} Members of both genera are responsible for health-care-associated episodes of colonization, bloodstream infections, pneumonia, and urinary tract infections among medically compromised patients, especially those in ICUs and burn therapy units.^{566, 572–583} Infections caused by *Acinetobacter spp.* represent a significant clinical problem. Average infection rates are higher from July through October compared with rates from November through June.⁵⁸⁴ Mortality rates associated with *Acinetobacter* bacteremia are 17%–52%, and rates as high as 71% have been reported for pneumonia caused by infection with either *Acinetobacter spp.* or

Pseudomonas spp.^{574–576} Multi-drug resistance, especially in third generation cephalosporins for *Enterobacter* spp., contributes to increased morbidity and mortality.^{569, 572}

Patients and health-care workers contribute significantly to the environmental contamination of surfaces and equipment with *Acinetobacter* spp. and *Enterobacter* spp., especially in intensive care areas, because of the nature of the medical equipment (e.g., ventilators) and the moisture associated with this equipment.^{549, 571, 572, 585} Hand carriage and hand transfer are commonly associated with health-care-associated transmission of these organisms and for *S. marcescens*.⁵⁸⁶ *Enterobacter* spp. are primarily spread in this manner among patients by the hands of health-care workers.^{567, 587} *Acinetobacter* spp. have been isolated from the hands of 4%–33% of health-care workers in some studies,^{585–590} and transfer of an epidemic strain of *Acinetobacter* from patients' skin to health-care workers' hands has been demonstrated experimentally.⁵⁹¹ *Acinetobacter* infections and outbreaks have also been attributed to medical equipment and materials (e.g., ventilators, cool mist humidifiers, vaporizers, and mist tents) that may have contact with water of uncertain quality (e.g., rinsing a ventilator circuit in tap water).^{549–556} Strict adherence to hand hygiene helps prevent the spread of both *Acinetobacter* spp. and *Enterobacter* spp.^{577, 592}

Acinetobacter spp. have also been detected on dry environmental surfaces (e.g., bed rails, counters, sinks, bed cupboards, bedding, floors, telephones, and medical charts) in the vicinity of colonized or infected patients; such contamination is especially problematic for surfaces that are frequently touched.^{557–564} In two studies, the survival periods of *Acinetobacter baumannii* and *Acinetobacter calcoaceticus* on dry surfaces approximated that for *S. aureus* (e.g., 26–27 days).^{593, 594} Because *Acinetobacter* spp. may come from numerous sources at any given time, laboratory investigation of health-care-associated *Acinetobacter* infections should involve techniques to determine biotype, antibiotype, plasmid profile, and genomic fingerprinting (i.e., macrorestriction analysis) to accurately identify sources and modes of transmission of the organism(s).⁵⁹⁵

c. Infections and Pseudo-Infections Due to Nontuberculous Mycobacteria

NTM are acid-fast bacilli (AFB) commonly found in potable water. NTM include both saprophytic and opportunistic organisms. Many NTM are of low pathogenicity, and some measure of host impairment is necessary to enhance clinical disease.⁵⁹⁶ The four most common forms of human disease associated with NTM are a) pulmonary disease in adults; b) cervical lymph node disease in children; c) skin, soft tissue, and bone infections; and d) disseminated disease in immunocompromised patients.^{596, 597} Person-to-person acquisition of NTM infection, especially among immunocompetent persons, does not appear to occur, and close contacts of patients are not readily infected, despite the high numbers of organisms harbored by such patients.^{596, 598–600} NTM are spread via all modes of transmission associated with water. In addition to health-care-associated outbreaks of clinical disease, NTM can colonize patients in health-care facilities through consumption of contaminated water or ice or through inhalation of aerosols.^{601–605} Colonization following NTM exposure, particularly of the respiratory tract, occurs when a patient's local defense mechanisms are impaired; overt clinical disease does not develop.⁶⁰⁶ Patients may have positive sputum cultures in the absence of clinical disease.

Using tap water during patient procedures and specimen collection and in the final steps of instrument reprocessing can result in pseudo-outbreaks of NTM contamination.^{607–609} NTM pseudo-outbreaks of *Mycobacterium chelonae*, *M. goodii*, and *M. xenopi* have been associated with both bronchoscopy and gastrointestinal endoscopy when a) tap water is used to provide irrigation to the site or to rinse off the viewing tip *in situ* or b) the instruments are inappropriately reprocessed with tap water in the final steps.^{610–612}

Table 14. Nontuberculous mycobacteria—environmental vehicles

Vehicles associated with infections or colonizations	References
<i>Mycobacterium abscessus</i>	
Inadequately sterilized medical instruments	613
<i>Mycobacterium avium</i> complex (MAC)	
Potable water	614–616
<i>Mycobacterium chelonae</i>	
Dialysis, reprocessed dialyzers	31, 32
Inadequately-sterilized medical instruments, jet injectors	617, 618
Contaminated solutions	619, 620
Hydrotherapy tanks	621
<i>Mycobacterium fortuitum</i>	
Aerosols from showers or other water sources	605, 606
Ice	602
Inadequately sterilized medical instruments	603
Hydrotherapy tanks	622
<i>Mycobacterium marinum</i>	
Hydrotherapy tanks	623
<i>Mycobacterium ulcerans</i>	
Potable water	624
Vehicles associated with pseudo-outbreaks	References
<i>Mycobacterium chelonae</i>	
Potable water used during bronchoscopy and instrument reprocessing	610
<i>Mycobacterium fortuitum</i>	
Ice	607
<i>Mycobacterium gordonae</i>	
Deionized water	611
Ice	603
Laboratory solution (intrinsically contaminated)	625
Potable water ingestion prior to sputum specimen collection	626
<i>Mycobacterium kansasii</i>	
Potable water	627
<i>Mycobacterium terrae</i>	
Potable water	608
<i>Mycobacterium xenopi</i>	
Potable water	609, 612, 627

NTM can be isolated from both natural and man-made environments. Numerous studies have identified various NTM in municipal water systems and in hospital water systems and storage tanks.^{615, 616, 624, 627–632} Some NTM species (e.g., *Mycobacterium xenopi*) can survive in water at 113°F (45°C), and can be isolated from hot water taps, which can pose a problem for hospitals that lower the temperature of their hot water systems.⁶²⁷ Other NTM (e.g., *Mycobacterium kansasii*, *M. gordonae*, *M. fortuitum*, and *M. chelonae*) cannot tolerate high temperatures and are associated more often with cold water lines and taps.⁶²⁹

NTM have a high resistance to chlorine; they can tolerate free chlorine concentrations of 0.05–0.2 mg/L (0.05–0.2 ppm) found at the tap.^{598, 633, 634} They are 20–100 times more resistant to chlorine compared with coliforms; slow-growing strains of NTM (e.g., *Mycobacterium avium* and *M. kansasii*) appear to be

more resistant to chlorine inactivation compared to fast-growing NTM.⁶³⁵ Slow-growing NTM species have also demonstrated some resistance to formaldehyde and glutaraldehyde, which has posed problems for reuse of hemodialyzers.³¹ The ability of NTM to form biofilms at fluid-surface interfaces (e.g., interior surfaces of water pipes) contributes to the organisms' resistance to chemical inactivation and provides a microenvironment for growth and proliferation.^{636, 637}

d. Cryptosporidiosis

Cryptosporidium parvum is a protozoan parasite that causes self-limiting gastroenteritis in normal hosts but can cause severe, life-threatening disease in immunocompromised patients. First recognized as a human pathogen in 1976, *C. parvum* can be present in natural and finished waters after fecal contamination from either human or animal sources.^{638–641}

The health risks associated with drinking potable water contaminated with minimal numbers of *C. parvum* oocysts are unknown.⁶⁴² It remains to be determined if immunosuppressed persons are more susceptible to lower doses of oocysts than are immunocompetent persons. One study demonstrated that a median 50% infectious dose (ID₅₀) of 132 oocysts of calf origin was sufficient to cause infection among healthy volunteers.⁶⁴³ In a second study, the same researchers found that oocysts obtained from infected foals (newborn horses) were infectious for human volunteers at median ID₅₀ of 10 oocysts, indicating that different strains or species of *Cryptosporidium* may vary in their infectivity for humans.⁶⁴⁴ In a small study population of 17 healthy adults with pre-existing antibody to *C. parvum*, the ID₅₀ was determined to be 1,880 oocysts, more than 20-fold higher than in seronegative persons.⁶⁴⁵ These data suggest that pre-existing immunity derived from previous exposures to *Cryptosporidium* offers some protection from infection and illness that ordinarily would result from exposure to low numbers of oocysts.^{645, 646}

Oocysts, particularly those with thick walls, are environmentally resistant, but their survival under natural water conditions is poorly understood. Under laboratory conditions, some oocysts remain viable and infectious in cold (41°F [5°C]) for months.⁶⁴¹ The prevalence of *Cryptosporidium* in the U.S. drinking water supply is notable. Two surveys of approximately 300 surface water supplies revealed that 55%–77% of the water samples contained *Cryptosporidium* oocysts.^{647, 648} Because the oocysts are highly resistant to common disinfectants (e.g., chlorine) used to treat drinking water, filtration of the water is important in reducing the risk of waterborne transmission. Coagulation-flocculation and sedimentation, when used with filtration, can collectively achieve approximately a 2.5 log₁₀ reduction in the number of oocysts.⁶⁴⁹ However, outbreaks have been associated with both filtered and unfiltered drinking water systems (e.g., the 1993 outbreak in Milwaukee, Wisconsin that affected 400,000 people).^{641, 650–652} The presence of oocysts in the water is not an absolute indicator that infection will occur when the water is consumed, nor does the absence of detectable oocysts guarantee that infection will not occur. Health-care-associated outbreaks of cryptosporidiosis primarily have been described among groups of elderly patients and immunocompromised persons.⁶⁵³

3. Water Systems in Health-Care Facilities

a. Basic Components and Point-of-Use Fixtures

Treated municipal water enters a health-care facility via the water mains and is distributed throughout the building(s) by a network of pipes constructed of galvanized iron, copper, and polyvinylchloride (PVC). The pipe runs should be as short as is practical. Where recirculation is employed, the pipe runs should be insulated and long dead legs avoided in efforts to minimize the potential for water stagnation, which favors the proliferation of *Legionella* spp. and NTM. In high-risk applications (e.g., PE areas for severely immunosuppressed patients), insulated recirculation loops should be incorporated as a design

feature. Recirculation loops prevent stagnation and insulation maintains return water temperature with minimal loss.

Each water service main, branch main, riser, and branch (to a group of fixtures) has a valve and a means to reach the valves via an access panel.¹²⁰ Each fixture has a stop valve. Valves permit the isolation of a portion of the water system within a facility during repairs or maintenance. Vacuum breakers and other similar devices in the lines prevent water from back-flowing into the system. All systems that supply water should be evaluated to determine risk for potential back siphonage and cross connections.

Health-care facilities generate hot water from municipal water using a boiler system. Hot water heaters and storage vessels for such systems should have a drainage facility at the lowest point, and the heating element should be located as close as possible to the bottom of the vessel to facilitate mixing and to prevent water temperature stratification. Those hot or cold water systems that incorporate an elevated holding tank should be inspected and cleaned annually. Lids should fit securely to exclude foreign materials.

The most common point-of-use fixtures for water in patient-care areas are sinks, faucets, aerators, showers, and toilets; eye-wash stations are found primarily in laboratories. The potential for these fixtures to serve as a reservoir for pathogenic microorganisms has long been recognized (Table 15).^{509, 654-656} Wet surfaces and the production of aerosols facilitate the multiplication and dispersion of microbes. The level of risk associated with aerosol production from point-of-use fixtures varies. Aerosols from shower heads and aerators have been linked to a limited number of clusters of gram-negative bacterial colonizations and infections, including Legionnaires disease, especially in areas where immunocompromised patients are present (e.g., surgical ICUs, transplant units, and oncology units).^{412, 415, 656-659} In one report, clinical infection was not evident among immunocompetent persons (e.g., hospital staff) who used hospital showers when *Legionella pneumophila* was present in the water system.⁶⁶⁰ Given the infrequency of reported outbreaks associated with faucet aerators, consensus has not been reached regarding the disinfection of or removal of these devices from general use. If additional clusters of infections or colonizations occur in high-risk patient-care areas, it may be prudent to clean and decontaminate the aerators or to remove them.^{658, 659} ASHRAE recommends cleaning and monthly disinfection of aerators in high-risk patient-care areas as part of *Legionella* control measures.⁶⁶¹ Although aerosols are produced with toilet flushing,^{662, 663} no epidemiologic evidence suggests that these aerosols pose a direct infection hazard.

Although not considered a standard point-of-use fixture, decorative fountains are being installed in increasing numbers in health-care facilities and other public buildings. Aerosols from a decorative fountain have been associated with transmission of *Legionella pneumophila* serogroup 1 infection to a small cluster of older adults.⁶⁶⁴ This hotel lobby fountain had been irregularly maintained, and water in the fountain may have been heated by submersed lighting, all of which favored the proliferation of *Legionella* in the system.⁶⁶⁴ Because of the potential for generations of infectious aerosols, a prudent prevention measure is to avoid locating these fixtures in or near high-risk patient-care areas and to adhere to written policies for routine fountain maintenance.¹²⁰

Table 15. Water and point-of-use fixtures as sources and reservoirs of waterborne pathogens*

Reservoir	Associated pathogens	Transmission	Strength of evidence+	Prevention and control	References
Potable water	<i>Pseudomonas</i> , gram-negative bacteria, NTM	Contact	Moderate	Follow public health guidelines.	(See Tables 12-14)

Reservoir	Associated pathogens	Transmission	Strength of evidence+	Prevention and control	References
Potable water	<i>Legionella</i>	Aerosol inhalation	Moderate	Provide supplemental treatment for water.	(See Table 11)
Holy water	Gram-negative bacteria	Contact	Low	Avoid contact with severe burn injuries. Minimize use among immunocompromised patients.	665
Dialysis water	Gram-negative bacteria	Contact	Moderate	Dialysate should be $\leq 2,000$ cfu/mL; water should be ≤ 200 cfu/mL.	2, 527, 666–668
Automated endoscope reprocessors and rinse water	Gram-negative bacteria	Contact	Moderate	Use and maintain equipment according to instructions; eliminate residual moisture by drying the channels (e.g., through alcohol rinse and forced air drying).	669–675
Water baths	<i>Pseudomonas</i> , <i>Burkholderia</i> , <i>Acinetobacter</i>	Contact	Moderate	Add germicide to the water; wrap transfusion products in protective plastic wrap if using the bath to modulate the temperature of these products.	29, 533, 676, 677
Tub immersion	<i>Pseudomonas</i> , <i>Enterobacter</i> , <i>Acinetobacter</i>	Contact	Moderate	Drain and disinfect tub after each use; consider adding germicide to the water; water in large hydrotherapy pools should be properly disinfected and filtered.	678–683
Ice and ice machines	NTM, <i>Enterobacter</i> , <i>Pseudomonas</i> , <i>Cryptosporidium</i>	Ingestion, contact	Moderate	Clean periodically; use automatic dispenser (avoid open chest storage compartments in patient areas).	601, 684–687
			Low		
Faucet aerators	<i>Legionella</i>	Aerosol inhalation	Moderate	Clean and disinfect monthly in high-risk patient areas; consider removing if additional infections occur.	415, 661
Faucet aerators	<i>Pseudomonas</i> , <i>Acinetobacter</i> , <i>Stenotrophomonas</i> , <i>Chryseobacterium</i>	Contact, droplet	Low	No precautions are necessary at present in immunocompetent patient-care areas.	658, 659, 688, 689
Sinks	<i>Pseudomonas</i>	Contact, droplet	Moderate	Use separate sinks for handwashing and disposal of contaminated fluids.	509, 653, 685–693
Showers	<i>Legionella</i>	Aerosol inhalation	Low	Provide sponge baths for hematopoietic stem cell transplant patients; avoid shower use for immunocompromised patients when <i>Legionella</i> is detected in facility water.	656

Reservoir	Associated pathogens	Transmission	Strength of evidence+	Prevention and control	References
Dental unit water lines	<i>Pseudomonas</i> , <i>Legionella</i> , <i>Sphingomonas</i> , <i>Acinetobacter</i>	Contact	Low	Clean water systems according to system manufacturer's instructions.	636, 694–696
Ice baths for thermodilution catheters	<i>Ewingella</i> , <i>Staphylococcus</i>	Contact	Low	Use sterile water.	697, 698
Decorative fountains	<i>Legionella</i>	Aerosol inhalation	Low	Perform regular maintenance, including water disinfection; avoid use in or near high-risk patient-care areas.	664
Eyewash stations	<i>Pseudomonas</i> , amoebae, <i>Legionella</i>	Contact	Low Minimum	Flush eyewash stations weekly; have sterile water available for eye flushes.	518, 699, 700
Toilets	Gram-negative bacteria	–	Minimum	Clean regularly; use good hand hygiene.	662
Flowers	Gram-negative bacteria, <i>Aspergillus</i>	–	Minimum	Avoid use in intensive care units and in immunocompromised patient-care settings.	515, 701, 702

* Modified from reference 654 and used with permission of the publisher (Slack, Inc.)

+ **Moderate:** occasional well-described outbreaks. **Low:** few well-described outbreaks. **Minimal:** actual infections not demonstrated.

b. Water Temperature and Pressure

Hot water temperature is usually measured at the point of use or at the point at which the water line enters equipment requiring hot water for proper operation.¹²⁰ Generally, the hot water temperature in hospital patient-care areas is no greater than a temperature within the range of 105°F–120°F (40.6°C–49°C), depending on the AIA guidance issued at the year in which the facility was built.¹²⁰ Hot water temperature in patient-care areas of skilled nursing-care facilities is set within a slightly lower range of 95°F–110°F (35°C–43.3°C) depending on the AIA guidance at the time of facility construction.¹²⁰ Many states have adopted a temperature setting in these ranges into their health-care regulations and building codes. ASHRAE, however, has recommended higher settings.⁶⁶¹ Steam jets or booster heaters are usually needed to meet the hot water temperature requirements in certain service areas of the hospital (e.g., the kitchen [120°F (49°C)] or the laundry [160°F (71°C)]).¹²⁰ Additionally, water lines may need to be heated to a particular temperature specified by manufacturers of specific hospital equipment. Hot-water distribution systems serving patient-care areas are generally operated under constant recirculation to provide continuous hot water at each hot-water outlet.¹²⁰ If a facility is or has a hemodialysis unit, then continuously circulated, cold treated water is provided to that unit.¹²⁰

To minimize the growth and persistence of gram-negative waterborne bacteria (e.g., thermophilic NTM and *Legionella* spp.),^{627, 703–709} cold water in health-care facilities should be stored and distributed at temperatures below 68°F (20°C); hot water should be stored above 140°F (60°C) and circulated with a minimum return temperature of 124°F (51°C),⁶⁶¹ or the highest temperature specified in state regulations and building codes. If the return temperature setting of 124°F (51°C) is permitted, then installation of preset thermostatic mixing valves near the point-of-use can help to prevent scalding. Valve maintenance is especially important in preventing valve failure, which can result in scalding. New shower systems in large buildings, hospitals, and nursing homes should be designed to permit mixing of hot and cold water near the shower head. The warm water section of pipe between the control valve and shower head should be self-draining. Where buildings can not be retrofitted, other

approaches to minimize the growth of *Legionella* spp. include a) periodically increasing the temperature to at least 150°F [66°C] at the point of use [i.e., faucets] and b) adding additional chlorine and flushing the water.^{661, 710, 711} Systems should be inspected annually to ensure that thermostats are functioning properly.

Adequate water pressure ensures sufficient water supplies for a) direct patient care; b) operation of water-cooled instruments and equipment [e.g., lasers, computer systems, telecommunications systems, and automated endoscope reprocessors⁷¹²]; c) proper function of vacuum suctioning systems; d) indoor climate control; and e) fire-protection systems. Maintaining adequate pressure also helps to ensure the integrity of the piping system.

c. Infection-Control Impact of Water System Maintenance and Repair

Corrective measures for water-system failures have not been studied in well-designed experiments; these measures are instead based on empiric engineering and infection-control principles. Health-care facilities can occasionally sustain both intentional cut-offs by the municipal water authority to permit new construction project tie-ins and unintentional disruptions in service when a water main breaks as a result of aging infrastructure or a construction accident. Vacuum breakers or other similar devices can prevent backflow of water in the facility's distribution system during water-disruption emergencies.¹¹ To be prepared for such an emergency, all health-care facilities need contingency plans that identify a) the total demand for potable water, b) the quantity of replacement water [e.g., bottled water] required for a minimum of 24 hours when the water system is down, c) mechanisms for emergency water distribution, and 4) procedures for correcting drops in water pressure that affect operation of essential devices and equipment that are driven or cooled by a water system [Table 16].⁷¹³

Table 16. Water demand in health-care facilities during water disruption emergencies

	Potable water	Bottled, sterile water
Water use needs	Drinking water Handwashing Cafeteria services Ice Manual flushing of toilets Patient baths, hygiene Hemodialysis Hydrotherapy Fire prevention (e.g., sprinkler systems) Surgery and critical care areas Laboratory services Laundry and central sterile services* Cooling towers+ Steam generation	Surgical scrub Emergency surgical procedures Pharmaceutical preparations Patient-care equipment (e.g., ventilators)§

* Arrange to have a contingency provision of these services from another resource, if possible (e.g., another health-care facility or contractor).

+ Some cooling towers may use a potable water source, but most units use non-potable water.

§ This item is included in the table under the assumption that electrical power is available during the water emergency.

Detailed, up-to-date plans for hot and cold water piping systems should be readily available for maintenance and repair purposes in case of system problems. Opening potable water systems for repair or construction and subjecting systems to water-pressure changes can result in water discoloration and dramatic increases in the concentrations of *Legionella* spp. and other gram-negative bacteria. The maintenance of a chlorine residual at all points within the piping system also offers some protection from entry of contamination to the pipes in the event of inadvertent cross-connection between potable and non-potable water lines. As a minimum preventive measure, ASHRAE recommends a thorough flushing of the system.⁶⁶¹ High-temperature flushing or hyperchlorination may also be appropriate

strategies to decrease potentially high concentrations of waterborne organisms. The decision to pursue either of these remediation strategies, however, should be made on a case-by-case basis. If only a portion of the system is involved, high temperature flushing or chlorination can be used on only that portion of the system.⁶⁶¹

When shock decontamination of hot water systems is necessary (e.g., after disruption caused by construction and after cross-connections), the hot water temperature should be raised to 160°F–170°F (71°C–77°C) and maintained at that level while each outlet around the system is progressively flushed. A minimum flush time of 5 minutes has been recommended;³ the optimal flush time is not known, however, and longer flush times may be necessary.⁷¹⁴ The number of outlets that can be flushed simultaneously depends on the capacity of the water heater and the flow capability of the system. Appropriate safety procedures to prevent scalding are essential. When possible, flushing should be performed when the fewest building occupants are present (e.g., during nights and weekends).

When thermal shock treatment is not possible, shock chlorination may serve as an alternative method.⁶⁶¹ Experience with this method of decontamination is limited, however, and high levels of free chlorine can corrode metals. Chlorine should be added, preferably overnight, to achieve a free chlorine residual of at least 2 mg/L (2 ppm) throughout the system.⁶⁶¹ This may require chlorination of the water heater or tank to levels of 20–50 mg/L (20–50 ppm). The pH of the water should be maintained at 7.0–8.0.⁶⁶¹ After completion of the decontamination, recolonization of the hot water system is likely to occur unless proper temperatures are maintained or a procedure such as continuous supplemental chlorination is continued.

Interruptions of the water supply and sewage spills are situations that require immediate recovery and remediation measures to ensure the health and safety of patients and staff.⁷¹⁵ When delivery of potable water through the municipal distribution system has been disrupted, the public water supplier must issue a “boil water” advisory if microbial contamination presents an immediate public health risk to customers. The hospital engineer should oversee the restoration of the water system in the facility and clear it for use when appropriate. Hospitals must maintain a high level of surveillance for waterborne disease among patients and staff after the advisory is lifted.⁶⁴²

Flooding from either external (e.g., from a hurricane) or internal sources (e.g., a water system break) usually results in property damage and a temporary loss of water and sanitation.^{716–718} JCAHO requires all hospitals to have plans that address facility response for recovery from both internal and external disasters.^{713, 719} The plans are required to discuss a) general emergency preparedness, b) staffing, c) regional planning among area hospitals, d) emergency supply of potable water, e) infection control and medical services needs, f) climate control, and g) remediation. The basic principles of structural recovery from flooding are similar to those for recovery from sewage contamination (Box 9 and 10). Following a major event (e.g., flooding), facilities may elect to conduct microbial sampling of water after the system is restored to verify that water quality has been returned to safe levels (<500 CFU/mL, heterotrophic plate count). This approach may help identify point-of-use fixtures that may harbor contamination as a result of design or engineering features.⁷²⁰ Medical records should be allowed to dry and then either photocopied or placed in plastic covers before returning them to the record.

Moisture meters can be used to assess water-damaged structural materials. If porous structural materials for walls have a moisture content of >20% after 72 hours, the affected material should be removed.^{266, 278, 313} The management of water-damaged structural materials is not strictly limited to major water catastrophes (e.g., flooding and sewage intrusions); the same principles are used to evaluate the damage from leaking roofs, point-of-use fixtures, and equipment. Additional sources of moisture include condensate on walls from boilers and poorly engineered humidification in HVAC systems.

Box 9. Recovery and remediation measures for water-related emergencies*

Potable water disruptions

Contingency plan items

- Ensure access to plumbing network so that repairs can be easily made.
- Provide sufficient potable water, either from bottled sources or truck delivery.
- Post advisory notices against consuming tap water, ice, or beverages made with water.
- Rope off or bag drinking fountains to designate these as being “out of service” until further notice.
- Rinse raw foods as needed in disinfected water.
- Disconnect ice machines whenever possible.+
- Postpone laundry services until after the water system is restored.

Water treatment

- Heat water to a rolling boil for ≥ 1 minute.

Remediation of the water system after the “boil water” advisory is rescinded

- Flush fixtures (e.g., faucets and drinking fountains) and equipment for several minutes and restart.
 - Run water softeners through a regeneration cycle.
 - Drain, disinfect, and refill water storage tanks, if needed.
 - Change pretreatment filters and disinfect the dialysis water system.
-

Sewage spills/malfunction

Overall strategy

- Move patients and clean/sterile supplies out of the area.
- Redirect traffic away from the area.
- Close the doors or use plastic sheeting to isolate the area prior to clean-up.
- Restore sewage system function first, then the potable water system (if both are malfunctioning).
- Remove sewage solids, drain the area, and let dry.

Remediation of the structure

- Hard surfaces: clean with detergent/disinfectant after the area has been drained.
- Carpeting, loose tiles, buckled flooring: remove and allow the support surface to dry; replace the items; wet down carpeting with a low-level disinfectant or sanitizer prior to removal to minimize dust dispersion to the air.
- Wallboard and other porous structural materials: remove and replace if they cannot be cleaned and dried within 72 hours.§

Furniture

- Hard surface furniture (e.g., metal or plastic furniture): clean and allow to dry.
- Wood furniture: let dry, sand the wood surface, and reapply varnish.
- Cloth furniture: replace.

Electrical equipment

- Replace if the item cannot be easily dismantled, cleaned, and reassembled.
-

* Material in this box is compiled from references 266, 278, 315, 713, 716–719, 721–729.

+ Ice machines should always be disconnected from the water source in advance of planned water disruptions.

§ Moisture meter readings should be <20% moisture content.

An exception to these recommendations is made for hemodialysis units where water is further treated either by portable water treatment or large-scale water treatment systems usually involving reverse osmosis (RO). In the United States, >97% of dialysis facilities use RO treatment for their water.⁷²¹ However, changing pre-treatment filters and disinfecting the system to prevent colonization of the RO membrane and microbial contamination down-stream of the pre-treatment filter are prudent measures.

Box 10. Contingency planning for flooding

General emergency preparedness

- Ensure that emergency electrical generators are not located in flood-prone areas of the facility.
- Develop alternative strategies for moving patients, water containers, medical records, equipment, and supplies in the event that the elevators are inoperable.
- Establish in advance a centralized base of operations with batteries, flashlights, and cellular phones.
- Ensure sufficient supplies of sandbags to place at the entrances and the area around boilers, incinerators, and generators.
- Establish alternative strategies for bringing core employees to the facility if high water prevents travel.

Staffing Patterns

- Temporarily reassign licensed staff as needed to critical care areas to provide manual ventilation and to perform vital assessments on patients.
- Designate a core group of employees to remain on site to keep all services operational if the facility remains open.
- Train all employees in emergency preparedness procedures.

Regional planning among are facilities for disaster management

- Incorporate community support and involvement (e.g., media alerts, news, and transportation).
- Develop in advance strategies for transferring patients, as needed.
- Develop strategies for sharing supplies and providing essential services among participating facilities (e.g., central sterile department services, and laundry services).
- Identify sources for emergency provisions (e.g., blood, emergency vehicles, and bottled water).

Medical services and infection control

- Use alcohol-based hand rubs in general patient-care areas.
- Postpone elective surgeries until full services are restored, or transfer these patients to other facilities.
- Consider using portable dialysis machines.+
- Provide an adequate supply of tetanus and hepatitis A immunizations for patients and staff.

Climate control

- Provide adequate water for cooling towers.§
-

* Material in this box was compiled from references 713, 716–719.

+ Portable dialysis machines require less water compared to the larger units situated in dialysis settings.

§ Water for cooling towers may need to be trucked in, especially if the tower uses a potable water source.

4. Strategies for Controlling Waterborne Microbial Contamination

a. Supplemental Treatment of Water with Heat and/or Chemicals

In addition to using supplemental treatment methods as remediation measures after inadvertent contamination of water systems, health-care facilities sometimes use special measures to control waterborne microorganisms on a sustained basis. This decision is most often associated with outbreaks of legionellosis and subsequent efforts to control legionellae,⁷²² although some facilities have tried supplemental measures to better control thermophilic NTM.⁶²⁷

The primary disinfectant for both cold and hot water systems is chlorine. However, chlorine residuals are expected to be low, and possibly nonexistent, in hot water tanks because of extended retention time in the tank and elevated water temperature. Flushing, especially that which removes sludge from the bottom of the tank, probably provides the most effective treatment of water systems. Unlike the situation for disinfecting cooling towers, no equivalent recommendations have been made for potable water systems, although specific intervention strategies have been published.^{403, 723} The principal approaches to disinfection of potable systems are heat flushing using temperatures 160°F–170°F (71°C–77°C), hyperchlorination, and physical cleaning of hot-water tanks.^{3, 403, 661} Potable systems are easily recolonized and may require continuous intervention (e.g., raising of hot water temperatures or continuous chlorination).^{403, 711} Chlorine solutions lose potency over time, thereby rendering the stocking of large quantities of chlorine impractical.

Some hospitals with hot water systems identified as the source of *Legionella* spp. have performed emergency decontamination of their systems by pulse (i.e., one-time) thermal disinfection/superheating or hyperchlorination.^{711, 714, 724, 725} After either of these procedures, hospitals either maintain their heated water with a minimum return temperature of 124°F (51°C) and cold water at <68°F (<20°C) or chlorinate their hot water to achieve 1–2 mg/L (1–2 ppm) of free residual chlorine at the tap.^{26, 437, 709–711, 726, 727} Additional measures (e.g., physical cleaning or replacement of hot-water storage tanks, water heaters, faucets, and shower heads) may be required to help eliminate accumulations of scale and sediment that protect organisms from the biocidal effects of heat and chlorine.^{457, 711} Alternative methods for controlling and eradicating legionellae in water systems (e.g., treating water with chlorine dioxide, heavy metal ions [i.e., copper/silver ions], ozone, and UV light) have limited the growth of legionellae under laboratory and operating conditions.^{728–742} Further studies on the long-term efficacy of these treatments are needed before these methods can be considered standard applications.

Renewed interest in the use of chloramines stems from concerns about adverse health effects associated with disinfectants and disinfection by-products.⁷⁴³ Monochloramine usage minimizes the formation of disinfection by-products, including trihalomethanes and haloacetic acids. Monochloramine can also reach distal points in a water system and can penetrate into bacterial biofilms more effectively than free chlorine.⁷⁴⁴ However, monochloramine use is limited to municipal water treatment plants and is currently not available to health-care facilities as a supplemental water-treatment approach. A recent study indicated that 90% of Legionnaires disease outbreaks associated with drinking water could have been prevented if monochloramine rather than free chlorine has been used for residual disinfection.⁷⁴⁵ In a retrospective comparison of health-care–associated Legionnaires disease incidence in central Texas hospitals, the same research group documented an absence of cases in facilities located in communities with monochloramine-treated municipal water.⁷⁴⁶ Additional data are needed regarding the effectiveness of using monochloramine before its routine use as a disinfectant in water systems can be recommended. No data have been published regarding the effectiveness of monochloramine installed at the level of the health-care facility.

Additional filtration of potable water systems is not routinely necessary. Filters are used in water lines in dialysis units, however, and may be inserted into the lines for specific equipment (e.g., endoscope washers and disinfectors) for the purpose of providing bacteria-free water for instrument reprocessing. Additionally, an RO unit is usually added to the distribution system leading to PE areas.

b. Primary Prevention of Legionnaires Disease (No Cases Identified)

The primary and secondary environmental infection-control strategies described in this section on the guideline pertain to health-care facilities without transplant units. Infection-control measures specific to PE or transplant units (i.e., patient-care areas housing patients at the highest risk for morbidity and mortality from *Legionella* spp. infection) are described in the subsection titled *Preventing Legionnaires Disease in Protective Environments*.

Health-care facilities use at least two general strategies to prevent health-care–associated legionellosis when no cases or only sporadic cases have been detected. The first is an environmental surveillance approach involving periodic culturing of water samples from the hospital’s potable water system to monitor for *Legionella* spp.^{747–750} If any sample is culture-positive, diagnostic testing is recommended for all patients with health-care–associated pneumonia.^{748, 749} In-house testing is recommended for facilities with transplant programs as part of a comprehensive treatment/management program. If $\geq 30\%$ of the samples are culture-positive for *Legionella* spp., decontamination of the facility’s potable water system is warranted.⁷⁴⁸ The premise for this approach is that no cases of health-care–associated legionellosis can occur if *Legionella* spp. are not present in the potable water system, and, conversely, cases of health-care–associated legionellosis could potentially occur if *Legionella* spp. are cultured from the water.^{26, 751} Physicians who are informed that the hospital’s potable water system is culture-positive

for *Legionella* spp. are more likely to order diagnostic tests for legionellosis.

A potential advantage of the environmental surveillance approach is that periodic culturing of water is less costly than routine laboratory diagnostic testing for all patients who have health-care-associated pneumonia. The primary argument against this approach is that, in the absence of cases, the relationship between water-culture results and legionellosis risk remains undefined.³ *Legionella* spp. can be present in the water systems of buildings⁷⁵² without being associated with known cases of disease.^{437, 707, 753} In a study of 84 hospitals in Québec, 68% of the water systems were found to be colonized with *Legionella* spp., and 26% were colonized at >30% of sites sampled; cases of Legionnaires disease, however, were infrequently reported from these hospitals.⁷⁰⁷

Other factors also argue against environmental surveillance. Interpretation of results from periodic water culturing might be confounded by differing results among the sites sampled in a single water system and by fluctuations in the concentration of *Legionella* spp. at the same site.^{709, 754} In addition, the risk for illness after exposure to a given source might be influenced by several factors other than the presence or concentration of organisms, including a) the degree to which contaminated water is aerosolized into respirable droplets, b) the proximity of the infectious aerosol to the potential host, c) the susceptibility of the host, and d) the virulence properties of the contaminating strain.^{755–757} Thus, data are insufficient to assign a level of disease risk even on the basis of the number of colony-forming units detected in samples from areas for immunocompetent patients. Conducting environmental surveillance would obligate hospital administrators to initiate water-decontamination programs if *Legionella* spp. are identified. Therefore, periodic monitoring of water from the hospital's potable water system and from aerosol-producing devices is not widely recommended in facilities that have not experienced cases of health-care-associated legionellosis.^{661, 758}

The second strategy to prevent and control health-care-associated legionellosis is a clinical approach, in which providers maintain a high index of suspicion for legionellosis and order appropriate diagnostic tests (i.e., culture, urine antigen, and direct fluorescent antibody [DFA] serology) for patients with health-care-associated pneumonia who are at high risk for legionellosis and its complications.^{437, 759, 760} The testing of autopsy specimens can be included in this strategy should a death resulting from health-care-associated pneumonia occur. Identification of one case of definite or two cases of possible health-care-associated Legionnaires disease should prompt an epidemiologic investigation for a hospital source of *Legionella* spp., which may involve culturing the facility's water for *Legionella*. Routine maintenance of cooling towers, and use of sterile water for the filling and terminal rinsing of nebulization devices and ventilation equipment can help to minimize potential sources of contamination. Circulating potable water temperatures should match those outlined in the subsection titled *Water Temperature and Pressure*, as permitted by state code.

c. Secondary prevention of Legionnaires Disease (With Identified Cases)

The indications for a full-scale environmental investigation to search for and subsequently decontaminate identified sources of *Legionella* spp. in health-care facilities without transplant units have not been clarified; these indications would likely differ depending on the facility. Case categories for health-care-associated Legionnaires disease in facilities without transplant units include definite cases (i.e., laboratory-confirmed cases of legionellosis that occur in patients who have been hospitalized continuously for ≥ 10 days before the onset of illness) and possible cases (i.e., laboratory-confirmed infections that occur 2–9 days after hospital admission).³ In settings in which as few as one to three health-care-associated cases are recognized over several months, intensified surveillance for Legionnaires disease has frequently identified numerous additional cases.^{405, 408, 432, 453, 739, 759, 760} This finding suggests the need for a low threshold for initiating an investigation after laboratory confirmation of cases of health-care-associated legionellosis. When developing a strategy for responding to such a finding, however, infection-control personnel should consider the level of risk for health-care-

associated acquisition of, and mortality from, *Legionella* spp. infection at their particular facility.

An epidemiologic investigation conducted to determine the source of *Legionella* spp. involves several important steps (Box 11). Laboratory assessment is crucial in supporting epidemiologic evidence of a link between human illness and a specific environmental source.⁷⁶¹ Strain determination from subtype analysis is most frequently used in these investigations.^{410, 762–764} Once the environmental source is established and confirmed with laboratory support, supplemental water treatment strategies can be initiated as appropriate.

Box 11. Steps in an epidemiologic investigation for legionellosis

Review medical and microbiologic records.

Initiate active surveillance to identify all recent or ongoing cases.

Develop a line listing of cases by time, place, and person.

Determine the type of epidemiologic investigation needed for assessing risk factors:

- Case-control study,
- Cohort study.

Gather and analyze epidemiologic information:

- Evaluate risk factors associated with potential environmental exposures (e.g., showers, cooling towers, and respiratory-therapy equipment).

Collect water samples:

- Sample environmental sources implicated by epidemiologic investigation,
- Sample other potential source of water aerosols.

Subtype strains of *Legionella* spp. cultured from both patients and environmental sources.

Review autopsy records and include autopsy specimens in diagnostic testing.

The decision to search for hospital environmental sources of *Legionella* spp. and the choice of procedures to eradicate such contamination are based on several considerations, as follows: a) the hospital's patient population; b) the cost of an environmental investigation and institution of control measures to eradicate *Legionella* spp. from the water supply,^{765–768} and c) the differential risk, based on host factors, for acquiring health-care-associated legionellosis and developing severe and fatal infection.

d. Preventing Legionnaires Disease in Protective Environments

This subsection outlines infection-control measures applicable to those health-care facilities providing care to severely immunocompromised patients. Indigenous microorganisms in the tap water of these facilities may pose problems for such patients. These measures are designed to prevent the generation of potentially infectious aerosols from water and the subsequent exposure of PE patients or other immunocompromised patients (e.g., transplant patients) (Table 17). Infection-control measures that address the use of water with medical equipment (e.g., ventilators, nebulizers, and equipment humidifiers) are described in other guidelines and publications.^{3, 455}

If one case of laboratory-confirmed, health-care-associated Legionnaires disease is identified in a patient in a solid-organ transplant program or in PE (i.e., an inpatient in PE for all or part of the 2–10 days prior to onset of illness) or if two or more laboratory-confirmed cases occur among patients who had visited an outpatient PE setting, the hospital should report the cases to the local and state health departments. The hospital should then initiate a thorough epidemiologic and environmental investigation to determine the likely environmental sources of *Legionella* spp.⁹ The source of *Legionella* should be decontaminated or removed. Isolated cases may be difficult to investigate. Because transplant recipients are at substantially higher risk for disease and death from legionellosis

compared with other hospitalized patients, periodic culturing for *Legionella* spp. in water samples from the potable water in the solid-organ transplant and/or PE unit can be performed as part of an overall strategy to prevent Legionnaires disease in PE units.^{9, 431, 710, 769} The optimal methodology (i.e., frequency and number of sites) for environmental surveillance cultures in PE units has not been determined, and the cost-effectiveness of this strategy has not been evaluated. Because transplant recipients are at high risk for Legionnaires disease and because no data are available to determine a safe concentration of legionellae organisms in potable water, the goal of environmental surveillance for *Legionella* spp. should be to maintain water systems with no detectable organisms.^{9, 431} Culturing for legionellae may be used to assess the effectiveness of water treatment or decontamination methods, a practice that provides benefits to both patients and health-care workers.^{767, 770}

Table 17. Additional infection-control measures to prevent exposure of high-risk patients to waterborne pathogens

Measures	References
<ul style="list-style-type: none"> • Restrict patients from taking showers if the water is contaminated with <i>Legionella</i> spp. • Use water that is not contaminated with <i>Legionella</i> spp. for patients' sponge baths. • Provide sterile water for drinking, tooth brushing, or for flushing nasogastric tubes. • Perform supplemental treatment of the water for the unit. • Consider periodic monitoring (i.e., culturing) of the unit water supply for <i>Legionella</i> spp. • Remove shower heads and faucet aerators monthly for cleaning.* • Use a 500–600 ppm (1:100 v/v dilution) solution of sodium hypochlorite to disinfect shower heads and faucet aerators.* • Do not use large-volume room air humidifiers that create aerosols unless these are subjected to cleaning and high-level disinfection daily and filled with distilled water. • Eliminate water-containing bath toys.+ 	<ul style="list-style-type: none"> • 407, 412, 654, 655, 658 • 9 • 9, 412 • 732 • 9, 431 • 661 • 661 • 3 • 30

* These measures can be considered in settings where legionellosis cases have occurred. These measures are not generally recommended in routine patient-care setting..

+ These items have been associated with outbreaks of *Pseudomonas*.

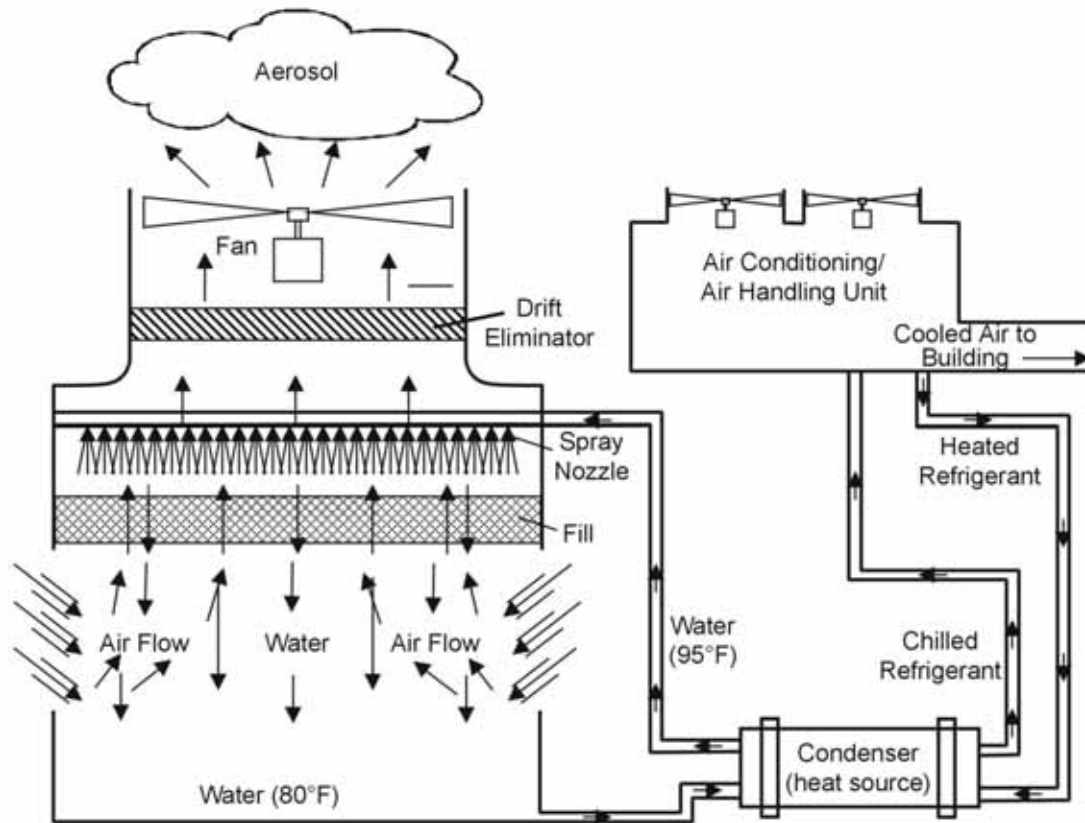
Protecting patient-care devices and instruments from inadvertent tap water contamination during room cleaning procedures is also important in any immunocompromised patient-care area. In a recent outbreak of gram-negative bacteremias among open-heart-surgery patients, pressure-monitoring equipment that was assembled and left uncovered overnight prior to the next day's surgeries was inadvertently contaminated with mists and splashing water from a hose-disinfectant system used for cleaning.⁷⁷¹

5. Cooling Towers and Evaporative Condensers

Modern health-care facilities maintain indoor climate control during warm weather by use of cooling towers (large facilities) or evaporative condensers (smaller buildings). A cooling tower is a wet-type, evaporative heat transfer device used to discharge to the atmosphere waste heat from a building's air conditioning condensers (Figure 5).^{772, 773} Warm water from air-conditioning condensers is piped to the cooling tower where it is sprayed downward into a counter- or cross-current air flow. To accelerate heat transfer to the air, the water passes over the fill, which either breaks water into droplets or causes it to spread into a thin film.^{772, 773} Most systems use fans to move air through the tower, although some large industrial cooling towers rely on natural draft circulation of air. The cooled water from the tower is piped back to the condenser, where it again picks up heat generated during the process of chilling the system's refrigerant. The water is cycled back to the cooling tower to be cooled. Closed-circuit cooling towers and evaporative condensers are also evaporative heat-transfer devices. In these systems, the

process fluid (e.g., water, ethylene glycol/water mixture, oil, or a condensing refrigerant) does not directly contact the cooling air, but is contained inside a coil assembly.⁶⁶¹

Figure 5. Diagram of a typical air conditioning (induced draft) cooling tower*



Water temperatures are approximate and may differ substantially according to system use and design. Warm water from the condenser (or chiller) is sprayed downward into a counter- or cross-current air flow. Water passes over the fill (a component of the system designed to increase the surface area of the water exposed to air), and heat from the water is transferred to the air. Some of the water becomes aerosolized during this process, although the volume of aerosol discharged to the air can be reduced by the placement of a drift eliminator. Water cooled in the tower returns to the heat source to cool refrigerant from the air conditioning unit.

* This figure is reprinted with permission of the publisher of reference 773 (Plenum Medical).

Cooling towers and evaporative condensers incorporate inertial stripping devices called drift eliminators to remove water droplets generated within the unit. Although the effectiveness of these eliminators varies substantially depending on design and condition, some water droplets in the size range of $<5 \mu\text{m}$ will likely leave the unit, and some larger droplets leaving the unit may be reduced to $\leq 5 \mu\text{m}$ by evaporation. Thus, even with proper operation, a cooling tower or evaporative condenser can generate and expel respirable water aerosols. If either the water in the unit's basin or the make-up water (added to replace water lost to evaporation) contains *Legionella* spp. or other waterborne microorganisms, these organisms can be aerosolized and dispersed from the unit.⁷⁷⁴ Clusters of both Legionnaires disease and Pontiac fever have been traced to exposure to infectious water aerosols originating from cooling towers and evaporative condensers contaminated with *Legionella* spp. Although most of these outbreaks have been community-acquired episodes of pneumonia,⁷⁷⁵⁻⁷⁸² health-care-associated Legionnaires disease

has been linked to cooling tower aerosol exposure.^{404, 405} Contaminated aerosols from cooling towers on hospital premises gained entry to the buildings either through open windows or via air handling system intakes located near the tower equipment.

Cooling towers and evaporative condensers provide ideal ecological niches for *Legionella* spp. The typical temperature of the water in cooling towers ranges from 85°F–95°F (29°C–35°C), although temperatures can be above 120°F (49°C) and below 70°F (21°C) depending on system heat load, ambient temperature, and operating strategy.⁶⁶¹ An Australian study of cooling towers found that legionellae colonized or multiplied in towers with basin temperatures above 60.8°F (16°C), and multiplication became explosive at temperatures above 73.4°F (23°C).⁷⁸³ Water temperature in closed-circuit cooling towers and evaporative condensers is similar to that in cooling towers. Considerable variation in the piping arrangement occurs. In addition, stagnant areas or dead legs may be difficult to clean or penetrate with biocides.

Several documents address the routine maintenance of cooling towers, evaporative condensers, and whirlpool spas.^{661, 784–787} They suggest following manufacturer's recommendations for cleaning and biocide treatment of these devices; all health-care facilities should ensure proper maintenance for their cooling towers and evaporative condensers, even in the absence of *Legionella* spp (Appendix C). Because cooling towers and evaporative condensers can be shut down during periods when air conditioning is not needed, this maintenance cleaning and treatment should be performed before starting up the system for the first time in the warm season.⁷⁸² Emergency decontamination protocols describing cleaning procedures and hyperchlorination for cooling towers have been developed for towers implicated in the transmission of legionellosis.^{786, 787}

6. Dialysis Water Quality and Dialysate

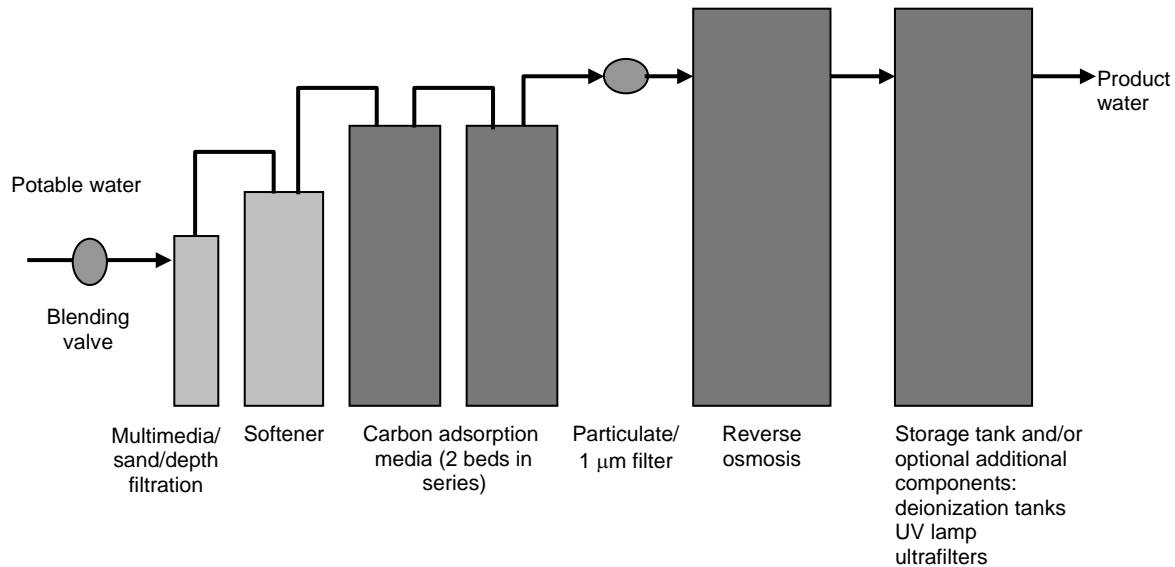
a. Rationale for Water Treatment in Hemodialysis

Hemodialysis, hemofiltration, and hemodiafiltration require special water-treatment processes to prevent adverse patient outcomes of dialysis therapy resulting from improper formulation of dialysate with water containing high levels of certain chemical or biological contaminants. The Association for the Advancement of Medical Instrumentation (AAMI) has established chemical and microbiologic standards for the water used to prepare dialysate, substitution fluid, or to reprocess hemodialyzers for renal replacement therapy.^{788–792} The AAMI standards address: a) equipment and processes used to purify water for the preparation of concentrates and dialysate and the reprocessing of dialyzers for multiple use and b) the devices used to store and distribute this water. Future revisions to these standards may include hemofiltration and hemodiafiltration.

Water treatment systems used in hemodialysis employ several physical and/or chemical processes either singly or in combination (Figure 6). These systems may be portable units or large systems that feed several rooms. In the United States, >97% of maintenance hemodialysis facilities use RO alone or in combination with deionization.⁷⁹³ Many acute-care facilities use portable hemodialysis machines with attached portable water treatment systems that use either deionization or RO. These machines were exempted from earlier versions of AAMI recommendations, but given current knowledge about toxic exposures to and inflammatory processes in patients new to dialysis, these machines should now come into compliance with current AAMI recommendations for hemodialysis water and dialysate quality.^{788, 789} Previous recommendations were based on the assumption that acute-care patients did not experience the same degree of adverse effects from short-term, cumulative exposures to either chemicals or microbiologic agents present in hemodialysis fluids compared with the effects encountered by patients during chronic, maintenance dialysis.^{788, 789} Additionally, JCAHO is reviewing inpatient

practices and record-keeping for dialysis (acute and maintenance) for adherence to AAMI standards and recommended practices.

Figure 6. Dialysis water treatment system*



* See text for description of the placement and function of these components.

Neither the water used to prepare dialysate nor the dialysate itself needs to be sterile, but tap water can not be used without additional treatment. Infections caused by rapid-growing NTM (e.g., *Mycobacterium chelonae* and *M. abscessus*) present a potential risk to hemodialysis patients (especially those in hemodialyzer reuse programs) if disinfection procedures to inactivate mycobacteria in the water (low-level disinfection) and the hemodialyzers (high-level disinfection) are inadequate.^{31, 32, 633} Other factors associated with microbial contamination in dialysis systems could involve the water treatment system, the water and dialysate distribution systems, and the type of hemodialyzer.^{666, 667, 794-799} Understanding the various factors and their influence on contamination levels is the key to preventing high levels of microbial contamination in dialysis therapy.

In several studies, pyrogenic reactions were demonstrated to have been caused by lipopolysaccharide or endotoxin associated with gram-negative bacteria.^{794, 800-803} Early studies demonstrated that parenteral exposure to endotoxin at a concentration of 1 ng/kg body weight/hour was the threshold dose for producing pyrogenic reactions in humans, and that the relative potencies of endotoxin differ by bacterial species.^{804, 805} Gram-negative water bacteria (e.g., *Pseudomonas* spp.) have been shown to multiply rapidly in a variety of hospital-associated fluids that can be used as supply water for hemodialysis (e.g., distilled water, deionized water, RO water, and softened water) and in dialysate (a balanced salt solution made with this water).⁸⁰⁶ Several studies have demonstrated that the attack rates of pyrogenic reactions are directly associated with the number of bacteria in dialysate.^{666, 667, 807} These studies provided the rationale for setting the heterotrophic bacteria standards in the first AAMI hemodialysis guideline at $\leq 2,000$ CFU/mL in dialysate and one log lower (≤ 200 CFU/mL) for the water used to prepare dialysate.^{668, 788} If the level of bacterial contamination exceeded 200 CFU/mL in water, this level could be amplified in the system and effectively constitute a high inoculum for dialysate at the start of a

dialysis treatment.^{807, 808} Pyrogenic reactions did not appear to occur when the level of contamination was below 2,000 CFU/mL in dialysate unless the source of the endotoxin was exogenous to the dialysis system (i.e., present in the community water supply). Endotoxins in a community water supply have been linked to the development of pyrogenic reactions among dialysis patients.⁷⁹⁴

Whether endotoxin actually crosses the dialyzer membrane is controversial. Several investigators have shown that bacteria growing in dialysate-generated products that could cross the dialyzer membrane.^{809,}

⁸¹⁰ Gram-negative bacteria growing in dialysate have produced endotoxins that in turn stimulated the production of anti-endotoxin antibodies in hemodialysis patients;^{801, 811} these data suggest that bacterial endotoxins, although large molecules, cross dialyzer membranes either intact or as fragments. The use of the very permeable membranes known as high-flux membranes (which allow large molecules [e.g., β_2 microglobulin] to traverse the membrane) increases the potential for passage of endotoxins into the blood path. Several studies support this contention. In one such study, an increase in plasma endotoxin concentrations during dialysis was observed when patients were dialyzed against dialysate containing 10^3 – 10^4 CFU/mL *Pseudomonas* spp.⁸¹² *In vitro* studies using both radiolabeled lipopolysaccharide and biologic assays have demonstrated that biologically active substances derived from bacteria found in dialysate can cross a variety of dialyzer membranes.^{802, 813–816} Patients treated with high-flux membranes have had higher levels of anti-endotoxin antibodies than subjects or patients treated with conventional membranes.⁸¹⁷ Finally, since 1989, 19%–22% of dialysis centers have reported pyrogenic reactions in the absence of septicemia.^{818, 819}

Investigations of adverse outcomes among patients using reprocessed dialyzers have demonstrated a greater risk for developing pyrogenic reactions when the water used to reprocess these devices contained >6 ng/mL endotoxin and $>10^4$ CFU/mL bacteria.⁸²⁰ In addition to the variability in endotoxin assays, host factors also are involved in determining whether a patient will mount a response to endotoxin.⁸⁰³ Outbreak investigations of pyrogenic reactions and bacteremias associated with hemodialyzer reuse have demonstrated that pyrogenic reactions are prevented once the endotoxin level in the water used to reprocess the dialyzers is returned to below the AAMI standard level.⁸²¹

Reuse of dialyzers and use of bicarbonate dialysate, high-flux dialyzer membranes, or high-flux dialysis may increase the potential for pyrogenic reactions if the water in the dialysis setting does not meet standards.^{796–798} Although investigators have been unable to demonstrate endotoxin transfer across dialyzer membranes,^{803, 822, 823} the preponderance of reports now supports the ability of endotoxin to transfer across at least some high-flux membranes under some operating conditions. In addition to the acute risk of pyrogenic reactions, indirect evidence is increasingly demonstrating that chronic exposure to low amounts of endotoxin may play a role in some of the long-term complications of hemodialysis therapy. Patients treated with ultrafiltered dialysate for 5–6 months have demonstrated a decrease in serum β_2 microglobulin concentrations and a decrease in markers of an inflammatory response.^{824–826} In studies of longer duration, use of microbiologically ultrapure dialysate has been associated with a decreased incidence of β_2 microglobulin-associated amyloidosis.^{827, 828}

Although patient benefit likely is associated with the use of ultrapure dialysate, no consensus has been reached regarding the potential adoption of this as standard in the United States. Debate continues regarding the bacterial and endotoxin limits for dialysate. As advances in water treatment and hemodialysis processes occur, efforts are underway to move improved technology from the manufacturer out into the user community. Cost-benefit studies, however, have not been done, and substantially increased costs to implement newer water treatment modalities are anticipated.

To reconcile AAMI documents with current International Organization for Standardization (ISO) format, AAMI has determined that its hemodialysis standards will be discussed in the following four installments: RD 5 for hemodialysis equipment, RD 62 for product water quality, RD 47 for dialyzer

reprocessing, and RD 52 for dialysate quality. The Renal Diseases and Dialysis Committee of AAMI is expected to finalize and promulgated the dialysate standard pertinent to the user community (RD 52), adopting by reference the bacterial and endotoxin limits in product water as currently outlined in the AAMI standard that applies to systems manufacturers (RD 62). At present, the user community should continue to observe water quality and dialysate standards as outlined in AAMI RD 5 (Hemodialysis Systems, 1992) and AAMI RD 47 (Reuse of Hemodialyzers, 1993) until the new RD 52 standard becomes available (Table 18).^{789, 791}

Table 18. Microbiologic limits for hemodialysis fluids*

Hemodialysis fluid	Maximum total heterotrophs (CFU/mL)+	Maximum endotoxin level (EU/mL)§
<i>Present standard</i>		
Product water¶		
Used to prepare dialysate	200	No standard
Used to reprocess dialyzers	200	5
Dialysate	2,000	No standard
<i>Proposed standard**</i>		
Product water	200	2
Dialysate	200	2

* The material in this table was compiled from references 789 and 791 (ANSI/AAMI standards RD 5-1992 and ANSI/AAMI RD 47-1993).

+ Colony forming units per milliliter.

§ Endotoxin units per milliliter.

¶ Product water presently includes water used to prepare dialysate and water used to reprocess dialyzers.

** Dialysate for hemodialysis, RD 52, under development, American National Standards Institute, Association for the Advancement of Medical Instrumentation (AAMI).

The current AAMI standard directed at systems manufacturers (RD 62 [Water Treatment Equipment for Hemodialysis Applications, 2001]) now specifies that all product water used to prepare dialysate or to reprocess dialyzers for multiple use should contain <2 endotoxin units per milliliter (EU/mL).⁷⁹² A level of 2 EU/mL was chosen as the upper limit for endotoxin because this level is easily achieved with contemporary water treatment systems using RO and/or ultrafiltration. CDC has advocated monthly endotoxin testing along with microbiologic assays of water, because endotoxin activity may not correspond to the total heterotrophic plate counts.⁸²⁹ Additionally, the current AAMI standard RD 62 for manufacturers includes action levels for product water. Because 48 hours can elapse between the time of sampling water for microbial contamination and the time when results are received, and because bacterial proliferation can be rapid, action levels for microbial counts and endotoxin concentrations are reported as 50 CFU/mL and 1 EU/mL, respectively, in this revision of the standard.⁷⁹² These recommendations will allow users to initiate corrective action before levels exceed the maximum levels established by the standard.

In hemodialysis, the net movement of water is from the blood to the dialysate, although within the dialyzer, local movement of water from the dialysate to the blood through the phenomenon of back-filtration may occur, particularly in dialyzers with highly permeable membranes.⁸³⁰ In contrast, hemofiltration and hemodiafiltration feature infusion of large volumes of electrolyte solution (20–70 L) into the blood. Increasingly, this electrolyte solution is being prepared on-line from water and concentrate. Because of the large volumes of fluid infused, AAMI considered the necessity of setting more stringent requirements for water to be used in this application, but this organization has not yet established these because of lack of expert consensus and insufficient experience with on-line therapies in the United States. On-line hemofiltration and hemodiafiltration systems use sequential ultrafiltration as the final step in the preparation of infusion fluid. Several experts from AAMI concur that these

point-of-use ultrafiltration systems should be capable of further reducing the bacteria and endotoxin burden of solutions prepared from water meeting the requirements of the AAMI standard to a safe level for infusion.

b. Microbial Control Strategies

The strategy for controlling massive accumulations of gram-negative water bacteria and NTM in dialysis systems primarily involves preventing their growth through proper disinfection of water-treatment systems and hemodialysis machines. Gram-negative water bacteria, their associated lipopolysaccharides (bacterial endotoxins), and NTM ultimately come from the community water supply, and levels of these bacteria can be amplified depending on the water treatment system, dialysate distribution system, type of dialysis machine, and method of disinfection (Table 19).^{634, 794, 831} Control strategies are designed to reduce levels of microbial contamination in water and dialysis fluid to relatively low levels but not to completely eradicate it.

Table 19. Factors influencing microbial contamination in hemodialysis systems

Factors	Comments
<u>Water supply</u> Source of community water Ground water Surface water	Contains endotoxin and bacteria Contains high levels of endotoxin and bacteria
<u>Water treatment at the dialysis center</u> None Filtration Prefilter Absolute filter (depth or membrane filter) Activated carbon filter	Not recommended Particulate filter to protect equipment; does not remove microorganisms Removes bacteria, however, unless the filter is changed frequently or disinfected, bacteria will accumulate and grow through the filter; acts as a significant reservoir of bacteria and endotoxin Removes organics and available chlorine or chloramines; acts as a significant reservoir of bacteria and endotoxin
<u>Water treatment devices</u> Deionization/ion-exchange softener Reverse osmosis (RO) Ultraviolet light Ultrafilter	Both softeners and deionizers are significant reservoirs of bacteria and do not remove endotoxin. Removes bacteria and endotoxin, but must be disinfected; operates at high water pressure Kills some bacteria, but there is no residual; ultraviolet-resistant bacteria can develop if the unit is not properly maintained Removes bacteria and endotoxin; operates on normal line pressure; can be positioned distal to deionizer; must be disinfected
<u>Water and dialysate distribution system</u> Distribution pipes Size Construction Elevation Storage tanks	Oversized diameter and length decrease fluid flow and increase bacterial reservoir for both treated water and centrally-prepared dialysate. Rough joints, dead ends, unused branches, and polyvinyl chloride (PVC) piping can act as bacterial reservoirs. Outlet taps should be located at the highest elevation to prevent loss of disinfectant; keep a recirculation loop in the system; flush unused ports routinely. Tanks are undesirable because they act as a reservoir for water bacteria; if tanks are present, they must be routinely scrubbed and disinfected.
<u>Dialysis machines</u> Single-pass Recirculating single-pass or recirculating (batch)	Disinfectant should have contact with all parts of the machine that are exposed to water or dialysis fluid. Recirculating pumps and machine design allow for massive contamination levels if not properly disinfected; overnight chemical germicide treatment is recommended.

Two components of hemodialysis water distribution systems – pipes (particularly those made of polyvinyl chloride [PVC]) and storage tanks – can serve as reservoirs of microbial contamination. Hemodialysis systems frequently use pipes that are wider and longer than are needed to handle the required flow, which slows the fluid velocity and increases both the total fluid volume and the wetted surface area of the system. Gram-negative bacteria in fluids remaining in pipes overnight multiply rapidly and colonize the wet surfaces, producing bacterial populations and endotoxin quantities in proportion to the volume and surface area. Such colonization results in formation of protective biofilm that is difficult to remove and protects the bacteria from disinfection.⁸³² Routine (i.e., monthly), low-level disinfection of the pipes can help to control bacterial contamination of the distribution system. Additional measures to protect pipes from contaminations include a) situating all outlet taps at equal elevation and at the highest point of the system so that the disinfectant cannot drain from pipes by gravity before adequate contact time has elapsed and b) eliminating rough joints, dead-end pipes, and unused branches and taps that can trap fluid and serve as reservoirs of bacteria capable of continuously inoculating the entire volume of the system.⁸⁰⁰ Maintain a flow velocity of 3–5 ft/sec.

A storage tank in the distribution system greatly increases the volume of fluid and surface area available and can serve as a niche for water bacteria. Storage tanks are therefore not recommended for use in dialysis systems unless they are frequently drained and adequately disinfected, including scrubbing the sides of the tank to remove bacterial biofilm. An ultrafilter should be used distal to the storage tank.^{808, 833}

Microbiologic sampling of dialysis fluids is recommended because gram-negative bacteria can proliferate rapidly in water and dialysate in hemodialysis systems; high levels of these organisms place patients at risk for pyrogenic reactions or health-care–associated infection.^{667, 668, 808}

Health-care facilities are advised to sample dialysis fluids at least monthly using standard microbiologic assay methods for waterborne microorganisms.^{788, 793, 799, 834–836} Product water used to prepare dialysate and to reprocess hemodialyzers for reuse on the same patient should also be tested for bacterial endotoxin on a monthly basis.^{792, 829, 837} (See Appendix C for information about water sampling methods for dialysis.)

Cross-contamination of dialysis machines and inadequate disinfection measures can facilitate the spread of waterborne organisms to patients. Steps should be taken to ensure that dialysis equipment is performing correctly and that all connectors, lines, and other components are specific for the equipment, in good repair, and properly in place. A recent outbreak of gram-negative bacteremias among dialysis patients was attributed to faulty valves in a drain port of the machine that allowed backflow of saline used to flush the dialyzer before patient use.^{838, 839} This backflow contaminated the drain priming connectors, which contaminated the blood lines and exposed the patients to high concentrations of gram-negative bacteria. Environmental infection control in dialysis settings also includes low-level disinfection of housekeeping surfaces and spot decontamination of spills of blood (see Environmental Services in Part I of this guideline for further information).

c. Infection-Control Issues in Peritoneal Dialysis

Peritoneal dialysis (PD), most commonly administered as continuous ambulatory peritoneal dialysis (CAPD) and continual cycling peritoneal dialysis (CCPD), is the third most common treatment for end-stage renal disease (ESRD) in the United States, accounting for 12% of all dialysis patients.⁸⁴⁰ Peritonitis is the primary complication of CAPD, with coagulase-negative staphylococci the most clinically significant causative organisms.⁸⁴¹ Other organisms that have been found to produce peritonitis include *Staphylococcus aureus*, *Mycobacterium fortuitum*, *M. mucogenicum*, *Stenotrophomonas maltophilia*, *Burkholderia cepacia*, *Corynebacterium jeikeium*, *Candida* spp., and

other fungi.⁸⁴²⁻⁸⁵⁰ Substantial morbidity is associated with peritoneal dialysis infections. Removal of peritoneal dialysis catheters usually is required for treatment of peritonitis caused by fungi, NTM, or other bacteria that are not cleared within the first several days of effective antimicrobial treatment. Furthermore, recurrent episodes of peritonitis may lead to fibrosis and loss of the dialysis membrane.

Many reported episodes of peritonitis are associated with exit-site or tunneled catheter infections. Risk factors for the development of peritonitis in PD patients include a) under dialysis, b) immune suppression, c) prolonged antimicrobial treatment, d) patient age [more infections occur in younger patients and older hospitalized patients], e) length of hospital stay, and f) hypoalbuminemia.^{844, 851, 852} Concern has been raised about infection risk associated with the use of automated cyclers in both inpatient and outpatient settings; however, studies suggest that PD patients who use automated cyclers have much lower infection rates.⁸⁵³ One study noted that a closed-drainage system reduced the incidence of system-related peritonitis among intermittent peritoneal dialysis (IPD) patients from 3.6 to 1.5 cases/100 patient days.⁸⁵⁴ The association of peritonitis with management of spent dialysate fluids requires additional study. Therefore, ensuring that the tip of the waste line is not submerged beneath the water level in a toilet or in a drain is prudent.

7. Ice Machines and Ice

Microorganisms may be present in ice, ice-storage chests, and ice-making machines. The two main sources of microorganisms in ice are the potable water from which it is made and a transferral of organisms from hands (Table 20). Ice from contaminated ice machines has been associated with patient colonization, blood stream infections, pulmonary and gastrointestinal illnesses, and pseudoinfections.^{602, 603, 683, 684, 854, 855} Microorganisms in ice can secondarily contaminate clinical specimens and medical solutions that require cold temperatures for either transport or holding.^{601, 620} An outbreak of surgical-site infections was interrupted when sterile ice was used in place of tap water ice to cool cardioplegia solutions.⁶⁰¹

Table 20. Microorganisms and their sources in ice and ice machines

Sources of microorganisms	References
From potable water	
<i>Legionella</i> spp.	684, 685, 857, 858
Nontuberculous mycobacteria (NTM)	602, 603, 859
<i>Pseudomonas aeruginosa</i>	859
<i>Burkholderia cepacia</i>	859, 860
<i>Stenotrophomonas maltophilia</i>	860
<i>Flavobacterium</i> spp.	860
From fecally-contaminated water	
Norwalk virus	861-863
<i>Giardia lamblia</i>	864
<i>Cryptosporidium parvum</i>	685
From hand-transfer of organisms	
<i>Acinetobacter</i> spp.	859
Coagulase-negative staphylococci	859
<i>Salmonella enteritidis</i>	865
<i>Cryptosporidium parvum</i>	685

In a study comparing the microbial populations of hospital ice machines with organisms recovered from ice samples gathered from the community, samples from 27 hospital ice machines yielded low numbers (<10 CFU/mL) of several potentially opportunistic microorganisms, mainly gram-negative bacilli.⁸⁵⁹ During the survey period, no health-care-associated infections were attributed to the use of ice. Ice from community sources had higher levels of microbial contamination (75%–95% of 194 samples had total heterotrophic plate counts <500 CFU/mL, with the proportion of positive cultures dependent on the incubation temperature) and showed evidence of fecal contamination from the source water.⁸⁵⁹ Thus, ice machines in health-care settings are no more heavily contaminated compared with ice machines in the community. If the source water for ice in a health-care facility is not fecally contaminated, then ice from clean ice machines and chests should pose no increased hazard for immunocompetent patients. Some waterborne bacteria found in ice could potentially be a risk to immunocompromised patients if they consume ice or drink beverages with ice. For example, *Burkholderia cepacia* in ice could present an infection risk for cystic fibrosis patients.^{859, 860} Therefore, protecting immunosuppressed and otherwise medically at-risk patients from exposure to tap water and ice potentially contaminated with opportunistic pathogens is prudent.⁹

No microbiologic standards for ice, ice-making machines, or ice storage equipment have been established, although several investigators have suggested the need for such standards.^{859, 866} Culturing of ice machines is not routinely recommended, but it may be useful as part of an epidemiologic investigation.^{867–869} Sampling might also help determine the best schedule for cleaning open ice-storage chests. Recommendations for a regular program of maintenance and disinfection have been published.^{866–869} Health-care facilities are advised to clean ice-storage chests on a regular basis. Open ice chests may require a more frequent cleaning schedule compared with chests that have covers. Portable ice chests and containers require cleaning and low-level disinfection before the addition of ice intended for consumption. Ice-making machines may require less frequent cleaning, but their maintenance is important to proper performance. The manufacturer's instructions for both the proper method of cleaning and/or maintenance should be followed. These instructions may also recommend an EPA-registered disinfectant to ensure chemical potency, materials compatibility, and safety. In the event that instructions and suitable EPA-registered disinfectants are not available for this process, then a generic approach to cleaning, disinfecting, and maintaining ice machines and dispensers can be used (Box 12).

Ice and ice-making machines also may be contaminated via improper storage or handling of ice by patients and/or staff.^{684–686, 855–858, 870} Suggested steps to avoid this means of contamination include a) minimizing or avoiding direct hand contact with ice intended for consumption, b) using a hard-surface scoop to dispense ice, and c) installing machines that dispense ice directly into portable containers at the touch of a control.^{687, 869}

Box 12. General steps for cleaning and maintaining ice machines, dispensers, and storage chests*+

-
- 1. Disconnect unit from power supply.**
 - 2. Remove and discard ice from bin or storage chest.**
 - 3. Allow unit to warm to room temperature.**
 - 4. Disassemble removable parts of machine that make contact with water to make ice.**
 - 5. Thoroughly clean machine and parts with water and detergent.**
 - 6. Dry external surfaces of removable parts before reassembling.**
 - 7. Check for any needed repair.**
 - 8. Replace feeder lines, as appropriate (e.g., when damaged, old, or difficult to clean).**
 - 9. Ensure presence of an air space in tubing leading from water inlet into water distribution system of machine.**

(Box 12. continued)

10. Inspect for rodent or insect infestations under the unit and treat, as needed.
11. Check door gaskets (open compartment models) for evidence of leakage or dripping into the storage chest.
12. Clean the ice-storage chest or bin with fresh water and detergent; rinse with fresh tap water.
13. Sanitize machine by circulating a 50–100 parts per million (ppm) solution of sodium hypochlorite (i.e., 4–8 mL sodium hypochlorite/gallon of water) through the ice-making and storage systems for 2 hours (100 ppm solution), or 4 hours (50 ppm solution).
14. Drain sodium hypochlorite solutions and flush with fresh tap water.
15. Allow all surfaces of equipment to dry before returning to service.

* Material in this box is adapted from reference 869.

+ These general guidelines should be used only where manufacturer-recommended methods and EPA-registered disinfectants are not available.

8. Hydrotherapy Tanks and Pools

a. General Information

Hydrotherapy equipment (e.g., pools, whirlpools, whirlpool spas, hot tubs, and physiotherapy tanks) traditionally has been used to treat patients with certain medical conditions (e.g., burns,^{871, 872} septic ulcers, lesions, amputations,⁸⁷³ orthopedic impairments and injuries, arthritis,⁸⁷⁴ and kidney lithotripsy).⁶⁵⁴ Wound-care medicine is increasingly moving away from hydrotherapy, however, in favor of bedside pulsed-lavage therapy using sterile solutions for cleaning and irrigation.^{492, 875–878}

Several episodes of health-care-associated infections have been linked to use of hydrotherapy equipment (Table 21). Potential routes of infection include incidental ingestion of the water, sprays and aerosols, and direct contact with wounds and intact skin (folliculitis). Risk factors for infection include a) age and sex of the patient, b) underlying medical conditions, c) length of time spent in the hydrotherapy water, and d) portals of entry.⁸⁷⁹

Table 21. Infections associated with use of hydrotherapy equipment

Microorganisms	Medical conditions	References
<i>Acinetobacter baumannii</i>	Sepsis	572
<i>Citrobacter freundii</i>	Cellulitis	880
<i>Enterobacter cloacae</i>	Sepsis	881
<i>Legionella</i> spp.	Legionellosis	882
<i>Mycobacterium abscessus</i> , <i>Mycobacterium fortuitum</i> , <i>Mycobacterium marinum</i>	Skin ulcers and soft tissue infections	621–623, 883
<i>Pseudomonas aeruginosa</i>	Sepsis, soft tissue infections, folliculitis, and wound infections	492, 493, 506, 679, 884–888
Adenovirus, adeno-associated virus	Conjunctivitis	889

Infection control for hydrotherapy tanks, pools, or birthing tanks presents unique challenges because indigenous microorganisms are always present in the water during treatments. In addition, some studies have found free living amoebae (i.e., *Naegleria lovaniensis*), which are commonly found in association with *Naegleria fowleri*, in hospital hydrotherapy pools.⁸⁹⁰ Although hydrotherapy is at times appropriate for patients with wounds, burns, or other types of non-intact skin conditions (determined on a case-by-case basis), this equipment should not be considered “semi-critical” in accordance with the Spaulding classification.⁸⁹¹ Microbial data to evaluate the risk of infection to patients using hydrotherapy pools and birthing tanks are insufficient. Nevertheless, health-care facilities should maintain stringent cleaning and disinfection practices in accordance with the manufacturer’s instructions

and with relevant scientific literature until data supporting more rigorous infection-control measures become available. Factors that should be considered in therapy decisions in this situation would include a) availability of alternative aseptic techniques for wound management and b) a risk-benefit analysis of using traditional hydrotherapy.

b. Hydrotherapy Tanks

Hydrotherapy tanks (e.g., whirlpools, Hubbard tanks and whirlpool bath tubs) are shallow tanks constructed of stainless steel, plexiglass, or tile. They are closed-cycle water systems with hydrojets to circulate, aerate, and agitate the water. The maximum water temperature range is 50°F–104°F (10°C–40°C). The warm water temperature, constant agitation and aeration, and design of the hydrotherapy tanks provide ideal conditions for bacterial proliferation if the equipment is not properly maintained, cleaned, and disinfected. The design of the hydrotherapy equipment should be evaluated for potential infection-control problems that can be associated with inaccessible surfaces that can be difficult to clean and/or remain wet in between uses (i.e., recessed drain plates with fixed grill plates).⁸⁸⁷ Associated equipment (e.g., parallel bars, plinths, Hoyer lifts, and wheelchairs) can also be potential reservoirs of microorganisms, depending on the materials used in these items (i.e., porous vs. non-porous materials) and the surfaces that may become wet during use. Patients with active skin colonizations and wound infections can serve as sources of contamination for the equipment and the water. Contamination from spilled tub water can extend to drains, floors, and walls.^{680–683} Health-care-associated colonization or infection can result from exposure to endogenous sources of microorganisms (autoinoculation) or exogenous sources (via cross-contamination from other patients previously receiving treatment in the unit).

Although some facilities have used tub liners to minimize environmental contamination of the tanks, the use of a tub liner does not eliminate the need for cleaning and disinfection. Draining these small pools and tanks after each patient use, thoroughly cleaning with a detergent, and disinfecting according to manufacturers' instructions have reduced bacterial contamination levels in the water from 10⁴ CFU/mL to <10 CFU/mL.⁸⁹² A chlorine residual of 15 ppm in the water should be obtained prior to the patient's therapy session (e.g., by adding 15 grams of calcium hypochlorite 70% [e.g., HTH®] per 100 gallons of water).⁸⁹² A study of commercial and residential whirlpools found that superchlorination or draining, cleaning, disinfection, and refilling of whirlpools markedly reduced densities of *Pseudomonas aeruginosa* in whirlpool water.⁸⁹³ The bacterial populations were rapidly replenished, however, when disinfectant concentrations dropped below recommended levels for recreational use (i.e., chlorine at 3.0 ppm or bromine at 6.0 ppm). When using chlorine, however, knowing whether the community drinking-water system is disinfected with chloramine is important, because municipal utilities adjust the pH of the water to the basic side to enhance chloramine formation. Because chlorine is not very effective at pH levels above 8, it may be necessary to re-adjust the pH of the water to a more acidic level.⁸⁹⁴

A few reports describe the addition of antiseptic chemicals to hydrotherapy tank water, especially for burn patient therapy.^{895–897} One study involving a minimal number of participants demonstrated a reduction in the number of *Pseudomonas* spp. and other gram-negative bacteria from both patients and equipment surfaces when chloramine-T ("chlorazene") was added to the water.⁸⁹⁸ Chloramine-T has not, however, been approved for water treatment in the United States.

c. Hydrotherapy Pools

Hydrotherapy pools typically serve large numbers of patients and are usually heated to 91.4°F–98.6°F (31°C–37°C). The temperature range is more narrow (94°F–96.8°F [35°C–36°C]) for pediatric and geriatric patient use.⁸⁹⁹ Because the size of hydrotherapy pools precludes draining after patient use, proper management is required to maintain the proper balance of water conditioning (i.e., alkalinity, hardness, and temperature) and disinfection. The most widely used chemicals for disinfection of pools

are chlorine and chlorine compounds – calcium hypochlorite, sodium hypochlorite, lithium hypochlorite, chloroisocyanurates, and chlorine gas. Solid and liquid formulations of chlorine chemicals are the easiest and safest to use.⁹⁰⁰ Other halogenated compounds have also been used for pool-water disinfection, albeit on a limited scale. Bromine, which forms bactericidal bromamines in the presence of ammonia, has limited use because of its association with contact dermatitis.⁹⁰¹ Iodine does not bleach hair, swim suits, or cause eye irritation, but when introduced at proper concentrations, it gives water a greenish-yellowish cast.⁸⁹²

In practical terms, maintenance of large hydrotherapy pools (e.g., those used for exercise) is similar to that for indoor public pools (i.e., continuous filtration, chlorine residuals no less than 0.4 ppm, and pH of 7.2–7.6).^{902,903} Supply pipes and pumps also need to be maintained to eliminate the possibility of this equipment serving as a reservoir for waterborne organisms.⁹⁰⁴ Specific standards for chlorine residual and pH of the water are addressed in local and state regulations. Patients who are fecally incontinent or who have draining wounds should refrain from using these pools until their condition improves.

d. Birthing Tanks and Other Equipment

The use of birthing tanks, whirlpool spas, and whirlpools is a recent addition to obstetrical practice.⁹⁰⁵ Few studies on the potential risks associated with these pieces of equipment have been conducted. In one study of 32 women, a newborn contracted a *Pseudomonas* infection after being birthed in such a tank, the strain of which was identical to the organism isolated from the tank water.⁹⁰⁶ Another report documented identical strains of *P. aeruginosa* isolates from a newborn with sepsis and on the environmental surfaces of a tub that the mother used for relaxation while in labor.⁹⁰⁷ Other studies have shown no significant increases in the rates of post-immersion infections among mothers and infants.^{908,909}

Because the water and the tub surfaces routinely become contaminated with the mother's skin flora and blood during labor and delivery, birthing tanks and other tub equipment must be drained after each patient use and the surfaces thoroughly cleaned and disinfected. Health-care facilities are advised to follow the manufacturer's instructions for selection of disinfection method and chemical germicide. The range of chlorine residuals for public whirlpools and whirlpool spas is 2–5 ppm.⁹¹⁰ Use of an inflatable tub is an alternative solution, but this item must be cleaned and disinfected between patients if it is not considered a single-use unit.

Recreational tanks and whirlpool spas are increasingly being used as hydrotherapy equipment. Although such home equipment appears to be suitable for hydrotherapy, they are neither designed nor constructed to function in this capacity. Additionally, manufacturers generally are not obligated to provide the health-care facility with cleaning and disinfecting instructions appropriate for medical equipment use, and the U.S. Food and Drug Administration (FDA) does not evaluate recreational equipment. Health-care facilities should therefore carefully evaluate this “off-label” use of home equipment before proceeding with a purchase.

9. Miscellaneous Medical/Dental Equipment Connected to Main Water Systems

a. Automated Endoscope Reprocessors

The automated endoscopic reprocessor (AER) is classified by the FDA as an accessory for the flexible endoscope.⁶⁵⁴ A properly operating AER can provide a more consistent, reliable method of decontaminating and terminal reprocessing for endoscopes between patient procedures than manual reprocessing methods alone.⁹¹¹ An endoscope is generally subjected to high-level disinfection using a

liquid chemical sterilant or a high-level disinfectant. Because the instrument is a semi-critical device, the optimal rinse fluid for a disinfected endoscope would be sterile water.³ Sterile water, however, is expensive and difficult to produce in sufficient quantities and with adequate quality assurance for instrument rinsing in an AER.^{912, 913} Therefore, one option to be used for AERs is rinse water that has been passed through filters with a pore size of 0.1–0.2 μm to render the water “bacteria-free.” These filters usually are located in the water line at or near the port where the mains water enters the equipment. The product water (i.e., tap water passing through these filters) in these applications is not considered equivalent in microbial quality to that for membrane-filtered water as produced by pharmaceutical firms. Membrane filtration in pharmaceutical applications is intended to ensure the microbial quality of polished product water.

Water has been linked to the contamination of flexible fiberoptic endoscopes in the following two scenarios: a) rinsing a disinfected endoscope with unfiltered tap water, followed by storage of the instrument without drying out the internal channels and b) contamination of AERs from tap water inadvertently introduced into the equipment. In the latter instance, the machine’s water reservoirs and fluid circuitry become contaminated with waterborne, heterotrophic bacteria (e.g., *Pseudomonas aeruginosa* and NTM), which can survive and persist in biofilms attached to these components.^{914–917} Colonization of the reservoirs and water lines of the AER becomes problematic if the required cleaning, disinfection, and maintenance are not performed on the equipment as recommended by the manufacturer.^{669, 916, 917} Use of the 0.1–0.2- μm filter in the water line helps to keep bacterial contamination to a minimum,^{670, 911, 917} but filters may fail and allow bacteria to pass through to the equipment and then to the instrument undergoing reprocessing.^{671–674, 913, 918} Filters also require maintenance for proper performance.^{670, 911, 912, 918, 919} Heightened awareness of the proper disinfection of the connectors that hook the instrument to the AER may help to further reduce the potential for contaminating endoscopes during reprocessing.⁹²⁰ An emerging issue in the field of endoscopy is that of the possible role of rinse water monitoring and its potential to help reduce endoscopy/bronchoscopy-associated infections.⁹¹⁸

Studies have linked deficiencies in endoscope cleaning and/or disinfecting processes to the incidence of post-endoscopic adverse outcomes.^{921–924} Several clusters have been traced to AERs of older designs and these were associated with water quality.^{675, 914–916} Regardless of whether manual or automated terminal reprocessing is used for endoscopes, the internal channels of the instrument should be dried before storage.⁹²⁵ The presence of residual moisture in the internal channels encourages the proliferation of waterborne microorganisms, some of which may be pathogenic. One of the most frequently used methods employs 70% isopropyl alcohol to flush the internal channels, followed by forced air drying of these channels and hanging the endoscope vertically in a protected cabinet; this method ensures internal drying of the endoscope, lessens the potential for proliferation of waterborne microorganisms,^{669, 913, 917, 922, 926, 927} and is consistent with professional organization guidance for endoscope reprocessing.⁹²⁸

An additional problem with waterborne microbial contamination of AERs centers on increased microbial resistance to alkaline glutaraldehyde, a widely used liquid chemical sterilant/high-level disinfectant.^{669, 929} Opportunistic waterborne microorganisms (e.g., *Mycobacterium chelonae*, *Methylobacterium* spp.) have been associated with pseudo-outbreaks and colonization; infection caused by these organisms has been associated with procedures conducted in clinical settings (e.g., bronchoscopy).^{669, 913, 929–931} Increasing microbial resistance to glutaraldehyde has been attributed to improper use of the disinfectant in the equipment, allowing the dilution of glutaraldehyde to fall below the manufacturer’s recommended minimal use concentration.⁹²⁹

b. Dental Unit Water Lines

Dental unit water lines (DUWLs) consist of small-bore plastic tubing that delivers water used for general, non-surgical irrigation and as a coolant to dental handpieces, sonic and ultrasonic scalers, and air-water syringes; municipal tap water is the source water for these lines. The presence of biofilms of waterborne bacteria and fungi (e.g., *Legionella* spp., *Pseudomonas aeruginosa*, and NTM) in DUWLs has been established.^{636, 637, 694, 695, 932–954} Biofilms continually release planktonic microorganisms into the water, the titers of which can exceed 1×10^6 CFU/mL.⁶⁹⁴ However, scientific evidence indicates that immunocompetent persons are only at minimal risk for substantial adverse health effects after contact with water from a dental unit. Nonetheless, exposing patients or dental personnel to water of uncertain microbiological quality is not consistent with universally accepted infection-control principles.⁹³⁵

In 1993, CDC issued guidelines relative to water quality in a dental setting. These guidelines recommend that all dental instruments that use water (including high-speed handpieces) should be run to discharge water for 20–30 seconds after each patient and for several minutes before the start of each clinic day.⁹³⁶ This practice can help to flush out any patient materials that may have entered the turbine, air, or waterlines.^{937, 938} The 1993 guidance also indicated that waterlines be flushed at the beginning of the clinic day. Although these guidelines are designed to help reduce the number of microorganisms present in treatment water, they do not address the issue of reducing or preventing biofilm formation in the waterlines. Research published subsequent to the 1993 dental infection control guideline suggests that flushing the lines at the beginning of the day has only minimal effect on the status of the biofilm in the lines and does not reliably improve the quality of water during dental treatment.^{939–941} Updated recommendations on infection-control practices for water line use in dentistry will be available in late 2003.⁹⁴²

The numbers of microorganisms in water used as coolant or irrigant for non-surgical dental treatment should be as low as reasonably achievable and, at a minimum, should meet nationally recognized standards for safe drinking water.^{935, 943} Only minimal evidence suggests that water meeting drinking water standards poses a health hazard for immunocompetent persons. The EPA, the American Public Health Association (APHA), and the American Water Works Association (AWWA) have set a maximum limit of 500 CFU/mL for aerobic, heterotrophic, mesophilic bacteria in drinking water in municipal distribution systems.^{944, 945} This standard is achievable, given improvements in water-line technology. Dentists should consult with the manufacturer of their dental unit to determine the best equipment and method for maintaining and monitoring good water quality.^{935, 946}

E. Environmental Services

1. Principles of Cleaning and Disinfecting Environmental Surfaces

Although microbiologically contaminated surfaces can serve as reservoirs of potential pathogens, these surfaces generally are not directly associated with transmission of infections to either staff or patients. The transferral of microorganisms from environmental surfaces to patients is largely via hand contact with the surface.^{947, 948} Although hand hygiene is important to minimize the impact of this transfer, cleaning and disinfecting environmental surfaces as appropriate is fundamental in reducing their potential contribution to the incidence of healthcare-associated infections.

The principles of cleaning and disinfecting environmental surfaces take into account the intended use of the surface or item in patient care. CDC retains the Spaulding classification for medical and surgical instruments, which outlines three categories based on the potential for the instrument to transmit infection if the instrument is microbiologically contaminated before use.^{949, 950} These categories are

“critical,” “semicritical,” and “noncritical.” In 1991, CDC proposed an additional category designated “environmental surfaces” to Spaulding’s original classification⁹⁵¹ to represent surfaces that generally do not come into direct contact with patients during care. Environmental surfaces carry the least risk of disease transmission and can be safely decontaminated using less rigorous methods than those used on medical instruments and devices. Environmental surfaces can be further divided into medical equipment surfaces (e.g., knobs or handles on hemodialysis machines, x-ray machines, instrument carts, and dental units) and housekeeping surfaces (e.g., floors, walls, and tabletops).⁹⁵¹

The following factors influence the choice of disinfection procedure for environmental surfaces: a) the nature of the item to be disinfected, b) the number of microorganisms present, c) the innate resistance of those microorganisms to the inactivating effects of the germicide, d) the amount of organic soil present, e) the type and concentration of germicide used, f) duration and temperature of germicide contact, and g) if using a proprietary product, other specific indications and directions for use.^{952, 953}

Cleaning is the necessary first step of any sterilization or disinfection process. Cleaning is a form of decontamination that renders the environmental surface safe to handle or use by removing organic matter, salts, and visible soils, all of which interfere with microbial inactivation.⁹⁵⁴⁻⁹⁶⁰ The physical action of scrubbing with detergents and surfactants and rinsing with water removes large numbers of microorganisms from surfaces.⁹⁵⁷ If the surface is not cleaned before the terminal reprocessing procedures are started, the success of the sterilization or disinfection process is compromised.

Spaulding proposed three levels of disinfection for the treatment of devices and surfaces that do not require sterility for safe use. These disinfection levels are “high-level,” “intermediate-level,” and “low-level.”^{949, 950} The basis for these levels is that microorganisms can usually be grouped according to their innate resistance to a spectrum of physical or chemical germicidal agents (Table 22). This information, coupled with the instrument/surface classification, determines the appropriate level of terminal disinfection for an instrument or surface.

Table 22. Levels of disinfection by type of microorganism*

Disinfection level	Bacteria			Fungi+	Viruses	
	Vegetative	Tubercle bacillus	Spores		Lipid and medium size	Nonlipid and small size
High	+ §	+	+ ¶	+	+	+
Intermediate	+	+	—**	+	+	± ⁺⁺
Low	+	—	—	±	+	±

* Material in this table compiled from references 2 and 951.

+ This class of microorganisms includes asexual spores but not necessarily chlamydo spores or sexual spores.

§ The “plus” sign indicates that a killing effect can be expected when the normal use-concentrations of chemical disinfectants or pasteurization are properly employed; a “negative” sign indicates little or no killing effect.

¶ Only with extended exposure times are high-level disinfectant chemicals capable of killing high numbers of bacterial spores in laboratory tests; they are, however, capable of sporicidal activity.

** Some intermediate-level disinfectants (e.g., hypochlorites) can exhibit some sporicidal activity; others (e.g., alcohols and phenolics) have no demonstrable sporicidal activity.

++ Some intermediate-level disinfectants, although they are tuberculocidal, may have limited virucidal activity.

The process of high-level disinfection, an appropriate standard of treatment for heat-sensitive, semi-critical medical instruments (e.g., flexible, fiberoptic endoscopes), inactivates all vegetative bacteria, mycobacteria, viruses, fungi, and some bacterial spores. High-level disinfection is accomplished with powerful, sporicidal chemicals (e.g., glutaraldehyde, peracetic acid, and hydrogen peroxide) that are not appropriate for use on housekeeping surfaces. These liquid chemical sterilants/high-level disinfectants

are highly toxic.^{961–963} Use of these chemicals for applications other than those indicated in their label instructions (i.e., as immersion chemicals for treating heat-sensitive medical instruments) is not appropriate.⁹⁶⁴ Intermediate-level disinfection does not necessarily kill bacterial spores, but it does inactivate *Mycobacterium tuberculosis* var. *bovis*, which is substantially more resistant to chemical germicides than ordinary vegetative bacteria, fungi, and medium to small viruses (with or without lipid envelopes). Chemical germicides with sufficient potency to achieve intermediate-level disinfection include chlorine-containing compounds (e.g., sodium hypochlorite), alcohols, some phenolics, and some iodophors. Low-level disinfection inactivates vegetative bacteria, fungi, enveloped viruses (e.g., human immunodeficiency virus [HIV], and influenza viruses), and some non-enveloped viruses (e.g., adenoviruses). Low-level disinfectants include quaternary ammonium compounds, some phenolics, and some iodophors. Sanitizers are agents that reduce the numbers of bacterial contaminants to safe levels as judged by public health requirements, and are used in cleaning operations, particularly in food service and dairy applications. Germicidal chemicals that have been approved by FDA as skin antiseptics are not appropriate for use as environmental surface disinfectants.⁹⁵¹

The selection and use of chemical germicides are largely matters of judgment, guided by product label instructions, information, and regulations. Liquid sterilant chemicals and high-level disinfectants intended for use on critical and semi-critical medical/dental devices and instruments are regulated exclusively by the FDA as a result of recent memoranda of understanding between FDA and the EPA that delineates agency authority for chemical germicide regulation.^{965, 966} Environmental surface germicides (i.e., primarily intermediate- and low-level disinfectants) are regulated by the EPA and labeled with EPA registration numbers. The labels and technical data or product literature of these germicides specify indications for product use and provide claims for the range of antimicrobial activity. The EPA requires certain pre-registration laboratory potency tests for these products to support product label claims. EPA verifies (through laboratory testing) manufacturers' claims to inactivate microorganisms for selected products and organisms. Germicides labeled as "hospital disinfectant" have passed the potency tests for activity against three representative microorganisms – *Pseudomonas aeruginosa*, *Staphylococcus aureus*, and *Salmonella cholerae suis*. Low-level disinfectants are often labeled "hospital disinfectant" without a tuberculocidal claim, because they lack the potency to inactivate mycobacteria. Hospital disinfectants with demonstrated potency against mycobacteria (i.e., intermediate-level disinfectants) may list "tuberculocidal" on the label as well. Other claims (e.g., "fungicidal," "pseudomonocidal," and "virucidal") may appear on labels of environmental surface germicides, but the designations of "tuberculocidal hospital disinfectant" and "hospital disinfectant" correlate directly to Spaulding's assessment of intermediate-level disinfectants and low-level disinfectants, respectively.⁹⁵¹

A common misconception in the use of surface disinfectants in health-care settings relates to the underlying purpose for use of proprietary products labeled as a "tuberculocidal" germicide. Such products will not interrupt and prevent the transmission of TB in health-care settings because TB is not acquired from environmental surfaces. The tuberculocidal claim is used as a benchmark by which to measure germicidal potency. Because mycobacteria have the highest intrinsic level of resistance among the vegetative bacteria, viruses, and fungi, any germicide with a tuberculocidal claim on the label (i.e., an intermediate-level disinfectant) is considered capable of inactivating a broad spectrum of pathogens, including much less resistant organisms such as the bloodborne pathogens (e.g., hepatitis B virus [HBV], hepatitis C virus [HCV], and HIV). It is this broad spectrum capability, rather than the product's specific potency against mycobacteria, that is the basis for protocols and OSHA regulations indicating the appropriateness of using tuberculocidal chemicals for surface disinfection.⁹⁶⁷

2. General Cleaning Strategies for Patient-Care Areas

The number and types of microorganisms present on environmental surfaces are influenced by the following factors: a) number of people in the environment, b) amount of activity, c) amount of moisture, d) presence of material capable of supporting microbial growth, e) rate at which organisms suspended in the air are removed, and f) type of surface and orientation [i.e., horizontal or vertical].⁹⁶⁸ Strategies for cleaning and disinfecting surfaces in patient-care areas take into account a) potential for direct patient contact, b) degree and frequency of hand contact, and c) potential contamination of the surface with body substances or environmental sources of microorganisms (e.g., soil, dust, and water).

a. Cleaning of Medical Equipment

Manufacturers of medical equipment should provide care and maintenance instructions specific to their equipment. These instructions should include information about a) the equipments' compatibility with chemical germicides, b) whether the equipment is water-resistant or can be safely immersed for cleaning, and c) how the equipment should be decontaminated if servicing is required.⁹⁶⁷ In the absence of manufacturers' instructions, non-critical medical equipment (e.g., stethoscopes, blood pressure cuffs, dialysis machines, and equipment knobs and controls) usually only require cleansing followed by low- to intermediate-level disinfection, depending on the nature and degree of contamination. Ethyl alcohol or isopropyl alcohol in concentrations of 60%–90% (v/v) is often used to disinfect small surfaces (e.g., rubber stoppers of multiple-dose medication vials, and thermometers)^{952, 969} and occasionally external surfaces of equipment (e.g., stethoscopes and ventilators). However, alcohol evaporates rapidly, which makes extended contact times difficult to achieve unless items are immersed, a factor that precludes its practical use as a large-surface disinfectant.⁹⁵¹ Alcohol may cause discoloration, swelling, hardening, and cracking of rubber and certain plastics after prolonged and repeated use and may damage the shellac mounting of lenses in medical equipment.⁹⁷⁰

Barrier protection of surfaces and equipment is useful, especially if these surfaces are a) touched frequently by gloved hands during the delivery of patient care, b) likely to become contaminated with body substances, or c) difficult to clean. Impervious-backed paper, aluminum foil, and plastic or fluid-resistant covers are suitable for use as barrier protection. An example of this approach is the use of plastic wrapping to cover the handle of the operatory light in dental-care settings.^{936, 942} Coverings should be removed and discarded while the health-care worker is still gloved.^{936, 942} The health-care worker, after ungloving and performing hand hygiene, must cover these surfaces with clean materials before the next patient encounter.

b. Cleaning Housekeeping Surfaces

Housekeeping surfaces require regular cleaning and removal of soil and dust. Dry conditions favor the persistence of gram-positive cocci (e.g., coagulase-negative *Staphylococcus* spp.) in dust and on surfaces, whereas moist, soiled environments favor the growth and persistence of gram-negative bacilli.^{948, 971, 972} Fungi are also present on dust and proliferate in moist, fibrous material.

Most, if not all, housekeeping surfaces need to be cleaned only with soap and water or a detergent/disinfectant, depending on the nature of the surface and the type and degree of contamination. Cleaning and disinfection schedules and methods vary according to the area of the health-care facility, type of surface to be cleaned, and the amount and type of soil present. Disinfectant/detergent formulations registered by EPA are used for environmental surface cleaning, but the actual physical removal of microorganisms and soil by wiping or scrubbing is probably as important, if not more so, than any antimicrobial effect of the cleaning agent used.⁹⁷³ Therefore, cost, safety, product-surface compatibility, and acceptability by housekeepers can be the main criteria for selecting a registered agent. If using a proprietary detergent/disinfectant, the manufacturers' instructions for appropriate use

of the product should be followed.⁹⁷⁴ Consult the products' material safety data sheets (MSDS) to determine appropriate precautions to prevent hazardous conditions during product application. Personal protective equipment (PPE) used during cleaning and housekeeping procedures should be appropriate to the task.

Housekeeping surfaces can be divided into two groups – those with minimal hand-contact (e.g., floors, and ceilings) and those with frequent hand-contact (“high touch surfaces”). The methods, thoroughness, and frequency of cleaning and the products used are determined by health-care facility policy.⁶ However, high-touch housekeeping surfaces in patient-care areas (e.g., doorknobs, bedrails, light switches, wall areas around the toilet in the patient's room, and the edges of privacy curtains) should be cleaned and/or disinfected more frequently than surfaces with minimal hand contact. Infection-control practitioners typically use a risk-assessment approach to identify high-touch surfaces and then coordinate an appropriate cleaning and disinfecting strategy and schedule with the housekeeping staff.

Horizontal surfaces with infrequent hand contact (e.g., window sills and hard-surface flooring) in routine patient-care areas require cleaning on a regular basis, when soiling or spills occur, and when a patient is discharged from the facility.⁶ Regular cleaning of surfaces and decontamination, as needed, is also advocated to protect potentially exposed workers.⁹⁶⁷ Cleaning of walls, blinds, and window curtains is recommended when they are visibly soiled.^{972, 973, 975} Disinfectant fogging is not recommended for general infection control in routine patient-care areas.^{2, 976} Further, paraformaldehyde, which was once used in this application, is no longer registered by EPA for this purpose. Use of paraformaldehyde in these circumstances requires either registration or an exemption issued by EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). Infection control, industrial hygienists, and environmental services supervisors should assess the cleaning procedures, chemicals used, and the safety issues to determine if a temporary relocation of the patient is needed when cleaning in the room.

Extraordinary cleaning and decontamination of floors in health-care settings is unwarranted. Studies have demonstrated that disinfection of floors offers no advantage over regular detergent/water cleaning and has minimal or no impact on the occurrence of health-care-associated infections.^{947, 948, 977–980} Additionally, newly cleaned floors become rapidly recontaminated from airborne microorganisms and those transferred from shoes, equipment wheels, and body substances.^{971, 975, 981} Nevertheless, health-care institutions or contracted cleaning companies may choose to use an EPA-registered detergent/disinfectant for cleaning low-touch surfaces (e.g., floors) in patient-care areas because of the difficulty that personnel may have in determining if a spill contains blood or body fluids (requiring a detergent/disinfectant for clean-up) or when a multi-drug resistant organism is likely to be in the environment. Methods for cleaning non-porous floors include wet mopping and wet vacuuming, dry dusting with electrostatic materials, and spray buffing.^{973, 982–984} Methods that produce minimal mists and aerosols or dispersion of dust in patient-care areas are preferred.^{9, 20, 109, 272}

Part of the cleaning strategy is to minimize contamination of cleaning solutions and cleaning tools. Bucket solutions become contaminated almost immediately during cleaning, and continued use of the solution transfers increasing numbers of microorganisms to each subsequent surface to be cleaned.^{971, 981, 985} Cleaning solutions should be replaced frequently. A variety of “bucket” methods have been devised to address the frequency with which cleaning solutions are replaced.^{986, 987} Another source of contamination in the cleaning process is the cleaning cloth or mop head, especially if left soaking in dirty cleaning solutions.^{971, 988–990} Laundering of cloths and mop heads after use and allowing them to dry before re-use can help to minimize the degree of contamination.⁹⁹⁰ A simplified approach to cleaning involves replacing soiled cloths and mop heads with clean items each time a bucket of detergent/disinfectant is emptied and replaced with fresh, clean solution (B. Stover, Kosair Children's Hospital, 2000). Disposable cleaning cloths and mop heads are an alternative option, if costs permit.

Another reservoir for microorganisms in the cleaning process may be dilute solutions of the detergents or disinfectants, especially if the working solution is prepared in a dirty container, stored for long periods of time, or prepared incorrectly.⁵⁴⁷ Gram-negative bacilli (e.g., *Pseudomonas* spp. and *Serratia marcescens*) have been detected in solutions of some disinfectants (e.g., phenolics and quaternary ammonium compounds).^{547, 991} Contemporary EPA registration regulations have helped to minimize this problem by asking manufacturers to provide potency data to support label claims for detergent/disinfectant properties under real-use conditions (e.g., diluting the product with tap water instead of distilled water). Application of contaminated cleaning solutions, particularly from small-quantity aerosol spray bottles or with equipment that might generate aerosols during operation, should be avoided, especially in high-risk patient areas.^{992, 993} Making sufficient fresh cleaning solution for daily cleaning, discarding any remaining solution, and drying out the container will help to minimize the degree of bacterial contamination. Containers that dispense liquid as opposed to spray-nozzle dispensers (e.g., quart-sized dishwashing liquid bottles) can be used to apply detergent/disinfectants to surfaces and then to cleaning cloths with minimal aerosol generation. A pre-mixed, “ready-to-use” detergent/disinfectant solution may be used if available.

c. Cleaning Special Care Areas

Guidelines have been published regarding cleaning strategies for isolation areas and operating rooms.^{6, 7} The basic strategies for areas housing immunosuppressed patients include a) wet dusting horizontal surfaces daily with cleaning cloths pre-moistened with detergent or an EPA-registered hospital disinfectant or disinfectant wipes;^{94, 98463} b) using care when wet dusting equipment and surfaces above the patient to avoid patient contact with the detergent/disinfectant; c) avoiding the use of cleaning equipment that produces mists or aerosols; d) equipping vacuums with HEPA filters, especially for the exhaust, when used in any patient-care area housing immunosuppressed patients,^{9, 94, 986} and e) regular cleaning and maintenance of equipment to ensure efficient particle removal. When preparing the cleaning cloths for wet-dusting, freshly prepared solutions of detergents or disinfectants should be used rather than cloths that have soaked in such solutions for long periods of time. Dispersal of microorganisms in the air from dust or aerosols is more problematic in these settings than elsewhere in health-care facilities. Vacuum cleaners can serve as dust disseminators if they are not operating properly.⁹⁹⁴ Doors to immunosuppressed patients’ rooms should be closed when nearby areas are being vacuumed.⁹ Bacterial and fungal contamination of filters in cleaning equipment is inevitable, and these filters should be cleaned regularly or replaced as per equipment manufacturer instructions.

Mats with tacky surfaces placed in operating rooms and other patient-care areas only slightly minimize the overall degree of contamination of floors and have little impact on the incidence rate of health-care-associated infection in general.^{351, 971, 983} An exception, however, is the use of tacky mats inside the entry ways of cordoned-off construction areas inside the health-care facility; these mats help to minimize the intrusion of dust into patient-care areas.

Special precautions for cleaning incubators, mattresses, and other nursery surfaces have been recommended to address reports of hyperbilirubinemia in newborns linked to inadequately diluted solutions of phenolics and poor ventilation.⁹⁹⁵⁻⁹⁹⁷ These medical conditions have not, however, been associated with the use of properly prepared solutions of phenolics. Non-porous housekeeping surfaces in neonatal units can be disinfected with properly diluted or pre-mixed phenolics, followed by rinsing with clean water.⁹⁹⁷ However, phenolics are not recommended for cleaning infant bassinets and incubators during the stay of the infant. Infants who remain in the nursery for an extended period should be moved periodically to freshly cleaned and disinfected bassinets and incubators.⁹⁹⁷ If phenolics are used for cleaning bassinets and incubators after they have been vacated, the surfaces should be rinsed thoroughly with water and dried before either piece of equipment is reused. Cleaning

and disinfecting protocols should allow for the full contact time specified for the product used. Bassinet mattresses should be replaced, however, if the mattress cover surface is broken.⁹⁹⁷

3. Cleaning Strategies for Spills of Blood and Body Substances

Neither HBV, HCV, nor HIV has ever been transmitted from a housekeeping surface (i.e., floors, walls, or countertops). Nonetheless, prompt removal and surface disinfection of an area contaminated by either blood or body substances are sound infection-control practices and OSHA requirements.⁹⁶⁷

Studies have demonstrated that HIV is inactivated rapidly after being exposed to commonly used chemical germicides at concentrations that are much lower than those used in practice.^{998–1003} HBV is readily inactivated with a variety of germicides, including quaternary ammonium compounds.¹⁰⁰⁴ Embalming fluids (e.g., formaldehyde) are also capable of completely inactivating HIV and HBV.^{1005, 1006} OSHA has revised its regulation for disinfecting spills of blood or other potentially infectious material to include proprietary products whose label includes inactivation claims for HBV and HIV, provided that such surfaces have not become contaminated with agent(s) or volumes of or concentrations of agent(s) for which a higher level of disinfection is recommended.¹⁰⁰⁷ These registered products are listed in EPA's List D – *Registered Antimicrobials Effective Against Hepatitis B Virus and Human HIV-1*, which may include products tested against duck hepatitis B virus (DHBV) as a surrogate for HBV.^{1008, 1009} Additional lists of interest include EPA's List C – *Registered Antimicrobials Effective Against Human HIV-1* and EPA's List E – *Registered Antimicrobials Effective Against Mycobacterium spp., Hepatitis B Virus, and Human HIV-1*.

Sodium hypochlorite solutions are inexpensive and effective broad-spectrum germicidal solutions.^{1010, 1011} Generic sources of sodium hypochlorite include household chlorine bleach or reagent grade chemical. Concentrations of sodium hypochlorite solutions with a range of 5,000–6,150 ppm (1:10 v/v dilution of household bleaches marketed in the United States) to 500–615 ppm (1:100 v/v dilution) free chlorine are effective depending on the amount of organic material (e.g., blood, mucus, and urine) present on the surface to be cleaned and disinfected.^{1010, 1011} EPA-registered chemical germicides may be more compatible with certain materials that could be corroded by repeated exposure to sodium hypochlorite, especially the 1:10 dilution. Appropriate personal protective equipment (e.g., gloves and goggles) should be worn when preparing and using hypochlorite solutions or other chemical germicides.⁹⁶⁷

Despite laboratory evidence demonstrating adequate potency against bloodborne pathogens (e.g., HIV and HBV), many chlorine bleach products available in grocery and chemical-supply stores are not registered by the EPA for use as surface disinfectants. Use of these chlorine products as surface disinfectants is considered by the EPA to be an “unregistered use.” EPA encourages the use of registered products because the agency reviews them for safety and performance when the product is used according to label instructions. When unregistered products are used for surface disinfection, users do so at their own risk.

Strategies for decontaminating spills of blood and other body fluids differ based on the setting in which they occur and the volume of the spill.¹⁰¹⁰ In patient-care areas, workers can manage small spills with cleaning and then disinfecting using an intermediate-level germicide or an EPA-registered germicide from the EPA List D or E.^{967, 1007} For spills containing large amounts of blood or other body substances, workers should first remove visible organic matter with absorbent material (e.g., disposable paper towels discarded into leak-proof, properly labeled containment) and then clean and decontaminate the area.^{1002, 1003, 1012} If the surface is nonporous and a generic form of a sodium hypochlorite solution is used (e.g., household bleach), a 1:100 dilution is appropriate for decontamination assuming that a) the

worker assigned to clean the spill is wearing gloves and other personal protective equipment appropriate to the task, b) most of the organic matter of the spill has been removed with absorbent material, and c) the surface has been cleaned to remove residual organic matter. A recent study demonstrated that even strong chlorine solutions (i.e., 1:10 dilution of chlorine bleach) may fail to totally inactivate high titers of virus in large quantities of blood, but in the absence of blood these disinfectants can achieve complete viral inactivation.¹⁰¹¹ This evidence supports the need to remove most organic matter from a large spill before final disinfection of the surface. Additionally, EPA-registered proprietary disinfectant label claims are based on use on a pre-cleaned surface.^{951, 954}

Managing spills of blood, body fluids, or other infectious materials in clinical, public health, and research laboratories requires more stringent measures because of a) the higher potential risk of disease transmission associated with large volumes of blood and body fluids and b) high numbers of microorganisms associated with diagnostic cultures. The use of an intermediate-level germicide for routine decontamination in the laboratory is prudent.⁹⁵⁴ Recommended practices for managing large spills of concentrated infectious agents in the laboratory include a) confining the contaminated area, b) flooding the area with a liquid chemical germicide before cleaning, and c) decontaminating with fresh germicidal chemical of at least intermediate-level disinfectant potency.¹⁰¹⁰ A suggested technique when flooding the spill with germicide is to lay absorbent material down on the spill and apply sufficient germicide to thoroughly wet both the spill and the absorbent material.¹⁰¹³ If using a solution of household chlorine bleach, a 1:10 dilution is recommended for this purpose. EPA-registered germicides should be used according to the manufacturers' instructions for use dilution and contact time. Gloves should be worn during the cleaning and decontamination procedures in both clinical and laboratory settings. PPE in such a situation may include the use of respiratory protection (e.g., an N95 respirator) if clean-up procedures are expected to generate infectious aerosols. Protocols for cleaning spills should be developed and made available on record as part of good laboratory practice.¹⁰¹³ Workers in laboratories and in patient-care areas of the facility should receive periodic training in environmental-surface infection-control strategies and procedures as part of an overall infection-control and safety curriculum.

4. Carpeting and Cloth Furnishings

a. Carpeting

Carpeting has been used for more than 30 years in both public and patient-care areas of health-care facilities. Advantages of carpeting in patient-care areas include a) its noise-limiting characteristics; b) the "humanizing" effect on health care; and c) its contribution to reductions in falls and resultant injuries, particularly for the elderly.¹⁰¹⁴⁻¹⁰¹⁶ Compared to hard-surface flooring, however, carpeting is harder to keep clean, especially after spills of blood and body substances. It is also harder to push equipment with wheels (e.g., wheelchairs, carts, and gurneys) on carpeting.

Several studies have documented the presence of diverse microbial populations, primarily bacteria and fungi, in carpeting;^{111, 1017-1024} the variety and number of microorganisms tend to stabilize over time. New carpeting quickly becomes colonized, with bacterial growth plateauing after about 4 weeks.¹⁰¹⁹ Vacuuming and cleaning the carpeting can temporarily reduce the numbers of bacteria, but these populations soon rebound and return to pre-cleaning levels.^{1019, 1020, 1023} Bacterial contamination tends to increase with higher levels of activity.^{1018-1020, 1025} Soiled carpeting that is or remains damp or wet provides an ideal setting for the proliferation and persistence of gram-negative bacteria and fungi.¹⁰²⁶ Carpeting that remains damp should be removed, ideally within 72 hours.

Despite the evidence of bacterial growth and persistence in carpeting, only limited epidemiologic evidence demonstrates that carpets influence health-care-associated infection rates in areas housing

immunocompetent patients.^{1023, 1025, 1027} This guideline, therefore, includes no recommendations against the use of carpeting in these areas. Nonetheless, avoiding the use of carpeting is prudent in areas where spills are likely to occur (e.g., laboratories, areas around sinks, and janitor closets) and where patients may be at greater risk of infection from airborne environmental pathogens (e.g., HSCT units, burn units, ICUs, and ORs).^{111, 1028} An outbreak of aspergillosis in an HSCT unit was recently attributed to carpet contamination and a particular method of carpet cleaning.¹¹¹ A window in the unit had been opened repeatedly during the time of a nearby building fire, which allowed fungal spore intrusion into the unit. After the window was sealed, the carpeting was cleaned using a “bonnet buffing” machine, which dispersed *Aspergillus* spores into the air.¹¹¹ Wet vacuuming was instituted, replacing the dry cleaning method used previously; no additional cases of invasive aspergillosis were identified.

The care setting and the method of carpet cleaning are important factors to consider when attempting to minimize or prevent production of aerosols and dispersal of carpet microorganisms into the air.^{94, 111} Both vacuuming and shampooing or wet cleaning with equipment can disperse microorganisms to the air.^{111, 994} Vacuum cleaners should be maintained to minimize dust dispersal in general, and be equipped with HEPA filters, especially for use in high-risk patient-care areas.^{9, 94, 986} Some formulations of carpet-cleaning chemicals, if applied or used improperly, can be dispersed into the air as a fine dust capable of causing respiratory irritation in patients and staff.¹⁰²⁹ Cleaning equipment, especially those that engage in wet cleaning and extraction, can become contaminated with waterborne organisms (e.g., *Pseudomonas aeruginosa*) and serve as a reservoir for these organisms if this equipment is not properly maintained. Substantial numbers of bacteria can then be transferred to carpeting during the cleaning process.¹⁰³⁰ Therefore, keeping the carpet cleaning equipment in good repair and allowing such equipment to dry between uses is prudent.

Carpet cleaning should be performed on a regular basis determined by internal policy. Although spills of blood and body substances on non-porous surfaces require prompt spot cleaning using standard cleaning procedures and application of chemical germicides,⁹⁶⁷ similar decontamination approaches to blood and body substance spills on carpeting can be problematic from a regulatory perspective.¹⁰³¹ Most, if not all, modern carpet brands suitable for public facilities can tolerate the activity of a variety of liquid chemical germicides. However, according to OSHA, carpeting contaminated with blood or other potentially infectious materials can not be fully decontaminated.¹⁰³² Therefore, facilities electing to use carpeting for high-activity patient-care areas may choose carpet tiles in areas at high risk for spills.^{967, 1032} In the event of contamination with blood or other body substances, carpet tiles can be removed, discarded, and replaced. OSHA also acknowledges that only minimal direct skin contact occurs with carpeting, and therefore, employers are expected to make reasonable efforts to clean and sanitize carpeting using carpet detergent/cleaner products.¹⁰³²

Over the last few years, some carpet manufacturers have treated their products with fungicidal and/or bactericidal chemicals. Although these chemicals may help to reduce the overall numbers of bacteria or fungi present in carpet, their use does not preclude the routine care and maintenance of the carpeting. Limited evidence suggests that chemically treated carpet may have helped to keep health-care–associated aspergillosis rates low in one HSCT unit,¹¹¹ but overall, treated carpeting has not been shown to prevent the incidence of health-care–associated infections in care areas for immunocompetent patients.

b. Cloth Furnishings

Upholstered furniture and furnishings are becoming increasingly common in patient-care areas. These furnishings range from simple cloth chairs in patients’ rooms to a complete decorating scheme that gives the interior of the facility more the look of an elegant hotel.¹⁰³³ Even though pathogenic microorganisms have been isolated from the surfaces of cloth chairs, no epidemiologic evidence suggests that general patient-care areas with cloth furniture pose increased risks of health-care–

associated infection compared with areas that contain hard-surfaced furniture.^{1034, 1035} Allergens (e.g., dog and cat dander) have been detected in or on cloth furniture in clinics and elsewhere in hospitals in concentrations higher than those found on bed linens.^{1034, 1035} These allergens presumably are transferred from the clothing of visitors. Researchers have therefore suggested that cloth chairs should be vacuumed regularly to keep the dust and allergen levels to a minimum. This recommendation, however, has generated concerns that aerosols created from vacuuming could place immunocompromised patients or patients with preexisting lung disease (e.g., asthma) at risk for development of health-care–associated, environmental airborne disease.^{9, 20, 109, 988} Recovering worn, upholstered furniture (especially the seat cushion) with covers that are easily cleaned (e.g., vinyl), or replacing the item is prudent; minimizing the use of upholstered furniture and furnishings in any patient-care areas where immunosuppressed patients are located (e.g., HSCT units) reduces the likelihood of disease.⁹

5. Flowers and Plants in Patient-Care Areas

Fresh flowers, dried flowers, and potted plants are common items in health-care facilities. In 1974, clinicians isolated an *Erwinia* sp. post mortem from a neonate diagnosed with fulminant septicemia, meningitis, and respiratory distress syndrome.¹⁰³⁸ Because *Erwinia* spp. are plant pathogens, plants brought into the delivery room were suspected to be the source of the bacteria, although the case report did not definitively establish a direct link. Several subsequent studies evaluated the numbers and diversity of microorganisms in the vase water of cut flowers. These studies revealed that high concentrations of bacteria, ranging from 10^4 – 10^{10} CFU/mL, were often present, especially if the water was changed infrequently.^{515, 702, 1039} The major group of microorganisms in flower vase water was gram-negative bacteria, with *Pseudomonas aeruginosa* the most frequently isolated organism.^{515, 702, 1039, 1040} *P. aeruginosa* was also the primary organism directly isolated from chrysanthemums and other potted plants.^{1041, 1042} However, flowers in hospitals were not significantly more contaminated with bacteria compared with flowers in restaurants or in the home.⁷⁰² Additionally, no differences in the diversity and degree of antibiotic resistance of bacteria have been observed in samples isolated from hospital flowers versus those obtained from flowers elsewhere.⁷⁰²

Despite the diversity and large numbers of bacteria associated with flower-vase water and potted plants, minimal or no evidence indicates that the presence of plants in immunocompetent patient-care areas poses an increased risk of health-care–associated infection.⁵¹⁵ In one study involving a limited number of surgical patients, no correlation was observed between bacterial isolates from flowers in the area and the incidence and etiology of postoperative infections among the patients.¹⁰⁴⁰ Similar conclusions were reached in a study that examined the bacteria found in potted plants.¹⁰⁴² Nonetheless, some precautions for general patient-care settings should be implemented, including a) limiting flower and plant care to staff with no direct patient contact, b) advising health-care staff to wear gloves when handling plants, c) washing hands after handling plants, d) changing vase water every 2 days and discharging the water into a sink outside the immediate patient environment, and e) cleaning and disinfecting vases after use.⁷⁰²

Some researchers have examined the possibility of adding a chemical germicide to vase water to control bacterial populations. Certain chemicals (e.g., hydrogen peroxide and chlorhexidine) are well tolerated by plants.^{1040, 1043, 1044} Use of these chemicals, however, was not evaluated in studies to assess impact on health-care–associated infection rates. Modern florists now have a variety of products available to add to vase water to extend the life of cut flowers and to minimize bacterial clouding of the water.

Flowers (fresh and dried) and ornamental plants, however, may serve as a reservoir of *Aspergillus* spp., and dispersal of conidiospores into the air from this source can occur.¹⁰⁹ Health-care–associated outbreaks of invasive aspergillosis reinforce the importance of maintaining an environment as free of

Aspergillus spp. spores as possible for patients with severe, prolonged neutropenia. Potted plants, fresh-cut flowers, and dried flower arrangements may provide a reservoir for these fungi as well as other fungal species (e.g., *Fusarium* spp.).^{109, 1045, 1046} Researchers in one study of bacteria and flowers suggested that flowers and vase water should be avoided in areas providing care to medically at-risk patients (e.g., oncology patients and transplant patients), although this study did not attempt to correlate the observations of bacterial populations in the vase water with the incidence of health-care–associated infections.⁵¹⁵ Another study using molecular epidemiology techniques demonstrated identical *Aspergillus terreus* types among environmental and clinical specimens isolated from infected patients with hematological malignancies.¹⁰⁴⁶ Therefore, attempts should be made to exclude flowers and plants from areas where immunosuppressed patients are located (e.g., HSCT units).^{9, 1046}

6. Pest Control

Cockroaches, flies and maggots, ants, mosquitoes, spiders, mites, midges, and mice are among the typical arthropod and vertebrate pest populations found in health-care facilities. Insects can serve as agents for the mechanical transmission of microorganisms, or as active participants in the disease transmission process by serving as a vector.^{1047–1049} Arthropods recovered from health-care facilities have been shown to carry a wide variety of pathogenic microorganisms.^{1050–1056} Studies have suggested that the diversity of microorganisms associated with insects reflects the microbial populations present in the indoor health-care environment; some pathogens encountered in insects from hospitals were either absent from or present to a lesser degree in insects trapped from residential settings.^{1057–1060} Some of the microbial populations associated with insects in hospitals have demonstrated resistance to antibiotics.^{1048, 1059, 1061–1063}

Insect habitats are characterized by warmth, moisture, and availability of food.¹⁰⁶⁴ Insects forage in and feed on substrates, including but not limited to food scraps from kitchens/cafeteria, foods in vending machines, discharges on dressings either in use or discarded, other forms of human detritus, medical wastes, human wastes, and routine solid waste.^{1057–1061} Cockroaches, in particular, have been known to feed on fixed sputum smears in laboratories.^{1065, 1066} Both cockroaches and ants are frequently found in the laundry, central sterile supply departments, and anywhere in the facility where water or moisture is present (e.g., sink traps, drains and janitor closets). Ants will often find their way into sterile packs of items as they forage in a warm, moist environment.¹⁰⁵⁷ Cockroaches and other insects frequent loading docks and other areas with direct access to the outdoors.

Although insects carry a wide variety of pathogenic microorganisms on their surfaces and in their gut, the direct association of insects with disease transmission (apart from vector transmission) is limited, especially in health-care settings; the presence of insects in itself likely does not contribute substantially to health-care–associated disease transmission in developed countries. However, outbreaks of infection attributed to microorganisms carried by insects may occur because of infestation coupled with breaks in standard infection-control practices.¹⁰⁶³ Studies have been conducted to examine the role of houseflies as possible vectors for shigellosis and other forms of diarrheal disease in non-health-care settings.^{1046, 1067} When control measures aimed at reducing the fly population density were implemented, a concomitant reduction in the incidence of diarrheal infections, carriage of *Shigella* organisms, and mortality caused by diarrhea among infants and young children was observed.

Myiasis is defined as a parasitosis in which the larvae of any of a variety of flies use living or necrotic tissue or body substances of the host as a nutritional source.¹⁰⁶⁸ Larvae from health-care–acquired myiasis have been observed in nares, wounds, eyes, ears, sinuses, and the external urogenital structures.^{1069–1071} Patients with this rare condition are typically older adults with underlying medical conditions (e.g., diabetes, chronic wounds, and alcoholism) who have a decreased capacity to ward off

the flies. Persons with underlying conditions who live or travel to tropical regions of the world are especially at risk.^{1070, 1071} Cases occur in the summer and early fall months in temperate climates when flies are most active.¹⁰⁷¹ An environmental assessment and review of the patient's history are necessary to verify that the source of the myiasis is health-care-acquired and to identify corrective measures.^{1069, 1072} Simple prevention measures (e.g., installing screens on windows) are important in reducing the incidence of myiasis.¹⁰⁷²

From a public health and hygiene perspective, arthropod and vertebrate pests should be eradicated from all indoor environments, including health-care facilities.^{1073, 1074} Modern approaches to institutional pest management usually focus on a) eliminating food sources, indoor habitats, and other conditions that attract pests; b) excluding pests from the indoor environments; and c) applying pesticides as needed.¹⁰⁷⁵ Sealing windows in modern health-care facilities helps to minimize insect intrusion. When windows need to be opened for ventilation, ensuring that screens are in good repair and closing doors to the outside can help with pest control. Insects should be kept out of all areas of the health-care facility, especially ORs and any area where immunosuppressed patients are located. A pest-control specialist with appropriate credentials can provide a regular insect-control program that is tailored to the needs of the facility and uses approved chemicals and/or physical methods. Industrial hygienists can provide information on possible adverse reactions of patients and staff to pesticides and suggest alternative methods for pest control, as needed.

7. Special Pathogen Concerns

a. Antibiotic-Resistant Gram-Positive Cocci

Vancomycin-resistant enterococci (VRE), methicillin-resistant *Staphylococcus aureus* (MRSA), and *S. aureus* with intermediate levels of resistance to glycopeptide antibiotics (vancomycin intermediate resistant *S. aureus* [VISA] or glycopeptide intermediate resistant *S. aureus* [GISA]) represent crucial and growing concerns for infection control. Although the term GISA is technically a more accurate description of the strains isolated to date (most of which are classified as having intermediate resistance to both vancomycin and teicoplanin), the term “glycopeptide” may not be recognized by many clinicians. Thus, the label of VISA, which emphasizes a change in minimum inhibitory concentration (MICs) to vancomycin, is similar to that of VRE and is more meaningful to clinicians.¹⁰⁷⁶ According to National Nosocomial Infection Surveillance (NNIS) statistics for infections acquired among ICU patients in the United States in 1999, 52.3% of infections resulting from *S. aureus* were identified as MRSA infections, and 25.2% of enterococcal infections were attributed to VRE. These figures reflect a 37% and a 43% increase, respectively, since 1994–1998.¹⁰⁷⁷

People represent the primary reservoir of *S. aureus*.¹⁰⁷⁸ Although *S. aureus* has been isolated from a variety of environmental surfaces (e.g., stethoscopes, floors, charts, furniture, dry mops, and hydrotherapy tanks), the role of environmental contamination in transmission of this organism in health care appears to be minimal.^{1079–1082} *S. aureus* contamination of surfaces and tanks within burn therapy units, however, may be a major factor in the transmission of infection among burn patients.¹⁰⁸³

Colonized patients are the principal reservoir of VRE, and patients who are immunosuppressed (e.g., transplant patients) or otherwise medically at-risk (e.g., ICU patients, cardio-thoracic surgical patients, patients previously hospitalized for extended periods, and those having received multi-antimicrobial or vancomycin therapy) are at greatest risk for VRE colonization.^{1084–1087} The mechanisms by which cross-colonization take place are not well defined, although recent studies have indicated that both MRSA and VRE may be transmitted either a) directly from patient to patient, b) indirectly by transient carriage on the hands of health-care workers,^{1088–1091} or c) by hand transfer of these gram-positive organisms from contaminated environmental surfaces and patient-care equipment.^{1084, 1087, 1092–1097} In

one survey, hand carriage of VRE in workers in a long-term care facility ranged from 13%–41%.¹⁰⁹⁸ Many of the environmental surfaces found to be contaminated with VRE in outbreak investigations have been those that are touched frequently by the patient or the health-care worker.¹⁰⁹⁹ Such high-touch surfaces include bedrails, doorknobs, bed linens, gowns, overbed tables, blood pressure cuffs, computer table, bedside tables, and various medical equipment.^{22, 1087, 1094, 1095, 1100–1102} Contamination of environmental surfaces with VRE generally occurs in clinical laboratories and areas where colonized patients are present,^{1087, 1092, 1094, 1095, 1103} but the potential for contamination increases when such patients have diarrhea¹⁰⁸⁷ or have multiple body-site colonization.¹¹⁰⁴ Additional factors that can be important in the dispersion of these pathogens to environmental surfaces are misuse of glove techniques by health-care workers (especially when cleaning fecal contamination from surfaces) and patient, family, and visitor hygiene.

Interest in the importance of environmental reservoirs of VRE increased when laboratory studies demonstrated that enterococci can persist in a viable state on dry environmental surfaces for extended periods of time (7 days to 4 months)^{1099, 1105} and multiple strains can be identified during extensive periods of surveillance.¹¹⁰⁴ VRE can be recovered from inoculated hands of health-care workers (with or without gloves) for up to 60 minutes.²² The presence of either MRSA, VISA, or VRE on environmental surfaces, however, does not mean that patients in the contaminated areas will become colonized. Strict adherence to hand hygiene/handwashing and the proper use of barrier precautions help to minimize the potential for spread of these pathogens. Published recommendations for preventing the spread of vancomycin resistance address isolation measures, including patient cohorting and management of patient-care items.⁵ Direct patient-care items (e.g., blood pressure cuffs) should be disposable whenever possible when used in contact isolation settings for patients with multiply resistant microorganisms.¹¹⁰²

Careful cleaning of patient rooms and medical equipment contributes substantially to the overall control of MRSA, VISA, or VRE transmission. The major focus of a control program for either VRE or MRSA should be the prevention of hand transfer of these organisms. Routine cleaning and disinfection of the housekeeping surfaces (e.g., floors and walls) and patient-care surfaces (e.g., bedrails) should be adequate for inactivation of these organisms. Both MRSA and VRE are susceptible to several EPA-registered low- and intermediate-level disinfectants (e.g., alcohols, sodium hypochlorite, quaternary ammonium compounds, phenolics, and iodophors) at recommended use dilutions for environmental surface disinfection.^{1103, 1106–1109} Additionally, both VRE and vancomycin-sensitive enterococci are equally sensitive to inactivation by chemical germicides,^{1106, 1107, 1109} and similar observations have been made when comparing the germicidal resistance of MRSA to that of either methicillin-sensitive *S. aureus* (MSSA) or VISA.¹¹¹⁰ The use of stronger solutions of disinfectants for inactivation of either VRE, MRSA, or VISA is not recommended based on the organisms' resistance to antibiotics.^{1110–1112} VRE from clinical specimens have exhibited some measure of increased tolerance to heat inactivation in temperature ranges <212°F (<100°C),^{1106, 1113} however, the clinical significance of these observations is unclear because the role of cleaning the surface or item prior to heat treatment was not evaluated. Although routine environmental sampling is not recommended, laboratory surveillance of environmental surfaces during episodes when VRE contamination is suspected can help determine the effectiveness of the cleaning and disinfecting procedures. Environmental culturing should be approved and supervised by the infection-control program in collaboration with the clinical laboratory.^{1084, 1087, 1088, 1092, 1096}

Two cases of wound infections associated with vancomycin-resistant *Staphylococcus aureus* (VRSA) determined to be resistant by NCCLS standards for sensitivity/resistance testing were identified in Michigan and Pennsylvania in 2002.^{1114, 1115} These represented isolated cases, and neither the family members nor the health-care providers of these case-patients had evidence of colonization or infection with VRSA. Conventional environmental infection-control measures (i.e., cleaning and then

disinfecting surfaces using EPA-registered disinfectants with label claims for *S. aureus*) were used during the environmental investigation of these two cases;^{1110–1112} however, studies have yet to evaluate the potential intrinsic resistance of these VRSA strains to surface disinfectants.

Standard procedures during terminal cleaning and disinfection of surfaces, if performed incorrectly, may be inadequate for the elimination of VRE from patient rooms.^{1113, 1116–1118} Given the sensitivity of VRE to hospital disinfectants, current disinfecting protocols should be effective if they are diligently carried out and properly performed. Health-care facilities should be sure that housekeeping staff use correct procedures for cleaning and disinfecting surfaces in VRE-contaminated areas, which include using sufficient amounts of germicide at proper use dilution and allowing adequate contact time.¹¹¹⁸

b. Clostridium difficile

Clostridium difficile is the most frequent etiologic agent for health-care–associated diarrhea.^{1119, 1120} In one hospital, 30% of adults who developed health-care–associated diarrhea were positive for *C. difficile*.¹¹²¹ One recent study employing PCR-ribotyping techniques demonstrated that cases of *C. difficile*-acquired diarrhea occurring in the hospital included patients whose infections were attributed to endogenous *C. difficile* strains and patients whose illnesses were considered to be health-care–associated infections.¹¹²² Most patients remain asymptomatic after infection, but the organism continues to be shed in their stools. Risk factors for acquiring *C. difficile*-associated infection include a) exposure to antibiotic therapy, particularly with beta-lactam agents;¹¹²³ b) gastrointestinal procedures and surgery;¹¹²⁴ c) advanced age; and d) indiscriminate use of antibiotics.^{1125–1128} Of all the measures that have been used to prevent the spread of *C. difficile*-associated diarrhea, the most successful has been the restriction of the use of antimicrobial agents.^{1129, 1130}

C. difficile is an anaerobic, gram-positive bacterium. Normally fastidious in its vegetative state, it is capable of sporulating when environmental conditions no longer support its continued growth. The capacity to form spores enables the organism to persist in the environment (e.g., in soil and on dry surfaces) for extended periods of time. Environmental contamination by this microorganism is well known, especially in places where fecal contamination may occur.¹¹³¹ The environment (especially housekeeping surfaces) rarely serves as a direct source of infection for patients.^{1024, 1132–1136} However, direct exposure to contaminated patient-care items (e.g., rectal thermometers) and high-touch surfaces in patients' bathrooms (e.g., light switches) have been implicated as sources of infection.^{1130, 1135, 1136, 1138}

Transfer of the pathogen to the patient via the hands of health-care workers is thought to be the most likely mechanism of exposure.^{24, 1133, 1139} Standard isolation techniques intended to minimize enteric contamination of patients, health-care–workers' hands, patient-care items, and environmental surfaces have been published.¹¹⁴⁰ Handwashing remains the most effective means of reducing hand contamination. Proper use of gloves is an ancillary measure that helps to further minimize transfer of these pathogens from one surface to another.

The degree to which the environment becomes contaminated with *C. difficile* spores is proportional to the number of patients with *C. difficile*-associated diarrhea,^{24, 1132, 1135} although asymptomatic, colonized patients may also serve as a source of contamination. Few studies have examined the use of specific chemical germicides for the inactivation of *C. difficile* spores, and no well-controlled trials have been conducted to determine efficacy of surface disinfection and its impact on health-care–associated diarrhea. Some investigators have evaluated the use of chlorine-containing chemicals (e.g., 1,000 ppm hypochlorite at recommended use-dilution, 5,000 ppm sodium hypochlorite [1:10 v/v dilution], 1:100 v/v dilutions of unbuffered hypochlorite, and phosphate-buffered hypochlorite [1,600 ppm]). One of the studies demonstrated that the number of contaminated environmental sites was reduced by half,¹¹³⁵ whereas another two studies demonstrated declines in health-care–associated *C. difficile* infections in a HSCT unit¹¹⁴¹ and in two geriatric medical units¹¹⁴² during a period of hypochlorite use. The presence

of confounding factors, however, was acknowledged in one of these studies.¹¹⁴² The recommended approach to environmental infection control with respect to *C. difficile* is meticulous cleaning followed by disinfection using hypochlorite-based germicides as appropriate.^{952, 1130, 1143} However, because no EPA-registered surface disinfectants with label claims for inactivation of *C. difficile* spores are available, the recommendation is based on the best available evidence from the scientific literature.

c. Respiratory and Enteric Viruses in Pediatric-Care Settings

Although the viruses mentioned in this guideline are not unique to the pediatric-care setting in health-care facilities, their prevalence in these areas, especially during the winter months, is substantial. Children (particularly neonates) are more likely to develop infection and substantial clinical disease from these agents compared with adults and therefore are more likely to require supportive care during their illness.

Common respiratory viruses in pediatric-care areas include rhinoviruses, respiratory syncytial virus (RSV), adenoviruses, influenza viruses, and parainfluenza viruses. Transmission of these viruses occurs primarily via direct contact with small-particle aerosols or via hand contamination with respiratory secretions that are then transferred to the nose or eyes. Because transmission primarily requires close personal contact, contact precautions are appropriate to interrupt transmission.⁶ Hand contamination can occur from direct contact with secretions or indirectly from touching high-touch environmental surfaces that have become contaminated with virus from large droplets. The indirect transfer of virus from one person to other via hand contact with frequently-touched fomites was demonstrated in a study using a bacteriophage whose environmental stability approximated that of human viral pathogens (e.g., poliovirus and parvovirus).¹¹⁴⁴ The impact of this mode of transmission with respect to human respiratory- and enteric viruses is dependent on the ability of these agents to survive on environmental surfaces. Infectious RSV has been recovered from skin, porous surfaces, and non-porous surfaces after 30 minutes, 1 hour, and 7 hours, respectively.¹¹⁴⁵ Parainfluenza viruses are known to persist for up to 4 hours on porous surfaces and up to 10 hours on non-porous surfaces.¹¹⁴⁶ Rhinoviruses can persist on porous surfaces and non-porous surfaces for approximately 1 and 3 hours respectively; study participants in a controlled environment became infected with rhinoviruses after first touching a surface with dried secretions and then touching their nasal or conjunctival mucosa.¹¹⁴⁷ Although the efficiency of direct transmission of these viruses from surfaces in uncontrolled settings remains to be defined, these data underscore the basis for maintaining regular protocols for cleaning and disinfecting of high-touch surfaces.

The clinically important enteric viruses encountered in pediatric care settings include enteric adenovirus, astroviruses, caliciviruses, and rotavirus. Group A rotavirus is the most common cause of infectious diarrhea in infants and children. Transmission of this virus is primarily fecal-oral, however, the role of fecally contaminated surfaces and fomites in rotavirus transmission is unclear. During one epidemiologic investigation of enteric disease among children attending day care, rotavirus contamination was detected on 19% of inanimate objects in the center.^{1148, 1149} In an outbreak in a pediatric unit, secondary cases of rotavirus infection clustered in areas where children with rotaviral diarrhea were located.¹¹⁵⁰ Astroviruses cause gastroenteritis and diarrhea in newborns and young children and can persist on fecally contaminated surfaces for several months during periods of relatively low humidity.^{1151, 1152} Outbreaks of small round-structured viruses (i.e., caliciviruses [Norwalk virus and Norwalk-like viruses]) can affect both patients and staff, with attack rates of $\geq 50\%$.¹¹⁵³ Routes of person-to-person transmission include fecal-oral spread and aerosols generated from vomiting.¹¹⁵⁴⁻¹¹⁵⁶ Fecal contamination of surfaces in care settings can spread large amounts of virus to the environment. Studies that have attempted to use low- and intermediate-level disinfectants to inactivate rotavirus suspended in feces have demonstrated a protective effect of high concentrations of organic matter.^{1157, 1158} Intermediate-level disinfectants (e.g., alcoholic quaternary ammonium compounds, and chlorine solutions) can be effective in inactivating enteric viruses provided that a cleaning step to remove most of

the organic matter precedes terminal disinfection.¹¹⁵⁸ These findings underscore the need for proper cleaning and disinfecting procedures where contamination of environmental surfaces with body substances is likely. EPA-registered surface disinfectants with label claims for these viral agents should be used in these settings. Using disposable, protective barrier coverings may help to minimize the degree of surface contamination.⁹³⁶

d. Severe Acute Respiratory Syndrome (SARS) Virus

In November 2002 an atypical pneumonia of unknown etiology emerged in Asia and subsequently developed into an international outbreak of respiratory illness among persons in 29 countries during the first six months of 2003. “Severe acute respiratory syndrome” (SARS) is a viral upper respiratory infection associated with a newly described coronavirus (SARS-associated Co-V [SARS-CoV]). SARS-CoV is an enveloped RNA virus. It is present in high titers in respiratory secretions, stool, and blood of infected persons. The modes of transmission determined from epidemiologic investigations were primarily forms of direct contact (i.e., large droplet aerosolization and person-to-person contact). Respiratory secretions were presumed to be the major source of virus in these situations; airborne transmission of virus has not been completely ruled out. Little is known about the impact of fecal-oral transmission and SARS.

The epidemiology of SARS-CoV infection is not completely understood, and therefore recommended infection control and prevention measures to contain the spread of SARS will evolve as new information becomes available.¹¹⁵⁹ At present there is no indication that established strategies for cleaning (i.e., to remove the majority of bioburden) and disinfecting equipment and environmental surfaces need to be changed for the environmental infection control of SARS. In-patient rooms housing SARS patients should be cleaned and disinfected at least daily and at the time of patient transfer or discharge. More frequent cleaning and disinfection may be indicated for high-touch surfaces and following aerosol-producing procedures (e.g., intubation, bronchoscopy, and sputum production). While there are presently no disinfectant products registered by EPA specifically for inactivation of SARS-CoV, EPA-registered hospital disinfectants that are equivalent to low- and intermediate-level germicides may be used on pre-cleaned, hard, non-porous surfaces in accordance with manufacturer’s instructions for environmental surface disinfection. Monitoring adherence to guidelines established for cleaning and disinfection is an important component of environmental infection control to contain the spread of SARS.

e. Creutzfeldt-Jakob Disease (CJD) in Patient-Care Areas

Creutzfeldt-Jakob disease (CJD) is a rare, invariably fatal, transmissible spongiform encephalopathy (TSE) that occurs worldwide with an average annual incidence of 1 case per million population.¹¹⁶⁰⁻¹¹⁶² CJD is one of several TSEs affecting humans; other diseases in this group include kuru, fatal familial insomnia, and Gerstmann-Sträussler-Scheinker syndrome. A TSE that affects a younger population (compared to the age range of CJD cases) has been described primarily in the United Kingdom since 1996.¹¹⁶³ This variant form of CJD (vCJD) is clinically and neuropathologically distinguishable from classic CJD; epidemiologic and laboratory evidence suggests a causal association for bovine spongiform encephalopathy (BSE [Mad Cow disease]) and vCJD.¹¹⁶³⁻¹¹⁶⁶

The agent associated with CJD is a prion, which is an abnormal isoform of a normal protein constituent of the central nervous system.¹¹⁶⁷⁻¹¹⁶⁹ The mechanism by which the normal form of the protein is converted to the abnormal, disease-causing prion is unknown. The tertiary conformation of the abnormal prion protein appears to confer a heightened degree of resistance to conventional methods of sterilization and disinfection.^{1170, 1171}

Although about 90% of CJD cases occur sporadically, a limited number of cases are the result of a direct exposure to prion-containing material (usually central nervous system tissue or pituitary

hormones) acquired as a result of health care (iatrogenic cases). These cases have been linked to a) pituitary hormone therapy [from human sources as opposed to hormones prepared through the use of recombinant technology],^{1170–1174} b) transplants of either dura mater or corneas,^{1175–1181} and c) neurosurgical instruments and depth electrodes.^{1182–1185} In the cases involving instruments and depth electrodes, conventional cleaning and terminal reprocessing methods of the day failed to fully inactivate the contaminating prions and are considered inadequate by today's standards.

Prion inactivation studies involving whole tissues and tissue homogenates have been conducted to determine the parameters of physical and chemical methods of sterilization or disinfection necessary for complete inactivation,^{1170, 1186–1191} however, the application of these findings to environmental infection control in health-care settings is problematic. No studies have evaluated the effectiveness of medical instrument reprocessing in inactivating prions. Despite a consensus that abnormal prions display some extreme measure of resistance to inactivation by either physical or chemical methods, scientists disagree about the exact conditions needed for sterilization. Inactivation studies utilizing whole tissues present extraordinary challenges to any sterilizing method.¹¹⁹² Additionally, the experimental designs of these studies preclude the evaluation of surface cleaning as a part of the total approach to pathogen inactivation.^{951, 1192}

Some researchers have recommended the use of either a 1:2 v/v dilution of sodium hypochlorite (approximately 20,000 ppm), full-strength sodium hypochlorite (50,000–60,000 ppm), or 1–2 N sodium hydroxide (NaOH) for the inactivation of prions on certain surfaces (e.g., those found in the pathology laboratory).^{1170, 1188} Although these chemicals may be appropriate for the decontamination of laboratory, operating-room, or autopsy-room surfaces that come into contact with central nervous system tissue from a known or suspected patient, this approach is not indicated for routine or terminal cleaning of a room previously occupied by a CJD patient. Both chemicals pose hazards for the health-care worker doing the decontamination. NaOH is caustic and should not make contact with the skin. Sodium hypochlorite solutions (i.e., chlorine bleach) can corrode metals (e.g., aluminum). MSDS information should be consulted when attempting to work with concentrated solutions of either chemical. Currently, no EPA-registered products have label claims for prion inactivation; therefore, this guidance is based on the best available evidence from the scientific literature.

Environmental infection-control strategies must be based on the principles of the “chain of infection,” regardless of the disease of concern.¹³ Although CJD is transmissible, it is not highly contagious. All iatrogenic cases of CJD have been linked to a direct exposure to prion-contaminated central nervous system tissue or pituitary hormones. The six documented iatrogenic cases associated with instruments and devices involved neurosurgical instruments and devices that introduced residual contamination directly to the recipient's brain. No evidence suggests that vCJD has been transmitted iatrogenically or that either CJD or vCJD has been transmitted from environmental surfaces (e.g., housekeeping surfaces). Therefore, routine procedures are adequate for terminal cleaning and disinfection of a CJD patient's room. Additionally, in epidemiologic studies involving highly transfused patients, blood was not identified as a source for prion transmission.^{1193–1198} Routine procedures for containing, decontaminating, and disinfecting surfaces with blood spills should be adequate for proper infection control in these situations.^{951, 1199}

Guidance for environmental infection control in ORs and autopsy areas has been published.^{1197, 1199} Hospitals should develop risk-assessment procedures to identify patients with known or suspected CJD in efforts to implement prion-specific infection-control measures for the OR and for instrument reprocessing.¹²⁰⁰ This assessment also should be conducted for older patients undergoing non-lesionous neurosurgery when such procedures are being done for diagnosis. Disposable, impermeable coverings should be used during these autopsies and neurosurgeries to minimize surface contamination. Surfaces that have become contaminated with central nervous system tissue or cerebral spinal fluid should be

cleaned and decontaminated by a) removing most of the tissue or body substance with absorbent materials, b) wetting the surface with a sodium hypochlorite solution containing $\geq 5,000$ ppm or a 1 N NaOH solution, and c) rinsing thoroughly.^{951, 1197–1199, 1201} The optimum duration of contact exposure in these instances is unclear. Some researchers recommend a 1-hour contact time on the basis of tissue-inactivation studies,^{1197, 1198, 1201} whereas other reviewers of the subject draw no conclusions from this research.¹¹⁹⁹ Factors to consider before cleaning a potentially contaminated surface are a) the degree to which gross tissue/body substance contamination can be effectively removed and b) the ease with which the surface can be cleaned.

F. Environmental Sampling

This portion of Part I addresses the basic principles and methods of sampling environmental surfaces and other environmental sources for microorganisms. The applied strategies of sampling with respect to environmental infection control have been discussed in the appropriate preceding subsections.

1. General Principles: Microbiologic Sampling of the Environment

Before 1970, U.S. hospitals conducted regularly scheduled culturing of the air and environmental surfaces (e.g., floors, walls, and table tops).¹²⁰² By 1970, CDC and the American Hospital Association (AHA) were advocating the discontinuation of routine environmental culturing because rates of health-care–associated infection had not been associated with levels of general microbial contamination of air or environmental surfaces, and because meaningful standards for permissible levels of microbial contamination of environmental surfaces or air did not exist.^{1203–1205} During 1970–1975, 25% of U.S. hospitals reduced the extent of such routine environmental culturing — a trend that has continued.^{1206, 1207}

Random, undirected sampling (referred to as “routine” in previous guidelines) differs from the current practice of targeted sampling for defined purposes.^{2, 1204} Previous recommendations against routine sampling were not intended to discourage the use of sampling in which sample collection, culture, and interpretation are conducted in accordance with defined protocols.² In this guideline, targeted microbiologic sampling connotes a monitoring process that includes a) a written, defined, multidisciplinary protocol for sample collection and culturing; b) analysis and interpretation of results using scientifically determined or anticipatory baseline values for comparison; and c) expected actions based on the results obtained. Infection control, in conjunction with laboratorians, should assess the health-care facility’s capability to conduct sampling and determine when expert consultation and/or services are needed.

Microbiologic sampling of air, water, and inanimate surfaces (i.e., environmental sampling) is an expensive and time-consuming process that is complicated by many variables in protocol, analysis, and interpretation. It is therefore indicated for only four situations.¹²⁰⁸ The first is to support an investigation of an outbreak of disease or infections when environmental reservoirs or fomites are implicated epidemiologically in disease transmission.^{161, 1209, 1210} It is important that such culturing be supported by epidemiologic data. Environmental sampling, as with all laboratory testing, should not be conducted if there is no plan for interpreting and acting on the results obtained.^{11, 1211, 1212} Linking microorganisms from environmental samples with clinical isolates by molecular epidemiology is crucial whenever it is possible to do so.

The second situation for which environmental sampling may be warranted is in research. Well-designed and controlled experimental methods and approaches can provide new information about the spread of health-care–associated diseases.^{126, 129} A classic example is the study of environmental microbial

contamination that compared health-care–associated infection rates in an old hospital and a new facility before and shortly after occupancy.⁹⁴⁷

The third indication for sampling is to monitor a potentially hazardous environmental condition, confirm the presence of a hazardous chemical or biological agent, and validate the successful abatement of the hazard. This type of sampling can be used to: a) detect bioaerosols released from the operation of health-care equipment (e.g., an ultrasonic cleaner) and determine the success of repairs in containing the hazard,¹²¹³ b) detect the release of an agent of bioterrorism in an indoor environmental setting and determine its successful removal or inactivation, and c) sample for industrial hygiene or safety purposes (e.g., monitoring a “sick building”).

The fourth indication is for quality assurance to evaluate the effects of a change in infection-control practice or to ensure that equipment or systems perform according to specifications and expected outcomes. Any sampling for quality-assurance purposes must follow sound sampling protocols and address confounding factors through the use of properly selected controls. Results from a single environmental sample are difficult to interpret in the absence of a frame of reference or perspective. Evaluations of a change in infection-control practice are based on the assumption that the effect will be measured over a finite period, usually of short duration. Conducting quality-assurance sampling on an extended basis, especially in the absence of an adverse outcome, is usually unjustified. A possible exception might be the use of air sampling during major construction periods to qualitatively detect breaks in environmental infection-control measures. In one study, which began as part of an investigation of an outbreak of health-care–associated aspergillosis, airborne concentrations of *Aspergillus* spores were measured in efforts to evaluate the effectiveness of sealing hospital doors and windows during a period of construction of a nearby building.⁵⁰ Other examples of sampling for quality-assurance purposes may include commissioning newly constructed space in special care areas (i.e., ORs and units for immunosuppressed patients) or assessing a change in housekeeping practice. However, the only types of routine environmental microbiologic sampling recommended as part of a quality-assurance program are a) the biological monitoring of sterilization processes by using bacterial spores¹²¹⁴ and b) the monthly culturing of water used in hemodialysis applications and for the final dialysate use dilution. Some experts also advocate periodic environmental sampling to evaluate the microbial/particulate quality for regular maintenance of the air handling system (e.g., filters) and to verify that the components of the system meet manufacturer’s specifications (A. Streifel, University of Minnesota, 2000). Certain equipment in health-care settings (e.g., biological safety cabinets) may also be monitored with air flow and particulate sampling to determine performance or as part of adherence to a certification program; results can then be compared with a predetermined standard of performance. These measurements, however, usually do not require microbiologic testing.

2. Air Sampling

Biological contaminants occur in the air as aerosols and may include bacteria, fungi, viruses, and pollens.^{1215, 1216} Aerosols are characterized as solid or liquid particles suspended in air. Talking for 5 minutes and coughing each can produce 3,000 droplet nuclei; sneezing can generate approximately 40,000 droplets which then evaporate to particles in the size range of 0.5–12 μm .^{137, 1217} Particles in a biological aerosol usually vary in size from $<1 \mu\text{m}$ to $\geq 50 \mu\text{m}$. These particles may consist of a single, unattached organism or may occur in the form of clumps composed of a number of bacteria. Clumps can also include dust and dried organic or inorganic material. Vegetative forms of bacterial cells and viruses may be present in the air in a lesser number than bacterial spores or fungal spores. Factors that determine the survival of microorganisms within a bioaerosol include a) the suspending medium, b) temperature, c) relative humidity, d) oxygen sensitivity, and e) exposure to UV or electromagnetic radiation.¹²¹⁵ Many vegetative cells will not survive for lengthy periods of time in the air unless the

relative humidity and other factors are favorable for survival and the organism is enclosed within some protective cover (e.g., dried organic or inorganic matter).¹²¹⁶ Pathogens that resist drying (e.g., *Staphylococcus* spp., *Streptococcus* spp., and fungal spores) can survive for long periods and can be carried considerable distances via air and still remain viable. They may also settle on surfaces and become airborne again as secondary aerosols during certain activities (e.g., sweeping and bed making).^{1216, 1218}

Microbiologic air sampling is used as needed to determine the numbers and types of microorganisms, or particulates, in indoor air.²⁸⁹ Air sampling for quality control is, however, problematic because of lack of uniform air-quality standards. Although airborne spores of *Aspergillus* spp. can pose a risk for neutropenic patients, the critical number (i.e., action level) of these spores above which outbreaks of aspergillosis would be expected to occur has not been defined. Health-care professionals considering the use of air sampling should keep in mind that the results represent indoor air quality at singular points in time, and these may be affected by a variety of factors, including a) indoor traffic, b) visitors entering the facility, c) temperature, d) time of day or year, e) relative humidity, f) relative concentration of particles or organisms, and g) the performance of the air-handling system components. To be meaningful, air-sampling results must be compared with those obtained from other defined areas, conditions, or time periods.

Several preliminary concerns must be addressed when designing a microbiologic air sampling strategy (Box 13). Because the amount of particulate material and bacteria retained in the respiratory system is largely dependent on the size of the inhaled particles, particle size should be determined when studying airborne microorganisms and their relation to respiratory infections. Particles $>5\ \mu\text{m}$ are efficiently trapped in the upper respiratory tract and are removed primarily by ciliary action.¹²¹⁹ Particles $\leq 5\ \mu\text{m}$ in diameter reach the lung, but the greatest retention in the alveoli is of particles 1–2 μm in diameter.^{1220–1222}

Box 13. Preliminary concerns for conducting air sampling

-
- Consider the possible characteristics and conditions of the aerosol, including size range of particles, relative amount of inert material, concentration of microorganisms, and environmental factors.
 - Determine the type of sampling instruments, sampling time, and duration of the sampling program.
 - Determine the number of samples to be taken.
 - Ensure that adequate equipment and supplies are available.
 - Determine the method of assay that will ensure optimal recovery of microorganisms.
 - Select a laboratory that will provide proper microbiologic support.
 - Ensure that samples can be refrigerated if they cannot be assayed in the laboratory promptly.
-

Bacteria, fungi, and particulates in air can be identified and quantified with the same methods and equipment (Table 23). The basic methods include a) impingement in liquids, b) impaction on solid surfaces, c) sedimentation, d) filtration, e) centrifugation, f) electrostatic precipitation, and g) thermal precipitation.¹²¹⁸ Of these, impingement in liquids, impaction on solid surfaces, and sedimentation (on settle plates) have been used for various air-sampling purposes in health-care settings.²⁸⁹

Several instruments are available for sampling airborne bacteria and fungi (Box 14). Some of the samplers are self-contained units requiring only a power supply and the appropriate collecting medium, but most require additional auxiliary equipment (e.g., a vacuum pump and an airflow measuring device [i.e., a flowmeter or anemometer]). Sedimentation or depositional methods use settle plates and

therefore need no special instruments or equipment. Selection of an instrument for air sampling requires a clear understanding of the type of information desired and the particular determinations that must be made (Box 14). Information may be needed regarding a) one particular organism or all organisms that may be present in the air, b) the concentration of viable particles or of viable organisms, c) the change in concentration with time, and d) the size distribution of the collected particles. Before sampling begins, decisions should be made regarding whether the results are to be qualitative or quantitative. Comparing quantities of airborne microorganisms to those of outdoor air is also standard operating procedure. Infection-control professionals, hospital epidemiologists, industrial hygienists, and laboratory supervisors, as part of a multidisciplinary team, should discuss the potential need for microbial air sampling to determine if the capacity and expertise to conduct such sampling exists within the facility and when it is appropriate to enlist the services of an environmental microbiologist consultant.

Table 23. Air sampling methods and examples of equipment*

Method	Principle	Suitable for measuring:	Collection media or surface	Rate of collection (L/min.)	Auxilliary equipment needed+	Points to consider	Prototype samplers§
Impingement in liquids	Air drawn through a small jet and directed against a liquid surface	Viable organisms, and concentration over time. Example use: sampling water aerosols to <i>Legionella</i> spp.	Buffered gelatin, tryptose saline, peptone, nutrient broth	12.5	Yes	Antifoaming agent may be needed. Ambient temperature and humidity will influence length of collection time	Chemical Corps. All Glass Impinger (AGI)
Impaction on solid surfaces	Air drawn into the sampler; particles deposited on a dry surface	Viable particles; viable organisms (on non-nutrient surfaces, limited to organisms that resist drying and spores); size measurement, and concentration over time. Example use: sampling air for <i>Aspergillus</i> spp., fungal spores	Dry surface, coated surfaces, and agar	28 (sieve) 30–800 (slit)	Yes	Available as sieve impactors or slit impactors. Sieve impactors can be set up to measure particle size. Slit impactors have a rotating support stage for agar plates to allow for measurement of concentration over time.	Andersen Air Sampler (sieve impactor); TDL, Cassella MK-2 (slit impactors)
Sedimentation	Particles and micro-organisms settle onto surfaces via gravity	Viable particles. Example uses: sampling air for bacteria in the vicinity of and during a medical procedure; general measurements of microbial air quality.	Nutrient media (agars) on plates or slides	—	No	Simple and inexpensive; best suited for qualitative sampling; significant airborne fungal spores are too buoyant to settle efficiently for collection using this method.	Settle plates

Method	Principle	Suitable for measuring:	Collection media or surface	Rate of collection (L/min.)	Auxilliary equipment needed+	Points to consider	Prototype samplers§
Filtration	Air drawn through a filter unit; particles trapped; 0.2 µm pore size	Viable particles; viable organisms (on non-nutrient surfaces, limited to spores and organisms that resist drying); concentration over time. Example use: air sampling for <i>Aspergillus</i> spp., fungal spores, and dust	Paper, cellulose, glass wool, gelatin foam, and membrane filters	1–50	Yes	Filter must be agitated first in rinse fluid to remove and disperse trapped micro-organisms; rinse fluid is assayed; used more for sampling dust and chemicals.	—
Centrifugation	Aerosols subjected to centrifugal force; particles impacted onto a solid surface	Viable particles; viable organisms (on non-nutrient surfaces, limited to spores and organisms that resist drying); concentration over time. Example use: air sampling for <i>Aspergillus</i> spp., and fungal spores	Coated glass or plastic slides, and agar surfaces	40–50	Yes	Calibration is difficult and is done only by the factory; relative comparison of airborne contamination is its general use.	Biotest RCS Plus
Electrostatic precipitation	Air drawn over an electrostatically charged surface; particles become charged	Viable particles; viable organisms (on non-nutrient surfaces, limited to spores and organisms that resist drying); concentration over time	Solid collecting surfaces (glass, and agar)	85	Yes	High volume sampling rate, but equipment is complex and must be handled carefully; not practical for use in health-care settings.	—
Thermal precipitation	Air drawn over a thermal gradient; particles repelled from hot surfaces, settle on colder surfaces	Size measurements	Glass coverslip, and electron microscope grid	0.003–0.4	Yes	Determine particle size by direct observation; not frequently used because of complex adjustments and low sampling rates.	—

* Material in this table is compiled from references 289, 1218, 1223, and 1224.

+ Most samplers require a flow meter or anemometer and a vacuum source as auxiliary equipment.

§ Trade names listed are for identification purposes only and are not intended as endorsements by the U.S. Public Health Service.

Box 14. Selecting an air sampling device*

The following factors must be considered when choosing an air sampling instrument:

- Viability and type of the organism to be sampled
- Compatibility with the selected method of analysis
- Sensitivity of particles to sampling
- Assumed concentrations and particle size
- Whether airborne clumps must be broken (i.e., total viable organism count vs. particle count)
- Volume of air to be sampled and length of time sampler is to be continuously operated
- Background contamination
- Ambient conditions
- Sampler collection efficiency
- Effort and skill required to operate sampler
- Availability and cost of sampler, plus back-up samplers in case of equipment malfunction
- Availability of auxiliary equipment and utilities (e.g., vacuum pumps, electricity, and water)

* Material in this box is compiled from reference 1218.

Liquid impinger and solid impactor samplers are the most practical for sampling bacteria, particles, and fungal spores, because they can sample large volumes of air in relatively short periods of time.²⁸⁹ Solid impactor units are available as either “slit” or “sieve” designs. Slit impactors use a rotating disc as support for the collecting surface, which allows determinations of concentration over time. Sieve impactors commonly use stages with calibrated holes of different diameters. Some impactor-type samplers use centrifugal force to impact particles onto agar surfaces. The interior of either device must be made sterile to avoid inadvertent contamination from the sampler. Results obtained from either sampling device can be expressed as organisms or particles per unit volume of air (CFU/m³).

Sampling for bacteria requires special attention, because bacteria may be present as individual organisms, as clumps, or mixed with or adhering to dust or covered with a protective coating of dried organic or inorganic substances. Reports of bacterial concentrations determined by air sampling therefore must indicate whether the results represent individual organisms or particles bearing multiple cells. Certain types of samplers (e.g., liquid impingers) will completely or partially disintegrate clumps and large particles; the sampling result will therefore reflect the total number of individual organisms present in the air.

The task of sizing a bioaerosol is simplified through the use of sieves or slit impactors because these samplers will separate the particles and microorganisms into size ranges as the sample is collected. These samplers must, however, be calibrated first by sampling aerosols under similar use conditions.¹²²⁵

The use of settle plates (i.e., the sedimentation or depositional method) is not recommended when sampling air for fungal spores, because single spores can remain suspended in air indefinitely.²⁸⁹ Settle plates have been used mainly to sample for particulates and bacteria either in research studies or during epidemiologic investigations.^{161, 1226–1229} Results of sedimentation sampling are typically expressed as numbers of viable particles or viable bacteria per unit area per the duration of sampling time (i.e., CFU/area/time); this method can not quantify the volume of air sampled. Because the survival of microorganisms during air sampling is inversely proportional to the velocity at which the air is taken into the sampler,¹²¹⁵ one advantage of using a settle plate is its reliance on gravity to bring organisms and particles into contact with its surface, thus enhancing the potential for optimal survival of collected organisms. This process, however, takes several hours to complete and may be impractical for some situations.

Air samplers are designed to meet differing measurement requirements. Some samplers are better suited for one form of measurement than others. No one type of sampler and assay procedure can be used to collect and enumerate 100% of airborne organisms. The sampler and/or sampling method chosen should, however, have an adequate sampling rate to collect a sufficient number of particles in a reasonable time period so that a representative sample of air is obtained for biological analysis. Newer analytical techniques for assaying air samples include PCR methods and enzyme-linked immunosorbent assays (ELISAs).

3. Water Sampling

A detailed discussion of the principles and practices of water sampling has been published.⁹⁴⁵ Water sampling in health-care settings is used to detect waterborne pathogens of clinical significance or to determine the quality of finished water in a facility's distribution system. Routine testing of the water in a health-care facility is usually not indicated, but sampling in support of outbreak investigations can help determine appropriate infection-control measures. Water-quality assessments in dialysis settings have been discussed in this guideline (see Water, Dialysis Water Quality and Dialysate, and Appendix C).

Health-care facilities that conduct water sampling should have their samples assayed in a laboratory that uses established methods and quality-assurance protocols. Water specimens are not "static specimens" at ambient temperature; potential changes in both numbers and types of microbial populations can occur during transport. Consequently, water samples should be sent to the testing laboratory cold (i.e., at approximately 39.2°F [4°C]) and testing should be done as soon as practical after collection (preferably within 24 hours).

Because most water sampling in health-care facilities involves the testing of finished water from the facility's distribution system, a reducing agent (i.e., sodium thiosulfate [Na₂S₂O₃]) needs to be added to neutralize residual chlorine or other halogen in the collected sample. If the water contains elevated levels of heavy metals, then a chelating agent should be added to the specimen. The minimum volume of water to be collected should be sufficient to complete any and all assays indicated; 100 mL is considered a suitable minimum volume. Sterile collection equipment should always be used.

Sampling from a tap requires flushing of the water line before sample collection. If the tap is a mixing faucet, attachments (e.g., screens and aerators) must be removed, and hot and then cold water must be run through the tap before collecting the sample.⁹⁴⁵ If the cleanliness of the tap is questionable, disinfection with 500–600 ppm sodium hypochlorite (1:100 v/v dilution of chlorine bleach) and flushing the tap should precede sample collection.

Microorganisms in finished or treated water often are physically damaged ("stressed") to the point that growth is limited when assayed under standard conditions. Such situations lead to false-negative readings and misleading assessments of water quality. Appropriate neutralization of halogens and chelation of heavy metals are crucial to the recovery of these organisms. The choice of recovery media and incubation conditions will also affect the assay. Incubation temperatures should be closer to the ambient temperature of the water rather than at 98.6°F (37°C), and recovery media should be formulated to provide appropriate concentrations of nutrients to support organisms exhibiting less than rigorous growth.⁹⁴⁵ High-nutrient content media (e.g., blood agar and tryptic soy agar [TSA]) may actually inhibit the growth of these damaged organisms. Reduced nutrient media (e.g., diluted peptone and R2A) are preferable for recovery of these organisms.⁹⁴⁵

Use of aerobic, heterotrophic plate counts allows both a qualitative and quantitative measurement for water quality. If bacterial counts in water are expected to be high in number (e.g., during waterborne outbreak investigations), assaying small quantities using pour plates or spread plates is appropriate.⁹⁴⁵ Membrane filtration is used when low-count specimens are expected and larger sampling volumes are required (≥ 100 mL). The sample is filtered through the membrane, and the filter is applied directly face-up onto the surface of the agar plate and incubated.

Unlike the testing of potable water supplies for coliforms (which uses standardized test and specimen collection parameters and conditions), water sampling to support epidemiologic investigations of disease outbreaks may be subjected to modifications dictated by the circumstances present in the facility. Assay methods for waterborne pathogens may also not be standardized. Therefore, control or comparison samples should be included in the experimental design. Any departure from a standard method should be fully documented and should be considered when interpreting results and developing strategies. Assay methods specific for clinically significant waterborne pathogens (e.g., *Legionella* spp., *Aeromonas* spp, *Pseudomonas* spp., and *Acinetobacter* spp.) are more complicated and costly compared with both methods used to detect coliforms and other standard indicators of water quality.

4. Environmental Surface Sampling

Routine environmental-surface sampling (e.g., surveillance cultures) in health-care settings is neither cost-effective nor warranted.^{951, 1225} When indicated, surface sampling should be conducted with multidisciplinary approval in adherence to carefully considered plans of action and policy (Box 15).

Box 15. Undertaking environmental-surface sampling*

The following factors should be considered before engaging in environmental-surface sampling:

- **Background information from the literature and present activities (i.e., preliminary results from an epidemiologic investigation)**
 - **Location of surfaces to be sampled**
 - **Method of sample collection and the appropriate equipment for this task**
 - **Number of replicate samples needed and which control or comparison samples are required**
 - **Parameters of the sample assay method and whether the sampling will be qualitative, quantitative, or both**
 - **An estimate of the maximum allowable microbial numbers or types on the surface(s) sampled (refer to the Spaulding classification for devices and surfaces)**
 - **Some anticipation of a corrective action plan**
-

* The material in this box is compiled from reference 1214.

Surface sampling is used currently for research, as part of an epidemiologic investigation, or as part of a comprehensive approach for specific quality assurance purposes. As a research tool, surface sampling has been used to determine a) potential environmental reservoirs of pathogens,^{564, 1230–1232} b) survival of microorganisms on surfaces,^{1232, 1233} and c) the sources of the environmental contamination.¹⁰²³ Some or all of these approaches can also be used during outbreak investigations.¹²³² Discussion of surface sampling of medical devices and instruments is beyond the scope of this document and is deferred to future guidelines on sterilization and disinfection issues.

Meaningful results depend on the selection of appropriate sampling and assay techniques.¹²¹⁴ The media, reagents, and equipment required for surface sampling are available from any well-equipped

microbiology laboratory and laboratory supplier. For quantitative assessment of surface organisms, non-selective, nutrient-rich agar media and broth (e.g., TSA and brain-heart infusion broth [BHI] with or without 5% sheep or rabbit blood supplement) are used for the recovery of aerobic bacteria. Broth media are used with membrane-filtration techniques. Further sample work-up may require the use of selective media for the isolation and enumeration of specific groups of microorganisms. Examples of selective media are MacConkey agar (MAC [selects for gram-negative bacteria]), Cetrimide agar (selects for *Pseudomonas aeruginosa*), or Sabouraud dextrose- and malt extract agars and broths (select for fungi). Qualitative determinations of organisms from surfaces require only the use of selective or non-selective broth media.

Effective sampling of surfaces requires moisture, either already present on the surface to be sampled or via moistened swabs, sponges, wipes, agar surfaces, or membrane filters.^{1214, 1234–1236} Dilution fluids and rinse fluids include various buffers or general purpose broth media (Table 24). If disinfectant residuals are expected on surfaces being sampled, specific neutralizer chemicals should be used in both the growth media and the dilution or rinse fluids. Lists of the neutralizers, the target disinfectant active ingredients, and the use concentrations have been published.^{1214, 1237} Alternatively, instead of adding neutralizing chemicals to existing culture media (or if the chemical nature of the disinfectant residuals is unknown), the use of either a) commercially available media including a variety of specific and non-specific neutralizers or b) double-strength broth media will facilitate optimal recovery of microorganisms. The inclusion of appropriate control specimens should be included to rule out both residual antimicrobial activity from surface disinfectants and potential toxicity caused by the presence of neutralizer chemicals carried over into the assay system.¹²¹⁴

Table 24. Examples of eluents and diluents for environmental-surface sampling* +

Solutions	Concentration in water
Ringer	¼ strength
Peptone water	0.1%–1.0%
Buffered peptone water	0.067 M phosphate, 0.43% NaCl, 0.1% peptone
Phosphate-buffered saline	0.02 M phosphate, 0.9% NaCl
Sodium chloride (NaCl)	0.25%–0.9%
Calgon Ringer§	¼ strength
Thiosulfate Ringer¶	¼ strength
Water	–
Tryptic soy broth (TSB)	–
Brain-heart infusion broth (BHI) supplemented with 0.5% beef extract	–

* Material in this table is compiled from references 1214 and 1238.

+ A surfactant (e.g., polysorbate [i.e., Tween® 80]) may be added to eluents and diluents. A concentration ranging from 0.01%–0.1% is generally used, depending on the specific application. Foaming may occur during use.

§ This solution is used for dissolution of calcium alginate swabs.

¶ This solution is used for neutralization of residual chlorine.

Several methods can be used for collecting environmental surface samples (Table 25). Specific step-by-step discussions of each of the methods have been published.^{1214, 1239} For best results, all methods should incorporate aseptic techniques, sterile equipment, and sterile recovery media.

Table 25. Methods of environmental-surface sampling

Method	Suitable for appropriate surface(s)	Assay technique	Procedural notes	Points of interpretation	Available standards	References
Sample/rinse Moistened swab/rinse	Non-absorbent surfaces, corners, crevices, devices, and instruments	Dilutions; qualitative or quantitative assays	Assay multiple measures areas or devices with separate swabs	Report results per measured areas or if assaying an object, per the entire sample site	YES – food industry; NO – health care	1214, 1239–1242
Moistened sponge/rinse	Large areas and housekeeping surfaces (e.g., floors or walls)	Dilutions; qualitative or quantitative assays	Vigorously rub a sterile sponge over the surface	Report results per measured area	YES – food industry; NO – health care	1214, 1239–1242
Moistened wipe/rinse	Large areas and housekeeping surfaces (e.g., countertops)	Dilutions; qualitative or quantitative assays	Use a sterile wipe	Report results per measured area	YES – food industry; NO – health care	1214, 1239–1242
Direct immersion	Small items capable of being immersed	Dilutions; qualitative or quantitative assays	Use membrane filtration if rinse volume is large and anticipated microbiological concentration is low	Report results per item	NO	1214
Containment	Interior surfaces of containers, tubes, or bottles	Dilutions; qualitative or quantitative assays	Use membrane filtration if rinse volume is large	Evaluate both the types and numbers of microorganisms	YES – food and industrial applications for containers prior to fill	1214
RODAC*	Previously cleaned and sanitized flat, non-absorbent surfaces; not suitable for irregular surfaces	Direct assay	Overgrowth occurs if used on heavily contaminated surfaces; use neutralizers in the agar if surface disinfectant residuals are present	Provides direct, quantitative results; use a minimum of 15 plates per an average hospital room	NO	1214, 1237, 1239, 1243, 1244

* RODAC stands for “replicate organism direct agar contact.”

Sample/rinse methods are frequently chosen because of their versatility. However, these sampling methods are the most prone to errors caused by manipulation of the swab, gauze pad, or sponge.¹²³⁸ Additionally, no microbiocidal or microbiostatic agents should be present in any of these items when used for sampling.¹²³⁸ Each of the rinse methods requires effective elution of microorganisms from the item used to sample the surface. Thorough mixing of the rinse fluids after elution (e.g., via manual or mechanical mixing using a vortex mixer, shaking with or without glass beads, and ultrasonic bath) will help to remove and suspend material from the sampling device and break up clumps of organisms for a more accurate count.¹²³⁸ In some instances, the item used to sample the surface (e.g., gauze pad and sponge) may be immersed in the rinse fluids in a sterile bag and subjected to stomaching.¹²³⁸ This technique, however, is suitable only for soft or absorbent items that will not puncture the bag during the elution process.

If sampling is conducted as part of an epidemiologic investigation of a disease outbreak, identification of isolates to species level is mandatory, and characterization beyond the species level is preferred.¹²¹⁴ When interpreting the results of the sampling, the expected degree of microbial contamination

associated with the various categories of surfaces in the Spaulding classification must be considered. Environmental surfaces should be visibly clean; recognized pathogens in numbers sufficient to result in secondary transfer to other animate or inanimate surfaces should be absent from the surface being sampled.¹²¹⁴ Although the interpretation of a sample with positive microbial growth is self-evident, an environmental surface sample, especially that obtained from housekeeping surfaces, that shows no growth does not represent a “sterile” surface. Sensitivities of the sampling and assay methods (i.e., level of detection) must be taken into account when no-growth samples are encountered. Properly collected control samples will help rule out extraneous contamination of the surface sample.

G. Laundry and Bedding

1. General Information

Laundry in a health-care facility may include bed sheets and blankets, towels, personal clothing, patient apparel, uniforms, scrub suits, gowns, and drapes for surgical procedures.¹²⁴⁵ Although contaminated textiles and fabrics in health-care facilities can be a source of substantial numbers of pathogenic microorganisms, reports of health-care–associated diseases linked to contaminated fabrics are so few in number that the overall risk of disease transmission during the laundry process likely is negligible. When the incidence of such events are evaluated in the context of the volume of items laundered in health-care settings (estimated to be 5 billion pounds annually in the United States),¹²⁴⁶ existing control measures (e.g., standard precautions) are effective in reducing the risk of disease transmission to patients and staff. Therefore, use of current control measures should be continued to minimize the contribution of contaminated laundry to the incidence of health-care–associated infections. The control measures described in this section of the guideline are based on principles of hygiene, common sense, and consensus guidance; they pertain to laundry services utilized by health-care facilities, either in-house or contract, rather than to laundry done in the home.

2. Epidemiology and General Aspects of Infection Control

Contaminated textiles and fabrics often contain high numbers of microorganisms from body substances, including blood, skin, stool, urine, vomitus, and other body tissues and fluids. When textiles are heavily contaminated with potentially infective body substances, they can contain bacterial loads of 10^6 – 10^8 CFU/100 cm² of fabric.¹²⁴⁷ Disease transmission attributed to health-care laundry has involved contaminated fabrics that were handled inappropriately (i.e., the shaking of soiled linens). Bacteria (*Salmonella* spp., *Bacillus cereus*), viruses (hepatitis B virus [HBV]), fungi (*Microsporium canis*), and ectoparasites (scabies) presumably have been transmitted from contaminated textiles and fabrics to workers via a) direct contact or b) aerosols of contaminated lint generated from sorting and handling contaminated textiles.^{1248–1252} In these events, however, investigations could not rule out the possibility that some of these reported infections were acquired from community sources. Through a combination of soil removal, pathogen removal, and pathogen inactivation, contaminated laundry can be rendered hygienically clean. Hygienically clean laundry carries negligible risk to health-care workers and patients, provided that the clean textiles, fabric, and clothing are not inadvertently contaminated before use.

OSHA defines contaminated laundry as “laundry which has been soiled with blood or other potentially infectious materials or may contain sharps.”⁹⁶⁷ The purpose of the laundry portion of the standard is to protect the worker from exposure to potentially infectious materials during collection, handling, and sorting of contaminated textiles through the use of personal protective equipment, proper work practices, containment, labeling, hazard communication, and ergonomics.

Experts are divided regarding the practice of transporting clothes worn at the workplace to the health-care worker's home for laundering. Although OSHA regulations prohibit home laundering of items that are considered personal protective apparel or equipment (e.g., laboratory coats),⁹⁶⁷ experts disagree about whether this regulation extends to uniforms and scrub suits that are not contaminated with blood or other potentially infectious material. Health-care facility policies on this matter vary and may be inconsistent with recommendations of professional organizations.^{1253, 1254} Uniforms without blood or body substance contamination presumably do not differ appreciably from street clothes in the degree and microbial nature of soilage. Home laundering would be expected to remove this level of soil adequately. However, if health-care facilities require the use of uniforms, they should either make provisions to launder them or provide information to the employee regarding infection control and cleaning guidelines for the item based on the tasks being performed at the facility. Health-care facilities should address the need to provide this service and should determine the frequency for laundering these items. In a recent study examining the microbial contamination of medical students' white coats, the students perceived the coats as "clean" as long as the garments were not visibly contaminated with body substances, even after wearing the coats for several weeks.¹²⁵⁵ The heaviest bacterial load was found on the sleeves and the pockets of these garments; the organisms most frequently isolated were *Staphylococcus aureus*, diphtheroids, and *Acinetobacter* spp.¹²⁵⁵ Presumably, the sleeves of the coat may make contact with a patient and potentially serve to transfer environmentally stable microorganisms among patients. In this study, however, surveillance was not conducted among patients to detect new infections or colonizations. The students did, however, report that they would likely replace their coats more frequently and regularly if clean coats were provided.¹²⁵⁵ Apart from this study, which documents the presence of pathogenic bacteria on health-care facility clothing, reports of infections attributed to either the contact with such apparel or with home laundering have been rare.^{1256, 1257}

Laundry services for health-care facilities are provided either in-house (i.e., on-premise laundry [OPL]), co-operatives (i.e., those entities owned and operated by a group of facilities), or by off-site commercial laundries. In the latter, the textiles may be owned by the health-care facility, in which case the processor is paid for laundering only. Alternatively, the textiles may be owned by the processor who is paid for every piece laundered on a "rental" fee. The laundry facility in a health-care setting should be designed for efficiency in providing hygienically clean textiles, fabrics, and apparel for patients and staff. Guidelines for laundry construction and operation for health-care facilities, including nursing facilities, have been published.^{120, 1258} The design and engineering standards for existing facilities are those cited in the AIA edition in effect during the time of the facility's construction.¹²⁰ A laundry facility is usually partitioned into two separate areas - a "dirty" area for receiving and handling the soiled laundry and a "clean" area for processing the washed items.¹²⁵⁹ To minimize the potential for recontaminating cleaned laundry with aerosolized contaminated lint, areas receiving contaminated textiles should be at negative air pressure relative to the clean areas.¹²⁶⁰⁻¹²⁶² Laundry areas should have handwashing facilities readily available to workers. Laundry workers should wear appropriate personal protective equipment (e.g., gloves and protective garments) while sorting soiled fabrics and textiles.⁹⁶⁷ Laundry equipment should be used and maintained according to the manufacturer's instructions to prevent microbial contamination of the system.^{1250, 1263} Damp textiles should not be left in machines overnight.¹²⁵⁰

3. Collecting, Transporting, and Sorting Contaminated Textiles and Fabrics

The laundry process starts with the removal of used or contaminated textiles, fabrics, and/or clothing from the areas where such contamination occurred, including but not limited to patients' rooms, surgical/operating areas, and laboratories. Handling contaminated laundry with a minimum of agitation

can help prevent the generation of potentially contaminated lint aerosols in patient-care areas.^{967, 1259} Sorting or rinsing contaminated laundry at the location where contamination occurred is prohibited by OSHA.⁹⁶⁷ Contaminated textiles and fabrics are placed into bags or other appropriate containment in this location; these bags are then securely tied or otherwise closed to prevent leakage.⁹⁶⁷ Single bags of sufficient tensile strength are adequate for containing laundry, but leak-resistant containment is needed if the laundry is wet and capable of soaking through a cloth bag.¹²⁶⁴ Bags containing contaminated laundry must be clearly identified with labels, color-coding, or other methods so that health-care workers handle these items safely, regardless of whether the laundry is transported within the facility or destined for transport to an off-site laundry service.⁹⁶⁷

Typically, contaminated laundry originating in isolation areas of the hospital is segregated and handled with special practices; however, few, if any, cases of health-care-associated infection have been linked to this source.¹²⁶⁵ Single-blinded studies have demonstrated that laundry from isolation areas is no more heavily contaminated with microorganisms than laundry from elsewhere in the hospital.¹²⁶⁶ Therefore, adherence to standard precautions when handling contaminated laundry in isolation areas and minimizing agitation of the contaminated items are considered sufficient to prevent the dispersal of potentially infectious aerosols.⁶

Contaminated textiles and fabrics in bags can be transported by cart or chute.^{1258, 1262} Laundry chutes require proper design, maintenance, and use, because the piston-like action of a laundry bag traveling in the chute can propel airborne microbial contaminants throughout the facility.^{1267–1269} Laundry chutes should be maintained under negative air pressure to prevent the spread of microorganisms from floor to floor. Loose, contaminated pieces of laundry should not be tossed into chutes, and laundry bags should be closed or otherwise secured to prevent the contents from falling out into the chute.¹²⁷⁰ Health-care facilities should determine the point in the laundry process at which textiles and fabrics should be sorted. Sorting after washing minimizes the exposure of laundry workers to infective material in soiled fabrics, reduces airborne microbial contamination in the laundry area, and helps to prevent potential percutaneous injuries to personnel.¹²⁷¹ Sorting laundry before washing protects both the machinery and fabrics from hard objects (e.g., needles, syringes, and patients' property) and reduces the potential for recontamination of clean textiles.¹²⁷² Sorting laundry before washing also allows for customization of laundry formulas based on the mix of products in the system and types of soils encountered. Additionally, if work flow allows, increasing the amount of segregation by specific product types will usually yield the greatest amount of work efficiency during inspection, folding, and pack-making operations.¹²⁵³ Protective apparel for the workers and appropriate ventilation can minimize these exposures.^{967, 1258–1260} Gloves used for the task of sorting laundry should be of sufficient thickness to minimize sharps injuries.⁹⁶⁷ Employee safety personnel and industrial hygienists can help to determine the appropriate glove choice.

4. Parameters of the Laundry Process

Fabrics, textiles, and clothing used in health-care settings are disinfected during laundering and generally rendered free of vegetative pathogens (i.e., hygienically clean), but they are not sterile.¹²⁷³ Laundering cycles consist of flush, main wash, bleaching, rinsing, and souring.¹²⁷⁴ Cleaned wet textiles, fabrics, and clothing are then dried, pressed as needed, and prepared (e.g., folded and packaged) for distribution back to the facility. Clean linens provided by an off-site laundry must be packaged prior to transport to prevent inadvertent contamination from dust and dirt during loading, delivery, and unloading. Functional packaging of laundry can be achieved in several ways, including a) placing clean linen in a hamper lined with a previously unused liner, which is then closed or covered; b) placing clean linen in a properly cleaned cart and covering the cart with disposable material or a properly cleaned reusable textile material that can be secured to the cart; and c) wrapping individual bundles of clean

textiles in plastic or other suitable material and sealing or taping the bundles.

The antimicrobial action of the laundering process results from a combination of mechanical, thermal, and chemical factors.^{1271, 1275, 1276} Dilution and agitation in water remove substantial quantities of microorganisms. Soaps and detergents function to suspend soils and also exhibit some microbiocidal properties. Hot water provides an effective means of destroying microorganisms.¹²⁷⁷ A temperature of at least 160°F (71°C) for a minimum of 25 minutes is commonly recommended for hot-water washing.² Water of this temperature can be provided by steam jet or separate booster heater.¹²⁰ The use of chlorine bleach assures an extra margin of safety.^{1278, 1279} A total available chlorine residual of 50–150 ppm is usually achieved during the bleach cycle.¹²⁷⁷ Chlorine bleach becomes activated at water temperatures of 135°F–145°F (57.2°C–62.7°C). The last of the series of rinse cycles is the addition of a mild acid (i.e., sour) to neutralize any alkalinity in the water supply, soap, or detergent. The rapid shift in pH from approximately 12 to 5 is an effective means to inactivate some microorganisms.¹²⁴⁷ Effective removal of residual alkali from fabrics is an important measure in reducing the risk for skin reactions among patients.

Chlorine bleach is an economical, broad-spectrum chemical germicide that enhances the effectiveness of the laundering process. Chlorine bleach is not, however, an appropriate laundry additive for all fabrics. Traditionally, bleach was not recommended for laundering flame-retardant fabrics, linens, and clothing because its use diminished the flame-retardant properties of the treated fabric.¹²⁷³ However, some modern-day flame retardant fabrics can now tolerate chlorine bleach. Flame-retardant fabrics, whether topically treated or inherently flame retardant, should be thoroughly rinsed during the rinse cycles, because detergent residues are capable of supporting combustion. Chlorine alternatives (e.g., activated oxygen-based laundry detergents) provide added benefits for fabric and color safety in addition to antimicrobial activity. Studies comparing the antimicrobial potencies of chlorine bleach and oxygen-based bleach are needed. Oxygen-based bleach and detergents used in health-care settings should be registered by EPA to ensure adequate disinfection of laundry. Health-care workers should note the cleaning instructions of textiles, fabrics, drapes, and clothing to identify special laundering requirements and appropriate hygienic cleaning options.¹²⁷⁸

Although hot-water washing is an effective laundry disinfection method, the cost can be substantial. Laundries are typically the largest users of hot water in hospitals. They consume 50%–75% of the total hot water,¹²⁸⁰ representing an average of 10%–15% of the energy used by a hospital. Several studies have demonstrated that lower water temperatures of 71°F–77°F (22°C–25°C) can reduce microbial contamination when the cycling of the washer, the wash detergent, and the amount of laundry additive are carefully monitored and controlled.^{1247, 1281–1285} Low-temperature laundry cycles rely heavily on the presence of chlorine- or oxygen-activated bleach to reduce the levels of microbial contamination. The selection of hot- or cold-water laundry cycles may be dictated by state health-care facility licensing standards or by other regulation. Regardless of whether hot or cold water is used for washing, the temperatures reached in drying and especially during ironing provide additional significant microbiocidal action.¹²⁴⁷ Dryer temperatures and cycle times are dictated by the materials in the fabrics. Man-made fibers (i.e., polyester and polyester blends) require shorter times and lower temperatures.

After washing, cleaned and dried textiles, fabrics, and clothing are pressed, folded, and packaged for transport, distribution, and storage by methods that ensure their cleanliness until use.² State regulations and/or accrediting standards may dictate the procedures for this activity. Clean/sterile and contaminated textiles should be transported from the laundry to the health-care facility in vehicles (e.g., trucks, vans, and carts) that allow for separation of clean/sterile and contaminated items. Clean/sterile textiles and contaminated textiles may be transported in the same vehicle, provided that the use of physical barriers and/or space separation can be verified to be effective in protecting the clean/sterile items from

contamination. Clean, uncovered/unwrapped textiles stored in a clean location for short periods of time (e.g., uncovered and used within a few hours) have not been demonstrated to contribute to increased levels of health-care–acquired infection. Such textiles can be stored in convenient places for use during the provision of care, provided that the textiles can be maintained dry and free from soil and body-substance contamination.

In the absence of microbiologic standards for laundered textiles, no rationale exists for routine microbiologic sampling of cleaned health-care textiles and fabrics.¹²⁸⁶ Sampling may be used as part of an outbreak investigation if epidemiologic evidence suggests that textiles, fabrics, or clothing are a suspected vehicle for disease transmission. Sampling techniques include aseptically macerating the fabric into pieces and adding these to broth media or using contact plates (RODAC plates) for direct surface sampling.^{1271, 1286} When evaluating the disinfecting properties of the laundering process specifically, placing pieces of fabric between two membrane filters may help to minimize the contribution of the physical removal of microorganisms.¹²⁸⁷

Washing machines and dryers in residential-care settings are more likely to be consumer items rather than the commercial, heavy-duty, large volume units typically found in hospitals and other institutional health-care settings. Although all washing machines and dryers in health-care settings must be properly maintained for performance according to the manufacturer's instructions, questions have been raised about the need to disinfect washers and dryers in residential-care settings. Disinfection of the tubs and tumblers of these machines is unnecessary when proper laundry procedures are followed; these procedures involve a) the physical removal of bulk solids (e.g., feces) before the wash/dry cycle and b) proper use of temperature, detergent, and laundry additives. Infection has not been linked to laundry procedures in residential-care facilities, even when consumer versions of detergents and laundry additives are used.

5. Special Laundry Situations

Some textile items (e.g., surgical drapes and reusable gowns) must be sterilized before use and therefore require steam autoclaving after laundering.⁷ Although the American Academy of Pediatrics in previous guidelines recommended autoclaving for linens in neonatal intensive care units (NICUs), studies on the microbial quality of routinely cleaned NICU linen have not identified any increased risk for infection among the neonates receiving care.¹²⁸⁸ Consequently, hygienically clean linens are suitable for use in this setting.⁹⁹⁷ The use of sterile linens in burn therapy units remains unresolved.

Coated or laminated fabrics are often used in the manufacture of PPE. When these items become contaminated with blood or other body substances, the manufacturer's instructions for decontamination and cleaning take into account the compatibility of the rubber backing with the chemical germicides or detergents used in the process. The directions for decontaminating these items should be followed as indicated; the item should be discarded when the backing develops surface cracks.

Dry cleaning, a cleaning process that utilizes organic solvents (e.g., perchloroethylene) for soil removal, is an alternative means of cleaning fabrics that might be damaged in conventional laundering and detergent washing. Several studies, however, have shown that dry cleaning alone is relatively ineffective in reducing the numbers of bacteria and viruses on contaminated linens;^{1289, 1290} microbial populations are significantly reduced only when dry-cleaned articles are heat pressed. Dry cleaning should therefore not be considered a routine option for health-care facility laundry and should be reserved for those circumstances in which fabrics can not be safely cleaned with water and detergent.¹²⁹¹

6. Surgical Gowns, Drapes, and Disposable Fabrics

An issue of recent concern involves the use of disposable (i.e., single use) versus reusable (i.e., multiple use) surgical attire and fabrics in health-care settings.¹²⁹² Regardless of the material used to manufacture gowns and drapes, these items must be resistant to liquid and microbial penetration.^{7, 1293–1297} Surgical gowns and drapes must be registered with FDA to demonstrate their safety and effectiveness. Repellency and pore size of the fabric contribute to gown performance, but performance capability can be influenced by the item's design and construction.^{1298, 1299} Reinforced gowns (i.e., gowns with double-layered fabric) generally are more resistant to liquid strike-through.^{1300, 1301} Reinforced gowns may, however, be less comfortable. Guidelines for selection and use of barrier materials for surgical gowns and drapes have been published.¹³⁰² When selecting a barrier product, repellency level and type of barrier should be compatible for the exposure expected.⁹⁶⁷ However, data are limited regarding the association between gown or drape characteristics and risk for surgical site infections.^{7, 1303} Health-care facilities must ensure optimal protection of patients and health-care workers. Not all fabric items in health care lend themselves to single-use. Facilities exploring options for gowns and drapes should consider the expense of disposable items and the impact on the facility's waste-management costs once these items are discarded. Costs associated with the use of durable goods involve the fabric or textile items; staff expenses to collect, sort, clean, and package the laundry; and energy costs to operate the laundry if on-site or the costs to contract with an outside service.^{1304, 1305}

7. Antimicrobial-Impregnated Articles and Consumer Items Bearing Antimicrobial Labeling

Manufacturers are increasingly incorporating antibacterial or antimicrobial chemicals into consumer and health-care items. Some consumer products bearing labels that indicate treatment with antimicrobial chemicals have included pens, cutting boards, toys, household cleaners, hand lotions, cat litter, soaps, cotton swabs, toothbrushes, and cosmetics. The “antibacterial” label on household cleaning products, in particular, gives consumers the impression that the products perform “better” than comparable products without this labeling, when in fact all household cleaners have antibacterial properties.

In the health-care setting, treated items may include children's pajamas, mattresses, and bed linens with label claims of antimicrobial properties. These claims require careful evaluation to determine whether they pertain to the use of antimicrobial chemicals as preservatives for the fabric or other components or whether they imply a health claim.^{1306, 1307} No evidence is available to suggest that use of these products will make consumers and patients healthier or prevent disease. No data support the use of these items as part of a sound infection-control strategy, and therefore, the additional expense of replacing a facility's bedding and sheets with these treated products is unwarranted.

EPA has reaffirmed its position that manufacturers who make public health claims for articles containing antimicrobial chemicals must provide evidence to support those claims as part of the registration process.¹³⁰⁸ Current EPA regulations outlined in the Treated Articles Exemption of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) require manufacturers to register both the antimicrobial chemical used in or on the product and the finished product itself if a public health claim is maintained for the item. The exemption applies to the use of antimicrobial chemicals for the purpose of preserving the integrity of the product's raw material(s). The U.S. Federal Trade Commission (FTC) is evaluating manufacturer advertising of products with antimicrobial claims.¹³⁰⁹

8. Standard Mattresses, Pillows, and Air-Fluidized Beds

Standard mattresses and pillows can become contaminated with body substances during patient care if the integrity of the covers of these items is compromised. The practice of sticking needles into the mattress should be avoided. A mattress cover is generally a fitted, protective material, the purpose of which is to prevent the mattress from becoming contaminated with body fluids and substances. A linen sheet placed on the mattress is not considered a mattress cover. Patches for tears and holes in mattress covers do not provide an impermeable surface over the mattress. Mattress covers should be replaced when torn; the mattress should be replaced if it is visibly stained. Wet mattresses, in particular, can be a substantial environmental source of microorganisms. Infections and colonizations caused by *Acinetobacter* spp., MRSA, and *Pseudomonas aeruginosa* have been described, especially among burn patients.^{1310–1315} In these reports, the removal of wet mattresses was an effective infection-control measure. Efforts were made to ensure that pads and covers were cleaned and disinfected between patients using disinfectant products compatible with mattress-cover materials to ensure that these covers remained impermeable to fluids.^{1310–1314} Pillows and their covers should be easily cleanable, preferably in a hot water laundry cycle.¹³¹⁵ These should be laundered between patients or if contaminated with body substances.

Air-fluidized beds are used for the care of patients immobilized for extended periods of time because of therapy or injury (e.g., pain, decubitus ulcers, and burns).¹³¹⁶ These specialized beds consist of a base unit filled with microsphere beads fluidized by warm, dry air flowing upward from a diffuser located at the bottom of the unit. A porous, polyester filter sheet separates the patient from direct contact with the beads but allows body fluids to pass through to the beads. Moist beads aggregate into clumps which settle to the bottom where they are removed as part of routine bed maintenance.

Because the beads become contaminated with the patient's body substances, concerns have been raised about the potential for these beds to serve as an environmental source of pathogens. Certain pathogens (e.g., *Enterococcus* spp., *Serratia marcescens*, *Staphylococcus aureus*, and *Streptococcus fecalis*) have been recovered either from the microsphere beads or the polyester sheet after cleaning.^{1317, 1318} Reports of cross-contamination of patients, however, are few.¹³¹⁸ Nevertheless, routine maintenance and between-patient decontamination procedures can minimize potential risks to patients. Regular removal of bead clumps, coupled with the warm, dry air of the bed, can help to minimize bacterial growth in the unit.^{1319–1321} Beads are decontaminated between patients by high heat (113°F–194°F [45°C–90°C], depending on the manufacturer's specifications) for at least 1 hour; this procedure is particularly important for the inactivation of *Enterococcus* spp. which are relatively resistant to heat.^{1322, 1323} The polyester filter sheet requires regular changing and thorough cleaning and disinfection, especially between patients.^{1317, 1318, 1322, 1323}

Microbial contamination of the air space in the immediate vicinity of a properly maintained air-fluidized bed is similar to that found in air around conventional bedding, despite the air flow out of the base unit and around the patient.^{1320, 1324, 1325} An operational air-fluidized bed can, however, interfere with proper pressure differentials, especially in negative-pressure rooms;¹³²⁶ the effect varies with the location of the bed relative to the room's configuration and supply and exhaust vent locations. Use of an air-fluidized bed in a negative-pressure room requires consultation with a facility engineer to determine appropriate placement of the bed.

H. Animals in Health-Care Facilities

1. General Information

Animals in health-care facilities traditionally have been limited to laboratories and research areas. However, their presence in patient-care areas is now more frequent, both in acute-care and long-term care settings, prompting consideration for the potential transmission of zoonotic pathogens from animals to humans in these settings. Although dogs and cats may be commonly encountered in health-care settings, other animals (e.g., fish, birds, non-human primates, rabbits, rodents, and reptiles) also can be present as research, resident, or service animals. These animals can serve as sources of zoonotic pathogens that could potentially infect patients and health-care workers (Table 26).¹³²⁷⁻¹³⁴⁰ Animals potentially can serve as reservoirs for antibiotic-resistant microorganisms, which can be introduced to the health-care setting while the animal is present. VRE have been isolated from both farm animals and pets,¹³⁴¹ and a cat in a geriatric care center was found to be colonized with MRSA.¹³⁴²

Table 26. Examples of diseases associated with zoonotic transmission*+

Infectious disease	Cats	Dogs	Fish	Birds	Rabbits	Reptiles§	Primates	Rodents§
Virus								
Lymphocytic choriomeningitis								+¶
Rabies	+	+						
Bacteria								
Campylobacteriosis	+	+				+	+	+
<i>Capnocytophaga canimorsus</i> infection	+	+						
Cat scratch disease (<i>Bartonella henselae</i>)	+							
Leptospirosis	+						+	+
Mycobacteriosis			+	+				
Pasteurellosis	+	+			+			
Plague	+			+			+	+
Psittacosis				+				
Q fever (<i>Coxiella burnetti</i>)	+							
Rat bite fever (<i>Spirillum minus</i> , <i>Streptobacillus moniliformis</i>)								+
Salmonellosis	+	+		+	+	+	+	+
Tularemia	+				+			+
Yersiniosis					+	+	+	+
Parasites								
Ancylostomiasis	+	+					+	
Cryptosporidiosis	+							
Giardiasis	+	+					+	
Toxocariasis	+	+					+	
Toxoplasmosis	+	+					+	
Fungi								
Blastomycosis		+						
Dermatophytosis		+			+		+	+

* Material in this table is adapted from reference 1331 and used with permission of the publisher (Lippincott Williams and Wilkins).

+ This table does not include vectorborne diseases.

§ Reptiles include lizards, snakes, and turtles. Rodents include hamsters, mice, and rats.

¶ The + symbol indicates that the pathogen associated with the infection has been isolated from animals and is considered to pose potential risk to humans.

Zoonoses can be transmitted from animals to humans either directly or indirectly via bites, scratches, aerosols, ectoparasites, accidental ingestion, or contact with contaminated soil, food, water, or unpasteurized milk.^{1331, 1332, 1343–1345} Colonization and hand transferral of pathogens acquired from pets in health-care workers' homes represent potential sources and modes of transmission of zoonotic pathogens in health-care settings. An outbreak of infections caused by a yeast (*Malassezia pachydermatis*) among newborns was traced to transfer of the yeast from the hands of health-care workers with pet dogs at home.¹³⁴⁶ In addition, an outbreak of ringworm in a NICU caused by *Microsporium canis* was associated with a nurse and her cat,¹³⁴⁷ and an outbreak of *Rhodococcus (Gordona) bronchialis* sternal SSIs after coronary-artery bypass surgery was traced to a colonized nurse whose dogs were culture-positive for the organism.¹³⁴⁸ In the latter outbreak, whether the dogs were the sole source of the organism and whether other environmental reservoirs contributed to the outbreak are unknown. Nonetheless, limited data indicate that outbreaks of infectious disease have occurred as a result of contact with animals in areas housing immunocompetent patients. However, the low frequency of outbreaks may result from a) the relatively limited presence of the animals in health-care facilities and b) the immunocompetency of the patients involved in the encounters. Formal scientific studies to evaluate potential risks of transmission of zoonoses in health-care settings outside of the laboratory are lacking.

2. Animal-Assisted Activities, Animal-Assisted Therapy, and Resident Animals

Animal-Assisted Activities (AAA) are those programs that enhance the patients' quality of life. These programs allow patients to visit animals in either a common, central location in the facility or in individual patient rooms. A group session with the animals enhances opportunities for ambulatory patients and facility residents to interact with caregivers, family members, and volunteers.^{1349–1351} Alternatively, allowing the animals access to individual rooms provides the same opportunity to non-ambulatory patients and patients for whom privacy or dignity issues are a consideration. The decision to allow this access to patients' rooms should be made on a case-by-case basis, with the consultation and consent of the attending physician and nursing staff.

Animal-Assisted Therapy (AAT) is a goal-directed intervention that incorporates an animal into the treatment process provided by a credentialed therapist.^{1330, 1331} The concept for AAT arose from the observation that some patients with pets at home recover from surgical and medical procedures more rapidly than patients without pets.^{1352, 1353} Contact with animals is considered beneficial for enhancing wellness in certain patient populations (e.g., children, the elderly, and extended-care hospitalized patients).^{1349, 1354–1357} However, evidence supporting this benefit is largely derived from anecdotal reports and observations of patient/animal interactions.^{1357–1359} Guidelines for establishing AAT programs are available for facilities considering this option.¹³⁶⁰

The incorporation of non-human primates into an AAA or AAT program is not encouraged because of concerns regarding potential disease transmission from and unpredictable behavior of these animals.^{1361, 1362} Animals participating in either AAA or AAT sessions should be in good health and up-to-date with recommended immunizations and prophylactic medications (e.g., heartworm prevention) as determined by a licensed veterinarian based on local needs and recommendations. Regular re-evaluation of the animal's health and behavior status is essential.¹³⁶⁰ Animals should be routinely screened for enteric parasites and/or have evidence of a recently completed antihelminthic regimen.¹³⁶³ They should also be free of ectoparasites (e.g., fleas and ticks) and should have no sutures, open wounds, or obvious dermatologic lesions that could be associated with bacterial, fungal, or viral infections or parasitic infestations. Incorporating young animals (i.e., those aged <1 year) into these programs is not encouraged because of issues regarding unpredictable behavior and elimination control. Additionally,

the immune systems of very young puppies and kittens is not completely developed, thereby placing the health of these animals at risk. Animals should be clean and well-groomed. The visits must be supervised by persons who know the animals and their behavior. Animal handlers should be trained in these activities and receive site-specific orientation to ensure that they work efficiently with the staff in the specific health-care environment.¹³⁶⁰ Additionally, animal handlers should be in good health.¹³⁶⁰

The most important infection-control measure to prevent potential disease transmission is strict enforcement of hand-hygiene measures (e.g., using either soap and water or an alcohol-based hand rub) for all patients, staff, and residents after handling the animals.^{1355, 1364} Care should also be taken to avoid direct contact with animal urine or feces. Clean-up of these substances from environmental surfaces requires gloves and the use of leak-resistant plastic bags to discard absorbent material used in the process.² The area must be cleaned after visits according to standard cleaning procedures.

The American Academy of Allergy, Asthma, and Immunology estimates that dog or cat allergies occur in approximately 15% of the population.¹³⁶⁵ Minimizing contact with animal saliva, dander, and/or urine helps to mitigate allergic responses.^{1365–1367} Some facilities may not allow animal visitation for patients with a) underlying asthma, b) known allergies to cat or dog hair, c) respiratory allergies of unknown etiology, and d) immunosuppressive disorders. Hair shedding can be minimized by processes that remove dead hair (e.g., grooming) and that prevent the shedding of dead hair (e.g., therapy capes for dogs). Allergens can be minimized by bathing therapy animals within 24 hours of a visit.^{1333, 1368}

Animal therapists and handlers must take precautions to prevent animal bites. Common pathogens associated with animal bites include *Capnocytophaga canimorsus*, *Pasteurella* spp., *Staphylococcus* spp., and *Streptococcus* spp. Selecting well-behaved and well-trained animals for these programs greatly decreases the incidence of bites. Rodents, exotic species, wild/domestic animals (i.e., wolf-dog hybrids), and wild animals whose behavior is unpredictable should be excluded from AAA or AAT programs. A well-trained animal handler should be able to recognize stress in the animal and to determine when to terminate a session to minimize risk. When an animal bites a person during AAA or AAT, the animal is to be permanently removed from the program. If a bite does occur, the wound must be cleansed immediately and monitored for subsequent infection. Most infections can be treated with antibiotics, and antibiotics often are prescribed prophylactically in these situations.

The health-care facility's infection-control staff should participate actively in planning for and coordinating AAA and AAT sessions. Many facilities do not offer AAA or AAT programs for severely immunocompromised patients (e.g., HSCT patients and patients on corticosteroid therapy).¹³³⁹ The question of whether family pets or companion animals can visit terminally-ill HSCT patients or other severely immunosuppressed patients is best handled on a case-by-case basis, although animals should not be brought into the HSCT unit or any other unit housing severely immunosuppressed patients. An in-depth discussion of this issue is presented elsewhere.¹³⁶⁶

Immunocompromised patients who have been discharged from a health-care facility may be at higher risk for acquiring some pet-related zoonoses. Although guidelines have been developed to minimize the risk of disease transmission to HIV-infected patients,⁸ these recommendations may be applicable for patients with other immunosuppressive disorders. In addition to handwashing or hand hygiene, these recommendations include avoiding contact with a) animal feces and soiled litter box materials, b) animals with diarrhea, c) very young animals (i.e., dogs <6 months of age and cats <1 year of age), and d) exotic animals and reptiles.⁸ Pets or companion animals with diarrhea should receive veterinary care to resolve their condition.

Many health-care facilities are adopting more home-like environments for residential-care or extended-stay patients in acute-care settings, and resident animals are one element of this approach.¹³⁶⁹ One

concept, the “Eden Alternative,” incorporates children, plants, and animals (e.g., dogs, cats, fish, birds, rabbits, and rodents) into the daily care setting.^{1370, 1371} The concept of working with resident animals has not been scientifically evaluated. Several issues beyond the benefits of therapy must be considered before embarking on such a program, including a) whether the animals will come into direct contact with patients and/or be allowed to roam freely in the facility; b) how the staff will provide care for the animals; c) the management of patients’ or residents’ allergies, asthma, and phobias; d) precautionary measures to prevent bites and scratches; and e) measures to properly manage the disposal of animal feces and urine, thereby preventing environmental contamination by zoonotic microorganisms (e.g., *Toxoplasma* spp., *Toxocara* spp., and *Ancylostoma* spp.).^{1372, 1373} Few data document a link between health-care–acquired infection rates and frequency of cleaning fish tanks or rodent cages. Skin infections caused by *Mycobacterium marinum* have been described among persons who have fish aquariums at home.^{1374, 1375} Nevertheless, immunocompromised patients should avoid direct contact with fish tanks and cages and the aerosols that these items produce. Further, fish tanks should be kept clean on a regular basis as determined by facility policy, and this task should be performed by gloved staff members who are not responsible for patient care. The use of the infection-control risk assessment can help determine whether a fish tank poses a risk for patient or resident safety and health in these situations. No evidence, however, links the incidence of health-care–acquired infections among immunocompetent patients or residents with the presence of a properly cleaned and maintained fish tank, even in dining areas. As a general preventive measure, resident animal programs are advised to restrict animals from a) food preparation kitchens, b) laundries, c) central sterile supply and any storage areas for clean supplies, and d) medication preparation areas. Resident-animal programs in acute-care facilities should not allow the animals into the isolation areas, protective environments, ORs, or any area where immunocompromised patients are housed. Patients and staff routinely should wash their hands or use waterless, alcohol-based hand-hygiene products after contact with animals.

3. Service Animals

Although this section provides an overview about service animals in health-care settings, it cannot address every situation or question that may arise (see Appendix E - Information Resources). A service animal is any animal individually trained to do work or perform tasks for the benefit of a person with a disability.^{1366, 1376} A service animal is not considered a pet but rather an animal trained to provide assistance to a person because of a disability. Title III of the “Americans with Disabilities Act” (ADA) of 1990 mandates that persons with disabilities accompanied by service animals be allowed access with their service animals into places of public accommodation, including restaurants, public transportation, schools, and health-care facilities.^{1366, 1376} In health-care facilities, a person with a disability requiring a service animal may be an employee, a visitor, or a patient.

An overview of the subject of service animals and their presence in health-care facilities has been published.¹³⁶⁶ No evidence suggests that animals pose a more significant risk of transmitting infection than people; therefore, service animals should not be excluded from such areas, unless an individual patient’s situation or a particular animal poses greater risk that cannot be mitigated through reasonable measures. If health-care personnel, visitors, and patients are permitted to enter care areas (e.g., in-patient rooms, some ICUs, and public areas) without taking additional precautions to prevent transmission of infectious agents (e.g., donning gloves, gowns, or masks), a clean, healthy, well-behaved service animal should be allowed access with its handler.¹³⁶⁶ Similarly, if immunocompromised patients are able to receive visitors without using protective garments or equipment, an exclusion of service animals from this area would not be justified.¹³⁶⁶

Because health-care facilities are covered by the ADA or the Rehabilitation Act, a person with a disability may be accompanied by a service animal within the facility unless the animal’s presence or

behavior creates a fundamental alteration in the nature of a facility's services in a particular area or a direct threat to other persons in a particular area.¹³⁶⁶ A "direct threat" is defined as a significant risk to the health or safety of others that cannot be mitigated or eliminated by modifying policies, practices, or procedures.¹³⁷⁶ The determination that a service animal poses a direct threat in any particular health-care setting must be based on an individualized assessment of the service animal, the patient, and the health-care situation. When evaluating risk in such situations, health-care personnel should consider the nature of the risk (including duration and severity); the probability that injury will occur; and whether reasonable modifications of policies, practices, or procedures will mitigate the risk (J. Wodatch, U.S. Department of Justice, 2000). The person with a disability should contribute to the risk-assessment process as part of a pre-procedure health-care provider/patient conference.

Excluding a service animal from an OR or similar special care areas (e.g., burn units, some ICUs, PE units, and any other area containing equipment critical for life support) is appropriate if these areas are considered to have "restricted access" with regards to the general public. General infection-control measures that dictate such limited access include a) the area is required to meet environmental criteria to minimize the risk of disease transmission, b) strict attention to hand hygiene and absence of dermatologic conditions, and c) barrier protective measures [e.g., using gloves, wearing gowns and masks] are indicated for persons in the affected space. No infection-control measures regarding the use of barrier precautions could be reasonably imposed on the service animal. Excluding a service animal that becomes threatening because of a perceived danger to its handler during treatment also is appropriate; however, exclusion of such an animal must be based on the actual behavior of the particular animal, not on speculation about how the animal might behave.

Another issue regarding service animals is whether to permit persons with disabilities to be accompanied by their service animals during all phases of their stay in the health-care facility. Health-care personnel should discuss all aspects of anticipatory care with the patient who uses a service animal. Health-care personnel may not exclude a service animal because health-care staff may be able to perform the same services that the service animal does (e.g., retrieving dropped items and guiding an otherwise ambulatory person to the restroom). Similarly, health-care personnel can not exclude service animals because the health-care staff perceive a lack of need for the service animal during the person's stay in the health-care facility. A person with a disability is entitled to independent access (i.e., to be accompanied by a service animal unless the animal poses a direct threat or a fundamental alteration in the nature of services); "need" for the animal is not a valid factor in either analysis. For some forms of care (e.g., ambulation as physical therapy following total hip replacement or knee replacement), the service animal should not be used in place of a credentialed health-care worker who directly provides therapy. However, service animals need not be restricted from being in the presence of its handler during this time; in addition, rehabilitation and discharge planning should incorporate the patient's future use of the animal. The health-care personnel and the patient with a disability should discuss both the possible need for the service animal to be separated from its handler for a period of time during non-emergency care and an alternate plan of care for the service animal in the event the patient is unable or unwilling to provide that care. This plan might include family members taking the animal out of the facility several times a day for exercise and elimination, the animal staying with relatives, or boarding off-site. Care of the service animal, however, remains the obligation of the person with the disability, not the health-care staff.

Although animals potentially carry zoonotic pathogens transmissible to man, the risk is minimal with a healthy, clean, vaccinated, well-behaved, and well-trained service animal, the most common of which are dogs and cats. No reports have been published regarding infectious disease that affects humans originating in service dogs. Standard cleaning procedures are sufficient following occupation of an area by a service animal.¹³⁶⁶ Clean-up of spills of animal urine, feces, or other body substances can be accomplished with blood/body substance procedures outlined in the Environmental Services section of

this guideline. No special bathing procedures are required prior to a service animal accompanying its handler into a health-care facility.

Providing access to exotic animals (e.g., reptiles and non-human primates) that are used as service animals is problematic. Concerns about these animals are discussed in two published reviews.^{1331, 1366} Because some of these animals exhibit high-risk behaviors that may increase the potential for zoonotic disease transmission (e.g., herpes B infection), providing health-care facility access to nonhuman primates used as service animals is discouraged, especially if these animals might come into contact with the general public.^{1361, 1362} Health-care administrators should consult the Americans with Disabilities Act for guidance when developing policies about service animals in their facilities.^{1366, 1376}

Requiring documentation for access of a service animal to an area generally accessible to the public would impose a burden on a person with a disability. When health-care workers are not certain that an animal is a service animal, they may ask the person who has the animal if it is a service animal required because of a disability; however, no certification or other documentation of service animal status can be required.¹³⁷⁷

4. Animals as Patients in Human Health-Care Facilities

The potential for direct and indirect transmission of zoonoses must be considered when rooms and equipment in human health-care facilities are used for the medical or surgical treatment or diagnosis of animals.¹³⁷⁸ Inquiries should be made to veterinary medical professionals to determine an appropriate facility and equipment to care for an animal.

The central issue associated with providing medical or surgical care to animals in human health-care facilities is whether cross-contamination occurs between the animal patient and the human health-care workers and/or human patients. The fundamental principles of infection control and aseptic practice should differ only minimally, if at all, between veterinary medicine and human medicine. Health-care-associated infections can and have occurred in both patients and workers in veterinary medical facilities when lapses in infection-control procedures are evident.^{1379–1384} Further, veterinary patients can be at risk for acquiring infection from veterinary health-care workers if proper precautions are not taken.¹³⁸⁵

The issue of providing care to veterinary patients in human health-care facilities can be divided into the following three areas of infection-control concerns: a) whether the room/area used for animal care can be made safe for human patients, b) whether the medical/surgical instruments used on animals can be subsequently used on human patients, and c) which disinfecting or sterilizing procedures need to be done for these purposes. Studies addressing these concerns are lacking. However, with respect to disinfection or sterilization in veterinary settings, only minimal evidence suggests that zoonotic microbial pathogens are unusually resistant to inactivation by chemical or physical agents (with the exception of prions). Ample evidence supports the contrary observation (i.e., that pathogens from human- and animal sources are similar in their relative intrinsic resistance to inactivation).^{1386–1391} Further, no evidence suggests that zoonotic pathogens behave differently from human pathogens with respect to ventilation. Despite this knowledge, an aesthetic and sociologic perception that animal care must remain separate from human care persists. Health-care facilities, however, are increasingly faced with requests from the veterinary medical community for access to human health-care facilities for reasons that are largely economical (e.g., costs of acquiring sophisticated diagnostic technology and complex medical instruments). If hospital guidelines allow treatment of animals, alternate veterinary resources (including veterinary hospitals, clinics, and universities) should be exhausted before using human health-care settings. Additionally, the hospital's public/media relations should be notified of the situation. The goal is to develop policies and procedures to proactively and positively discuss and

disclose this activity to the general public.

An infection-control risk assessment (ICRA) must be undertaken to evaluate the circumstances specific to providing care to animals in a human health-care facility. Individual hospital policies and guidelines should be reviewed before any animal treatment is considered in such facilities. Animals treated in human health-care facilities should be under the direct care and supervision of a licensed veterinarian; they also should be free of known infectious diseases, ectoparasites, and other external contaminants (e.g., soil, urine, and feces). Measures should be taken to avoid treating animals with a known or suspected zoonotic disease in a human health-care setting (e.g., lambs being treated for Q fever).

If human health-care facilities must be used for animal treatment or diagnostics, the following general infection-control actions are suggested: a) whenever possible, the use of ORs or other rooms used for invasive procedures should be avoided [e.g., cardiac catheterization labs and invasive nuclear medicine areas]; b) when all other space options are exhausted and use of the aforementioned rooms is unavoidable, the procedure should be scheduled late in the day as the last procedure for that particular area such that patients are not present in the department/unit/area; c) environmental surfaces should be thoroughly cleaned and disinfected using procedures discussed in the Environmental Services portion of this guideline after the animal is removed from the care area; d) sufficient time should be allowed for ACH to help prevent allergic reactions by human patients [Table B.1. in Appendix B]; e) only disposable equipment or equipment that can be thoroughly and easily cleaned, disinfected, or sterilized should be used; f) when medical or surgical instruments, especially those invasive instruments that are difficult to clean [e.g., endoscopes], are used on animals, these instruments should be reserved for future use only on animals; and g) standard precautions should be followed.

5. Research Animals in Health-Care Facilities

The risk of acquiring a zoonotic infection from research animals has decreased in recent years because many small laboratory animals (e.g., mice, rats, and rabbits) come from quality stock and have defined microbiologic profiles.¹³⁹² Larger animals (e.g., nonhuman primates) are still obtained frequently from the wild and may harbor pathogens transmissible to humans. Primates, in particular, benefit from vaccinations to protect their health during the research period provided the vaccination does not interfere with the study of the particular agent. Animals serving as models for human disease studies pose some risk for transmission of infection to laboratory or health-care workers from percutaneous or mucosal exposure. Exposures can occur either through a) direct contact with an infected animal or its body substances and secretions or b) indirect contact with infectious material on equipment, instruments, surfaces, or supplies.¹³⁹² Uncontained aerosols generated during laboratory procedures can also transmit infection.

Infection-control measures to prevent transmission of zoonotic infections from research animals are largely derived from the following basic laboratory safety principles: a) purchasing pathogen-free animals, b) quarantining incoming animals to detect any zoonotic pathogens, c) treating infected animals or removing them from the facility, d) vaccinating animal carriers and high-risk contacts if possible, e) using specialized containment caging or facilities, and f) using protective clothing and equipment [e.g., gloves, face shields, gowns, and masks].¹³⁹² An excellent resource for detailed discussion of these safety measures has been published.¹⁰¹³

The animal research unit within a health-care facility should be engineered to provide a) adequate containment of animals and pathogens; b) daily decontamination and transport of equipment and waste; c) proper ventilation and air filtration, which prevents recirculation of the air in the unit to other areas of the facility; and d) negative air pressure in the animal rooms relative to the corridors. To ensure

adequate security and containment, no through traffic to other areas of the health-care facility should flow through this unit; access should be restricted to animal-care staff, researchers, environmental services, maintenance, and security personnel.

Occupational health programs for animal-care staff, researchers, and maintenance staff should take into consideration the animals' natural pathogens and research pathogens. Components of such programs include a) prophylactic vaccines, b) TB skin testing when primates are used, c) baseline serums, and d) hearing and respiratory testing. Work practices, PPE, and engineering controls specific for each of the four animal biosafety levels have been published.^{1013, 1393} The facility's occupational or employee health clinic should be aware of the appropriate post-exposure procedures involving zoonoses and have available the appropriate post-exposure biologicals and medications.

Animal-research-area staff should also develop standard operating procedures for a) daily animal husbandry [e.g., protection of the employee while facilitating animal welfare]; b) pathogen containment and decontamination; c) management, cleaning, disinfecting and/or sterilizing equipment and instruments; and d) employee training for laboratory safety and safety procedures specific to animal research worksites.¹⁰¹³ The federal Animal Welfare Act of 1966 and its amendments serve as the regulatory basis for ensuring animal welfare in research.^{1394, 1395}

I. Regulated Medical Waste

1. Epidemiology

No epidemiologic evidence suggests that most of the solid- or liquid wastes from hospitals, other health-care facilities, or clinical/research laboratories is any more infective than residential waste. Several studies have compared the microbial load and the diversity of microorganisms in residential wastes and wastes obtained from a variety of health-care settings.¹³⁹⁹⁻¹⁴⁰² Although hospital wastes had a greater number of different bacterial species compared with residential waste, wastes from residences were more heavily contaminated.^{1397, 1398} Moreover, no epidemiologic evidence suggests that traditional waste-disposal practices of health-care facilities (whereby clinical and microbiological wastes were decontaminated on site before leaving the facility) have caused disease in either the health-care setting or the general community.^{1400, 1401} This statement excludes, however, sharps injuries sustained during or immediately after the delivery of patient care before the sharp is "discarded." Therefore, identifying wastes for which handling and disposal precautions are indicated is largely a matter of judgment about the relative risk of disease transmission, because no reasonable standards on which to base these determinations have been developed. Aesthetic and emotional considerations (originating during the early years of the HIV epidemic) have, however, figured into the development of treatment and disposal policies, particularly for pathology and anatomy wastes and sharps.¹⁴⁰²⁻¹⁴⁰⁵ Public concerns have resulted in the promulgation of federal, state, and local rules and regulations regarding medical waste management and disposal.¹⁴⁰⁶⁻¹⁴¹⁴

2. Categories of Medical Waste

Precisely defining medical waste on the basis of quantity and type of etiologic agents present is virtually impossible. The most practical approach to medical waste management is to identify wastes that represent a sufficient potential risk of causing infection during handling and disposal and for which some precautions likely are prudent.² Health-care facility medical wastes targeted for handling and disposal precautions include microbiology laboratory waste (e.g., microbiologic cultures and stocks of microorganisms), pathology and anatomy waste, blood specimens from clinics and laboratories, blood

products, and other body-fluid specimens.² Moreover, the risk of either injury or infection from certain sharp items (e.g., needles and scalpel blades) contaminated with blood also must be considered. Although any item that has had contact with blood, exudates, or secretions may be potentially infective, treating all such waste as infective is neither practical nor necessary. Federal, state, and local guidelines and regulations specify the categories of medical waste that are subject to regulation and outline the requirements associated with treatment and disposal. The categorization of these wastes has generated the term “regulated medical waste.” This term emphasizes the role of regulation in defining the actual material and as an alternative to “infectious waste,” given the lack of evidence of this type of waste’s infectivity. State regulations also address the degree or amount of contamination (e.g., blood-soaked gauze) that defines the discarded item as a regulated medical waste. The EPA’s *Manual for Infectious Waste Management* identifies and categorizes other specific types of waste generated in health-care facilities with research laboratories that also require handling precautions.¹⁴⁰⁶

3. Management of Regulated Medical Waste in Health-Care Facilities

Medical wastes require careful disposal and containment before collection and consolidation for treatment. OSHA has dictated initial measures for discarding regulated medical-waste items. These measures are designed to protect the workers who generate medical wastes and who manage the wastes from point of generation to disposal.⁹⁶⁷ A single, leak-resistant biohazard bag is usually adequate for containment of regulated medical wastes, provided the bag is sturdy and the waste can be discarded without contaminating the bag’s exterior. The contamination or puncturing of the bag requires placement into a second biohazard bag. All bags should be securely closed for disposal. Puncture-resistant containers located at the point of use (e.g., sharps containers) are used as containment for discarded slides or tubes with small amounts of blood, scalpel blades, needles and syringes, and unused sterile sharps.⁹⁶⁷ To prevent needlestick injuries, needles and other contaminated sharps should not be recapped, purposefully bent, or broken by hand. CDC has published general guidelines for handling sharps.^{6, 1415} Health-care facilities may need additional precautions to prevent the production of aerosols during the handling of blood-contaminated items for certain rare diseases or conditions (e.g., Lassa fever and Ebola virus infection).²⁰³

Transporting and storing regulated medical wastes within the health-care facility prior to terminal treatment is often necessary. Both federal and state regulations address the safe transport and storage of on- and off-site regulated medical wastes.^{1406–1408} Health-care facilities are instructed to dispose medical wastes regularly to avoid accumulation. Medical wastes requiring storage should be kept in labeled, leak-proof, puncture-resistant containers under conditions that minimize or prevent foul odors. The storage area should be well ventilated and be inaccessible to pests. Any facility that generates regulated medical wastes should have a regulated medical waste management plan to ensure health and environmental safety as per federal, state, and local regulations.

4. Treatment of Regulated Medical Waste

Regulated medical wastes are treated or decontaminated to reduce the microbial load in or on the waste and to render the by-products safe for further handling and disposal. From a microbiologic standpoint, waste need not be rendered “sterile” because the treated waste will not be deposited in a sterile site. In addition, waste need not be subjected to the same reprocessing standards as are surgical instruments. Historically, treatment methods involved steam-sterilization (i.e., autoclaving), incineration, or interment (for anatomy wastes). Alternative treatment methods developed in recent years include chemical disinfection, grinding/shredding/disinfection methods, energy-based technologies (e.g., microwave or radiowave treatments), and disinfection/encapsulation methods.¹⁴⁰⁹ State medical waste regulations specify appropriate treatment methods for each category of regulated medical waste.

The recommendations in this guideline for Ebola Virus Disease has been superseded by CDC’s Infection Prevention and Control Recommendations for Hospitalized Patients with Known or Suspected Ebola Virus Disease in U.S. Hospitals and by CDC’s Interim Guidance for Environmental Infection Control in Hospitals for Ebola Virus issued on August 1, 2014. [Click here for current information on how Ebola virus is transmitted.](#)

Of all the categories comprising regulated medical waste, microbiologic wastes (e.g., untreated cultures, stocks, and amplified microbial populations) pose the greatest potential for infectious disease transmission, and sharps pose the greatest risk for injuries. Untreated stocks and cultures of microorganisms are subsets of the clinical laboratory or microbiologic waste stream. If the microorganism must be grown and amplified in culture to high concentration to permit work with the specimen, this item should be considered for on-site decontamination, preferably within the laboratory unit. Historically, this was accomplished effectively by either autoclaving (steam sterilization) or incineration. If steam sterilization in the health-care facility is used for waste treatment, exposure of the waste for up to 90 minutes at 250°F (121°C) in an autoclave (depending on the size of the load and type container) may be necessary to ensure an adequate decontamination cycle.^{1416–1418} After steam sterilization, the residue can be safely handled and discarded with all other nonhazardous solid waste in accordance with state solid-waste disposal regulations. On-site incineration is another treatment option for microbiologic, pathologic, and anatomic waste, provided the incinerator is engineered to burn these wastes completely and stay within EPA emissions standards.¹⁴¹⁰ Improper incineration of waste with high moisture and low energy content (e.g., pathology waste) can lead to emission problems. State medical-waste regulatory programs identify acceptable methods for inactivating amplified stocks and cultures of microorganisms, some of which may employ technology rather than steam sterilization or incineration.

Concerns have been raised about the ability of modern health-care facilities to inactivate microbiologic wastes on-site, given that many of these institutions have decommissioned their laboratory autoclaves. Current laboratory guidelines for working with infectious microorganisms at biosafety level (BSL) 3 recommend that all laboratory waste be decontaminated before disposal by an approved method, preferably within the laboratory.¹⁰¹³ These same guidelines recommend that all materials removed from a BSL 4 laboratory (unless they are biological materials that are to remain viable) are to be decontaminated before they leave the laboratory.¹⁰¹³ Recent federal regulations for laboratories that handle certain biological agents known as “select agents” (i.e., those that have the potential to pose a severe threat to public health and safety) require these agents (and those obtained from a clinical specimen intended for diagnostic, reference, or verification purposes) to be destroyed on-site before disposal.¹⁴¹² Although recommendations for laboratory waste disposal from BSL 1 or 2 laboratories (e.g., most health-care clinical and diagnostic laboratories) allow for these materials to be decontaminated off-site before disposal, on-site decontamination by a known effective method is preferred to reduce the potential of exposure during the handling of infectious material.

A recent outbreak of TB among workers in a regional medical-waste treatment facility in the United States demonstrated the hazards associated with aerosolized microbiologic wastes.^{1419, 1420} The facility received diagnostic cultures of *Mycobacterium tuberculosis* from several different health-care facilities before these cultures were chemically disinfected; this facility treated this waste with a grinding/shredding process that generated aerosols from the material.^{1419, 1420} Several operational deficiencies facilitated the release of aerosols and exposed workers to airborne *M. tuberculosis*. Among the suggested control measures was that health-care facilities perform on-site decontamination of laboratory waste containing live cultures of microorganisms before release of the waste to a waste management company.^{1419, 1420} This measure is supported by recommendations found in the CDC/NIH guideline for laboratory workers.¹⁰¹³ This outbreak demonstrates the need to avoid the use of any medical-waste treatment method or technology that can aerosolize pathogens from live cultures and stocks (especially those of airborne microorganisms) unless aerosols can be effectively contained and workers can be equipped with proper PPE.^{1419–1421} Safe laboratory practices, including those addressing waste management, have been published.^{1013, 1422}

In an era when local, state, and federal health-care facilities and laboratories are developing bioterrorism

response strategies and capabilities, the need to reinstate in-laboratory capacity to destroy cultures and stocks of microorganisms becomes a relevant issue.¹⁴²³ Recent federal regulations require health-care facility laboratories to maintain the capability of destroying discarded cultures and stocks on-site if these laboratories isolate from a clinical specimen any microorganism or toxin identified as a “select agent” from a clinical specimen (Table 27).^{1412, 1413} As an alternative, isolated cultures of select agents can be transferred to a facility registered to accept these agents in accordance with federal regulations.¹⁴¹² State medical waste regulations can, however, complicate or completely prevent this transfer if these cultures are determined to be medical waste, because most states regulate the inter-facility transfer of untreated medical wastes.

Table 27. Microorganisms and biologicals identified as select agents*+

<i>HHS Non-overlap select agents and toxins (42 CFR Part 73 §73.4)</i>	
Viruses	Crimean-Congo hemorrhagic fever virus; Ebola viruses; Cercopithecine herpesvirus 1 (herpes B virus); Lassa fever virus; Marburg virus; monkeypox virus; South American hemorrhagic fever viruses (Junin, Machupo, Sabia, Flexal, Guanarito); tick-borne encephalitis complex (flavi) viruses (Central European tick-borne encephalitis, Far Eastern tick-borne encephalitis [Russian spring and summer encephalitis, Kyasnaur Forest disease, Omsk hemorrhagic fever]); variola major virus (smallpox virus); and variola minor virus (alastrim)
Exclusions¶	Vaccine strain of Junin virus (Candid. #1)
Bacteria	<i>Rickettsia prowazekii</i> , <i>R. rickettsii</i> , <i>Yersinia pestis</i>
Fungi	<i>Coccidioides posadasii</i>
Toxins	Abrin; conotoxins; diacetoxyscirpenol; ricin; saxitoxin; Shiga-like ribosome inactivating proteins; tetrodotoxin
Exclusions¶	The following toxins (in purified form or in combinations of pure and impure forms) if the aggregate amount under the control of a principal investigator does not, at any time, exceed the amount specified: 100 mg of abrin; 100 mg of conotoxins; 1,000 mg of diacetoxyscirpenol; 100 mg of ricin; 100 mg of saxitoxin; 100 mg of Shiga-like ribosome inactivating proteins; or 100 mg of tetrodotoxin
Genetic elements, recombinant nucleic acids, and recombinant organisms¶	<ul style="list-style-type: none"> • Select agent viral nucleic acids (synthetic or naturally-derived, contiguous or fragmented, in host chromosomes or in expression vectors) that can encode infectious and/or replication competent forms of any of the select agent viruses; • Nucleic acids (synthetic or naturally-derived) that encode for the functional form(s) of any of the toxins listed in this table if the nucleic acids: a) are in a vector or host chromosome; b) can be expressed <i>in vivo</i> or <i>in vitro</i>; or c) are in a vector or host chromosome and can be expressed <i>in vivo</i> or <i>in vitro</i>; • Viruses, bacteria, fungi, and toxins listed in this table that have been genetically modified.
<i>High consequence livestock pathogens and toxins/select agents (overlap agents) (42 CFR Part 73 §73.5 and USDA regulation 9 CFR Part 121)</i>	
Viruses	Eastern equine encephalitis virus; Nipah and Hendra complex viruses; Rift Valley fever virus; Venezuelan equine encephalitis virus
Exclusions¶	MP-12 vaccine strain of Rift Valley fever virus; TC-83 vaccine strain of Venezuelan equine encephalitis virus
Bacteria	<i>Bacillus anthracis</i> ; <i>Brucella abortus</i> , <i>B. melitensis</i> , <i>B. suis</i> ; <i>Burkholderia mallei</i> (formerly <i>Pseudomonas mallei</i>), <i>B. pseudomallei</i> (formerly <i>P. pseudomallei</i>); botulinum neurotoxin-producing species of <i>Clostridium</i> ; <i>Coxiella burnetii</i> ; <i>Francisella tularensis</i>
Fungi	<i>Coccidioides immitis</i>
Toxins	Botulinum neurotoxins; <i>Clostridium perfringens</i> epsilon toxin; Shigatoxin; staphylococcal enterotoxins; T-2 toxin
Exclusions¶	The following toxins (in purified form or in combinations of pure and impure forms) if the aggregate amount under the control of a principal investigator does not, at any time, exceed the amount specified: 0.5 mg of botulinum neurotoxins; 100 mg of <i>Clostridium perfringens</i> epsilon toxin; 100 mg of Shigatoxin; 5 mg of staphylococcal enterotoxins; or 1,000 mg of T-2 toxin

High consequence livestock pathogens and toxins/select agents (overlap agents) (42 CFR Part 73 §73.5 and USDA regulation 9 CFR Part 121) (continued)	
Genetic elements, recombinant nucleic acids, and recombinant organisms¶	<ul style="list-style-type: none"> • Select agent viral nuclei acids (synthetic or naturally derived, contiguous or fragmented, in host chromosomes or in expression vectors) that can encode infectious and/or replication competent forms of any of the select agent viruses; • Nucleic acids (synthetic or naturally derived) that encode for the functional form(s) of any of the toxins listed in this table if the nucleic acids: a) are in a vector or host chromosome; b) can be expressed <i>in vivo</i> or <i>in vitro</i>; or c) are in a vector or host chromosome and can be expressed <i>in vivo</i> or <i>in vitro</i>; • Viruses, bacteria, fungi, and toxins listed in this table that have been genetically modified

* Material in this table is compiled from references 1412, 1413, and 1424. Reference 1424 also contains lists of select agents that include plant pathogens and pathogens affecting livestock.

+ 42 CFR 73 §§73.4 and 73.5 do not include any select agent or toxin that is in its naturally-occurring environment, provided it has not been intentionally introduced, cultivated, collected, or otherwise extracted from its natural source. These sections also do not include non-viable select agent organisms or nonfunctional toxins. This list of select agents is current as of 3 October 2003 and is subject to change pending the final adoption of 42 CFR Part 73.

¶ These table entries are listed in reference 1412 and 1413, but were not included in reference 1424.

5. Discharging Blood, Fluids to Sanitary Sewers or Septic Tanks

The contents of all vessels that contain more than a few milliliters of blood remaining after laboratory procedures, suction fluids, or bulk blood can either be inactivated in accordance with state-approved treatment technologies or carefully poured down a utility sink drain or toilet.¹⁴¹⁴ State regulations may dictate the maximum volume allowable for discharge of blood/body fluids to the sanitary sewer. No evidence indicates that bloodborne diseases have been transmitted from contact with raw or treated sewage. Many bloodborne pathogens, particularly bloodborne viruses, are not stable in the environment for long periods of time;^{1425, 1426} therefore, the discharge of small quantities of blood and other body fluids to the sanitary sewer is considered a safe method of disposing of these waste materials.¹⁴¹⁴ The following factors increase the likelihood that bloodborne pathogens will be inactivated in the disposal process: a) dilution of the discharged materials with water; b) inactivation of pathogens resulting from exposure to cleaning chemicals, disinfectants, and other chemicals in raw sewage; and c) effectiveness of sewage treatment in inactivating any residual bloodborne pathogens that reach the treatment facility. Small amounts of blood and other body fluids should not affect the functioning of a municipal sewer system. However, large quantities of these fluids, with their high protein content, might interfere with the biological oxygen demand (BOD) of the system. Local municipal sewage treatment restrictions may dictate that an alternative method of bulk fluid disposal be selected. State regulations may dictate what quantity constitutes a small amount of blood or body fluids.

Although concerns have been raised about the discharge of blood and other body fluids to a septic tank system, no evidence suggests that septic tanks have transmitted bloodborne infections. A properly functioning septic system is adequate for inactivating bloodborne pathogens. System manufacturers' instructions specify what materials may be discharged to the septic tank without jeopardizing its proper operation.

6. Medical Waste and CJD

Concerns also have been raised about the need for special handling and treatment procedures for wastes generated during the care of patients with CJD or other transmissible spongiform encephalopathies (TSEs). Prions, the agents that cause TSEs, have significant resistance to inactivation by a variety of physical, chemical, or gaseous methods.¹⁴²⁷ No epidemiologic evidence, however, links acquisition of CJD with medical-waste disposal practices. Although handling neurologic tissue for pathologic examination and autopsy materials with care, using barrier precautions, and following specific

procedures for the autopsy are prudent measures,¹¹⁹⁷ employing extraordinary measures once the materials are discarded is unnecessary. Regulated medical wastes generated during the care of the CJD patient can be managed using the same strategies as wastes generated during the care of other patients. After decontamination, these wastes may then be disposed in a sanitary landfill or discharged to the sanitary sewer, as appropriate.

Part II. Recommendations for Environmental Infection Control in Health-Care Facilities

A. Rationale for Recommendations

As in previous CDC guidelines, each recommendation is categorized on the basis of existing scientific data, theoretic rationale, applicability, and possible economic benefit. The recommendations are evidence-based wherever possible. However, certain recommendations are derived from empiric infection-control or engineering principles, theoretic rationale, or from experience gained from events that cannot be readily studied (e.g., floods).

The HICPAC system for categorizing recommendations has been modified to include a category for engineering standards and actions required by state or federal regulations. Guidelines and standards published by the American Institute of Architects (AIA), American Society of Heating, Refrigeration, and Air-Conditioning Engineers (ASHRAE), and the Association for the Advancement in Medical Instrumentation (AAMI) form the basis of certain recommendations. These standards reflect a consensus of expert opinions and extensive consultation with agencies of the U.S. Department of Health and Human Services. Compliance with these standards is usually voluntary. However, state and federal governments often adopt these standards as regulations. For example, the standards from AIA regarding construction and design of new or renovated health-care facilities, have been adopted by reference by >40 states. Certain recommendations have two category ratings (e.g., Categories IA and IC or Categories IB and IC), indicating the recommendation is evidence-based as well as a standard or regulation.

B. Rating Categories

Recommendations are rated according to the following categories:

- **Category IA.** Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiologic studies.
- **Category IB.** Strongly recommended for implementation and supported by certain experimental, clinical, or epidemiologic studies and a strong theoretical rationale.
- **Category IC.** Required by state or federal regulation, or representing an established association standard. (Note: Abbreviations for governing agencies and regulatory citations are listed, where appropriate. Recommendations from regulations adopted at state levels are also noted. Recommendations from AIA guidelines cite the appropriate sections of the standard).
- **Category II.** Suggested for implementation and supported by suggestive clinical or epidemiologic studies, or a theoretical rationale.
- **Unresolved Issue.** No recommendation is offered. No consensus or insufficient evidence exists regarding efficacy.

C. Recommendations—Air

I. Air-Handling Systems in Health-Care Facilities

- A. Use AIA guidelines as minimum standards where state or local regulations are not in place for design and construction of ventilation systems in new or renovated health-care facilities. Ensure that existing structures continue to meet the specifications in effect at the time of construction.¹²⁰ **Category IC** (AIA: 1.1.A, 5.4)
- B. Monitor ventilation systems in accordance with engineers' and manufacturers' recommendations to ensure preventive engineering, optimal performance for removal of particulates, and elimination of excess moisture.^{18, 35, 106, 120, 220, 222, 333, 336} **Category IB, IC** (AIA: 7.2, 7.31.D, 8.31.D, 9.31.D, 10.31.D, 11.31.D, EPA guidance)
 1. Ensure that heating, ventilation, air conditioning (HVAC) filters are properly installed and maintained to prevent air leakages and dust overloads.^{17, 18, 106, 222} **Category IB**
 2. Monitor areas with special ventilation requirements (e.g., AII or PE) for ACH, filtration, and pressure differentials.^{21, 120, 249, 250, 273–275, 277, 333–344} **Category IB, IC** (AIA: 7.2.C7, 7.2.D6)
 - a. Develop and implement a maintenance schedule for ACH, pressure differentials, and filtration efficiencies using facility-specific data as part of the multidisciplinary risk assessment. Take into account the age and reliability of the system.
 - b. Document these parameters, especially the pressure differentials.
 3. Engineer humidity controls into the HVAC system and monitor the controls to ensure proper moisture removal.¹²⁰ **Category IC** (AIA: 7.31.D9)
 - a. Locate duct humidifiers upstream from the final filters.
 - b. Incorporate a water-removal mechanism into the system.
 - c. Locate all duct takeoffs sufficiently down-stream from the humidifier so that moisture is completely absorbed.
 4. Incorporate steam humidifiers, if possible, to reduce potential for microbial proliferation within the system, and avoid use of cool mist humidifiers. **Category II**
 5. Ensure that air intakes and exhaust outlets are located properly in construction of new facilities and renovation of existing facilities.^{3, 120} **Category IC** (AIA: 7.31.D3, 8.31.D3, 9.31.D3, 10.31.D3, 11.31.D3)
 - a. Locate exhaust outlets >25 ft. from air-intake systems.
 - b. Locate outdoor air intakes ≥6 ft. above ground or ≥3 ft. above roof level.
 - c. Locate exhaust outlets from contaminated areas above roof level to minimize recirculation of exhausted air.
 6. Maintain air intakes and inspect filters periodically to ensure proper operation.^{3, 120, 249, 250, 273–275, 277} **Category IC** (AIA: 7.31.D8)
 7. Bag dust-filled filters immediately upon removal to prevent dispersion of dust and fungal spores during transport within the facility.^{106, 221} **Category IB**
 - a. Seal or close the bag containing the discarded filter.
 - b. Discard spent filters as regular solid waste, regardless of the area from which they were removed.²²¹
 8. Remove bird roosts and nests near air intakes to prevent mites and fungal spores from entering the ventilation system.^{3, 98, 119} **Category IB**
 9. Prevent dust accumulation by cleaning air-duct grilles in accordance with facility-specific procedures and schedules when rooms are not occupied by patients.^{21, 120, 249, 250, 273–275, 277} **Category IC, II** (AIA: 7.31.D10)

10. Periodically measure output to monitor system function; clean ventilation ducts as part of routine HVAC maintenance to ensure optimum performance.^{120, 263, 264}
Category II (AIA: 7.31.D10)
- C. Use portable, industrial-grade HEPA filter units capable of filtration rates in the range of 300–800 ft³/min. to augment removal of respirable particles as needed.²¹⁹ **Category II**
 1. Select portable HEPA filters that can recirculate all or nearly all of the room air and provide the equivalent of ≥ 12 ACH.⁴ **Category II**
 2. Portable HEPA filter units previously placed in construction zones can be used later in patient-care areas, provided all internal and external surfaces are cleaned, and the filter's performance verified by appropriate particle testing. **Category II**
 3. Situate portable HEPA units with the advice of facility engineers to ensure that all room air is filtered.⁴ **Category II**
 4. Ensure that fresh-air requirements for the area are met.^{214, 219} **Category II**
- D. Follow appropriate procedures for use of areas with through-the-wall ventilation units.¹²⁰
Category IC (AIA: 8.31.D1, 8.31.D8, 9.31.D23, 10.31.D18, 11.31.D15)
 1. Do not use such areas as PE rooms.¹²⁰ **Category IC** (AIA: 7.2.D3)
 2. Do not use a room with a through-the-wall ventilation unit as an AII room unless it can be demonstrated that all required AII engineering controls required are met.^{4, 120}
Category IC (AIA: 7.2.C3)
- E. Conduct an infection-control risk assessment (ICRA) and provide an adequate number of AII and PE rooms (if required) or other areas to meet the needs of the patient population.^{4, 6, 9, 18, 19, 69, 94, 120, 142, 331–334, 336–338} **Category IA, IC** (AIA: 7.2.C, 7.2.D)
- F. When UVGI is used as a supplemental engineering control, install fixtures 1) on the wall near the ceiling or suspended from the ceiling as an upper air unit; 2) in the air-return duct of an AII room; or 3) in designated enclosed areas or booths for sputum induction.⁴
Category II
- G. Seal windows in buildings with centralized HVAC systems and especially with PE areas.^{35, 111, 120}
Category IB, IC (AIA: 7.2.D3)
- H. Keep emergency doors and exits from PE rooms closed except during an emergency; equip emergency doors and exits with alarms. **Category II**
- I. Develop a contingency plan for backup capacity in the event of a general power failure.⁷¹³
Category IC (Joint Commission on Accreditation of Healthcare Organizations [JCAHO]: Environment of Care [EC] 1.4)
 1. Emphasize restoration of proper air quality and ventilation conditions in AII rooms, PE rooms, operating rooms, emergency departments, and intensive care units.^{120, 713}
Category IC (AIA: 1.5.A1; JCAHO: EC 1.4)
 2. Deploy infection-control procedures to protect occupants until power and systems functions are restored.^{6, 120, 713} **Category IC** (AIA: 5.1, 5.2; JCAHO: EC 1.4)
- J. Do not shut down HVAC systems in patient-care areas except for maintenance, repair, testing of emergency backup capacity, or new construction.^{120, 206} **Category IB, IC** (AIA: 5.1, 5.2.B, C)
 1. Coordinate HVAC system maintenance with infection-control staff to allow for relocation of immunocompromised patients if necessary.¹²⁰ **Category IC** (AIA: 5.1, 5.2)
 2. Provide backup emergency power and air-handling and pressurization systems to maintain filtration, constant ACH, and pressure differentials in PE rooms, AII rooms, operating rooms, and other critical-care areas.^{9, 120, 278} **Category IC** (AIA: 1.5, 5.1, 5.2)
 3. For areas not served by installed emergency ventilation and backup systems, use portable units and monitor ventilation parameters and patients in those areas.²¹⁹
Category II
 4. Coordinate system startups with infection-control staff to protect patients in PE rooms from bursts of fungal spores.^{9, 35, 120, 278} **Category IC** (AIA: 5.1, 5.2)

5. Allow sufficient time for ACH to clean the air once the system is operational (Appendix B, Table B.1).^{4, 120} **Category IC** (AIA: 5.1, 5.2)
- K. HVAC systems serving offices and administration areas may be shut down for energy conservation purposes, but the shutdown must not alter or adversely affect pressure differentials maintained in laboratories or critical-care areas with specific ventilation requirements (i.e., PE rooms, AII rooms, operating rooms). **Category II**
- L. Whenever possible, avoid inactivating or shutting down the entire HVAC system at one time, especially in acute-care facilities. **Category II**
- M. Whenever feasible, design and install fixed backup ventilation systems for new or renovated construction for PE rooms, AII rooms, operating rooms, and other critical care areas identified by ICRA.¹²⁰ **Category IC** (AIA: 1.5.A1)

II. Construction, Renovation, Remediation, Repair, and Demolition

- A. Establish a multidisciplinary team that includes infection-control staff to coordinate demolition, construction, and renovation projects and consider proactive preventive measures at the inception; produce and maintain summary statements of the team's activities.^{17, 19, 20, 97, 109, 120, 249, 250, 273–277} **Category IB, IC** (AIA: 5.1)
- B. Educate both the construction team and the health-care staff in immunocompromised patient-care areas regarding the airborne infection risks associated with construction projects, dispersal of fungal spores during such activities, and methods to control the dissemination of fungal spores.^{3, 249, 250, 273–277, 1428–1432} **Category IB**
- C. Incorporate mandatory adherence agreements for infection control into construction contracts, with penalties for noncompliance and mechanisms to ensure timely correction of problems.^{3, 120, 249, 273–277} **Category IC** (AIA: 5.1)
- D. Establish and maintain surveillance for airborne environmental disease (e.g., aspergillosis) as appropriate during construction, renovation, repair, and demolition activities to ensure the health and safety of immunocompromised patients.^{3, 64, 65, 79} **Category IB**
 1. Using active surveillance, monitor for airborne fungal infections in immunocompromised patients.^{3, 9, 64, 65} **Category IB**
 2. Periodically review the facility's microbiologic, histopathologic, and postmortem data to identify additional cases.^{3, 9, 64, 65} **Category IB**
 3. If cases of aspergillosis or other health-care-associated airborne fungal infections occur, aggressively pursue the diagnosis with tissue biopsies and cultures as feasible.^{3, 64, 65, 79, 249, 273–277} **Category IB**
- E. Implement infection-control measures relevant to construction, renovation, maintenance, demolition, and repair.^{96, 97, 120, 276, 277} **Category IB, IC** (AIA: 5.1, 5.2)
 1. Before the project gets underway, perform an ICRA to define the scope of the project and the need for barrier measures.^{96, 97, 120, 249, 273–277} **Category IB, IC** (AIA: 5.1)
 - a. Determine if immunocompromised patients may be at risk for exposure to fungal spores from dust generated during the project.^{20, 109, 273–275, 277}
 - b. Develop a contingency plan to prevent such exposures.^{20, 109, 273–275, 277}
 2. Implement infection-control measures for external demolition and construction activities.^{50, 249, 273–277, 283} **Category IB**
 - a. Determine if the facility can operate temporarily on recirculated air; if feasible, seal off adjacent air intakes.
 - b. If this is not possible or practical, check the low-efficiency (roughing) filter banks frequently and replace as needed to avoid buildup of particulates.
 - c. Seal windows and reduce wherever possible other sources of outside air intrusion (e.g., open doors in stairwells and corridors), especially in PE areas.
 3. Avoid damaging the underground water distribution system (i.e., buried pipes) to prevent soil and dust contamination of the water.^{120, 305} **Category IB, IC** (AIA: 5.1)

4. Implement infection-control measures for internal construction activities.^{20, 49, 97, 120, 249, 273–277} **Category IB, IC** (AIA: 5.1, 5.2)
 - a. Construct barriers to prevent dust from construction areas from entering patient-care areas; ensure that barriers are impermeable to fungal spores and in compliance with local fire codes.^{20, 49, 97, 120, 284, 312, 713, 1431}
 - b. Block and seal off return air vents if rigid barriers are used for containment.^{120, 276, 277}
 - c. Implement dust control measures on surfaces and by diverting pedestrian traffic away from work zones.^{20, 49, 97, 120}
 - d. Relocate patients whose rooms are adjacent to work zones, depending upon their immune status, the scope of the project, the potential for generation of dust or water aerosols, and the methods used to control these aerosols.^{49, 120, 281}
5. Perform those engineering and work-site related infection-control measures as needed for internal construction, repairs, and renovations.^{20, 49, 97, 109, 120, 312} **Category IB, IC** (AIA: 5.1, 5.2)
 - a. Ensure proper operation of the air-handling system in the affected area after erection of barriers and before the room or area is set to negative pressure.^{49, 69, 276, 278} **Category IB**
 - b. Create and maintain negative air pressure in work zones adjacent to patient-care areas and ensure that required engineering controls are maintained.^{20, 49, 97, 109, 120, 312}
 - c. Monitor negative air flow inside rigid barriers.^{120, 281}
 - d. Monitor barriers and ensure the integrity of the construction barriers; repair gaps or breaks in barrier joints.^{120, 284, 307, 312}
 - e. Seal windows in work zones if practical; use window chutes for disposal of large pieces of debris as needed, but ensure that the negative pressure differential for the area is maintained.^{20, 120, 273}
 - f. Direct pedestrian traffic from construction zones away from patient-care areas to minimize the dispersion of dust.^{20, 49, 97, 109, 111, 120, 273–277}
 - g. Provide construction crews with 1) designated entrances, corridors, and elevators whenever practical; 2) essential services [e.g., toilet facilities], and convenience services [e.g., vending machines]; 3) protective clothing [e.g., coveralls, footwear, and headgear] for travel to patient-care areas; and 4) a space or anteroom for changing clothing and storing equipment.^{120, 249, 273–277}
 - h. Clean work zones and their entrances daily by 1) wet-wiping tools and tool carts before their removal from the work zone; 2) placing mats with tacky surfaces inside the entrance; and 3) covering debris and securing this covering before removing debris from the work zone.^{120, 249, 273–277}
 - i. In patient-care areas, for major repairs that include removal of ceiling tiles and disruption of the space above the false ceiling, use plastic sheets or prefabricated plastic units to contain dust; use a negative pressure system within this enclosure to remove dust; and either pass air through an industrial grade, portable HEPA filter capable of filtration rates ranging from 300–800 ft³/min., or exhaust air directly to the outside.^{49, 276, 277, 281, 309}
 - j. Upon completion of the project, clean the work zone according to facility procedures, and install barrier curtains to contain dust and debris before removal of rigid barriers.^{20, 97, 120, 249, 273–277}
 - k. Flush the water system to clear sediment from pipes to minimize waterborne microorganism proliferation.^{120, 305}
 - l. Restore appropriate ACH, humidity, and pressure differential; clean or replace air filters; dispose of spent filters.^{35, 106, 221, 278}

- F. Use airborne-particle sampling as a tool to evaluate barrier integrity.^{35, 100} **Category II**
- G. Commission the HVAC system for newly constructed health-care facilities and renovated spaces before occupancy and use, with emphasis on ensuring proper ventilation for operating rooms, AII rooms, and PE areas.^{100, 120, 288, 304} **Category IC** (AIA: 5.1; ASHRAE: 1-1996)
- H. **No recommendation is offered** on routine microbiologic air sampling before, during, or after construction or before or during occupancy of areas housing immunocompromised patients.^{17, 20, 49, 97, 109, 272, 1433} **Unresolved issue**
- I. If a case of health-care-acquired aspergillosis or other opportunistic environmental airborne fungal disease occurs during or immediately after construction, implement appropriate follow-up measures.^{20, 55, 62, 77, 94, 95} **Category IB**
1. Review pressure differential monitoring documentation to verify that pressure differentials in the construction zone and in PE rooms were appropriate for their settings.^{94, 95, 120} **Category IB, IC** (AIA: 5.1)
 2. Implement corrective engineering measures to restore proper pressure differentials as needed.^{94, 95, 120} **Category IB, IC** (AIA: 5.1)
 3. Conduct a prospective search for additional cases and intensify retrospective epidemiologic review of the hospital's medical and laboratory records.^{3, 20, 62, 63, 104} **Category IB**
 4. If there is no evidence of ongoing transmission, continue routine maintenance in the area to prevent health-care-acquired fungal disease.^{3, 55} **Category IB**
- J. If there is epidemiologic evidence of ongoing transmission of fungal disease, conduct an environmental assessment to determine and eliminate the source.^{3, 96, 97, 109, 111, 115, 249, 273-277} **Category IB**
1. Collect environmental samples from potential sources of airborne fungal spores, preferably using a high-volume air sampler rather than settle plates.^{3, 18, 44, 48, 49, 97, 106, 111, 112, 115, 249, 254, 273-277, 292, 312} **Category IB**
 2. If either an environmental source of airborne fungi or an engineering problem with filtration or pressure differentials is identified, promptly perform corrective measures to eliminate the source and route of entry.^{96, 97} **Category IB**
 3. Use an EPA-registered anti-fungal biocide (e.g., copper-8-quinolinolate) for decontaminating structural materials.^{50, 277, 312, 329} **Category IB**
 4. If an environmental source of airborne fungi is not identified, review infection control measures, including engineering controls, to identify potential areas for correction or improvement.^{73, 117} **Category IB**
 5. If possible, perform molecular subtyping of *Aspergillus* spp. isolated from patients and the environment to establish strain identities.^{252, 293-296} **Category II**
- K. If air-supply systems to high-risk areas (e.g., PE rooms) are not optimal, use portable, industrial-grade HEPA filters on a temporary basis until rooms with optimal air-handling systems become available.^{3, 120, 273-277} **Category II**

III. Infection-Control and Ventilation Requirements for PE Rooms

- A. Minimize exposures of severely immunocompromised patients (e.g., solid organ transplant patients or allogeneic neutropenic patients) to activities that might cause aerosolization of fungal spores (e.g., vacuuming or disruption of ceiling tiles).^{9, 20, 109, 272} **Category IB**
- B. Minimize the length of time that immunocompromised patients in PE are outside their rooms for diagnostic procedures and other activities.^{9, 283} **Category IB**
- C. Provide respiratory protection for severely immunocompromised patients when they must leave PE for diagnostic studies and other activities; consult the most recent revision of CDC's *Guidelines for Prevention of Health-Care-Associated Pneumonia* for information regarding the appropriate type of respiratory protection.^{3, 9} **Category II**

- D. Incorporate ventilation engineering specifications and dust-controlling processes into the planning and construction of new PE units. **Category IB, IC**
1. Install central or point-of-use HEPA filters for supply (incoming) air.^{3, 18, 20, 44, 99–104, 120, 254, 316–318, 1432, 1434} **Category IB, IC** (AIA: 5.1, 5.2, 7.2.D)
 2. Ensure that rooms are well sealed by 1) properly constructing windows, doors, and intake and exhaust ports; 2) maintaining ceilings that are smooth and free of fissures, open joints, and crevices; 3) sealing walls above and below the ceiling, and 4) monitoring for leakage and making necessary repairs.^{3, 111, 120, 317, 318} **Category IB, IC** (AIA: 7.2.D3)
 3. Ventilate the room to maintain ≥ 12 ACH.^{3, 9, 120, 241, 317, 318} **Category IC** (AIA: 7.2.D)
 4. Locate air supply and exhaust grilles so that clean, filtered air enters from one side of the room, flows across the patient's bed, and exits from the opposite side of the room.^{3, 120, 317, 318} **Category IC** (AIA: 7.31.D1)
 5. Maintain positive room air pressure (≥ 2.5 Pa [0.01-inch water gauge]) in relation to the corridor.^{3, 35, 120, 317, 318} **Category IB, IC** (AIA: Table 7.2)
 6. Maintain airflow patterns and monitor these on a daily basis by using permanently installed visual means of detecting airflow in new or renovated construction, or using other visual methods (e.g., flutter strips, or smoke tubes) in existing PE units. Document the monitoring results.^{120, 273} **Category IC** (AIA: 7.2.D6)
 7. Install self-closing devices on all room exit doors in protective environments.¹²⁰ **Category IC** (AIA: 7.2.D4)
- E. Do not use laminar air flow systems in newly constructed PE rooms.^{316, 318} **Category II**
- F. Take measures to protect immunocompromised patients who would benefit from a PE room and who also have an airborne infectious disease (e.g., acute VZV infection or tuberculosis).
1. Ensure that the patient's room is designed to maintain positive pressure.
 2. Use an anteroom to ensure appropriate air balance relationships and provide independent exhaust of contaminated air to the outside, or place a HEPA filter in the exhaust duct if the return air must be recirculated.^{120, 317} **Category IC** (AIA: 7.2.D1, A7.2.D)
 3. If an anteroom is not available, place the patient in AII and use portable, industrial-grade HEPA filters to enhance filtration of spores in the room.²¹⁹ **Category II**
- G. Maintain backup ventilation equipment (e.g., portable units for fans or filters) for emergency provision of ventilation requirements for PE areas and take immediate steps to restore the fixed ventilation system function.^{9, 120, 278} **Category IC** (AIA: 5.1)

IV. Infection-Control and Ventilation Requirements for AII Rooms

- A. Incorporate certain specifications into the planning, and construction or renovation of AII units.^{4, 107, 120, 317, 318} **Category IB, IC**
1. Maintain continuous negative air pressure (2.5 Pa [0.01-inch water gauge]) in relation to the air pressure in the corridor; monitor air pressure periodically, preferably daily, with audible manometers or smoke tubes at the door (for existing AII rooms) or with a permanently installed visual monitoring mechanism. Document the results of monitoring.^{120, 317, 318} **Category IB, IC** (AIA: 7.2.C7, Table 7.2)
 2. Ensure that rooms are well-sealed by properly constructing windows, doors, and air-intake and exhaust ports; when monitoring indicates air leakage, locate the leak and make necessary repairs.^{120, 317, 318} **Category IB, IC** (AIA: 7.2.C3)
 3. Install self-closing devices on all AII room exit doors.¹²⁰ **Category IC** (AIA: 7.2.C4)
 4. Provide ventilation to ensure ≥ 12 ACH for renovated rooms and new rooms, and ≥ 6 ACH for existing AII rooms.^{4, 107, 120} **Category IC** (AIA: Table 7.2)

5. Direct exhaust air to the outside, away from air-intake and populated areas. If this is not practical, air from the room can be recirculated after passing through a HEPA filter.^{4, 120} **Category IC** (AIA: Table 7.2)
- B. Where supplemental engineering controls for air cleaning are indicated from a risk assessment of the AII area, install UVGI units in the exhaust air ducts of the HVAC system to supplement HEPA filtration or install UVGI fixtures on or near the ceiling to irradiate upper room air.⁴ **Category II**
- C. Implement environmental infection-control measures for persons with known or suspected airborne infectious diseases.
 1. Use AII rooms for patients with or suspected of having an airborne infection who also require cough-inducing procedures, or use an enclosed booth that is engineered to provide 1) ≥ 12 ACH; 2) air supply and exhaust rate sufficient to maintain a 2.5 Pa [0.01-inch water gauge] negative pressure difference with respect to all surrounding spaces with an exhaust rate of ≥ 50 ft³/min.; and 3) air exhausted directly outside away from air intakes and traffic or exhausted after HEPA filtration prior to recirculation.^{4, 120, 348–350} **Category IB, IC** (AIA: 7.15.E, 7.31.D23, 9.10, Table 7.2)
 2. Although airborne spread of viral hemorrhagic fever (VHF) has not been documented in a health-care setting, prudence dictates placing a VHF patient in an AII room, preferably with an anteroom to reduce the risk of occupational exposure to aerosolized infectious material in blood, vomitus, liquid stool, and respiratory secretions present in large amounts during the end stage of a patient's illness.^{202–204} **Category II**
 - a. If an anteroom is not available, use portable, industrial-grade HEPA filters in the patient's room to provide additional ACH equivalents for removing airborne particulates.
 - b. Ensure that health-care workers wear face shields or goggles with appropriate respirators when entering the rooms of VHF patients with prominent cough, vomiting, diarrhea, or hemorrhage.²⁰³
 3. Place smallpox patients in negative pressure rooms at the onset of their illness, preferably using a room with an anteroom if available.⁶ **Category II**
- D. **No recommendation is offered** regarding negative pressure or isolation rooms for patients with *Pneumocystis carinii* pneumonia.^{126, 131, 152} **Unresolved issue**
- E. Maintain back-up ventilation equipment (e.g., portable units for fans or filters) for emergency provision of ventilation requirements for AII rooms and take immediate steps to restore the fixed ventilation system function.^{4, 120, 278} **Category IC** (AIA: 5.1)

V. Infection-Control and Ventilation Requirements for Operating Rooms

- A. Implement environmental infection-control and ventilation measures for operating rooms.
 1. Maintain positive-pressure ventilation with respect to corridors and adjacent areas.^{7, 120, 356} **Category IB, IC** (AIA: Table 7.2)
 2. Maintain ≥ 15 ACH, of which ≥ 3 ACH should be fresh air.^{120, 357, 358} **Category IC** (AIA: Table 7.2)
 3. Filter all recirculated and fresh air through the appropriate filters, providing 90% efficiency (dust-spot testing) at a minimum.^{120, 362} **Category IC** (AIA: Table 7.3)
 4. In rooms not engineered for horizontal laminar airflow, introduce air at the ceiling and exhaust air near the floor.^{120, 357, 359} **Category IC** (AIA: 7.31.D4)
 5. Do not use UV lights to prevent surgical-site infections.^{356, 364–370} **Category IB**
 6. Keep operating room doors closed except for the passage of equipment, personnel, and patients, and limit entry to essential personnel.^{351, 352} **Category IB**
- B. Follow precautionary procedures for TB patients who also require emergency surgery.^{4, 347, 371} **Category IB, IC**

The recommendations in this guideline for Ebola Virus Disease has been superseded by CDC's Infection Prevention and Control Recommendations for Hospitalized Patients with Known or Suspected Ebola Virus Disease in U.S. Hospitals and by CDC's Interim Guidance for Environmental Infection Control in Hospitals for Ebola Virus issued on August 1, 2014. [Click here for current information on how Ebola virus is transmitted.](#)

1. Use an N95 respirator approved by the National Institute for Occupational Safety and Health (NIOSH) without exhalation valves in the operating room.^{347, 372} **Category IC** (Occupational Safety and Health Administration [OSHA]; 29 Code of Federal Regulations [CFR] 1910.134,139)
 2. Intubate the patient in either the AII room or the operating room; if intubating the patient in the operating room, do not allow the doors to open until 99% of the airborne contaminants are removed (Appendix B, Table B.1).^{4, 358} **Category IB**
 3. When anesthetizing a patient with confirmed or suspected TB, place a bacterial filter between the anesthesia circuit and patient's airway to prevent contamination of anesthesia equipment or discharge of tubercle bacilli into the ambient air.^{371, 373}
Category IB
 4. Extubate and allow the patient to recover in an AII room.^{4, 358} **Category IB**
 5. If the patient has to be extubated in the operating room, allow adequate time for ACH to clean 99% of airborne particles from the air (Appendix B, Table B.1) because extubation is a cough-producing procedure.^{4, 358} **Category IB**
- C. Use portable, industrial-grade HEPA filters temporarily for supplemental air cleaning during intubation and extubation for infectious TB patients who require surgery.^{4, 219, 358}
Category II
1. Position the units appropriately so that all room air passes through the filter; obtain engineering consultation to determine the appropriate placement of the unit.⁴
Category II
 2. Switch the portable unit off during the surgical procedure. **Category II**
 3. Provide fresh air as per ventilation standards for operating rooms; portable units do not meet the requirements for the number of fresh ACH.^{120, 215, 219} **Category II**
- D. If possible, schedule infectious TB patients as the last surgical cases of the day to maximize the time available for removal of airborne contamination. **Category II**
- E. **No recommendation is offered** for performing orthopedic implant operations in rooms supplied with laminar airflow.^{362, 364} **Unresolved issue**
- F. Maintain backup ventilation equipment (e.g., portable units for fans or filters) for emergency provision of ventilation requirements for operating rooms, and take immediate steps to restore the fixed ventilation system function.^{68, 120, 278, 372} **Category IB, IC** (AIA: 5.1)

VI. Other Potential Infectious Aerosol Hazards in Health-Care Facilities

- A. In settings where surgical lasers are used, wear appropriate personal protective equipment, including N95 or N100 respirators, to minimize exposure to laser plumes.^{347, 378, 389}
Category IC (OSHA; 29 CFR 1910.134,139)
- B. Use central wall suction units with in-line filters to evacuate minimal laser plumes.^{378, 382, 386, 389}
Category II
- C. Use a mechanical smoke evacuation system with a high-efficiency filter to manage the generation of large amounts of laser plume, when ablating tissue infected with human papilloma virus (HPV) or performing procedures on a patient with extrapulmonary TB.^{4, 382, 389-392}
Category II

D. Recommendations—Water

I. Controlling the Spread of Waterborne Microorganisms

- A. Practice hand hygiene to prevent the hand transfer of waterborne pathogens, and use barrier precautions (e.g., gloves) as defined by other guidelines.^{6, 464, 577, 586, 592, 1364} **Category IA**

- B. Eliminate contaminated water or fluid environmental reservoirs (e.g., in equipment or solutions) wherever possible.^{464, 465} **Category IB**
- C. Clean and disinfect sinks and wash basins on a regular basis by using an EPA-registered product as set by facility policies. **Category II**
- D. Evaluate for possible environmental sources (e.g., potable water) of specimen contamination when waterborne microorganisms (e.g., NTM) of unlikely clinical importance are isolated from clinical cultures (e.g., specimens collected aseptically from sterile sites or, if post-procedural, colonization occurs after use of tap water in patient care).^{607, 610–612} **Category IB**
- E. Avoid placing decorative fountains and fish tanks in patient-care areas; ensure disinfection and fountain maintenance if decorative fountains are used in the public areas of the health-care facility.⁶⁶⁴ **Category IB**

II. Routine Prevention of Waterborne Microbial Contamination Within the Distribution System

- A. Maintain hot water temperature at the return at the highest temperature allowable by state regulations or codes, preferably $\geq 124^{\circ}\text{F}$ ($\geq 51^{\circ}\text{C}$), and maintain cold water temperature at $< 68^{\circ}\text{F}$ ($< 20^{\circ}\text{C}$).^{3, 661} **Category IC** (States; ASHRAE: 12:2000)
- B. If the hot water temperature can be maintained at $\geq 124^{\circ}\text{F}$ ($\geq 51^{\circ}\text{C}$), explore engineering options (e.g., install preset thermostatic valves in point-of-use fixtures) to help minimize the risk of scalding.⁶⁶¹ **Category II**
- C. When state regulations or codes do not allow hot water temperatures above the range of 105°F – 120°F (40.6°C – 49°C) for hospitals or 95°F – 110°F (35°C – 43.3°C) for nursing care facilities or when buildings cannot be retrofitted for thermostatic mixing valves, follow either of these alternative preventive measures to minimize the growth of *Legionella* spp. in water systems. **Category II**
 - 1. Periodically increase the hot water temperature to $\geq 150^{\circ}\text{F}$ ($\geq 66^{\circ}\text{C}$) at the point of use.⁶⁶¹ **Category II**
 - 2. Alternatively, chlorinate the water and then flush it through the system.^{661, 710, 711} **Category II**
- D. Maintain constant recirculation in hot-water distribution systems serving patient-care areas.¹²⁰ **Category IC** (AIA: 7.31.E.3)

III. Remediation Strategies for Distribution System Repair or Emergencies

- A. Whenever possible, disconnect the ice machine before planned water disruptions. **Category II**
- B. Prepare a contingency plan to estimate water demands for the entire facility in advance of significant water disruptions (i.e., those expected to result in extensive and heavy microbial or chemical contamination of the potable water), sewage intrusion, or flooding.^{713, 719} **Category IC** (JCAHO: EC 1.4)
- C. When a significant water disruption or an emergency occurs, adhere to any advisory to boil water issued by the municipal water utility.⁶⁴² **Category IB, IC** (Municipal order)
 - 1. Alert patients, families, staff, and visitors not to consume water from drinking fountains, ice, or drinks made from municipal tap water, while the advisory is in effect, unless the water has been disinfected (e.g., by bringing to a rolling boil for ≥ 1 minute).⁶⁴² **Category IB, IC** (Municipal order)
 - 2. After the advisory is lifted, run faucets and drinking fountains at full flow for ≥ 5 minutes, or use high-temperature water flushing or chlorination.^{642, 661} **Category IC, II** (Municipal order; ASHRAE 12:2000)
- D. Maintain a high level of surveillance for waterborne disease among patients after a boil water advisory is lifted. **Category II**

- E. Corrective decontamination of the hot water system might be necessary after a disruption in service or a cross-connection with sewer lines has occurred.
1. Decontaminate the system when the fewest occupants are present in the building (e.g., nights or weekends).^{3, 661} **Category IC** (ASHRAE: 12:2000)
 2. If using high-temperature decontamination, raise the hot-water temperature to 160°F–170°F (71°C–77°C) and maintain that level while progressively flushing each outlet around the system for ≥ 5 minutes.^{3, 661} **Category IC** (ASHRAE: 12:2000)
 3. If using chlorination, add enough chlorine, preferably overnight, to achieve a free chlorine residual of ≥ 2 mg/L (≥ 2 ppm) throughout the system.⁶⁶¹ **Category IC** (ASHRAE: 12:2000)
 - a. Flush each outlet until chlorine odor is detected.
 - b. Maintain the elevated chlorine concentration in the system for ≥ 2 hrs (but ≤ 24 hrs).
 4. Use a very thorough flushing of the water system instead of chlorination if a highly chlorine-resistant microorganism (e.g., *Cryptosporidium* spp.) is suspected as the water contaminant. **Category II**
- F. Flush and restart equipment and fixtures according to manufacturers' instructions. **Category II**
- G. Change the pretreatment filter and disinfect the dialysis water system with an EPA-registered product to prevent colonization of the reverse osmosis membrane and downstream microbial contamination.⁷²¹ **Category II**
- H. Run water softeners through a regeneration cycle to restore their capacity and function. **Category II**
- I. If the facility has a water-holding reservoir or water-storage tank, consult the facility engineer or local health department to determine whether this equipment needs to be drained, disinfected with an EPA-registered product, and refilled. **Category II**
- J. Implement facility management procedures to manage a sewage system failure or flooding (e.g., arranging with other health-care facilities for temporary transfer of patients or provision of services), and establish communications with the local municipal water utility and the local health department to ensure that advisories are received in a timely manner upon release.^{713, 719} **Category IC** (JCAHO: EC 1.4; Municipal order)
- K. Implement infection-control measures during sewage intrusion, flooding, or other water-related emergencies.
1. Relocate patients and clean or sterilize supplies from affected areas. **Category II**
 2. If hands are not visibly soiled or contaminated with proteinaceous material, include an alcohol-based hand rub in the hand hygiene process 1) before performing invasive procedures; 2) before and after each patient contact; and 3) whenever hand hygiene is indicated.¹³⁶⁴ **Category II**
 3. If hands are visibly soiled or contaminated with proteinaceous material, use soap and bottled water for handwashing.¹³⁶⁴ **Category II**
 4. If the potable water system is not affected by flooding or sewage contamination, process surgical instruments for sterilization according to standard procedures. **Category II**
 5. Contact the manufacturer of the automated endoscope reprocessor (AER) for specific instructions on the use of this equipment during a water advisory. **Category II**
- L. Remediate the facility after sewage intrusion, flooding, or other water-related emergencies.
1. Close off affected areas during cleanup procedures. **Category II**
 2. Ensure that the sewage system is fully functional before beginning remediation so contaminated solids and standing water can be removed. **Category II**

3. If hard-surface equipment, floors, and walls remain in good repair, ensure that these are dry within 72 hours; clean with detergent according to standard cleaning procedures. **Category II**
 4. Clean wood furniture and materials (if still in good repair); allow them to dry thoroughly before restoring varnish or other surface coatings. **Category II**
 5. Contain dust and debris during remediation and repair as outlined in air recommendations (Air: II G 4, 5). **Category II**
- M. Regardless of the original source of water damage (e.g., flooding versus water leaks from point-of-use fixtures or roofs), remove wet, absorbent structural items (e.g., carpeting, wallboard, and wallpaper) and cloth furnishings if they cannot be easily and thoroughly cleaned and dried within 72 hours (e.g., moisture content $\leq 20\%$ as determined by moisture meter readings); replace with new materials as soon as the underlying structure is declared by the facility engineer to be thoroughly dry.^{18, 266, 278, 1026} **Category IB**

IV. Additional Engineering Measures as Indicated by Epidemiologic Investigation for Controlling Waterborne, Health-Care–Associated Legionnaires Disease

- A. When using a pulse or one-time decontamination method, superheat the water by flushing each outlet for ≥ 5 minutes with water at 160°F–170°F (71°C–77°C) or hyperchlorinate the system by flushing all outlets for ≥ 5 minutes with water containing ≥ 2 mg/L (≥ 2 ppm) free residual chlorine using a chlorine-based product registered by the EPA for water treatment (e.g., sodium hypochlorite [chlorine bleach]).^{661, 711, 714, 724, 764, 766} **Category IB** (ASHRAE: 12:2000)
- B. After a pulse treatment, maintain both the heated water temperature at the return and the cold water temperature as per the recommendation (Water: IIA) wherever practical and permitted by state codes, or chlorinate heated water to achieve 1–2 mg/L (1–2 ppm) free residual chlorine at the tap using a chlorine-based product registered by the EPA for water treatment (e.g., sodium hypochlorite [bleach]).^{26, 437, 661, 709, 726, 727} **Category IC** (States; ASHRAE: 12:2000)
- C. Explore engineering or educational options (e.g., install preset thermostatic mixing valves in point-of-use fixtures or post warning signs at each outlet) to minimize the risk of scalding for patients, visitors, and staff. **Category II**
- D. **No recommendation is offered** for treating water in the facility’s distribution system with chlorine dioxide, heavy-metal ions (e.g., copper or silver), monochloramine, ozone, or UV light.^{728–746} **Unresolved issue**

V. General Infection-Control Strategies for Preventing Legionnaires Disease

- A. Conduct an infection-control risk assessment of the facility to determine if patients at risk or severely immunocompromised patients are present.^{3, 431, 432} **Category IB**
- B. Implement general strategies for detecting and preventing Legionnaires disease in facilities that do not provide care for severely immunocompromised patients (i.e., facilities that do not have HSCT or solid organ transplant programs).^{3, 431, 432} **Category IB**
 1. Establish a surveillance process to detect health-care–associated Legionnaires disease.^{3, 431, 432} **Category IB**
 2. Inform health-care personnel (e.g., infection control, physicians, patient-care staff, and engineering) regarding the potential for Legionnaires disease to occur and measures to prevent and control health-care–associated legionellosis.^{437, 759} **Category IB**
 3. Establish mechanisms to provide clinicians with laboratory tests (e.g., culture, urine antigen, direct fluorescence assay [DFA], and serology) for the diagnosis of Legionnaires disease.^{3, 431} **Category IB**

- C. Maintain a high index of suspicion for health-care–associated Legionnaires disease, and perform laboratory diagnostic tests for legionellosis on suspected cases, especially in patients at risk who do not require a PE for care (e.g., patients receiving systemic steroids; patients aged ≥ 65 years; or patients with chronic underlying disease [e.g., diabetes mellitus, congestive heart failure, or chronic obstructive lung disease]).^{3, 395, 417, 423–425, 432, 435, 437, 453}
Category IA
- D. Periodically review the availability and clinicians' use of laboratory diagnostic tests for Legionnaires disease in the facility; if clinicians' use of the tests on patients with diagnosed or suspected pneumonia is limited, implement measures (e.g., an educational campaign) to enhance clinicians' use of the test(s).⁴⁵³ **Category IB**
- E. If one case of laboratory-confirmed, health-care–associated Legionnaires disease is identified, or if two or more cases of laboratory-suspected, health-care–associated Legionnaires disease occur during a 6-month period, certain activities should be initiated.^{405, 408, 431, 453, 739, 759} **Category IB**
1. Report the cases to the state and local health departments where required. **Category IC** (States)
 2. If the facility does not treat severely immunocompromised patients, conduct an epidemiologic investigation, including retrospective review of microbiologic, serologic, and postmortem data to look for previously unidentified cases of health-care–associated Legionnaires disease, and begin intensive prospective surveillance for additional cases.^{3, 405, 408, 431, 453, 739, 759} **Category IB**
 3. If no evidence of continued health-care–associated transmission exists, continue intensive prospective surveillance for ≥ 2 months after the initiation of surveillance.^{3, 405, 408, 431, 453, 739, 759} **Category IB**
- F. If there is evidence of continued health-care–associated transmission (i.e., an outbreak), conduct an environmental assessment to determine the source of *Legionella* spp.^{403–410, 455} **Category IB**
1. Collect water samples from potential aerosolized water sources (Appendix C).¹²⁰⁹ **Category IB**
 2. Save and subtype isolates of *Legionella* spp. obtained from patients and the environment.^{403–410, 453, 763, 764} **Category IB**
 3. If a source is identified, promptly institute water system decontamination measures per recommendations (see Water IV).^{766, 767} **Category IB**
 4. If *Legionella* spp. are detected in ≥ 1 cultures (e.g., conducted at 2-week intervals during 3 months), reassess the control measures, modify them accordingly, and repeat the decontamination procedures; consider intensive use of techniques used for initial decontamination, or a combination of superheating and hyperchlorination.^{3, 767, 768} **Category IB**
- G. If an environmental source is not identified during a Legionnaires disease outbreak, continue surveillance for new cases for ≥ 2 months. Either defer decontamination pending identification of the source of *Legionella* spp., or proceed with decontamination of the hospital's water distribution system, with special attention to areas involved in the outbreak. **Category II**
- H. **No recommendation is offered** regarding routine culturing of water systems in health-care facilities that do not have patient-care areas (i.e., PE or transplant units) for persons at high risk for *Legionella* spp. infection.^{26, 453, 707, 709, 714, 747, 753} **Unresolved issue**
- I. **No recommendation is offered** regarding the removal of faucet aerators in areas for immunocompetent patients. **Unresolved issue**
- J. Keep adequate records of all infection-control measures and environmental test results for potable water systems. **Category II**

VI. Preventing Legionnaires Disease in Protective Environments and Transplant Units

- A. When implementing strategies for preventing Legionnaires disease among severely immunosuppressed patients housed in facilities with HSCT or solid-organ transplant programs, incorporate these specific surveillance and epidemiologic measures in addition to the steps previously outlined (Water: V and Appendix C).
1. Maintain a high index of suspicion for legionellosis in transplant patients even when environmental surveillance cultures do not yield legionellae.^{430, 431} **Category IB**
 2. If a case occurs in a severely immunocompromised patient, or if severely immunocompromised patients are present in high-risk areas of the hospital (e.g., PE or transplant units) and cases are identified elsewhere in the facility, conduct a combined epidemiologic and environmental investigation to determine the source of *Legionella* spp.^{431, 767} **Category IB**
- B. Implement culture strategies and potable water and fixture treatment measures in addition to those previously outlined (Water: V). **Category II**
1. Depending on state regulations on potable water temperature in public buildings,⁷²⁵ hospitals housing patients at risk for health-care-associated legionellosis should either maintain heated water with a minimum return temperature of $\geq 124^{\circ}\text{F}$ [$\geq 51^{\circ}\text{C}$] and cold water at $< 68^{\circ}\text{F}$ [$< 20^{\circ}\text{C}$], or chlorinate heated water to achieve 1–2 mg/L (1–2 ppm) of free residual chlorine at the tap.^{26, 441, 661, 709–711, 726, 727} **Category II**
 2. Periodic culturing for legionellae in potable water samples from HSCT or solid-organ transplant units can be performed as part of a comprehensive strategy to prevent Legionnaires disease in these units.^{9, 431, 710, 769} **Category II**
 3. **No recommendation is offered** regarding the optimal methodology (i.e., frequency or number of sites) for environmental surveillance cultures in HSCT or solid organ transplant units. **Unresolved issue**
 4. In areas with patients at risk, when *Legionella* spp. are not detectable in unit water, remove, clean, and disinfect shower heads and tap aerators monthly by using a chlorine-based, EPA-registered product. If an EPA-registered chlorine disinfectant is not available, use a chlorine bleach solution (500–615 ppm [1:100 v/v dilution]).^{661, 745} **Category II**
- C. If *Legionella* spp. are determined to be present in the water of a transplant unit, implement certain measures until *Legionella* spp. are no longer detected by culture.
1. Decontaminate the water supply as outlined previously (Water: IV).^{3, 9, 661, 766, 767} **Category IB**
 2. Do not use water from the faucets in patient-care rooms to avoid creating infectious aerosols.^{9, 412} **Category IB**
 3. Restrict severely immunocompromised patients from taking showers.^{9, 412} **Category IB**
 4. Use water that is not contaminated with *Legionella* spp. for HSCT patients' sponge baths.^{9, 412} **Category IB**
 5. Provide patients with sterile water for tooth brushing, drinking, and for flushing nasogastric tubing during legionellosis outbreaks.^{9, 412} **Category IB**
- D. Do not use large-volume room air humidifiers that create aerosols (e.g., by Venturi principle, ultrasound, or spinning disk) unless they are subjected to high-level disinfection and filled only with sterile water.^{3, 9, 402, 455} **Category IB**

VII. Cooling Towers and Evaporative Condensers

- A. When planning construction of new health-care facilities, locate cooling towers so that the drift is directed away from the air-intake system, and design the towers to minimize the volume of aerosol drift.^{404, 661, 786} **Category IC** (ASHRAE: 12:2000)

- B. Implement infection-control procedures for operational cooling towers.^{404, 661, 784}
Category IC (ASHRAE: 12:2000)
1. Install drift eliminators.^{404, 661, 784} **Category IC** (ASHRAE: 12:2000)
 2. Use an effective EPA-registered biocide on a regular basis.⁶⁶¹ **Category IC** (ASHRAE: 12:2000)
 3. Maintain towers according to manufacturers' recommendations, and keep detailed maintenance and infection control records, including environmental test results from legionellosis outbreak investigations.⁶⁶¹ **Category IC** (ASHRAE: 12:2000)
- C. If cooling towers or evaporative condensers are implicated in health-care-associated legionellosis, decontaminate the cooling-tower system.^{404, 405, 786, 787} **Category IB**

VIII. Dialysis Water Quality and Dialysate

- A. Adhere to current AAMI standards for quality assurance performance of devices and equipment used to treat, store, and distribute water in hemodialysis centers (both acute and maintenance [chronic] settings) and for the preparation of concentrates and dialysate.^{31, 32, 666-668, 789, 791, 800, 807, 809, 1454, 1455} **Category IA, IC** (AAMI: ANSI/AAMI RD5:1992, ANSI/AAMI RD 47:1993)
- B. **No recommendation is offered** regarding whether more stringent requirements for water quality should be imposed in hemofiltration and hemodiafiltration. **Unresolved issue**^{789, 791, 792, 834, 835}
- C. Conduct microbiological testing specific to water in dialysis settings. **Category IA, IC** (AAMI: ANSI/AAMI RD 5: 1992, ANSI/AAMI RD 47: 1993, ANSI/AAMI RD 62:2001)
1. Perform bacteriologic assays of water and dialysis fluids at least once a month and during outbreaks using standard quantitative methods.^{792, 834, 835} **Category IA, IC** (AAMI: ANSI/AAMI RD 62:2001)
 - a. Assay for heterotrophic, mesophilic bacteria (e.g., *Pseudomonas* spp).
 - b. Do not use nutrient-rich media (e.g., blood agar or chocolate agar).
 2. In conjunction with microbiological testing, perform endotoxin testing on product water used to reprocess dialyzers for multiple use.^{789, 791, 806, 811, 816, 829} **Category IA, IC** (AAMI: ANSI/AAMI RD 5:1992, ANSI/AAMI RD 47:1993)
 3. Ensure that water does not exceed the limits for microbial counts and endotoxin concentrations outlined in Table 18.^{789, 791, 800} **Category IA, IC** (AAMI: ANSI/AAMI RD 5:1992, ANSI/AAMI RD 47:1993)
- D. Disinfect water distribution systems in dialysis settings on a regular schedule. Monthly disinfection is recommended.^{666-668, 792, 800} **Category IA, IC** (AAMI: ANSI/AAMI RD62:2001)
- E. Whenever practical, design and engineer water systems in dialysis settings to avoid incorporating joints, dead-end pipes, and unused branches and taps that can harbor bacteria.^{666-668, 792, 800} **Category IA, IC** (AAMI: ANSI/AAMI RD62:2001)
- F. When storage tanks are used in dialysis systems, they should be routinely drained, disinfected with an EPA-registered product, and fitted with an ultrafilter or pyrogenic filter (membrane filter with a pore size sufficient to remove small particles and molecules ≥ 1 kilodalton) installed in the water line distal to the storage tank.⁷⁹² **Category IC** (AAMI: ANSI/AAMI RD62:2001)

IX. Ice Machines and Ice

- A. Do not handle ice directly by hand, and wash hands before obtaining ice. **Category II**
- B. Use a smooth-surface ice scoop to dispense ice.^{680, 863} **Category II**
1. Keep the ice scoop on a chain short enough the scoop cannot touch the floor, or keep the scoop on a clean, hard surface when not in use.^{680, 863} **Category II**
 2. Do not store the ice scoop in the ice bin. **Category II**
- C. Do not store pharmaceuticals or medical solutions on ice intended for consumption; use sterile ice to keep medical solutions cold, or use equipment specifically manufactured for this purpose.^{600, 863} **Category IB**

- D. Machines that dispense ice are preferred to those that require ice to be removed from bins or chests with a scoop.^{687, 869} **Category II**
- E. Limit access to ice-storage chests, and keep the container doors closed except when removing ice.⁸⁶³ **Category II**
- F. Clean, disinfect, and maintain ice-storage chests on a regular basis. **Category II**
 - 1. Follow the manufacturer's instructions for cleaning. **Category II**
 - 2. Use an EPA-registered disinfectant suitable for use on ice machines, dispensers, or storage chests in accordance with label instructions. **Category II**
 - 3. If instructions and EPA-registered disinfectants suitable for use on ice machines are not available, use a general cleaning/disinfecting regimen as outlined in Box 12.⁸⁶³ **Category II**
 - 4. Flush and clean the ice machines and dispensers if they have not been disconnected before anticipated lengthy water disruptions. **Category II**
- G. Install proper air gaps where the condensate lines meet the waste lines. **Category II**
- H. Conduct microbiologic sampling of ice, ice chests, and ice-making machines and dispensers where indicated during an epidemiologic investigation.^{861–863} **Category IB**

X. Hydrotherapy Tanks and Pools

- A. Drain and clean hydrotherapy equipment (e.g., Hubbard tanks, tubs, whirlpools, whirlpool spas, or birthing tanks) after each patient's use, and disinfect equipment surfaces and components by using an EPA-registered product in accordance with the manufacturer's instructions. **Category II**
- B. In the absence of an EPA-registered product for water treatment, add sodium hypochlorite to the water:
 - 1. Maintain a 15-ppm chlorine residual in the water of small hydrotherapy tanks, Hubbard tanks, and tubs.⁸⁸⁹ **Category II**
 - 2. Maintain a 2–5 ppm chlorine residual in the water of whirlpools and whirlpool spas.⁹⁰⁵ **Category II**
 - 3. If the pH of the municipal water is in the basic range (e.g., when chloramine is used as the primary drinking water disinfectant in the community), consult the facility engineer regarding the possible need to adjust the pH of the water to a more acid level before disinfection, to enhance the biocidal activity of chlorine.⁸⁹⁴ **Category II**
- C. Clean and disinfect hydrotherapy equipment after using tub liners. **Category II**
- D. Clean and disinfect inflatable tubs unless they are single-use equipment. **Category II**
- E. **No recommendation is offered** regarding the use of antiseptic chemicals (e.g., chloramine-T) in the water during hydrotherapy sessions. **Unresolved issue**
- F. Conduct a risk assessment of patients prior to their use of large hydrotherapy pools, deferring patients with draining wounds or fecal incontinence from pool use until their condition resolves. **Category II**
- G. For large hydrotherapy pools, use pH and chlorine residual levels appropriate for an indoor pool as provided by local and state health agencies. **Category IC** (States)
- H. **No recommendation is offered** regarding the use in health care of whirlpools or spa equipment manufactured for home or recreational use. **Unresolved issue**

XI. Miscellaneous Medical Equipment Connected to Water Systems

- A. Clean, disinfect, and maintain AER equipment according to the manufacturer's instructions and relevant scientific literature to prevent inadvertent contamination of endoscopes and bronchoscopes with waterborne microorganisms.^{911–915} **Category IB**
 - 1. To rinse disinfected endoscopes and bronchoscopes, use water of the highest quality practical for the system's engineering and design (e.g., sterile water or

- bacteriologically-filtered water [water filtered through 0.1–0.2- μm filters]).^{912, 914, 915, 918} **Category IB**
2. Dry the internal channels of the reprocessed endoscope or bronchoscope using a proven method (e.g., 70% alcohol followed by forced-air treatment) to lessen the potential for the proliferation of waterborne microorganisms and to help prevent biofilm formation.^{671, 921, 923, 925, 928} **Category IB**
- B. Use water that meets nationally recognized standards set by the EPA for drinking water (<500 CFU/mL for heterotrophic plate count) for routine dental treatment output water.^{935, 936, 943, 944} **Category IB, IC** (EPA: 40 CFR 1 Part 141, Subpart G).
- C. Take precautions to prevent waterborne contamination of dental unit water lines and instruments.
1. After each patient, discharge water and air for a minimum of 20–30 seconds from any dental device connected to the dental water system that enters the patient’s mouth (e.g., handpieces, ultrasonic scalers, and air/water syringe).^{936, 937} **Category II**
 2. Consult with dental water-line manufacturers to 1) determine suitable methods and equipment to obtain the recommended water quality; and 2) determine appropriate methods for monitoring the water to ensure quality is maintained.^{936, 946} **Category II**
 3. Consult with the dental unit manufacturer on the need for periodic maintenance of anti-retraction mechanisms.^{937, 946} **Category IB**

E. Recommendations—Environmental Services

I. Cleaning and Disinfecting Strategies for Environmental Surfaces in Patient-Care Areas

- A. Select EPA-registered disinfectants, if available, and use them in accordance with the manufacturer’s instructions.^{2, 974, 983} **Category IB, IC** (EPA: 7 United States Code [USC] § 136 et seq)
- B. Do not use high-level disinfectants/liquid chemical sterilants for disinfection of either noncritical instrument/devices or any environmental surfaces; such use is counter to label instructions for these toxic chemicals.^{951, 952, 961–964} **Category IB, IC** (FDA: 21 CFR 801.5, 807.87.e)
- C. Follow manufacturers’ instructions for cleaning and maintaining noncritical medical equipment. **Category II**
- D. In the absence of a manufacturer’s cleaning instructions, follow certain procedures.
 1. Clean noncritical medical equipment surfaces with a detergent/disinfectant. This may be followed with an application of an EPA-registered hospital disinfectant with or without a tuberculocidal claim (depending on the nature of the surface and the degree of contamination), in accordance with disinfectant label instructions.⁹⁵² **Category II**
 2. Do not use alcohol to disinfect large environmental surfaces.⁹⁵¹ **Category II**
 3. Use barrier protective coverings as appropriate for noncritical equipment surfaces that are 1) touched frequently with gloved hands during the delivery of patient care; 2) likely to become contaminated with blood or body substances; or 3) difficult to clean (e.g., computer keyboards).⁹³⁶ **Category II**
- E. Keep housekeeping surfaces (e.g., floors, walls, and tabletops) visibly clean on a regular basis and clean up spills promptly.⁹⁵⁴ **Category II**
 1. Use a one-step process and an EPA-registered hospital disinfectant/detergent designed for general housekeeping purposes in patient-care areas when 1) uncertainty exists as to the nature of the soil on these surfaces [e.g., blood or body fluid contamination versus routine dust or dirt]; or 2) uncertainty exists regarding the presence or absence of multi-drug resistant organisms on such surfaces.^{952, 983, 986, 987} **Category II**

2. Detergent and water are adequate for cleaning surfaces in nonpatient-care areas (e.g., administrative offices). **Category II**
3. Clean and disinfect high-touch surfaces (e.g., doorknobs, bed rails, light switches, and surfaces in and around toilets in patients' rooms) on a more frequent schedule than minimal touch housekeeping surfaces. **Category II**
4. Clean walls, blinds, and window curtains in patient-care areas when they are visibly dusty or soiled.^{2, 971, 972, 982} **Category II**
- F. Do not perform disinfectant fogging in patient-care areas.^{2, 976} **Category IB**
- G. Avoid large-surface cleaning methods that produce mists or aerosols or disperse dust in patient-care areas.^{9, 20, 109, 272} **Category IB**
- H. Follow proper procedures for effective use of mops, cloths, and solutions. **Category II**
 1. Prepare cleaning solutions daily or as needed, and replace with fresh solution frequently according to facility policies and procedures.^{986, 987} **Category II**
 2. Change the mop head at the beginning of the day and also as required by facility policy, or after cleaning up large spills of blood or other body substances. **Category II**
 3. Clean mops and cloths after use and allow to dry before reuse; or use single-use, disposable mop heads and cloths.^{971, 988-990} **Category II**
- I. After the last surgical procedure of the day or night, wet vacuum or mop operating room floors with a single-use mop and an EPA-registered hospital disinfectant.⁷ **Category IB**
- J. Do not use mats with tacky surfaces at the entrance to operating rooms or infection-control suites.⁷ **Category IB**
- K. Use appropriate dusting methods for patient-care areas designated for immunocompromised patients (e.g., HSCT patients).^{9, 94, 986} **Category IB**
 1. Wet-dust horizontal surfaces daily by moistening a cloth with a small amount of an EPA-registered hospital detergent/disinfectant.^{9, 94, 986} **Category IB**
 2. Avoid dusting methods that disperse dust (e.g., feather-dusting).⁹⁴ **Category IB**
- L. Keep vacuums in good repair, and equip vacuums with HEPA filters for use in areas with patients at risk.^{9, 94, 986, 994} **Category IB**
- M. Close the doors of immunocompromised patients' rooms when vacuuming, waxing, or buffing corridor floors to minimize exposure to airborne dust.^{9, 94, 994} **Category IB**
- N. When performing low- or intermediate-level disinfection of environmental surfaces in nurseries and neonatal units, avoid unnecessary exposure of neonates to disinfectant residues on environmental surfaces by using EPA-registered disinfectants in accordance with manufacturers' instructions and safety advisories.^{974, 995-997} **Category IB, IC** (EPA: 7 USC § 136 et seq.)
 1. Do not use phenolics or any other chemical germicide to disinfect bassinets or incubators during an infant's stay.^{952, 995-997} **Category IB**
 2. Rinse disinfectant-treated surfaces, especially those treated with phenolics, with water.⁹⁹⁵⁻⁹⁹⁷ **Category IB**
- O. When using phenolic disinfectants in neonatal units, prepare solutions to correct concentrations in accordance with manufacturers' instructions, or use premixed formulations.^{974, 995-997} **Category IB, IC** (EPA: 7 USC § 136 et seq.)

II. Cleaning Spills of Blood and Body Substances

- A. Promptly clean and decontaminate spills of blood or other potentially infectious materials.^{967, 998-1004} **Category IB, IC** (OSHA: 29 CFR 1910.1030 §d.4.ii.A)
- B. Follow proper procedures for site decontamination of spills of blood or blood-containing body fluids.^{967, 998-1004} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.ii.A)
 1. Use protective gloves and other PPE appropriate for this task.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.3.i, ii)

2. If the spill contains large amounts of blood or body fluids, clean the visible matter with disposable absorbent material, and discard the contaminated materials in appropriate, labeled containment.^{967, 1002, 1003, 1010, 1012} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iii.B)
3. Swab the area with a cloth or paper towels moderately wetted with disinfectant, and allow the surface to dry.^{967, 1010} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.ii.A)
- C. Use EPA-registered hospital disinfectants labeled tuberculocidal or registered germicides on the EPA Lists D and E (products with specific label claims for HIV or hepatitis B virus [HBV]) in accordance with label instructions to decontaminate spills of blood and other body fluids.^{967, 1007, 1010} **Category IC** (OSHA 29 CFR 1910.1030 § d.4.ii.A memorandum 2/28/97; compliance document CPL 2-2.44D [11/99])
- D. An EPA-registered sodium hypochlorite product is preferred, but if such products are not available, generic versions of sodium hypochlorite solutions (e.g., household chlorine bleach) may be used.
 1. Use a 1:100 dilution (500–615 ppm available chlorine) to decontaminate nonporous surfaces after cleaning a spill of either blood or body fluids in patient-care settings.^{1010, 1011} **Category II**
 2. If a spill involves large amounts of blood or body fluids, or if a blood or culture spill occurs in the laboratory, use a 1:10 dilution (5,000–6,150 ppm available chlorine) for the first application of germicide before cleaning.^{954, 1010} **Category II**

III. Carpeting and Cloth Furnishings

- A. Vacuum carpeting in public areas of health-care facilities and in general patient-care areas regularly with well-maintained equipment designed to minimize dust dispersion.⁹⁸⁶
Category II
- B. Periodically perform a thorough, deep cleaning of carpeting as determined by facility policy by using a method that minimizes the production of aerosols and leaves little or no residue.¹¹¹ **Category II**
- C. Avoid use of carpeting in high-traffic zones in patient-care areas or where spills are likely (e.g., burn therapy units, operating rooms, laboratories, and intensive care units).^{111, 1023, 1028}
Category IB
- D. Follow proper procedures for managing spills on carpeting.
 1. Spot-clean blood or body substance spills promptly.^{967, 1010, 1011, 1032} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.ii.A, interpretation)
 2. If a spill occurs on carpet tiles, replace any tiles contaminated by blood and body fluids or body substances.¹⁰³² **Category IC** (OSHA 29 CFR 1910.1030 § d.4.ii interpretation)
- E. Thoroughly dry wet carpeting to prevent the growth of fungi; replace carpeting that remains wet after 72 hours.^{9, 1026} **Category IB**
- F. **No recommendation is offered** regarding the routine use of fungicidal or bactericidal treatments for carpeting in public areas of a health-care facility or in general patient-care areas. **Unresolved issue**
- G. Do not use carpeting in hallways and patient rooms in areas housing immunosuppressed patients (e.g., PE areas).^{9, 111} **Category IB**
- H. Avoid the use of upholstered furniture and furnishings in high-risk patient-care areas and in areas with increased potential for body substance contamination (e.g., pediatrics units).⁹
Category II
- I. **No recommendation is offered** regarding whether upholstered furniture and furnishings should be avoided in general patient-care areas. **Unresolved issue**
- J. Maintain upholstered furniture in good repair. **Category II**
 1. Maintain the surface integrity of the upholstery by repairing tears and holes.
Category II

2. If upholstered furniture in a patient's room requires cleaning to remove visible soil or body substance contamination, move that item to a maintenance area where it can be adequately cleaned with a process appropriate for the type of upholstery and the nature of the soil. **Category II**

IV. Flowers and Plants in Patient-Care Areas

- A. Flowers and potted plants need not be restricted from areas for immunocompetent patients.^{515, 702, 1040, 1042} **Category II**
- B. Designate care and maintenance of flowers and potted plants to staff not directly involved with patient care.⁷⁰² **Category II**
- C. If plant or flower care by patient-care staff is unavoidable, instruct the staff to wear gloves when handling the plants and flowers and perform hand hygiene after glove removal.⁷⁰² **Category II**
- D. Do not allow fresh or dried flowers, or potted plants in patient-care areas for immunosuppressed patients.^{9, 109, 515, 1046} **Category II**

V. Pest Control

- A. Develop pest-control strategies, with emphasis on kitchens, cafeterias, laundries, central sterile supply areas, operating rooms, loading docks, construction activities, and other areas prone to infestations.^{1050, 1072, 1075} **Category II**
- B. Install screens on all windows that open to the outside; keep screens in good repair.¹⁰⁷² **Category IB**
- C. Contract for routine pest control service by a credentialed pest-control specialist who will tailor the application to the needs of a health-care facility.¹⁰⁷⁵ **Category II**
- D. Place laboratory specimens (e.g., fixed sputum smears) in covered containers for overnight storage.^{1065, 1066} **Category II**

VI. Special Pathogens

- A. Use appropriate hand hygiene, PPE (e.g., gloves), and isolation precautions during cleaning and disinfecting procedures.^{5, 952, 1130, 1364} **Category IB**
- B. Use standard cleaning and disinfection protocols to control environmental contamination with antibiotic-resistant gram-positive cocci (e.g., methicillin-resistant *Staphylococcus aureus*, vancomycin intermediate-resistant *Staphylococcus aureus*, or vancomycin-resistant *Enterococcus* [VRE]).^{5, 1116–1118} **Category IB**
 1. Pay close attention to cleaning and disinfection of high-touch surfaces in patient-care areas (e.g., bed rails, carts, bedside commodes, bedrails, doorknobs, or faucet handles).^{5, 1116–1118} **Category IB**
 2. Ensure compliance by housekeeping staff with cleaning and disinfection procedures.^{5, 1116–1118} **Category IB**
 3. Use EPA-registered hospital disinfectants appropriate for the surface to be disinfected (e.g., either low- or intermediate-level disinfection) as specified by the manufacturers' instructions.^{974, 1106–1110, 1118} **Category IB, IC** (EPA: 7 USC § 136 et seq.)
 4. When contact precautions are indicated for patient care, use disposable patient-care items (e.g., blood pressure cuffs) whenever possible to minimize cross-contamination with multiple-resistant microorganisms.¹¹⁰² **Category IB**
 5. Follow these same surface cleaning and disinfecting measures for managing the environment of VRSA patients.^{1110, 1116–1118} **Category II**
- C. Environmental-surface culturing can be used to verify the efficacy of hospital policies and procedures before and after cleaning and disinfecting rooms that house patients with VRE.^{5, 1084, 1087, 1088, 1092, 1096} **Category II**

1. Obtain prior approval from infection-control staff and the clinical laboratory before performing environmental surface culturing. **Category II**
 2. Infection-control staff, with clinical laboratory consultation, must supervise all environmental culturing. **Category II**
- D. Thoroughly clean and disinfect environmental and medical equipment surfaces on a regular basis using EPA-registered disinfectants in accordance with manufacturers' instructions.^{952, 974, 1130, 1143} **Category IB, IC** (EPA: 7 USC § 136 et seq.)
- E. Advise families, visitors, and patients about the importance of hand hygiene to minimize the spread of body substance contamination (e.g., respiratory secretions or fecal matter) to surfaces.⁹⁵² **Category II**
- F. Do not use high-level disinfectants (i.e., liquid chemical sterilants) on environmental surfaces; such use is inconsistent with label instructions and because of the toxicity of the chemicals.^{2, 951, 952, 964} **Category IC** (FDA: 21 CFR 801.5, 807.87.e)
- G. Because no EPA-registered products are specific for inactivating *Clostridium difficile* spores, use hypochlorite-based products for disinfection of environmental surfaces in those patient-care areas where surveillance and epidemiology indicate ongoing transmission of *C. difficile*.^{952, 1130, 1141} **Category II**
- H. **No recommendation is offered** regarding the use of specific EPA-registered hospital disinfectants with respect to environmental control of *C. difficile*. **Unresolved issue**
- I. Apply standard cleaning and disinfection procedures to control environmental contamination with respiratory and enteric viruses in pediatric-care units and care areas for immunocompromised patients.^{986, 1158} **Category IC** (EPA: 7 USC § 136 et seq.)
- J. Clean surfaces that have been contaminated with body substances; perform low- to intermediate-level disinfection on cleaned surfaces with an EPA-registered disinfectant in accordance with the manufacturer's instructions.^{967, 974, 1158} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.ii.A; EPA: 7 USC § 136 et seq.)
- K. Use disposable barrier coverings as appropriate to minimize surface contamination. **Category II**
- L. Develop and maintain cleaning and disinfection procedures to control environmental contamination with agents of Creutzfeldt-Jakob disease (CJD), for which no EPA-registered product exists. **Category II**
1. In the absence of contamination with central nervous system tissue, extraordinary measures (e.g., use of 2N sodium hydroxide [NaOH] or applying full-strength sodium hypochlorite) are not needed for routine cleaning or terminal disinfection of a room housing a confirmed or suspected CJD patient.^{951, 1199} **Category II**
 2. After removing gross tissue from the surface, use either 1N NaOH or a sodium hypochlorite solution containing approximately 10,000–20,000 ppm available chlorine (dilutions of 1:5 to 1:3 v/v, respectively, of U.S. household chlorine bleach; contact the manufacturers of commercially available sodium hypochlorite products for advice) to decontaminate operating room or autopsy surfaces with central nervous system or cerebral spinal fluid contamination from a diagnosed or suspected CJD patient.^{951, 1170, 1188, 1191, 1197–1199, 1201} **Category II**
 - a. The contact time for the chemical used during this process should be 30 min–1 hour.^{1191, 1197, 1201}
 - b. Blot up the chemical with absorbent material and rinse the treated surface thoroughly with water.
 - c. Discard the used, absorbent material into appropriate waste containment.
 3. Use disposable, impervious covers to minimize body substance contamination to autopsy tables and surfaces.^{1197, 1201} **Category IB**

- M. Use standard procedures for containment, cleaning, and decontamination of blood spills on surfaces as previously described (Environmental Services: II).⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 §d.4.ii.A)
1. Wear PPE appropriate for a surface decontamination and cleaning task.^{967, 1199}
Category IC (OSHA 29 CFR 1910.1030 §d.3.i, ii)
 2. Discard used PPE by using routine disposal procedures or decontaminate reusable PPE as appropriate.^{967, 1199} **Category IC** (OSHA 29 CFR 1910.1030 §d.3.viii)

F. Recommendations—Environmental Sampling

I. General Information

- A. Do not conduct random, undirected microbiologic sampling of air, water, and environmental surfaces in health-care facilities.^{2, 1214} **Category IB**
- B. When indicated, conduct microbiologic sampling as part of an epidemiologic investigation or during assessment of hazardous environmental conditions to detect contamination and verify abatement of a hazard.^{2, 1214} **Category IB**
- C. Limit microbiologic sampling for quality assurance purposes to 1) biological monitoring of sterilization processes; 2) monthly cultures of water and dialysate in hemodialysis units; and 3) short-term evaluation of the impact of infection-control measures or changes in infection-control protocols.^{2, 1214} **Category IB**

II. Air, Water, and Environmental-Surface Sampling

- A. When conducting any form of environmental sampling, identify existing comparative standards and fully document departures from standard methods.^{945, 1214, 1223, 1224, 1238}
Category II
- B. Select a high-volume air sampling device if anticipated levels of microbial airborne contamination are expected to be low.^{290, 1218, 1223, 1224} **Category II**
- C. Do not use settle plates to quantify the concentration of airborne fungal spores.²⁹⁰
Category II
- D. When sampling water, choose growth media and incubation conditions that will facilitate the recovery of waterborne organisms.⁹⁴⁵ **Category II**
- E. When using a sample/rinse method for sampling an environmental surface, develop and document a procedure for manipulating the swab, gauze, or sponge in a reproducible manner so that results are comparable.¹²³⁸ **Category II**
- F. When environmental samples and patient specimens are available for comparison, perform the laboratory analysis on the recovered microorganisms down to the species level at a minimum and beyond the species level if possible.¹²¹⁴ **Category II**

G. Recommendations—Laundry and Bedding

I. Employer Responsibilities

- A. Employers must launder workers' personal protective garments or uniforms that are contaminated with blood or other potentially infectious materials.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.3.iv)

II. Laundry Facilities and Equipment

- A. Maintain the receiving area for contaminated textiles at negative pressure compared with the clean areas of the laundry in accordance with AIA construction standards in effect during the time of facility construction.^{120, 1260–1262} **Category IC** (AIA: 7.23.B1, B2)
- B. Ensure that laundry areas have handwashing facilities and products and appropriate PPE available for workers.^{120, 967} **Category IC** (AIA: 7.23.D4; OSHA: 29 CFR 1910.1030 § d.2.iii)
- C. Use and maintain laundry equipment according to manufacturers' instructions.^{1250, 1263}
Category II
- D. Do not leave damp textiles or fabrics in machines overnight.¹²⁵⁰ **Category II**
- E. Disinfection of washing and drying machines in residential care is not needed as long as gross soil is removed before washing and proper washing and drying procedures are used.
Category II

III. Routine Handling of Contaminated Laundry

- A. Handle contaminated textiles and fabrics with minimum agitation to avoid contamination of air, surfaces, and persons.^{6, 967, 1258, 1259} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iv)
- B. Bag or otherwise contain contaminated textiles and fabrics at the point of use.⁹⁶⁷
Category IC (OSHA: 29 CFR 1910.1030 § d.4.iv)
 - 1. Do not sort or prerinse contaminated textiles or fabrics in patient-care areas.⁹⁶⁷
Category IC (OSHA: 29 CFR 1910.1030 § d.4.iv)
 - 2. Use leak-resistant containment for textiles and fabrics contaminated with blood or body substances.^{967, 1258} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iv)
 - 3. Identify bags or containers for contaminated textiles with labels, color coding, or other alternative means of communication as appropriate.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iv)
- C. Covers are not needed on contaminated textile hampers in patient-care areas. **Category II**
- D. If laundry chutes are used, ensure that they are properly designed, maintained, and used in a manner to minimize dispersion of aerosols from contaminated laundry.^{1253, 1267–1270}
Category IC (AAMI: ANSI/AAMI ST65:2000)
 - 1. Ensure that laundry bags are closed before tossing the filled bag into the chute.
Category II
 - 2. Do not place loose items in the chute. **Category II**
- E. Establish a facility policy to determine when textiles or fabrics should be sorted in the laundry facility (i.e., before or after washing).^{1271, 1272} **Category II**

IV. Laundry Process

- A. If hot-water laundry cycles are used, wash with detergent in water $\geq 160^{\circ}\text{F}$ ($\geq 71^{\circ}\text{C}$) for ≥ 25 minutes.^{2, 120} **Category IC** (AIA: 7.31.E3)
- B. **No recommendation is offered** regarding a hot-water temperature setting and cycle duration for items laundered in residence-style health-care facilities. **Unresolved issue**
- C. Follow fabric-care instructions and special laundering requirements for items used in the facility.¹²⁷⁸ **Category II**
- D. Choose chemicals suitable for low-temperature washing at proper use concentration if low-temperature ($< 160^{\circ}\text{F}$ [$< 71^{\circ}\text{C}$]) laundry cycles are used.^{1247, 1281–1285} **Category II**
- E. Package, transport, and store clean textiles and fabrics by methods that will ensure their cleanliness and protect them from dust and soil during interfacility loading, transport, and unloading.² **Category II**

V. Microbiologic Sampling of Textiles

- A. Do not conduct routine microbiological sampling of clean textiles.^{2, 1286} **Category IB**

- B. Use microbiological sampling during outbreak investigations if epidemiologic evidence suggests a role for health-care textiles and clothing in disease transmission.¹²⁸⁶ **Category IB**

VI. Special Laundry Situations

- A. Use sterilized textiles, surgical drapes, and gowns for situations requiring sterility in patient care.⁷ **Category IB**
- B. Use hygienically clean textiles (i.e., laundered, but not sterilized) in neonatal intensive care units.^{997, 1288} **Category IB**
- C. Follow manufacturers' recommendations for cleaning fabric products including those with coated or laminated surfaces. **Category II**
- D. Do not use dry cleaning for routine laundering in health-care facilities.^{1289–1291} **Category II**
- E. Use caution when considering the use of antimicrobial mattresses, textiles, and clothing as replacements for standard bedding and other fabric items; EPA has not approved public health claims asserting protection against human pathogens for treated articles.¹³⁰⁶ **Category II**
- F. **No recommendation is offered** regarding using disposable fabrics and textiles versus durable goods. **Unresolved issue**

VII. Mattresses and Pillows

- A. Keep mattresses dry; discard them if they become and remain wet or stained, particularly in burn units.^{1310–1315} **Category IB**
- B. Clean and disinfect mattress covers using EPA-registered disinfectants, if available, that are compatible with the cover materials to prevent the development of tears, cracks, or holes in the cover.^{1310–1315} **Category IB**
- C. Maintain the integrity of mattress and pillow covers. **Category II**
 - 1. Replace mattress and pillow covers if they become torn or otherwise in need of repair. **Category II**
 - 2. Do not stick needles into the mattress through the cover. **Category II**
- D. Clean and disinfect moisture-resistant mattress covers between patients using an EPA-registered product, if available.^{1310–1315} **Category IB**
- E. If using a mattress cover completely made of fabric, change these covers and launder between patients.^{1310–1315} **Category IB**
- F. Launder pillow covers and washable pillows in the hot-water cycle between patients or when they become contaminated with body substances.¹³¹⁵ **Category IB**

VIII. Air-Fluidized Beds

- A. Follow manufacturers' instructions for bed maintenance and decontamination. **Category II**
- B. Change the polyester filter sheet at least weekly or as indicated by the manufacturer.^{1317, 1318, 1322, 1323} **Category II**
- C. Clean and disinfect the polyester filter sheet thoroughly, especially between patients, using an EPA-registered product, if available.^{1317, 1318, 1322, 1323} **Category IB**
- D. Consult the facility engineer to determine the proper location of air-fluidized beds in negative-pressure rooms.¹³²⁶ **Category II**

H. Recommendations—Animals in Health-Care Facilities

I. General Infection-Control Measures for Animal Encounters

- A. Minimize contact with animal saliva, dander, urine, and feces.^{1365–1367} **Category II**
- B. Practice hand hygiene after any animal contact.^{2, 1364} **Category IB**
 - 1. Wash hands with soap and water, especially if hands are visibly soiled.¹³⁶⁴
Category IB
 - 2. Use either soap and water or alcohol-based hand rubs when hands are not visibly soiled.¹³⁶⁴ **Category IB**

II. Animal-Assisted Activities, Animal-Assisted Therapy, and Resident Animal Programs

- A. Avoid selection of nonhuman primates and reptiles in animal-assisted activities, animal-assisted therapy, or resident animal programs.^{1360–1362} **Category IB**
- B. Enroll animals that are fully vaccinated for zoonotic diseases and that are healthy, clean, well-groomed, and negative for enteric parasites or otherwise have completed recent antihelminthic treatment under the regular care of a veterinarian.^{1349, 1360} **Category II**
- C. Enroll animals that are trained with the assistance or under the direction of individuals who are experienced in this field.¹³⁶⁰ **Category II**
- D. Ensure that animals are handled by persons trained in providing activities or therapies safely, and who know the animals' health status and behavior traits.^{1349, 1360} **Category II**
- E. Take prompt action when an incident of biting or scratching by an animal occurs during an animal-assisted activity or therapy.
 - 1. Remove the animal permanently from these programs.¹³⁶⁰ **Category II**
 - 2. Report the incident promptly to appropriate authorities (e.g., infection-control staff, animal program coordinator, or local animal control).¹³⁶⁰ **Category II**
 - 3. Promptly clean and treat scratches, bites, or other accidental breaks in the skin.
Category II
- F. Perform an ICRA and work actively with the animal handler prior to conducting an animal-assisted activity or therapy to determine if the session should be held in a public area of the facility or in individual patient rooms.^{1349, 1360} **Category II**
- G. Take precautions to mitigate allergic responses to animals. **Category II**
 - 1. Minimize shedding of animal dander by bathing animals <24 hours before a visit.¹³⁶⁰
Category II
 - 2. Groom animals to remove loose hair before a visit, or using a therapy animal cape.¹³⁵⁸
Category II
- H. Use routine cleaning protocols for housekeeping surfaces after therapy sessions.
Category II
- I. Restrict resident animals, including fish in fish tanks, from access to or placement in patient-care areas, food preparation areas, dining areas, laundry, central sterile supply areas, sterile and clean supply storage areas, medication preparation areas, operating rooms, isolation areas, and PE areas. **Category II**
- J. Establish a facility policy for regular cleaning of fish tanks, rodent cages, bird cages, and any other animal dwellings and assign this cleaning task to a nonpatient-care staff member; avoid splashing tank water or contaminating environmental surfaces with animal bedding.
Category II

III. Protective Measures for Immunocompromised Patients

- A. Advise patients to avoid contact with animal feces and body fluids such as saliva, urine, or solid litter box material.⁸ **Category II**

- B. Promptly clean and treat scratches, bites, or other wounds that break the skin.⁸ **Category II**
- C. Advise patients to avoid direct or indirect contact with reptiles.¹³⁴⁰ **Category IB**
- D. Conduct a case-by-case assessment to determine if animal-assisted activities or animal-assisted therapy programs are appropriate for immunocompromised patients.¹³⁴⁹ **Category II**
- E. **No recommendation is offered** regarding permitting pet visits to terminally ill immunosuppressed patients outside their PE units. **Unresolved issue**

IV. Service Animals

- A. Avoid providing access to nonhuman primates and reptiles as service animals.^{1340, 1362} **Category IB**
- B. Allow service animals access to the facility in accordance with the Americans with Disabilities Act of 1990, unless the presence of the animal creates a direct threat to other persons or a fundamental alteration in the nature of services.^{1366, 1376} **Category IC** (U.S. Department of Justice: 28 CFR § 36.302)
- C. When a decision must be made regarding a service animal's access to any particular area of the health-care facility, evaluate the service animal, the patient, and the health-care situation on a case-by-case basis to determine whether significant risk of harm exists and whether reasonable modifications in policies and procedures will mitigate this risk.¹³⁷⁶ **Category IC** (Justice: 28 CFR § 36.208 and App.B)
- D. If a patient must be separated from his or her service animal while in the health-care facility
 - 1) ascertain from the person what arrangements have been made for supervision or care of the animal during this period of separation; and 2) make appropriate arrangements to address the patient's needs in the absence of the service animal. **Category II**

V. Animals as Patients in Human Health-Care Facilities

- A. Develop health-care facility policies to address the treatment of animals in human health-care facilities.
 1. Use the multidisciplinary team approach to policy development, including public media relations in order to disclose and discuss these activities. **Category II**
 2. Exhaust all veterinary facility, equipment, and instrument options before undertaking the procedure. **Category II**
 3. Ensure that the care of the animal is supervised by a licensed veterinarian. **Category II**
- B. When animals are treated in human health-care facilities, avoid treating animals in operating rooms or other patient-care areas where invasive procedures are performed (e.g., cardiac catheterization laboratories, or invasive nuclear medicine areas). **Category II**
- C. Schedule the animal procedure for the last case of the day for the area, at a time when human patients are not scheduled to be in the vicinity. **Category II**
- D. Adhere strictly to standard precautions. **Category II**
- E. Clean and disinfect environmental surfaces thoroughly using an EPA-registered product in the room after the animal is removed. **Category II**
- F. Allow sufficient ACH to clean the air and help remove airborne dander, microorganisms, and allergens [Appendix B, Table B.1.]). **Category II**
- G. Clean and disinfect using EPA-registered products or sterilize equipment that has been in contact with animals, or use disposable equipment. **Category II**
- H. If reusable medical or surgical instruments are used in an animal procedure, restrict future use of these instruments to animals only. **Category II**

VI. Research Animals in Health-Care Facilities

- A. Use animals obtained from quality stock, or quarantine incoming animals to detect zoonotic diseases. **Category II**
- B. Treat sick animals or remove them from the facility. **Category II**
- C. Provide prophylactic vaccinations, as available, to animal handlers and contacts at high risk. **Category II**
- D. Ensure proper ventilation through appropriate facility design and location.¹³⁹⁵ **Category IC** (U.S. Department of Agriculture [USDA]: 7 USC 2131)
 - 1. Keep animal rooms at negative pressure relative to corridors.¹³⁹⁵ **Category IC** (USDA: 7 USC 2131)
 - 2. Prevent air in animal rooms from recirculating elsewhere in the health-care facility.¹³⁹⁵ **Category IC** (USDA: 7 USC 2131)
- E. Keep doors to animal research rooms closed. **Category II**
- F. Restrict access to animal facilities to essential personnel. **Category II**
- G. Establish employee occupational health programs specific to the animal research facility, and coordinate management of postexposure procedures specific for zoonoses with occupational health clinics in the health-care facility.^{1013, 1378} **Category IC** (U.S. Department of Health and Human Services [DHHS]: BMBL; OSHA: 29 CFR 1910.1030.132-139)
- H. Document standard operating procedures for the unit.¹⁰¹³ **Category IC** (DHHS: BMBL)
- I. Conduct routine employee training on worker safety issues relevant to the animal research facility (e.g., working safely with animals and animal handling).^{1013, 1393} **Category IC** (DHHS: BMBL; OSHA: 29 CFR 1910.1030.132-139)
- J. Use precautions to prevent the development of animal-induced asthma in animal workers.¹⁰¹³ **Category IC** (DHHS: BMBL)

I. Recommendations—Regulated Medical Waste

I. Categories of Regulated Medical Waste

- A. Designate the following as major categories of medical waste that require special handling and disposal precautions: 1) microbiology laboratory wastes [e.g., cultures and stocks of microorganisms]; 2) bulk blood, blood products, blood, and bloody body fluid specimens; 3) pathology and anatomy waste; and 4) sharps [e.g., needles and scalpels].² **Category II**
- B. Consult federal, state, and local regulations to determine if other waste items are considered regulated medical wastes.^{967, 1407, 1408} **Category IC** (States; Authorities having jurisdiction [AHJ]; OSHA: 29 CFR 1910.1030 §g.2.1; U.S. Department of Transportation [DOT]: 49 CFR 171-180; U.S. Postal Service: CO23.8)

II. Disposal Plan for Regulated Medical Wastes

- A. Develop a plan for the collection, handling, predisposal treatment, and terminal disposal of regulated medical wastes.^{967, 1409} **Category IC** (States; AHJ; OSHA: 29 CFR 1910.1030 §g.2.i)
- B. Designate a person or persons to be responsible for establishing, monitoring, reviewing, and administering the plan. **Category II**

III. Handling, Transporting, and Storing Regulated Medical Wastes

- A. Inform personnel involved in the handling and disposal of potentially infective waste of the possible health and safety hazards; ensure that they are trained in appropriate handling and disposal methods.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § g.2.i)
- B. Manage the handling and disposal of regulated medical wastes generated in isolation areas by using the same methods as for regulated medical wastes from other patient-care areas.² **Category II**
- C. Use proper sharps disposal strategies.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iii.A)

1. Use a sharps container capable of maintaining its impermeability after waste treatment to avoid subsequent physical injuries during final disposal.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iii.A)
 2. Place disposable syringes with needles, including sterile sharps that are being discarded, scalpel blades, and other sharp items into puncture-resistant containers located as close as practical to the point of use.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iii.A)
 3. Do not bend, recap, or break used syringe needles before discarding them into a container.^{6, 967, 1415} **Category IC** (OSHA: 29 CFR 1910.1030 § d.2.vii and § d.2.vii.A)
- D. Store regulated medical wastes awaiting treatment in a properly ventilated area that is inaccessible to vertebrate pests; use waste containers that prevent the development of noxious odors. **Category IC** (States; AHJ)
- E. If treatment options are not available at the site where the medical waste is generated, transport regulated medical wastes in closed, impervious containers to the on-site treatment location or to another facility for treatment as appropriate. **Category IC** (States; AHJ)

IV. Treatment and Disposal of Regulated Medical Wastes

- A. Treat regulated medical wastes by using a method (e.g., steam sterilization, incineration, interment, or an alternative treatment technology) approved by the appropriate authority having jurisdiction (AHJ) (e.g., states, Indian Health Service [IHS], Veterans Affairs [VA]) before disposal in a sanitary landfill. **Category IC** (States, AHJ)
- B. Follow precautions for treating microbiological wastes (e.g., amplified cultures and stocks of microorganisms).¹⁰¹³ **Category IC** (DHHS: BMBL)
1. Biosafety level 4 laboratories must inactivate microbiological wastes in the laboratory by using an approved inactivation method (e.g., autoclaving) before transport to and disposal in a sanitary landfill.¹⁰¹³ **Category IC** (DHHS: BMBL)
 2. Biosafety level 3 laboratories must inactivate microbiological wastes in the laboratory by using an approved inactivation method (e.g., autoclaving) or incinerate them at the facility before transport to and disposal in a sanitary landfill.¹⁰¹³ **Category IC** (DHHS: BMBL)
- C. Biosafety levels 1 and 2 laboratories should develop strategies to inactivate amplified microbial cultures and stocks onsite by using an approved inactivation method (e.g., autoclaving) instead of packaging and shipping untreated wastes to an offsite facility for treatment and disposal.^{1013, 1419–1421} **Category II**
- D. Laboratories that isolate select agents from clinical specimens must comply with federal regulations for the receipt, transfer, management, and appropriate disposal of these agents.¹⁴¹² **Category IC** (DHHS: 42 CFR 73 § 73.6)
- E. Sanitary sewers may be used for the safe disposal of blood, suctioned fluids, ground tissues, excretions, and secretions, provided that local sewage discharge requirements are met and that the state has declared this to be an acceptable method of disposal.¹⁴¹⁴ **Category II**

V. Special Precautions for Wastes Generated During Care of Patients with Rare Diseases

- A. When discarding items contaminated with blood and body fluids from VHF patients, contain these regulated medical wastes with minimal agitation during handling.^{6, 203} **Category II**
- B. Manage properly contained wastes from areas providing care to VHF patients in accordance with recommendations for other isolation areas (Regulated Medical Waste: III B).^{2, 6, 203} **Category II**
- C. Decontaminate bulk blood and body fluids from VHF patients using approved inactivation methods (e.g., autoclaving or chemical treatment) before disposal.^{6, 203} **Category IC, II** (States; AHJ)

- D. When discarding regulated medical waste generated during the routine (i.e., non-surgical) care of CJD patients, contain these wastes and decontaminate them using approved inactivation methods (e.g., autoclaving or incineration) appropriate for the medical waste category (e.g., blood, sharps, pathological waste).^{2, 6, 948, 1199} **Category IC, II** (States; AHJ)
- E. Incinerate medical wastes (e.g., central nervous system tissues or contaminated disposable materials) from brain autopsy or biopsy procedures of diagnosed or suspected CJD patients.^{1197, 1201} **Category IB**

Part III. References

Note: The bold item in parentheses indicated the citation number or the location of this reference listed in the MMWR version of this guideline.

1. Simmons BP. Guideline for hospital environmental control. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, 1981.
2. **(270)** Garner JS, Favero MS. Guideline for handwashing and hospital environmental control. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, 1985. Document No. 99-1117 (Also available at Infect Control 1986; 7: 231-43.)
3. **(27)** CDC. Guidelines for prevention of nosocomial pneumonia. MMWR 1997;46(No. RR-1):1-79.
4. **(34)** CDC. Guidelines for preventing the transmission of *Mycobacterium tuberculosis* in health-care facilities. MMWR 1994;43(No. RR-13):1-132.
5. **(318)** CDC. Recommendations for preventing the spread of vancomycin resistance. Recommendations of the Hospital Infection Control Practices Advisory Committee (HICPAC). MMWR 1995;44(No. RR-12):1-13.
6. **(36)** Garner JS, Hospital Infection Control Practices Advisory Committee. Guideline for isolation precautions in hospitals. Infect Control Hosp Epidemiol 1996;17:53-80.
7. **(114)** Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR, Hospital Infection Control Practices Advisory Committee. Guideline for prevention of surgical site infection, 1999. Infect Control Hosp Epidemiol 1999;20:247-80.
8. **(396)** CDC. USPHS/IDSA guidelines for the prevention of opportunistic infections in persons infected with human immunodeficiency virus. MMWR 1999;48(No. RR-10):1-66.
9. **(37, Appendix; 5)** CDC. CDC/IDSA/ASBMT guidelines for the prevention of opportunistic infections in hematopoietic stem cell transplant recipients. MMWR 2000;49(No. RR-10):1-128.
10. Garner JS. The CDC Hospital Infection Control Practice Advisory Committee. Am J Infect Control 1993;21:160-2.
11. Bennett JV, Brachman PS, eds. The inanimate environment. In: Rhame FS. Hospital Infections, 4th ed. Philadelphia, PA: Lippincott-Raven, 1998;299-324.
12. Weber DJ, Rutala WA. Environmental issues and nosocomial infections. In: Wenzel RP, ed. Prevention and control of nosocomial infections, 3rd ed. Baltimore, MD: Williams & Wilkins, 1997;491-514.
13. Greene VW. Microbiological contamination control in hospitals. Hospitals JAHA 1969;43:78-88.
14. American Hospital Association. Hospital statistics, 2000 ed. Historical trends in utilization, personnel, and finances for selected years from 1946 through 1998 [Table]. Chicago, IL: Health Forum LLC, 2000;2-3.
15. McKee B. Neither bust nor boom. Architecture 1998. Available at: www.britannica.com/bcom/magazine/article/0,5744,39579,00.html
16. Crowell CL. Better, not bigger: construction costs soar on wings of patient demand, construction and design survey finds. Mod Healthc 1999;29:23-6, 28-34, 36-8.
17. **(9)** Sarubbi FA Jr, Kopf BB, Wilson NO, McGinnis MR, Rutala WA. Increased recovery of *Aspergillus flavus* from respiratory specimens during hospital construction. Am Rev Respir Dis 1982;125:33-8.
18. **(2)** Arnow PM, Sadigh MC, Weil D, Chudy R. Endemic and epidemic aspergillosis associated with inhospital replication of *Aspergillus* organisms. J Infect Dis 1991;164:998-1002.

19. (38) Flynn PM, Williams BG, Hethrington SV, Williams BF, Giannini MA, Pearson TA. *Aspergillus terreus* during hospital renovation [letter]. *Infect Control Hosp Epidemiol* 1993;14:363–5.
20. (48) Weems JJ Jr, Davis BJ, Tablan OC, Kaufman L, Martone WJ. Construction activity: an independent risk factor for invasive aspergillosis and zygomycosis in patients with hematologic malignancy. *Infect Control* 1987;8:71–5.
21. (10) Streifel AJ, Stevens PP, Rhame FS. In-hospital source of airborne *Penicillium* species spores. *J Clin Microbiol* 1987;25:1–4.
22. Noskin GA, Stosor V, Cooper J, Peterson L. Recovery of vancomycin-resistant enterococci on fingertips and environmental surfaces. *Infect Control Hosp Epidemiol* 1995;16:577–81.
23. Manian FA, Meyer L, Jenne J. *Clostridium difficile* contamination of blood pressure cuffs: a call for a closer look at gloving practices in the era of universal precautions. *Infect Control Hosp Epidemiol* 1996;17:180–2.
24. McFarland LV, Mulligan NE, Kwok RYY, et al. Nosocomial acquisition of *Clostridium difficile* infection. *N Engl J Med* 1989;320:204–10.
25. Nath SK, Thomely JH, Kelly M, et al. A sustained outbreak of *Clostridium difficile* in a general hospital: persistence of a toxigenic clone in four units. *Infect Control Hosp Epidemiol* 1994;15:382–9.
26. (165) Johnson JT, Yu VL, Best MG, et al. Nosocomial legionellosis in surgical patients with head and neck cancer: implications for epidemiological reservoir and mode of transmission. *Lancet* 1985;2:298–300.
27. Blatt SP, Parkinson MD, Pace E, et al. Nosocomial Legionnaires' disease: aspiration as a primary mode of disease acquisition. *Am J Med* 1993;95:16–22.
28. Bert F, Maubec E, Bruneau B, Berry P, Lambert-Zechovsky N. Multi-resistant *Pseudomonas aeruginosa* associated with contaminated tap-water in a neurosurgery intensive care unit. *J Hosp Infect* 1998;39:53–62.
29. Muylldermans G, de Smet F, Perrard D, et al. Neonatal infections with *Pseudomonas aeruginosa* associated with a water-bath used to thaw fresh frozen plasma. *J Hosp Infect* 1998;39:309–14.
30. Buttery JP, Alabaster SJ, Heine FG, et al. Multi-resistant *Pseudomonas aeruginosa* outbreak in a pediatric oncology ward related to bath toys. *Pediatr Infect Dis J* 1998;17:509–13.
31. (224) Bolan G, Reingold AL, Carson LA, et al. Infections with *Mycobacterium chelonae* in patients receiving dialysis and using processed hemodialyzers. *J Infect Dis* 1985;152:1013–9.
32. (225) Lowry PW, Beck-Sague CM, Bland LA, et al. *Mycobacterium chelonae* infection among patients receiving high-flux dialysis in a hemodialysis clinic in California. *J Infect Dis* 1990;161:85–90.
33. Schaal KP. Medical and microbiological problems arising from airborne infection in hospitals. *J Hosp Infect* 1991;18 (Suppl A):451–9.
34. Osterholm MT, Hedberg CW, Moore KA. Epidemiology of infectious diseases. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and practice of infectious diseases*, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;156–67.
35. (3) Streifel AJ. Design and maintenance of hospital ventilation systems and prevention of airborne nosocomial infections. In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999:1211–21.
36. Bodey GP, Vartivarian S. Aspergillosis. *Eur J Clin Microbiol Infect Dis* 1989;8:413–37.
37. Latgé JP. *Aspergillus fumigatus* and aspergillosis. *Clin Microbiol Rev* 1999;12:310–50.
38. Derouin F. Special issue on aspergillosis. *Pathol Biol* 1990;42:625–736.
39. Dixon DD, Walsh TJ. Human pathogenesis. In: Bennett JW, Klich MA, eds. *Aspergillus*, biology and industrial application. Boston, MA: Butterworth-Heinemann, 1992;249–67.
40. Kurup VP, Kumar A. Immunodiagnosis of aspergillosis. *Clin Microbiol Rev* 1991;4:439–56.
41. Latgé JP, Paris S, Sarfati J, et al. Infectivity of *Aspergillus fumigatus*. In: Vanden Bossche H, Stevens DA, Odds FC, eds. *Host-Fungus Interplay*. Bethesda, MD: National Foundation for Infectious Diseases, 1997;99–110.
42. Schaffner A. Host defense in aspergillosis. In: Bennett E, Hay RJ, Peterson PK, eds. *New Strategies in Fungal Disease*. Edinburgh, United Kingdom: Churchill Livingstone, 1992;98–112.
43. Vanden Bossche H, Mackenzie DWR, Cauwenbergh G, eds. *Aspergillus* and aspergillosis. New York, NY: Plenum Press, 1988.
44. (82) Sherertz RJ, Belani A, Kramer BS, et al. Impact of air filtration on nosocomial *Aspergillus* infections: unique risk of bone marrow transplant recipients. *Am J Med* 1987;83:709–18.
45. Young RC, Bennett JE, Vogel CL, Carbone PP, DeVita VT. Aspergillosis: the spectrum of the disease in 98 patients. *Medicine* 1970;49:147–73.
46. Rhame FS. Lessons from the Roswell Park bone marrow transplant aspergillosis outbreak. *Infect Control* 1985;6:345–6.

47. Rotsein C, Cummings KM, Tidings J, et al. An outbreak of invasive aspergillosis among allogeneic bone marrow transplants: a case-control study. *Infect Control* 1985;6:347-55.
48. (83) Aisner J, Schimpff SC, Bennett JE, Young VM, Wiernik PH. Aspergillus infections in cancer patients: association with fireproofing materials in a new hospital. *JAMA* 1976;235:411-2.
49. (64) Arnow PM, Anderson RL, Mainous PD, Smith EJ. Pulmonary aspergillosis during hospital renovation. *Am Rev Respir Dis* 1978;118:49-53.
50. (61) Streifel AJ, Lauer JL, Vesley D, Juni B, Rhame FS. *Aspergillus fumigatus* and other thermotolerant fungi generated by hospital building demolition. *Appl Environ Microbiol* 1983;46:375-8.
51. Hopkins CC, Weber DJ, Rubin RH. Invasive aspergillosis infection: possible non-ward common source within the hospital environment. *J Hosp Infect* 1989;13:19-25.
52. Denning DW. Invasive aspergillosis. *Clin Infect Dis* 1998;26:781-805.
53. Manuel RJ, Kibbler CC. The epidemiology and prevention of invasive aspergillosis. *J Hosp Infect* 1998;39:95-109.
54. Kennedy HF, Simpson EM, Wilson N, Richardson MD, Michie JR. *Aspergillus flavus* endocarditis in a child with neuroblastoma. *J Infect* 1998;36:126-7.
55. (75) McCarty JM, Flam MS, Pullen G, Jones R, Kassel SH. Outbreak of primary cutaneous aspergillosis related to intravenous arm boards. *J Pediatr* 1986;108(Pt.1):721-4.
56. Goldberg B, Eversmann WW, Eitzen EM Jr. Invasive aspergillosis of the hand. *J Hand Surg* 1982;7:38-42.
57. Grossman ME, Fithian EC, Behrens C, Bissinger J, Fracaro M, Neu HC. Primary cutaneous aspergillosis in six leukemic children. *J Am Acad Dermatol* 1985;12:313-8.
58. Panke TW, McManus AT, Spebar MJ. Infection of a burn wound by *Aspergillus niger*: gross appearance simulating ecthyma gangrenosa. *Am J Clin Pathol* 1979;72:230-2.
59. Fraser DW, Ward JL, Ajello L, Plikaytis BD. Aspergillosis and other systemic mycoses: the growing problem. *JAMA* 1979;242:1631-5.
60. Iwen PC, Reed EC, Armitage JO, et al. Nosocomial invasive aspergillosis in lymphoma patients treated with bone marrow or peripheral stem cell transplants. *Infect Control Hosp Epidemiol* 1993;14:131-9.
61. Cordonnier C, Bernaudin JF, Bierling P, Huet Y, Vernant JP. Pulmonary complications occurring after allogeneic bone marrow transplantation: a study of 130 consecutively transplanted patients. *Cancer* 1986;58:1047-54.
62. (76) Klimowski LL, Rotstein C, Cummings KM. Incidence of nosocomial aspergillosis in patients with leukemia over a twenty-year period. *Infect Control Hosp Epidemiol* 1989;10:299-305.
63. (79) Walmsley S, Devi S, King S, Schneider R, Richardson S, Ford-Jones L. Invasive *Aspergillus* infections in a pediatric hospital: a ten-year review. *Pediatr Infect Dis* 1993;12:673-82.
64. (57) Pannuti CS, Gingrich RD, Pfaller MA, Wenzel RP. Nosocomial pneumonia in adult patients undergoing bone marrow transplantation: a 9-year study. *J Clin Oncol* 1991;9:77-84.
65. (58) Wingard JR, Beals SU, Santos GW, Mertz WG, Saral R. *Aspergillus* infections in bone marrow transplant recipients. *Bone Marrow Transplant* 1987;2:175-81.
66. Humphreys H, Johnson EM, Warnock DW, Willatts SM, Winter RJ, Speller DCE. An outbreak of aspergillosis in a general ITU. *J Hosp Infect* 1991;18:167-77.
67. Sessa A, Meroni M, Battini G, et al. Nosocomial outbreak of *Aspergillus fumigatus* infection among patients in a renal unit? *Nephrol Dial Transplant* 1996;11:1322-4.
68. (134) Anderson K, Morris G, Kennedy H, et al. Aspergillosis in immunocompromised pediatric patients: associations with building hygiene, design, and indoor air. *Thorax* 1996;51:256-61.
69. (39) Tabbara KF, al Jabarti A. Hospital construction-associated outbreak of ocular aspergillosis after cataract surgery. *Ophthalmology* 1998;105:522-26.
70. Ferre A, Domingo P, Alonso C, Franquet T, Gurgui M, Verger G. Invasive pulmonary aspergillosis: A study of 33 cases. *Med Clin (Barc)* 1998;110:421-5. (Spanish)
71. Ewig S, Paar WD, Pakos E et al. Nosocomial ventilator-associated pneumonias caused by *Aspergillus fumigatus* in non-immunosuppressed, non-neutropenic patients. *Pneumologie* 1998;52:85-90. (German)
72. Singer S, Singer D, Ruchel R, Mergeryan H, Schmidt U, Harms K. Outbreak of systemic aspergillosis in a neonatal intensive care unit. *Mycoses* 1998;41:223-7.
73. (88) Allo MD, Miller J, Townsend T, Tan C. Primary cutaneous aspergillosis associated with Hickman intravenous catheters. *N Engl J Med* 1987;317:1105-8.
74. Boon AP, Adams DH, Buckels J, McMaster P. Cerebral aspergillosis in liver transplantation. *J Clin Pathol* 1990;43:114-8.

75. Pla MP, Berenguer J, Arzuaga JA, Banares R, Polo JR, Bouza E. Surgical wound infection by *Aspergillus fumigatus* in liver transplant recipients. *Diagn Microbiol Infect Dis* 1992;15:703–6.
76. Kanj SS, Welty-Wolf K, Madden J, et al. Fungal infections in lung and heart-lung transplant recipients: report of 9 cases and review of the literature. *Medicine* 1996;75:142–56.
77. (77) Pfundstein J. *Aspergillus* infection among solid organ transplant recipients: a case study. *J Transpl Coord* 1997;7:187–9.
78. Brenier-Pinchart MP, Lebeau B, Devouassoux G, et al. *Aspergillus* and lung transplant recipients: a mycologic and molecular epidemiologic study. *J Heart Lung Transplant* 1998;17:972–9.
79. (59) Gerson SL, Talbot GH, Hurwitz S, Strom B, Lusk EJ, Cassileth PA. Prolonged granulocytopenia: the major risk factor for invasive pulmonary aspergillosis in patients with leukemia. *Ann Intern Med* 1984;100:345–51.
80. Weber SF, Peacock JE Jr, Do KA, Cruz JM, Powell BL, Capizzi RL. Interaction of granulocytopenia and construction activity as risk factors for nosocomial invasive filamentous fungal disease in patients with hematologic disorders. *Infect Control Hosp Epidemiol* 1990;11:235–42.
81. Rees JR, Pinner RW, Hajjeh RA, Brandt ME, Reingold AL. The epidemiological features of invasive mycotic infections in the San Francisco Bay area, 1992–1993: results of population-based laboratory active surveillance. *Clin Infect Dis* 1998;27:1138–47.
82. McNeil MM, Nash SL, Hajjeh RA, Conn LA, Plikaytis BD. Trends in mortality due to invasive mycoses in the United States [abstract]. In: Program & Abstracts of the International Conference on Emerging Infectious Diseases. Atlanta, GA, 1998. Abstract No. S7.3.
83. Wald A, Leisenring W, van Burik JA, Bowden RA. Epidemiology of *Aspergillus* infections in a large cohort of patients undergoing bone marrow transplantation. *J Infect Dis* 1997;175:1459–66.
84. Gurwith MJ, Stinson EB, Remington JS. *Aspergillus* infection complicating cardiac transplantation: Report of five cases. *Arch Intern Med* 1971;128:541–5.
85. Weiland D, Ferguson RM, Peterson PK, Snover DC, Simmons RL, Najarian JS. Aspergillosis in 25 renal transplant patients. *Ann Surg* 1983;198:622–9.
86. Hofflin JM, Potasman I, Baldwin JC, Oyster PE, Stinson EB, Remington JS. Infectious complications in heart transplant recipients receiving cyclosporine and corticosteroids. *Ann Intern Med* 1987;106:209–16.
87. Schulman LL, Smith CR, Drusin R, Rose EA, Enson Y, Reemtsma K. Respiratory complications of cardiac transplantation. *Am J Med Sci* 1988;296:1–10.
88. Gustafson TL, Schaffner W, Lavelly GB, Stratton CW, Johnson HK, Hutcheson RH. Invasive aspergillosis in renal transplant recipients: correlation with corticosteroid therapy. *J Infect Dis* 1983;148:230–8.
89. Denning DW, Stevens DA. Antifungal and surgical treatment of invasive aspergillosis: review of 2121 published cases. *Rev Infect Dis* 1990;12:1147–201.
90. Weinberger M, Elattaar I, Marshall D, et al. Patterns of infection in patients with aplastic anemia and the emergence of *Aspergillus* as a major cause of death. *Medicine* 1992;71:24–43.
91. Noble WC, Clayton YM. Fungi in the air of hospital wards. *J Gen Microbiol* 1963;32:397–402.
92. Solomon WR, Burge HP, Boise JR. Airborne *Aspergillus fumigatus* levels outside and within a large clinical center. *J Allergy Clin Immunol* 1978;62:56–60.
93. Streifel AJ, Rhame FS. Hospital air filamentous fungal spore and particle counts in a specially designed hospital. In: *Indoor Air '93: Proceedings of the Sixth International Conference on Indoor Air and Climate*, Vol. 4. Helsinki, Finland:161–5.
94. (40) Rhame FS, Streifel AJ, Kersey JH Jr, McGlave PB. Extrinsic risk factors for pneumonia in the patient at high risk for infection. *Am J Med* 1984;76(5A):42–52.
95. (78) Rhame FS, Streifel A, Stevens P, et al. Endemic *Aspergillus* airborne spore levels are a major risk factor for aspergillosis in bone marrow transplant patients [abstract]. In: Abstracts of the 25th Interscience Conference on Antimicrobial Agents and Chemotherapy 1985. Abstract No. 147.
96. (60) Lentino JR, Rosenkranz MA, Michaels JA, Kurup VP, Rose HD, Rytel MW. Nosocomial aspergillosis: a retrospective review of airborne disease secondary to road construction and contaminated air conditioners. *Am J Epidemiol* 1982;116:430–7.
97. (49) Krasinski K, Holzman RS, Hanna B, Greco MA, Graff M, Bhogal M. Nosocomial fungal infection during hospital renovation. *Infect Control* 1985;6:278–82.
98. (29) Gage AA, Dean DC, Schimert G, Minsley N. *Aspergillus* infection after cardiac surgery. *Arch Surg* 1970;101:384–87.

99. **(95)** Siegler L, Kennedy MJ. *Aspergillus, Fusarium*, and other opportunistic moniliaceous fungi. In: Murray PR, Baron EJ, Pfaller MA, Tenoer FC, Tenover RH, eds. Manual of clinical microbiology, 7th ed. Washington, DC: American Society for Microbiology Press, 1999;1212–41.
100. **(70)** Overberger PA, Wadowsky RM, Schaper MM. Evaluation of airborne particulates and fungi during hospital renovation. *Am Ind Hyg Assoc J* 1995;56:706–12.
101. **(96)** Breton P, Germaud P, Morin O, Audoin AF, Milpied N, Harousseau JL. Unusual pulmonary mycoses in patients with hematologic disease. *Rev Pneumol Clin* 1998;54:253–7. (French)
102. **(97)** Guarro J, Nucci M, Akiti T, Gené J, Barreiro MDGC, Gonçalves RT. Fungemia due to *Fusarium sacchari* in an immunosuppressed patient. *J Clin Microbiol* 2000;38:419–21.
103. **(98)** Burton JR, Zachery JB, Bessin R, et al. Aspergillosis in four renal transplant patients: diagnosis and effective treatment with amphotericin B. *Ann Intern Med* 1972;77:383–8.
104. **(80)** Kyriakides GK, Zinneman HHA, Hall WH, et al. Immunologic monitoring and aspergillosis in renal transplant patients. *Am J Surg* 1976;131:246–52.
105. Simmons RB, Price DL, Noble JA, Crow SA, Ahearn DG. Fungal colonization of air filters from hospitals. *Am Ind Hyg Assoc J* 1997;58:900–4.
106. **(4)** Pittet D, Huguenin T, Dharan S, et al. Unusual case of lethal pulmonary aspergillosis in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 1996;154(2 Pt 1):541–4.
107. **(104)** Mahoney DH Jr, Steuber CP, Starling KA, Barrett FF, Goldberg J, Fernbach DJ. An outbreak of aspergillosis in children with acute leukemia. *J Pediatr* 1979;95:70–2.
108. Ruutu P, Valtonen V, Tiitonen L, et al. An outbreak of invasive aspergillosis in a hematologic unit. *Scand J Infect Dis* 1987;19:347–51.
109. **(51)** Walsh TJ, Dixon DM. Nosocomial aspergillosis: environmental microbiology, hospital epidemiology, diagnosis, and treatment. *Eur J Epidemiol* 1989;5:131–42.
110. Buffington J, Reporter R, Lasker BA, et al. Investigation of an epidemic of invasive aspergillosis: utility of molecular typing with the use of random amplified polymorphic DNA probes. *Pediatr Infect Dis J* 1994;13:386–93.
111. **(44)** Gerson SL, Parker P, Jacobs MR, Creger R, Lazarus HM. Aspergillosis due to carpet contamination [letter]. *Infect Control Hosp Epidemiol* 1994;15:221–3.
112. **(84)** Fox BC, Chamberlin L, Kulich P, Rae EJ, Webster LR. Heavy contamination of operating room air by *Penicillium* species: identification of the source and attempts at decontamination. *Am J Infect Control* 1990;18:300–6.
113. Chazalet V, Debeauvais J-P, Sarfati J, et al. Molecular typing of environmental and patient isolates of *Aspergillus fumigatus* from various hospital settings. *J Clin Microbiol* 1998;36:1494–500.
114. Loudon KW, Coke AP, Burnie JP, Shaw AJ, Oppenheim BA, Morris CQ. Kitchens as a source of *Aspergillus niger* infection. *J Hosp Infect* 1996;32:191–8.
115. **(81)** Abzug MJ, Gardner S, Glode MP, Cymanski M, Roe MH, Odom LF. Heliport-associated nosocomial mucormycoses [letter]. *Infect Control Hosp Epidemiol* 1992;13:325–6.
116. Alvarez M, Lopez Ponga B, Rayon C, et al. Nosocomial outbreak caused by *Scedosporium prolificans (inflatum)*: four fatal cases in leukemic patients. *J Clin Microbiol* 1995;33:3290–5.
117. **(89)** Schleupner CJ, Hamilton JR. A pseudoepidemic of pulmonary fungal infections related to fiberoptic bronchoscopy. *Infect Control* 1980;1:38–42.
118. Jackson L, Klotz SA, Normand RE. A pseudoepidemic of *Sporothrix cyanescens* pneumonia occurring during renovation of a bronchoscopy suite. *J Med Vet Mycol* 1990;28:455–9.
119. **(30)** Vargo JA, Ginsberg MM, Mizrahi M. Human infestation by the pigeon mite: a case report. *Am J Infect Control* 1983;11:24–5.
120. **(1)** American Institute of Architects. Guidelines for design and construction of hospital and health care facilities, 2001. Washington, DC: American Institute of Architects Press, 2001.
121. Diamond RD. *Cryptococcus neoformans*. In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of Infectious Diseases, 5th Ed. Philadelphia, PA: Churchill Livingstone, 2000;2707–18.
122. Deepe GS Jr. *Histoplasma capsulatum*. In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of Infectious Diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2718–33.
123. Brodsky AL, Gregg MB, Loewenstein MS, et al. Outbreak of histoplasmosis associated with the 1970 earth day activities. *Am J Med* 1973;54:333–42.
124. Ward JI, Weeks M, Allen D, et al. Acute histoplasmosis: clinical, epidemiologic, and serologic findings of an outbreak associated with exposure to a fallen tree. *Am J Med* 1979;66:587–95.

125. Galgiani JN. Coccidioidomycoses. In: Remington JS, Swartz MN, eds. Current clinical topics in infectious disease. Malden, MA: Blackwell Science, 1997;188–204.
126. **(111)** Gerberding JL. Nosocomial transmission of opportunistic infections. *Infect Control Hosp Epidemiol* 1998;19:574–7.
127. Hughes WT. Natural mode of acquisition for de novo infection with *Pneumocystis carinii*. *J Infect Dis* 1982;145:842–8.
128. Olsson M, Sukura A, Lindberg LA, et al. Detections of *Pneumocystis carinii* DNA by filtration of air. *Scand J Infect Dis* 1996;28:279–82.
129. Bartlett MS, Vermund SH, Jacobs R, et al. Detection of *Pneumocystis carinii* DNA in air samples: likely environmental risk to susceptible persons. *J Clin Microbiol* 1997;35:2511–3.
130. Lundgren B, Elvin K, Rothman LP, Ljungstrom I, Lidman C, Lundgren JD. Transmission of *Pneumocystis carinii* from patients to hospital staff. *Thorax* 1997;52:422–4.
131. **(112)** Vargas SL, Ponce CA, Gigliotti F, et al. Transmission of *Pneumocystis carinii* DNA from a patient with *P. carinii* pneumonia to immunocompetent contact health care workers. *J Clin Microbiol* 2000;38:1536–8.
132. **(113)** Walzer PD. *Pneumocystis carinii*. In: Mandell GL, Bennett JE, Dolin R. eds. Principles and practice of infectious diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2781–95.
133. CDC. Screening for tuberculosis and tuberculosis infection in high-risk populations: recommendations of the Advisory Committee for Elimination of Tuberculosis. *MMWR* 1995;44(No. RR-11):18–34.
134. CDC. Targeted tuberculin testing and treatment of latent tuberculosis infection. *MMWR* 2000;49(No. RR-6):1–51.
135. D'Agata EMC, Wise S, Stewart A, Lefkowitz LB Jr. Nosocomial transmission of *Mycobacterium tuberculosis* from an extrapulmonary site. *Infect Control Hosp Epidemiol* 2001;22:10–2.
136. CDC. Summary of notifiable diseases, United States 2001. *MMWR* 2001;50(53):1–108.
137. Haas DW. *Mycobacterium tuberculosis*. In: Mandell GL, Bennett JE, Dolin R. eds. Principles and practice of infectious diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2576–607.
138. American Public Health Association. Tuberculosis. In: Chin J, ed. Control of communicable diseases manual, 17th ed. Washington, DC: American Public Health Association Press, 2000:521–30.
139. American Thoracic Society/CDC. Treatment of tuberculosis. *Am J Respir Crit Care Med* 2003;167:603–62.
140. Matlow AG, Jarrison A, Monteath A, Roach P, Balfe JW. Nosocomial transmission of tuberculosis (TB) associated with care of an infant with peritoneal TB. *Infect Control Hosp Epidemiol* 2000;21:222–3.
141. Jensen PA. Airborne *Mycobacterium* spp. In: Hurst CJ, Knudsen GR, McInerney MJ, Stetzenbach LD, Walter MV, eds. Manual of environmental microbiology. Washington, DC: American Society for Microbiology Press, 1997;676–81.
142. **(41)** Wells WF. Aerodynamics of droplet nuclei. In: Airborne contagion and air hygiene. Cambridge, MA: Harvard University Press, 1955;13–9.
143. White A. Relation between quantitative nasal cultures and dissemination of staphylococci. *J Lab Clin Med* 1961;58:273–7.
144. Huijsmans-Evers AG. Results of routine tests for the detection of dispersers of *Staphylococcus aureus*. *Arch Chir Neerl* 1978;30:141–50.
145. Boyce JM, Opal SM, Potter-Bynoe G, Medeiros AA. Spread of methicillin-resistant *Staphylococcus aureus* in a hospital after exposure to a healthcare worker with chronic sinusitis. *Clin Infect Dis* 1993;17:496–504.
146. Hambraeus A, Sanderson HF. The control of ventilation of airborne bacterial transfer between hospital patients, and its assessment by means of a particle tracer. 3. Studies with an airborne-particle tracer in an isolation ward for burned patients. *J Hyg (Lond)* 1972;70:299–312.
147. Nakashima AK, Allen JR, Martone WJ, et al. Epidemic bullous impetigo in a nursery due to a nasal carrier of *Staphylococcus aureus*: role of epidemiology and control measures. *Infect Control* 1984;5:326–31.
148. Bethune DW, Blowers R, Parker M, Pask EA. Dispersal of *Staphylococcus aureus* by patients and surgical staff. *Lancet* 1965;1:480–3.
149. Sherertz RJ, Reagan DR, Hampton KD, et al. A cloud adult: the *Staphylococcus aureus* — virus interaction revisited. *Ann Intern Med* 1996;124:539–47.
150. Gryska PF, O'Dea AE. Postoperative streptococcal wound infection: the anatomy of an epidemic. *JAMA* 1970;213:1189–91.
151. Stamm WE, Feeley JC, Facklam RR. Wound infection due to group A *Streptococcus* traced to a vaginal carrier. *J Infect Dis* 1978;138:287–92.

152. Berkelman RL, Martin D, Graham DR. Streptococcal wound infection caused by a vaginal carrier. *JAMA* 1982;247:2680–2.
153. McIntyre DM. An epidemic of *Streptococcus pyrogenes* puerpural and postoperative sepsis with an unusual site — the anus. *Am J Obstet Gynecol* 1968;101:308–14.
154. Gaynes RP, Horan TC. Surveillance of nosocomial infections. In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;1285–318.
155. Wenzel RP, Veazey JM Jr, Townsend TR. Role of the inanimate environment in hospital-acquired infections. In: Cundy KR, Ball W, eds. *Infection control in healthcare facilities: microbiological surveillance*. Baltimore, MD: University Park Press, 1977;71–98.
156. Mortimer EA Jr, Wolinsky E, Gonzaga AJ, Rammelkamp CH Jr. Role of airborne transmission in staphylococcal infections. *Br Med J* 1966;5483:319–22.
157. Youngs ER, Roberts C, Kramer JM, Gilbert RJ. Dissemination of *Bacillus cereus* in a maternity unit. *J Infect* 1985;10:228–32.
158. Richardson AJ, Rothburn MM, Roberts C. Pseudo-outbreak of *Bacillus* species: related to fiberoptic bronchoscopy. *J Hosp Infect* 1986;7:208–10.
159. Bryce EA, Smith JA, Tweeddale M, Andruschak BJ, Maxwell MR. Dissemination of *Bacillus cereus* in an intensive care unit. *Infect Control Hosp Epidemiol* 1993;14:459–62.
160. Lie PY-F, Ke S-C, Chen S-L. Use of pulsed-field gel electrophoresis to investigate a pseudo-outbreak of *Bacillus cereus* in a pediatric unit. *J Clin Microbiol* 1997;35:1533–5.
161. McDonald LC, Walker M, Carson L, et al. Outbreak of *Acinetobacter* spp. bloodstream infections in a nursery associated with contaminated aerosols and air conditioners. *Pediatr Infect Dis J* 1998;17:716–22.
162. Leclair JM, Zaia JA, Levin MJ, Congdon RG, Goldmann DA. Airborne transmission of chickenpox in a hospital. *N Engl J Med* 1980;302:450–3.
163. Gustafson TL, Lavelly GB, Brawner ERJ, Hutcheson RHJ, Wright PF, Schaffner W. An outbreak of airborne nosocomial varicella. *Pediatrics* 1982;70:550–6.
164. Josephson A, Gombert ME. Airborne transmission of nosocomial varicella from localized zoster. *J Infect Dis* 1988;158:238–41.
165. Sawyer MJ, Chamberlin CJ, Wu YN, Aintablian N, Wallace MR. Detection of varicella-zoster virus DNA in air samples from hospital rooms. *J Infect Dis* 1994;169:91–4.
166. Menkhaus NA, Lamphear B, Linnemann CC. Airborne transmission of varicella-zoster virus in hospitals. *Lancet* 1990;226:1315.
167. CDC. Prevention of varicella: recommendations of the advisory committee on immunization practices (ACIP). *MMWR* 1996;45(No. RR-11):1–36.
168. Davis RM, Orenstein WA, Frank JAJ, et al. Transmission of measles in medical settings: 1980 through 1984. *JAMA* 1986;255:1295–8.
169. Watkins NM, Smith RPJ, St Germain DL, Mackay DN. Measles infection in a hospital setting. *Am J Infect Control* 1987;15:201–6.
170. Revera ME, Mason WH, Ross LA, Wright HT Jr. Nosocomial measles infection in a pediatric hospital during a community-wide epidemic. *J Pediatr* 1991;119:183–6.
171. Atkinson WL, Markowitz LE, Adams NC, Seastrom GR. Transmission of measles in medical settings — United States, 1985–1989. *Am J Med* 1991;91(suppl):320S–4S.
172. Patriarca PA, Weber JA, Parker RA, et al. Efficacy of influenza vaccine in nursing homes: reduction in illness and complications during influenza A (H3N2) epidemics. *JAMA* 1985;253:1136–9.
173. Arden NH, Patriarca PA, Fasano MB, et al. The roles of vaccination and amantadine prophylaxis in controlling an outbreak of influenza A (H3N2) in a nursing home. *Arch Intern Med* 1988;148:865–8.
174. CDC. Influenza A outbreaks — Louisiana, August 1993. *MMWR* 1993;42:132–4.
175. Drinka PJ, Gravenstein S, Krause P, Schilling M, Miller BA, Shult P. Outbreaks of influenza A and B in a highly immunized nursing home population. *J Family Practice* 1997;45:509–14.
176. Schilling M, Povinelli L, Krause P, et al. Efficacy of zanamivir for chemoprophylaxis of nursing home influenza outbreaks. *Vaccine* 1998;16:1771–4.
177. Hall CB. Nosocomial viral infections: perennial weeds on pediatric wards. *Am J Med* 1981;70:670–6.
178. Whimbey E, Elting LS, Couch RB, et al. Influenza A virus infections among hospitalized adult bone marrow transplant recipients. *Bone Marrow Transpl* 1994;13:437–40.
179. Evans ME, Hall KL, Berry SE. Influenza control in acute care hospitals. *Am J Infect Control* 1997;25:357–62.

180. Munoz FM, Campbell JR, Atmar RL, et al. Influenza A virus outbreak in a neonatal intensive care unit. *Pediatr Infect Dis J* 1999;18:811–5.
181. Alford RH, Kasel JA, Gerone PJ, Knight V. Human influenza resulting from aerosol inhalation. *Proc Soc Exp Biol Med* 1966;122:800–4.
182. Moser MR, Bender TR, Margolis HS, Noble GR, Kendal AP, Ritter DG. An outbreak of influenza aboard a commercial airliner. *Am J Epidemiol* 1979;110:1–6.
183. Chanock RW, Kim HW, Vargosko AJ, et al. Respiratory syncytial virus 1: virus recovery and other observations during 1960 — outbreak of bronchiolitis, pneumonia, and other minor respiratory illness in children. *JAMA* 1961;176:647–53.
184. Gardner DS, Court SDM, Brocklebank JT, et al. Virus cross-infection in paediatric wards. *Br Med J* 1973;2:571–75.
185. Sawyer LA, Murphy JJ, Kaplan JE, et al. 25–30 nm virus particle associated with a hospital outbreak of acute gastroenteritis with evidence for airborne transmission. *Am J Epidemiol* 1988;127:1261–71.
186. Baxby D. Poxviruses. In: Belshe RB, ed. *Textbook of human virology*, 2nd ed. St. Louis, MO: Mosby Year Book, 1991;930–46.
187. Neff JM. Variola (smallpox) and monkeypox viruses. In: Mandell GL, Bennett JE, Dolin R. eds. *Principles and practice of infectious diseases*, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;1555–6.
188. Wehrle PF, Posch J, Richter KH, Henderson DA. An airborne outbreak of smallpox in a German hospital and its significance with respect to other recent outbreaks in Europe. *Bull WHO* 1970;43:669–79.
189. Hawkes N. Science in Europe: smallpox death in Britain challenges presumption of laboratory safety. *Science* 1979;203:855–6.
190. Eickhoff TC. Airborne nosocomial infection: a contemporary perspective. *Infect Control Hosp Epidemiol* 1994;15:663–72.
191. Nuzum EO, Rossi CA, Stephenson EH, LeDuc JW. Aerosol transmission of Hantaan and related viruses to laboratory rats. *Am J Trop Med Hyg* 1988;38:636–40.
192. CDC. Hantavirus infection — southwestern United States: Interim recommendations for risk reduction. *CDC. MMWR* 1993;42(No. RR-11):1–13.
193. Vitek CR, Breiman RF, Ksiazek TG, et al. Evidence against person-to-person transmission of hantavirus to health care workers. *Clin Infect Dis* 1996;22:824–6.
194. Wells RM, Young J, Williams RJ, et al. Hantavirus transmission in the United States. *Emerg Infect Dis* 1997;3:361–5.
195. Chaparro J, Vega J, Terry W, et al. Assessment of person-to-person transmission of hantavirus pulmonary syndrome in a Chilean hospital setting. *J Hosp Infect* 1998;40:281–5.
196. Nolte KB, Foucar K, Richmond JY. Hantaviral biosafety issues in the autopsy room and laboratory: Concerns and recommendations. *Hum Pathol* 1996;27:1253–4.
197. Stephenson EH, Larson EW, Dominik JW. Effect of environmental factors on aerosol-induced Lassa virus infection. *J Med Virol* 1984;14:295–303.
198. Monath TP. Lassa fever: review of epidemiology and epizootiology. *Bull World Health Organ* 1975;52:577–92.
199. Monath TP, Casals J. Diagnosis of Lassa fever and the isolation and management of patients. *Bull WHO* 1975;52:707–15.
200. Zweighaft RM, Fraser DW, Hattwick MA, et al. Lassa fever: response to an imported case. *N Engl J Med* 1977;297:803–7.
201. Cooper CB, Gransden WR, Webster M, et al. A case of Lassa fever: experience at St Thomas' hospital. *Br Med J (Clin Res Ed)* 1982;285:1003–5.
202. **(108)** Monath TP. Yellow fever: Victor, victoria? Conqueror, conquest? Epidemics and research in the last forty years and prospects for the future. *Am J Trop Med Hyg* 1991;45:1–43.
203. **(109)** CDC. Update: management of patients with suspected viral hemorrhagic fever — United States. *MMWR* 1995;44:475–9.
204. **(110)** Weber DJ, Rutala WA. Risks and prevention of nosocomial transmission rare zoonotic diseases. *Clin Infect Dis* 2001;32:446–56.
205. Decker MD, Schaffner W. Nosocomial diseases of healthcare workers spread by the airborne or contact routes (other than tuberculosis). In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;1101–26.

206. **(46)** Fridkin SK, Kremer FB, Bland LA, Padhye A, McNeil MM, Jarvis WR. *Acremonium kiliense* endophthalmitis that occurred after cataract extraction in an ambulatory surgical center and was traced to an environmental reservoir. *Clin Infect Dis* 1996;22:222–7.
207. Loeb M, Wilcox L, Thornley D, et al. *Bacillus* species pseudobacteremia following hospital construction. *Can J Infect Control* 1995;10:37–40.
208. Olle-Goig JE, Canela-Soler J. An outbreak of *Brucella melitensis* infection by airborne transmission among laboratory workers. *Am J Public Health* 1987;77:335–8.
209. Kiel FW, Khan MY. Brucellosis among hospital employees in Saudi Arabia. *Infect Control Hosp Epidemiol* 1993;14:268–72.
210. Staszkiwicz J, Lewis CM, Colville J, Zervos M, Band J. Outbreak of *Brucella melitensis* among microbiology laboratory workers in a community hospital. *J Clin Microbiol* 1991;20:287–90.
211. Fiori PL, Mastrandrea S, Rappelli P, Cappuccinelli P. *Brucella abortus* infection acquired in microbiology laboratories. *J Clin Microbiol* 2000;38:2005–6.
212. Spinelli JS, Ascher MS, Brooks DL, et al. Q fever crisis in San Francisco: controlling a sheep zoonosis in a lab animal facility. *Lab Anim* 1981;10:29–38.
213. **(Table 1)** American Conference of Governmental Industrial Hygienists (ACGIH). HVAC components, functions and malfunctions (topic 8-4). In: *Industrial ventilation: a Manual of recommended practice*, 24th ed. Cincinnati, OH : American Conference of Governmental Industrial Hygienists, Inc., 2001.
214. **(35)** American Society of Heating, Refrigerating and Air-conditioning Engineers, Inc. Ventilation for Indoor Air Quality. ASHRAE Standard 62-1999. Atlanta, GA: ASHRAE 1999;1–27.
215. **(133)** Burroughs HEB. Sick building syndrome: fact, fiction, or facility? In: Hansen W, ed. *A guide to managing indoor air quality in health care organizations*. Oakbrook Terrace, IL: Joint Commission on Accreditation of Health Care Organizations, 1997;3–13.
216. American Society of Heating, Refrigerating and Air-conditioning Engineers, Inc. Gravimetric and dust spot procedures for testing air cleaning devices used in general ventilation for removing particulate matter. ANSI/ASHRAE Standard 52-1-1999. Atlanta, GA: ASHRAE 1999;1–25.
217. Robinson TJ, Ouellet AE. Filters and filtration. *ASHRAE J* 1999;65–70.
218. Dryden GE, Dryden SR, Brown DG, Schatzle KC, Godzeski C. Performance of bacteria filters. *Respir Care* 1980;25:1127–35.
219. **(33)** Rutala WA, Jones SM, Worthington JM, Reist PC, Weber DJ. Efficacy of portable filtration units in reducing aerosolized particles in the size range of *Mycobacterium tuberculosis*. *Infect Control Hosp Epidemiol* 1995;16:391–8.
220. **(5)** U.S. Environmental Protection Agency, Office of Air and Radiation and U.S. Department of Health & Human Services, National Institute of Occupational Safety and Health. *Building air quality: a guide for building owners and facilities managers*. Washington, DC: USEPA, 1991. EPA/400/1-91/033, or NIOSH 91-114. Available at: www.cdc.gov/niosh/baqtoc.html
221. **(28)** Ko G, Burge HA, Muileberg M, Rudnick S, First M. Survival of mycobacteria on HEPA filter material. *J Am Biol Safety Assoc* 1998;3:65–78.
222. **(6)** Rao CY, Burge HA, Chang JCS. Review of quantitative standards and guidelines for fungi in indoor air. *J Air & Waste Manage Assoc* 1996;46:899–906.
223. Riley RL, Wells WF, Mills CC, Nyka W, McLean RL. Air hygiene in tuberculosis: quantitative studies of infectivity and control in a pilot ward. *Am Rev Tuberc* 1957;75:420–31.
224. Riley RL, Nardell EA. Cleaning the air: the theory and application of UV air disinfection. *Am Rev Respir Dis* 1989;139:1286–94.
225. Riley RL. Ultraviolet air disinfection for control of respiratory contagion. In: Kundsinn RB, ed. *Architectural design and indoor microbial pollution*. New York, NY: Oxford University Press, 1988;174–97.
226. Willmon TL, Hollaender A, Langmuir AD. Studies of the control of acute respiratory diseases among naval recruits. I. A review of a four-year experience with ultraviolet irradiation and dust suppressive measures, 1943 to 1947. *Am J Hyg* 1948;48:227–32.
227. Wells WF, Wells MW, Wilder TS. The environmental control of epidemic contagion. I. An epidemiologic study of radiant disinfection of air in day schools. *Am J Hyg* 1942;35:97–121.
228. Perkins JE, Bahlke AM, Silverman HF. Effect of ultra-violet irradiation of classrooms on spread of measles in large rural central schools. *Am J Public Health Nations Health* 1947;37:529–37.
229. Nagy R. Application and measurement of ultraviolet radiation. *Am Ind Hyg Assoc J* 1964;25:274–81.
230. Illuminating Engineering Society. *IES Lighting handbook*, 4th ed. New York, NY: Illuminating Engineering Society, 1966;25–7.

231. Riley RL. Indoor spread of respiratory infection by recirculation of air. *Bull Physiopathol Respir* 1979;15:699–705.
232. Menzies D, Pasztor J, Rand T, Bourbeau J. Germicidal ultraviolet irradiation in air conditioning systems: effect on office worker health and wellbeing — a pilot study. *Occup Environ Med* 1999;56:397–402.
233. Riley RL, Permutt S. Room air disinfection by ultraviolet irradiation of upper air: air mixing and germicidal effectiveness. *Arch Environ Health* 1971;22:208–19.
234. Nicas M, Miller SL. A multi-zone model evaluation of the efficacy of upper-room air ultraviolet germicidal irradiation. *Appl Occup Environ Hyg* 1999;14:317–28.
235. Kethley TW, Branch K. Ultraviolet lamps for room air disinfection: effect of sampling location and particle size of bacterial aerosol. *Arch Environ Health* 1972;25:205–14.
236. Riley RL, Knight M, Middlebrook G. Ultraviolet susceptibility of BCG and virulent tubercle bacilli. *Am Rev Respir Dis* 1976;113:413–8.
237. Collins FM. Relative susceptibility of acid-fast and non-acid-fast bacteria to ultraviolet light. *Appl Microbiol* 1971;21:411–3.
238. Riley RL, Permutt S, Kaufman JE. Convection, air mixing, and ultraviolet air disinfection in rooms. *Arch Environ Health* 1971;22:200–7.
239. Nardell EA. Fans, filters, or rays? Pros and cons of the current environmental tuberculosis control technologies. *Infect Control Hosp Epidemiol* 1993;14:681–5.
240. ECRI. Health devices evaluation of mobile high efficiency filter air cleaners (MHEFACs). *ECRI* 1997;26:367–88.
241. **(103)** American Society of Heating, Refrigerating, and Air-Conditioning Engineers. 1999 ASHRAE Handbook: heating, ventilating, and air-conditioning applications. Chapter 7: Health care facilities. Atlanta, GA: ASHRAE, 1999;7.1–7.13.
242. Elovitz KM. Understanding what humidity does and why. *ASHRAE J* 1999;April:84–90.
243. Orme I. Patient impact. In: Hansen W, ed. *A guide to managing indoor air quality in health care organizations*. Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997:43–52.
244. Gundermann KO. Spread of microorganisms by air-conditioning systems — especially in hospitals. *Ann NY Acad Sci* 1980;209–17.
245. Arundel AV, Sterling EM, Biggin JH, Sterling TD. Indirect health effects of relative humidity in indoor environments. *Environ Health Perspect* 1986;65:351–61.
246. U.S. Environmental Protection Agency. *Ventilation and air quality in offices*. Washington, DC: EPA Document #402-F-94-003, Revision: July 1990.
247. Hermans RD, Streifel AJ. Ventilation design. In: Bierbaum PJ, Lippman M, eds. *Workshop on engineering controls for preventing airborne infections in workers in health care and related facilities*. Cincinnati, OH: NIOSH and CDC, 1993;107–46.
248. Memarzadeh F, Jiang J. A methodology for minimizing risk from airborne organisms in hospital isolation rooms. *ASHRAE Trans* 2000;106:731–47.
249. **(11)** Hansen W. The need for an integrated indoor air quality program. In: Hansen W, ed. *A guide to managing indoor air quality in health care organizations*. Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997;xiii – xviii.
250. **(12)** Bartley J. Ventilation. In: Pfeiffer J, ed. *APIC Text of infection control and epidemiology*. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc (APIC), 2000;77.1–77.11.
251. Levine AS, Siegel SE, Schreiber AD, et al. Protected environments and prophylactic antibiotics. *N Engl J Med* 1973;288:477–483.
252. **(90)** Denning DW, Clemons KV, Hanson LH, Stevens DA. Restriction endonuclease analysis of total cellular DNA of *Aspergillus fumigatus* isolates of geographically and epidemiologically diverse origin. *J Infect Dis* 1990;162:1151–8.
253. Rhame FS. Prevention of nosocomial aspergillosis. *J Hosp Infect* 1991;18 (Suppl. A):466–72.
254. **(85)** Barnes RA, Rogers TR. Control of an outbreak of nosocomial aspergillosis by laminar air-flow isolation. *J Hosp Infect* 1989;14:89–94.
255. Roy M-C. The operating theater: a special environmental area. In: Wenzel RP, ed. *Prevention and control of nosocomial infections*, 3rd ed. Baltimore, MD: William & Wilkins, 1997;515–38.
256. Pavelchak N, DePersis RP, London M, et al. Identification of factors that disrupt negative air pressurization of respiratory isolation rooms. *Infect Control Hosp Epidemiol* 2000;21:191–5.

257. Anderson K. *Pseudomonas pyocyanea* disseminated from an air cooling apparatus. *Med J Austr* 1959;529–32.
258. Shaffer JG, McDade JJ. Airborne *Staphylococcus aureus*: a possible source in air control equipment. *Arch Environ Health* 1963;5:547–51.
259. Morey PR. Building-related illness with a focus on fungal issues. In: Hansen W, ed. A guide to managing indoor air quality in health care organizations. Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997;15–25.
260. Streifel AJ. Recognizing IAQ risk and implementing an IAQ program. In: Hansen W, ed. A guide to managing indoor air quality in health care organizations; Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997;75–91.
261. Morey PR. Appendix B. Fungal growth checklist. In: Hansen W. ed. A guide to managing indoor air quality in health care organizations. Oakbrook Terrace, IL: Joint Commission on Accreditation of Health Care Organizations Publications, 1997;129–35.
262. Brock DL, Jiankang J, Rinaldi MG, Wickes BL, Huycke MM. Outbreak of invasive *Aspergillus* infection in surgical patients, associated with a contaminated air-handling system. *Clin Inf Dis* 2003;37:786–93.
263. (31) National Air Duct Cleaners Association. General specifications for the cleaning of commercial HVAC systems. Washington, DC: NADCA, 2002. Publication No. NAD-06. Available at: www.nadca.com/standards/standards.asp
264. (32) U.S. Environmental Protection Agency. Use of disinfectants and sanitizers in heating, ventilation, air conditioning, and refrigeration systems [letter]. March 14, 2002. Available at: www.epa.gov/oppad001/hvac.htm
265. U.S. Environmental Protection Agency. Should you have the air ducts in your home cleaned? Washington, DC: EPA, 1997. EPA Document No. 402-K-97-002.
266. (159) Vujanovic V, Smoragiewicz W, Krzysztyniak K. Airborne fungal ecological niche determination as one of the possibilities for indirect mycotoxin risk assessment in indoor air. *Environ Toxicol* 2001;16:1–8.
267. Soules WJ. Airflow management techniques. *Clean Rooms* 1993;2:18–20.
268. Lawson CN. Commissioning hospitals for compliance. *ASHRAE Trans* 1993;99(2).
269. Wadowsky R, Benner S. Distribution of the genus *Aspergillus* in hospital room air conditioners. *Infect Control* 1987;8:516–8.
270. Streifel AJ. Aspergillosis and construction. In: Kundsin RB, ed. Architectural design and indoor microbial pollution. New York, NY: Oxford University Press, 1988;198–217.
271. Streifel AJ, Vesley D, Rhame FS. Occurrence of transient high levels of airborne fungal spores. Proceedings of the 6th Conference on Indoor Air Quality and Climate. Toronto, ON: 1990.
272. (73) Morey R, Williams C. Porous insulation in buildings: a potential source of microorganisms. Proceedings - Indoor Air '90, 5th International Conference. Toronto, ON: 1990;1–6.
273. (13) Bartley J. Construction and renovation. In: Pfeiffer J, ed. APIC Text of infection control and epidemiology. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc., 2000; 72.1–72.11.
274. (14) Harvey MA. Critical-care-unit design and furnishing: Impact on nosocomial infections. *Infect Control Hosp Epidemiol* 1998;19:597–601.
275. (15) National Association of Children's Hospitals and Related Institutions. Patient Care Focus Groups 1998. Assessing organizational readiness for infection control issues related to construction, renovation, and physical plant projects.
276. (50) Bartley JM. APIC State-of-the-art report: the role of infection control during construction in health care facilities. *Am J Infect Control* 2000;28:156–9.
277. (16) Carter CD, Barr BA. Infection control issues in construction and renovation. *Infect Control Hosp Epidemiol* 1997;18:587–96.
278. (47) Streifel AJ. Maintenance and engineering. In: Pfeiffer J, ed. APIC Text of Infection Control and Epidemiology. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc., 2000;76.1-76.8.
279. Kennedy V, Barnard B, Hackett B. Use of a risk matrix to determine the level of barrier protection during construction activities. Atlanta, GA : Peachtree Publications, 1997;27–8.
280. Morey PR. Building-related illness with a focus on fungal issues. In: Hansen W, ed. A guide to managing indoor air quality in health care organizations. Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997;15–25.

281. (67) Bartley J, ed. Infection control tool kit series – construction and renovation. Washington, DC: Association for Professionals in Infection Control and Epidemiology, 1999.
282. Bryce EA, Walker M, Scharf S, et al. An outbreak of cutaneous aspergillosis in a tertiary-care hospital. *Infect Control Hosp Epidemiol* 1996;17:170–2.
283. (62) Thio CL, Smith D, Merz WG, et al. Refinements of environmental assessment during an outbreak investigation of invasive aspergillosis in a leukemia and bone marrow transplant unit. *Infect Control Hosp Epidemiol* 2000;21:18–23.
284. (65) Kuehn TH, Gacek B, Yang CH, et al. Final report: ASHRAE 804-RP Phase I identification of contaminants, exposures effects, and control options for construction/renovation activities. Atlanta, GA:ASHRAE, Inc. 1995.
285. Kennedy HF, Michie JR, Richardson MD. Air sampling for *Aspergillus* spp. during building activity in a paediatric hospital ward. *J Hosp Infect* 1995;31:322–25.
286. Leenders ACAP, van Belkum A, Behrendt M, Luijendijk AD, Verbrugh HA. Density and molecular epidemiology of *Aspergillus* in air and relationship to outbreaks of *Aspergillus* infection. *J Clin Microbiol* 1999;37:1752–7.
287. Rath PM, Ansorg R. Value of environmental sampling and molecular typing of aspergilli to assess nosocomial sources of aspergillosis. *J Hosp Infect* 1997;37:47–53.
288. (71) Streifel AJ, Marshall JW. Parameters for ventilation controlled environments in hospitals. In: Design, Construction, and Operation of Healthy Buildings. Atlanta, GA: ASHRAE Press, 1998.
289. (348) Streifel AJ. Air cultures for fungi. In: Gilchrist M, ed. Clinical microbiology procedures handbook. Washington, DC: American Society for Microbiology Press, 1992;11.8.1–11.8.7.
290. American Conference of Governmental Industrial Hygienists (ACGIH). 2000 Threshold limit Values and biological exposure indices. Cincinnati, OH: American Conference of Governmental Industrial Hygienists, Inc., 2000;1–184.
291. U.S. Department of Labor, Occupational Safety and Health Administration. Air contaminants standard. 29 CFR 1910.1000, §1910.1000, Tables Z-1, Z-3. *Federal Register* 1993;58:35338–51.
292. (86) Leenders A, vanBelkum A, Janssen S, et al. Molecular epidemiology of apparent outbreaks of invasive *Aspergillus* in a hematology ward. *J Clin Microbiol* 1996;34:345–51.
293. (91) James MJ, Lasker BA, McNeil MM, Shelton M, Warnock DW, Reiss E. Use of a repetitive DNA probe to type clinical and environmental isolates of *Aspergillus flavus* from a cluster of cutaneous infections in a neonatal intensive care unit. *J Clin Microbiol* 2000;38:3612–8.
294. (92) Skladny H, Buchheidt D, Baust C, et al. Specific detection of *Aspergillus* species in blood and bronchoalveolar lavage samples of immunocompromised patients by two-step PCR. *J Clin Microbiol* 1999;37:3865–71.
295. (93) Symoens F, Bouchara J-P, Heinemann S, Nolard N. Molecular typing of *Aspergillus terreus* isolates by random amplification of polymorphic DNA. *J Hosp Infect* 2000;44:273–80.
296. (94) Diaz-Guerra TM, Mellado E, Cuenca-Estrella M, Gaztelurrutia L, Villate Navarro JI, Rodríguez Tudela JL. Genetic similarity among one *Aspergillus flavus* strain isolated from a patient who underwent heart surgery and two environmental strains obtained from the operating room. *J Clin Microbiol* 2000;38:2419–22.
297. Buttner MP, Stetzenbach LD. Monitoring airborne fungal spores in an experimental indoor environment to evaluate sampling methods and the effects of human activity on air sampling. *Appl Environ Microbiol* 1993;59:219–26.
298. Sayer WJ, Shean DB, Ghosseiri J. Estimation of airborne fungal flora by the Anderson sampler versus the gravity settling culture plate. *J Allerg* 1969;44:214–27.
299. Hay RJ, Clayton YM, Goodley JM. Fungal aerobiology: how, when and where? *J Hosp Infect* 1995;30(Suppl):S352–7.
300. Morris G, Kokki MH, Anderson K, Richardson MD. Sampling of *Aspergillus* spores in air. *J Hosp Infect* 2000;44:81–92.
301. Iwen PC, Davis JC, Reed EC, Winfield BA, Hinrichs SH. Airborne fungal spore monitoring in a protective environment during hospital construction and correlation with an outbreak of invasive aspergillosis. *Infect Control Hosp Epidemiol* 1994;15:303–6.
302. Pegues DA, Lasker BA, McNeil MM, Hamm PM, Lundal JL, Kubak BM. Cluster of cases of invasive aspergillosis in a transplant intensive care unit: evidence of person-to-person airborne transmission. *Clin Infect Dis* 2002;34:412–6.

303. Goodley JM, Clayton YM, Hay RJ. Environmental sampling for aspergilli during building construction on a hospital site. *J Hosp Infect* 1994;26:27–35.
304. (72) American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE). The HVAC commissioning process. Atlanta, GA: ASHRAE, 1996;1–48. ASHRAE Guideline 1.
305. (63) Mermel LA, Josephson SL, Giorgio CH, Dempsey J, Parenteau S. Association of Legionnaires' disease with construction: contamination of potable water? *Infect Control Hosp Epidemiol* 1995;16:76–81.
306. Loo VG, Bertrand C, Dixon C, et al. Control of construction-associated nosocomial aspergillosis in an antiquated hematology unit. *Infect Control Hosp Epidemiol* 1996;17:360–4.
307. (68) Ottney TC. Particle management for HVAC systems. *ASHRAE J* 1993;35:26–28, 30, 32, 34.
308. Rautiala S, Reponen T, Nevalainen A, et al. Control of exposure to airborne viable microorganisms during remediation of moldy buildings: report of three case studies. *Am Ind Hyg Assoc J* 1998;59:455–60.
309. (69) Finkelstein LE, Mendelson MH. Infection control challenges during hospital renovation. *Am J Nursing* 1997;97:60–1.
310. Hruszkewycz V, Ruben B, Hypes CM, Bostic GD, Staszkiwicz J, Band JD. A cluster of pseudofungemia associated with hospital renovation adjacent to the microbiology laboratory. *Infect Control Hosp Epidemiol* 1992;13:147–50.
311. Laurel VL, Meier PA, Astorga A, Dolan D, Brockett R, Rinaldi MG. Pseudoepidemic of *Aspergillus niger* infections traced to specimen contamination in the microbiology laboratory. *J Clin Microbiol* 1999;37:1612–6.
312. (66) Opal SM, Asp AA, Cannady PB Jr, Morse PL, Burton LJ, Hammer II PG. Efficacy of infection control measures during a nosocomial outbreak of disseminated aspergillosis associated with hospital construction. *J Infect Dis* 1986;153: 634–7.
313. Fitzpatrick F, Prout S, Gilleece A, Fenelon LE, Murphy OM. Nosocomial aspergillosis during building work — a multidisciplinary approach. *J Hosp Infect* 1999;42:170–1.
314. Garrett DO, Jochimsen E, Jarvis W. Invasive *Aspergillus* spp. infections in rheumatology patients. *J Rheumatol* 1999;26:146–9.
315. Larsson L, Larsson PF. Analysis of chemical markers as a means of characterizing airborne microorganisms in indoor environments: a case study. *Indoor Built Environ* 2001;10:232–7.
316. (99) Buckner CD, Clift RA, Sanders JE, et al. Protective environment for marrow transplant recipients: a prospective study. *Ann Intern Med* 1978;89:893–901.
317. (100) Murray WA, Streifel AJ, O'Dea TJ, Rhame FS. Ventilation for protection of immune compromised patients. *ASHRAE Trans* 1988;94:1185–91.
318. (101) Streifel AJ, Vesley D, Rhame FS, Murray B. Control of airborne fungal spores in a university hospital. *Environment International* 1989;12:441–4.
319. Perry S, Penland WZ. The portable laminar flow isolator: new unit for patient protection in a germ-free environment. In: *Recent Results in Cancer Research*. New York, NY: Springer-Verlag, 1970.
320. Hayden CS, Fischbach M, Johnston OE. A model for calculating air leakage in negative pressure isolation areas. Cincinnati, OH: DHHS, 1997. NIOSH Report ECTR 212-05c.
321. DeLuga GF. Differential airflow, pressure, have key relationship in pressurization. *Lab Design* 1997;2:6–7.
322. Rhame FS. Nosocomial aspergillosis: How much protection for which patients? *Infect Control Hosp Epidemiol* 1989;10:296–8.
323. Hofflin JM, Potasman I, Baldwin JC, Oyster PE, Stinson EB, Remington JS. Infectious complications in heart transplant recipients receiving cyclosporine and corticosteroids. *Ann Intern Med* 1987;106:209–16.
324. Schulman LL, Smith CR, Drusin R, Rose EA, Enson Y, Reemtsma K. Respiratory complications of cardiac transplantation. *Am J Med Sci* 1988;296:1–10.
325. Dummer JS, Ho M. Risk factors and approaches to infections in transplant recipients. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and practice of infectious diseases*, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;3126–35.
326. Dummer JS, Ho M. Infections in solid organ transplant recipients. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and practice of infectious diseases*, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;3148–58.
327. Walsh TR, Guttendorf J, Dummer S, et al. The value of protective isolation procedures in cardiac transplant recipients. *Ann Thorac Surg* 1989;47:539–45.
328. Streifel AJ. Health-care IAQ: guidance for infection control. *HPAC Heating/Piping/Air Cond Eng* 2000; Oct:28–30, 33, 34, 36.

329. (87) Yeager CC. Copper and zinc preservatives. In: Block SS, ed. Disinfection, sterilization, and preservation, 4th ed. Philadelphia, PA: Lea & Febiger, 1991;358–61.
330. Cookson ST, Jarvis WR. Prevention of nosocomial transmission of *Mycobacterium tuberculosis*. Infect Dis Clin North Am 1997;11:367–409.
331. (42) CDC. Nosocomial transmission of multidrug-resistant tuberculosis among HIV-infected persons: Florida and New York, 1988–1991. MMWR 1991;40:585–91.
332. (43) CDC. Outbreak of multidrug-resistant tuberculosis at a hospital — New York City, 1991. MMWR 1993;42:427–34.
333. (7) Beck-Sague CM, Dooley SW, Hutton MD, et al. Hospital outbreak of multidrug-resistant *Mycobacterium tuberculosis*. JAMA 1992;268:1280–6.
334. (17) Coronado VG, Beck-Sague CM, Hutton MD, et al. Transmission of multidrug-resistant *Mycobacterium tuberculosis* among persons with human immunodeficiency virus infection in an urban hospital: Epidemiologic and restriction fragment length polymorphism analysis. J Infect Dis 1993;168:1052–5.
335. (18) Coronado VG, Valway S, Finelli L, et al. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis* among intravenous drug users with human immunodeficiency virus infection [abstract]. In: Abstracts of the Third Annual Meeting of the Society for Hospital Epidemiology of America. Chicago, IL: Infect Control Hosp Epidemiol 1993;14:428.
336. (8) Dooley SW, Villarino ME, Lawrence M, et al. Tuberculosis in a hospital unit for patients infected with the human immunodeficiency virus (HIV): evidence of nosocomial transmission. JAMA 1992;267:2632–4.
337. (19) Edlin BR, Tokars JI, Grieco MH, et al. An outbreak of multidrug-resistant tuberculosis among hospitalized patients with the acquired immunodeficiency syndrome: epidemiologic studies and restriction fragment length polymorphism analysis. N Engl J Med 1992;326:1514–22.
338. (20) Fischl MA, Uttamchandani RB, Daikos GL, et al. An outbreak of tuberculosis caused by multiple-drug-resistant tubercle bacilli among patients with HIV infection. Ann Intern Med 1992;117:177–83.
339. (21) Ikeda ARM, Birkhead GS, DeFerdinando Jr GT, et al. Nosocomial tuberculosis: an outbreak of a strain resistant to seven drugs. Infect Control Hosp Epidemiol 1995;16:152–9.
340. (22) Jarvis WR. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*. Res Microbiol 1992;144:117–22.
341. (23) Jarvis WR. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*. Am J Infect Control 1995;23:146–51.
342. (24) Jereb JA, Klevens RM, Privett TD, et al. Tuberculosis in health care workers at a hospital with an outbreak of multidrug-resistant *Mycobacterium tuberculosis*. Arch Intern Med 1995;155:854–9.
343. (25) Moran GJ, McCabe F, Morgan MT, Talan DA. Delayed recognition and infection control for tuberculosis patients in the emergency department. Ann Emerg Med 1995;26:283–9.
344. (26) Pearson ML, Jereb JA, Frieden TR, et al. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*: a risk to hospitalized patients and health-care workers. Ann Intern Med 1992;117:191–6.
345. Tokars JI, Jarvis WR, Edlin BR, et al. Tuberculin skin testing of hospital employees during an outbreak of multidrug-resistant tuberculosis in human immunodeficiency virus (HIV) infected patients. Infect Control Hosp Epidemiol 1992;13:509–10.
346. Macher JM. The use of germicidal lamps to control tuberculosis in healthcare facilities. Infect Control Hosp Epidemiol 1993;14:723–9.
347. (129) U.S. Department of Labor, Occupational Safety and Health Administration. Respiratory Protection, 29 CFR 1910.139. Federal Register 1998;63:1152–300.
348. (105) Ehrenkranz NJ, Kicklighter JL. Tuberculosis outbreak in a general hospital: evidence for airborne spread of infection. Ann Intern Med 1972;77:377–82.
349. (106) Calder RA, Duclos P, Wilder MH, et al. *Mycobacterium tuberculosis* transmission in a health clinic. Bull Int Union Tuberc Lung Dis 1991;66:103–6.
350. (107) Jereb JA, Burwen DR, Dooley SW, et al. Nosocomial outbreak of tuberculosis in a renal transplant unit: application of a new technique for restriction fragment length polymorphism analysis of *Mycobacterium tuberculosis* isolates. J Infect Dis 1993;168:1219–24.
351. (127) Ayliffe GAJ. Role of the environment of the operating suite in surgical wound infection. Rev Infect Dis 1991;13(suppl):S800–S804.
352. (128) Choux M, Genitori L, Lang D, Lena G. Shunt implantation: reducing the incidence of shunt infection. J Neurosurg 1992;77:875–80.

353. Edmiston CE Jr, Sinski S, Seabrook GR, Simons D, Goheen MP. Airborne particulates in the OR environment. *AORN J* 1999;69:1169–72.
354. Duhaime AC, Bonner K, McGowan KL, Schut L, Sutton LN, Plotkin S. Distribution of bacteria in the operating room environment and its relation to ventricular shunt infections: a prospective study. *Childs Nerv Syst* 1991;7:211–4.
355. Everett WD, Kipp H. Epidemiologic observations of operating room infections resulting from variations in ventilation and temperature. *Am J Infect Control* 1991;19:277–82.
356. **(115)** Lidwell OM. Clean air at operation and subsequent sepsis in the joint. *Clin Orthop* 1986;211:91–02.
357. **(116)** Nichols RL. The operating room. In: Bennett JV, Brachman PS, eds. *Hospital infections*, 3rd ed. Boston, MA: Little, Brown and Company, 1992;461–73.
358. **(117)** Clark RP, Reed PJ, Seal DV, Stephenson ML. Ventilation conditions and air-borne bacteria and particles in operating theatres: proposed safe economies. *J Hyg (Lond)* 1985;95:325–35.
359. **(119)** Laufman H. The operating room. In: Bennett JV, Brachman PS, eds. *Hospital infections*, 2nd ed. Boston, MA/Toronto, ON: Little, Brown and Company, 1986;315–23.
360. Pittet D, Ducle G. Infectious risk factors related to operating rooms. *Infect Control Hosp Epidemiol* 1994;15:456–62.
361. Hambræus A. Aerobiology in the operating room — a review. *J Hosp Infect* 1988;11(suppl. A):68–76.
362. **(118)** Babb JR, Lynam P, Ayliffe GAJ. Risk of airborne transmission in an operating theater containing four ultraclean air units. *J Hosp Infect* 1995;31:159–68.
363. Velesco, E, Thuler LCS, Martins CAS, deCastroDias LM, Conalves VMSC. Risk factors for infectious complications after abdominal surgery for malignant disease. *Am J Infect Control* 1996;24:1–6.
364. **(120)** National Academy of Sciences, National Research Council, Division of Medical Sciences, Ad Hoc Committee on Trauma. Postoperative wound infections: the influence of ultraviolet irradiation of the operating room and of various other factors. *Ann Surg* 1964;160 (suppl.):1–192.
365. **(121)** Charnley J. A clean-air operating enclosure. *Br J Surg* 1964;51:202–5.
366. **(122)** Lidwell OM, Lowbury EJJ, Whyte W, Blowers R, Stanley SJ, Lowe D. Effect of ultraclean air in operating rooms on deep sepsis in the joint after total hip or knee replacement: a randomized study. *Br Med J* 1982;285:10–4.
367. **(123)** Hill C, Flamant R, Mazas F, Evrard J. Prophylactic cefazolin versus placebo in total hip replacement: report of a multicentre double-blind randomized trial. *Lancet* 1981;1:795–6.
368. **(124)** Ha’eri GB, Wiley AM. Total hip replacement in a laminar flow environment with special reference to deep infections. *Clin Orthop* 1980;148:163–8.
369. **(125)** Collins DK, Steinhaus K. Total hip replacement without deep infection in a standard operating room. *J Bone Joint Surg* 1976;58A:446–50.
370. **(126)** Taylor GD, Bannister GC, Leeming JP. Wound disinfection with ultraviolet radiation. *J Hosp Infect* 1995;30:85–93.
371. **(130)** Langevin PB, Rand KH, Layton AJ. The potential for dissemination of *Mycobacterium tuberculosis* through the anesthesia breathing circuit. *Chest* 1999;115:1107–14.
372. **(131)** U.S. Department of Labor, Occupational Safety and Health Administration. Occupational exposure to tuberculosis: proposed rule. (29 CFR 1910). *Federal Register* 1997;62:54159–209.
373. **(132)** Aranha-Creado H, Prince D, Greene K, Brandwein H. Removal of *Mycobacterium* species by breathing circuit filters. *Infect Control Hosp Epidemiol* 1997;18:252–254.
374. Anesthesiology Society of America. *Infection Control for Practice of Anesthesiology*. 1999. Available at: www.asahq.org/Profinfo/Infection/Infection_TOC.html
375. McCarthy JF. Risk factors for occupational exposures in healthcare professionals. In: Hansen W, ed. *A guide to managing indoor air quality in health care organizations*. Oakbrook Terrace IL: Joint Commission on Accreditation of Healthcare Organizations, 1997;27–41.
376. National Institute for Occupational Safety and Health. NIOSH Health Hazard Evaluation and Technical Assistance Report: HETA 85-126-1932;1988.
377. National Institute for Occupational Safety and Health. NIOSH Health Hazard Evaluation and Technical Assistance Report: HETA 88-101-2008;1990.
378. **(135)** National Institute for Occupational Safety and Health. Control of smoke from laser/electric surgical procedures. DHHS (NIOSH) Publication 96-128;1996. Available at: www.cdc.gov/niosh/hc11.html
379. Taravella MJ, Weinberg A, Blackburn P, May M. Do intact viral particles survive excimer laser ablation? *Arch Ophthalmol* 1997;115:1028–30.

380. Hagen KB, Kettering JD, Aprecio RM, et al. Lack of virus transmission by the excimer laser plume. *Am J Ophthalmol* 1997;124:206–11.
381. Kunachak S, Sithisarn P, Kulapaditharom B. Are laryngeal papilloma virus-infected cells viable in the plume derived from a continuous mode carbon dioxide laser, and are they infectious? A preliminary report on one laser mode. *J Laryng Otol* 1996;110:1031–3.
382. (137) Hughes PS, Hughes AP. Absence of human papillomavirus DNA in the plume of erbium:YAG laser-treated warts. *J Am Acad Dermatol* 1998;38:426–8.
383. Garden JM, O'Bannon K, Sheinitz LS, et al. Papillomavirus in the vapor of carbon dioxide laser treated verrucae. *JAMA* 1988;125:1199–202.
384. Sawchuck WS, Weber JP, Lowry DR, et al. Infectious papillomavirus in the vapour of warts treated with carbon dioxide laser or electrocoagulation: detection and protection. *J Am Acad Dermatol* 1989;21:41–9.
385. Baggish MS, Poiesz BJ, Joret D, et al. Presence of human immunodeficiency virus DNA in laser smoke. *Lasers Surg Med* 1991;11:197–203.
386. (138) Capizzi PJ, Clay RP, Battey MJ. Microbiologic activity in laser resurfacing plume and debris. *Lasers Surg Med* 1998;23:172–4.
387. McKinley IB Jr, Ludlow MO. Hazards of laser smoke during endodontic therapy. *J Endodont* 1994;20:558.
388. Favero MS, Bolyard EA. Microbiologic considerations: disinfection and sterilization strategies and the potential for airborne transmission of bloodborne pathogens. *Surg Clin North Am* 1995;75:1071–89.
389. (136) Association of periOperative Registered Nurses. Recommended practices for laser safety in practice settings. In: Standards, Recommended Practices and Guidelines. Denver CO; AORN;2003;301–5.
390. (139) ECRI. Surgical smoke evacuation systems. *Health Devices* 1997;26:132–72.
391. (140) ECRI. Update evaluation: Surgical smoke evacuation systems. *Health Devices* 1999;28:333–62.
392. (141) ECRI. Stationary surgical smoke evacuation systems. *Health Devices* 2001;30:73–86.
393. American National Standards Institute. ANSI National standard for safe use of lasers in health care facilities. ANSI Z136.3-1996.
394. Kaufman AF, McDade J, Patton C, et al. Pontiac fever: isolation of the etiologic agent (*Legionella pneumophila*) and demonstration of its mode of transmission. *Am J Epidemiol* 1981;114:337–47.
395. (192) Marston BJ, Lipman HB, Breiman RF. Surveillance for Legionnaires' disease: risk factors for morbidity and mortality related to infection with *Legionella*. *Arch Intern Med* 1994;154:2417–22.
396. (Appendix; 4) Hoge CW, Breiman RF. Advances in the epidemiology and control of *Legionella* infections. *Epidemiol Rev* 1991;13:329–40.
397. Breiman RF, Butler JC. Legionnaires' disease: clinical, epidemiological, and public health perspectives. *Semin Respir Infect* 1998;13:84–9.
398. Yu, VL. *Legionella pneumophila* (Legionnaires' disease). In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of infectious diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2424–35.
399. Muder RR. Other *Legionella* species. In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of infectious diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2435–41.
400. Yu VL. Could aspiration be the major mode of transmission for *Legionella*? *Am J Med* 1993;95:13–5.
401. Jimenez P, Torres A, Rodriguez-Roisin R, et al. Incidence and etiology of pneumonia acquired during mechanical ventilation. *Crit Care Med* 1989;17:882–5.
402. (220) Zuravleff JJ, Yu VL, Shonnard JW, Rihs JD, Best M. *Legionella pneumophila* contamination of a hospital humidifier: demonstration of aerosol transmission and subsequent subclinical infection in exposed guinea pigs. *Am Rev Respir Dis* 1983;128:657–61.
403. (202) Mastro TD, Fields BS, Breiman RF, Campbell J, Plikaytis BD, Spika JS. Nosocomial Legionnaires' disease and use of medication nebulizers. *J Infect Dis* 1991;163:667–70.
404. (203) Dondero TJ Jr, Rendtorff RC, Mallison GF, et al. An outbreak of Legionnaires' disease associated with a contaminated air-conditioning cooling tower. *N Engl J Med* 1980;302:365–70.
405. (199) Garbe PL, Davis BJ, Weisfield JS, et al. Nosocomial Legionnaires' disease: epidemiologic demonstration of cooling towers as a source. *JAMA* 1985;254:521–4.
406. (204) O'Mahony MC, Stanwell-Smith RE, Tillett HE, et al. The Stafford outbreak of Legionnaires' disease. *Epidemiol Infect* 1990;104:361–80.
407. (205) Breiman RF, Fields BS, Sanden G, Volmer L, Meier A, Spika J. An outbreak of Legionnaires' disease associated with shower use: possible role of amoebae. *JAMA* 1990;263:2924–6.

408. (200) Hanrahan JP, Morse DL, Scharf VB, et al. A community hospital outbreak of legionellosis: transmission by potable hot water. *Am J Epidemiol* 1987;125:639–9.
409. (206) Breiman RF, VanLoock FL, Sion JP, et al. Association of “sink bathing” and Legionnaires’ disease [abstract]. In: Abstracts of the 91st Meeting of the American Society for Microbiology, 1991.
410. (207) Struelens MJ, Maes N, Rost F, et al. Genotypic and phenotypic methods for the investigation of a nosocomial *Legionella pneumophila* outbreak and efficacy of control measures. *J Infect Dis* 1992;166:22–30.
411. Terranova W, Cohen ML, Fraser DW. Outbreak of Legionnaires’ disease diagnosed in 1977. *Lancet* 1978;2:122–4.
412. (219) Marrie TJ, Haldane D, MacDonald S, et al. Control of endemic nosocomial Legionnaires’ disease by using sterile potable water for high risk patients. *Epidemiol Infect* 1991;107:591–605.
413. Nechwatal R, Ehret W, Klatte OJ, et al. Nosocomial outbreak of legionellosis in a rehabilitation center: demonstration of potable water as a source. *Infection* 1993;21:235–40.
414. Hoebe CJP, Cluitmanans JJM, Wagenvoort JHT. Two fatal cases of nosocomial *Legionella pneumophila* pneumonia associated with a contaminated cold water supply. *Eur J Clin Microbiol Infect Dis* 1998;17:740–9.
415. Helms CM, Viner JP, Sturm RH, et al. Comparative features of pneumococcal, *Mycoplasma*, and Legionnaires’ disease pneumonias. *Ann Intern Med* 1979;90:543–7.
416. Yu V, Kroboth FJ, Shonnard J, Brown A, McDearman S, Magnussen M. Legionnaires’ disease: new clinical perspectives from a prospective pneumonia study. *Am J Med* 1982;73:357–61.
417. (194) Jimenez ML, Aspa J, Padilla B, et al. Fiberoptic bronchoscopic diagnosis of pulmonary disease in 151 HIV-infected patients with pneumonitis. *Eur J Clin Microbiol Infect Dis* 1991;10:491–6.
418. Lowry PW, Blankenship RJ, Gridley W, et al. A cluster of *Legionella* sternal wound infections due to postoperative topical exposure to contaminated tap water. *N Engl J Med* 1991;324:109–12.
419. Shah A, Check F, Baskin S. Legionnaires’ disease and acute renal failure: case report and review. *Clin Infect Dis* 1992;14:204–7.
420. Lowry PW, Tompkins LS. Nosocomial legionellosis: a review of pulmonary and extrapulmonary syndromes. *Am J Infect Control* 1993;21:21–7.
421. Schlanger G, Lutwick LI, Kurzman M, et al. Sinusitis caused by *L. pneumophila* in a patient with acquired immune deficiency syndrome. *Am J Med* 1984;77:957–60.
422. Tompkins LS, Roessler BJ, Redd SC, et al. *Legionella* prosthetic-valve endocarditis. *N Engl J Med* 1988;318:530–5.
423. (195) Bock BV, Kirby BD, Edelstein PH, et al. Legionnaires’ disease in renal transplant recipients. *Lancet* 1978;1:410–3.
424. (196) Kirby BD, Snyder KM, Meyer RD, Finegold SM. Legionnaires’ disease: report of 65 nosocomially acquired cases and review of the literature. *Medicine* 1980;59:188–205.
425. (197) Brady MT. Nosocomial Legionnaires’ disease in a children’s hospital. *J Pediatr* 1989;115:46–50.
426. Horie H, Kawakami H, Minoshima K, et al. Neonatal Legionnaires’ disease: histologic findings in an autopsied neonate. *Acta Pathol Jpn* 1992;42:427–31.
427. Roig J, Aguilar X, Ruiz J, et al. Comparative study of *Legionella pneumophila* and other nosocomial pneumonias. *Chest* 1991;99:344–50.
428. Redd SC, Schuster DM, Quan J, et al. Legionellosis cardiac transplant recipients: results of a nationwide survey. *J Infect Dis* 1988;158:651–3.
429. Seu P, Winston DJ, Olthoft KM, et al. Legionnaires’ disease in liver transplant recipients. *Infect Dis Clin Pract* 1993;2:109–13.
430. (215) Chow JW, Yu VL. *Legionella*: a major opportunistic pathogen in transplant recipients. *Semin Respir Infect* 1998;13:132–9.
431. (189) Kool JL, Fiore AE, Kioski CM, et al. More than ten years of unrecognized nosocomial transmission of Legionnaires’ disease among transplant patients. *Infect Control Hosp Epidemiol* 1998;19:898–904.
432. (190) Le Saux NM, Sekla L, McLeod J, et al. Epidemic of nosocomial Legionnaires’ disease in renal transplant recipients: a case-control and environmental study. *Can Med Assoc J* 1989;140:1047–53.
433. Berendt RF, Young HW, Allen RG, Knutsen GL. Dose-response of guinea pigs experimentally infected with aerosols of *Legionella pneumophila*. *J Infect Dis* 1980;141:186–92.
434. Marston BJ, Plouffe JF, File TM, et al. Incidence of community-acquired pneumonia requiring hospitalization — results of a population-based active surveillance study in Ohio. *Arch Intern Med* 1997;157:1709–18.

435. **(198)** Muder RR, Yu VL, McClure JK, Kroboth FJ, Kominos SD, Lumish RN. Nosocomial Legionnaires' disease uncovered in a prospective pneumonia study: implications for underdiagnosis. *JAMA* 1983;249:3184–8.
436. Brennen C, Vickers JP, Yu VL, Puntereri A, Yee YC. Discovery of occult *Legionella pneumonia* in a long-stay hospital: Results of prospective serologic survey. *Br Med J* 1987;295:306–7.
437. **(166)** Marrie TJ, MacDonald S, Clarke K, Haldane D. Nosocomial Legionnaires' disease: lessons from a four-year prospective study. *Am J Infect Control* 1991;19:79–85.
438. Stout JE, Yu VL. Current concepts: legionellosis. *N Engl J Med* 1997;337:682–7.
439. Vergis EN, Yu VL. Macrolides are ideal for empiric therapy of community-acquired pneumonia in the immunocompromised host. *Semin Respir Infect* 1998;13:322–8.
440. Sopena N, Sabria-Leal M, Pedro-Botet ML, et al. Comparative study of the clinical presentation of *Legionella pneumonia* and other community-acquired pneumonias. *Chest* 1998;113:1195–200.
441. **(217)** Hirani NA, MacFarlane JT. Impact of management guidelines on the outcome of severe community acquired pneumonia. *Thorax* 1997;52:17–21.
442. Lieberman D, Porath A, Schlaeffer F, Boldur L. *L. pneumophila* species community-acquired pneumonia: a review of 56 hospitalized patients. *Chest* 1996;109:1243–9.
443. Ewig S, Bauer T, Hasper E, et al. Value of routine microbial investigation in community-acquired pneumonia treated in a tertiary care center. *Respiration* 1996;63:164–9.
444. Marrie TJ, Peeling RW, Fine MJ, et al. Ambulatory patients with community-acquired pneumonia: the frequency of atypical agents and clinical course. *Am J Med* 1996;101:508–15.
445. Benin AI, Benson RF, Besser RE. Trends in Legionnaires' disease, 1980–1998: declining mortality and new patterns of diagnosis. *Clin Infect Dis* 2002;35:1039–46.
446. Fliermans CD, Cherry WB, Orrison LH, Smith SJ, Tison DL, Pope DH. Ecologic distribution of *Legionella pneumophila*. *Appl Environ Microbiol* 1981;41:9–16.
447. Morris GK, Patton CM, Feeley JC, et al. Isolation of the Legionnaires' disease bacterium from environmental samples. *Ann Intern Med* 1979;90:664–6.
448. Hsu SC, Martin R, Wentworth BB. Isolation of *Legionella* species from drinking water. *Appl Environ Microbiol* 1984;48:830–2.
449. Tison DL, Seidler RJ. *Legionella* incidence and density in potable drinking water. *Appl Environ Microbiol* 1983;45:337–9.
450. Parry MF, Stampleman L, Hutchinson JH, et al. Waterborne *Legionella bozemanii* and nosocomial pneumonia in immunosuppressed patients. *Ann Intern Med* 1985;103:205–10.
451. England AC, Fraser DW. Sporadic and epidemic nosocomial legionellosis in the United States: epidemiologic features. *Am J Med* 1981;70:707–11.
452. Cohen ML, Broome CV, Paris AL, et al. Fatal nosocomial Legionnaires' disease: clinical and epidemiological characteristics. *Ann Intern Med* 1979;90:611–3.
453. **(193)** Haley CE, Cohen ML, Halter J, Meyer RD. Nosocomial Legionnaires' disease: a continuing common-source epidemic at Wadsworth Medical Center. *Ann Intern Med* 1979;90:583–6.
454. Stout JE, Yu VL, Vickers RM, Shonnard J. Potable water supply as the hospital reservoir for Pittsburgh pneumonia agent. *Lancet* 1982;1:471–2.
455. **(201)** Arnow PM, Chou T, Weil D, Shapiro EN, Kretzschmar C. Nosocomial Legionnaires' disease caused by aerosolized tap water from respiratory devices. *J Infect Dis* 1982;146:460–7.
456. Farrell ID, Barker JE, Miles EP, Hutchinson JCP. A field study of the survival of *Legionella pneumophila* in a hospital hot-water system. *Epidemiol Infect* 1990;104:381–7.
457. Stout JE, Yu VL, Best MG. Ecology of *Legionella pneumophila* within water distribution systems. *Appl Environ Microbiol* 1985;49:221–8.
458. Sanden GN, Fields BS, Barbaree JM, et al. Viability of *Legionella pneumophila* in chlorine-free water at elevated temperatures. *Curr Microbiol* 1989;61–5.
459. Schulze-Röbbecke R, Rodder M, Exner M. Multiplication and killing temperatures of naturally occurring legionellae. *Zbl Bakt Hyg B* 1987;184:495–500.
460. Habicht W, Muller HE. Occurrence and parameters of frequency of *Legionella* in warm water systems of hospitals and hotels in Lower Saxony. *Zbl Bakt Hyg B* 1988;186:79–88.
461. Ciesielski CA, Blaser MJ, Wang WL. Role of stagnation and obstruction of water flow in isolation of *Legionella pneumophila* from hospital plumbing. *Appl Environ Microbiol* 1984;48:984–7.
462. Rowbotham TJ. Preliminary report on the pathogenicity of *Legionella pneumophila* for freshwater and soil amoebae. *J Clin Path* 1980;33:179–83.

463. Fields BS, Sanden GN, Barbaree JM, et al. Intracellular multiplication of *Legionella pneumophila* in amoebae isolated from hospital hot water tanks. *Curr Microbiol* 1989;18:131–7.
464. (142) Villarino ME, Stevens LE, Schable B, et al. Risk factors for epidemic *Xanthomonas maltophilia* infection/colonization in intensive care unit patients. *Infect Control Hosp Epidemiol* 1992;13:201–6.
465. (147) Burdge DR, Nakielna EM, Noble MA. Case-control and vector studies of nosocomial acquisition of *Pseudomonas cepacia* in adult patients with cystic fibrosis. *Infect Control Hosp Epidemiol* 1993;14:127–30.
466. Stephenson JR, Heard SR, Richards MA, Tabaqchali S. Gastrointestinal colonization and septicaemia with *Pseudomonas aeruginosa* due to contaminated thymol mouthwash in immunocompromised patients. *J Hosp Infect* 1985;6:369–78.
467. Kolmos HJ, Thusen B, Neilsen SV, Lohmann M, Kristoffersen K, Rosdahl VT. Outbreak of infection in a burns unit due to *Pseudomonas aeruginosa* originating from contaminated tubing used for irrigating patients. *J Hosp Infect* 1993;24:11–21.
468. Vanholder R, Vanhaecke E, Ringoir S. Waterborne *Pseudomonas* septicemia. *ASAIO Trans* 1990;36:M215–6.
469. Ehni WF, Reller LB, Ellison RT III. Bacteremia in granulocytopenic patients in a tertiary-care general hospital. *Rev Infect Dis* 1991;13:613–9.
470. Gallagher PG, Watanakunakorn C. *Pseudomonas* bacteremia in a community teaching hospital, 1980–1984. *Rev Infect Dis* 1989;11:846–52.
471. Centers for Disease Control. Nosocomial infection and pseudoinfection from contaminated endoscopes and bronchoscopes — Wisconsin and Missouri. *MMWR* 1991;40:675–8.
472. Kerr JR, Moore JE, Curran MD, et al. Investigation of a nosocomial outbreak of *Pseudomonas aeruginosa* pneumonia in an intensive care unit by random amplification of polymorphic DNA assay. *J Hosp Infect* 1995;30:125–31.
473. Brewer SC, Wunderink RG, Jones CB, Leeper KV. Ventilator-associated pneumonia due to *Pseudomonas aeruginosa*. *Chest* 1996;109:1019–22.
474. Rello J, Jubert P, Valles J, et al. Evaluation of outcome for intubated patients with pneumonia due to *Pseudomonas aeruginosa*. *Clin Infect Dis* 1996;23:973–8.
475. Henderson A, Kelly W, Wright M. Fulminant primary *Pseudomonas aeruginosa* pneumonia and septicaemia in previously well adults. *Intensive Care Med* 1992;18:430–2.
476. Torres A, Serra-Battles J, Ferrer A, et al. Severe community acquired pneumonia: epidemiology and prognostic factors. *Am Rev Respir Dis* 1991;144:312–8.
477. Pedersen SS, Koch C, Høiby N, Rosendal K. An epidemic spread of multiresistant *Pseudomonas aeruginosa* in a cystic fibrosis center. *J Antimicrob Chemother* 1986;17:505–6.
478. Kubesch P, Dörk T, Wulbrand U, et al. Genetic determinants of airways' colonization with *Pseudomonas aeruginosa* in cystic fibrosis. *Lancet* 1993;341:189–93.
479. Koch C, Høiby N. Pathogenesis of cystic fibrosis. *Lancet* 1993;341:1065–9.
480. Worlitzsch D, Wolz C, Botzenart K, et al. Molecular epidemiology of *Pseudomonas aeruginosa* – urinary tract infections in paraplegic patients. *Zentrabl Hyg Umweltmed* 1989;189:175–84.
481. Glenister H, Holton J, Teall A. Urinary tract pressure recording equipment as a source for infection. *J Hosp Infect* 1985;6:224–6.
482. Ferroni A., Nguyen L, Pron B, Quense G, Brusset MC, Berche P. Outbreak of nosocomial urinary tract infections due to *Pseudomonas aeruginosa* in a paediatric surgical unit associated with tap water contamination. *J Hosp infect* 1998;39:301–7.
483. Marrie TJ, Major H, Gurwith M, et al. Prolonged outbreak of nosocomial urinary tract infection with a single strain of *Pseudomonas aeruginosa*. *Can Med Assoc J* 1978;119:593–8.
484. Moore B, Forman A. An outbreak of urinary *Pseudomonas aeruginosa* infection acquired during urological operations. *Lancet* 1966;2:929–31.
485. Anderson RJ, Schafer LA, Olin DB, Eickhoff TC. Septicemia in renal transplant recipients. *Arch Surg* 1973;106:692–4.
486. Fang G, Brennen C, Wagener M, et al. Use of ciprofloxacin versus use of aminoglycosides for therapy of complicated urinary tract infection: prospective, randomized clinical and pharmacokinetic study. *Antimicrob Agents Chemother* 1991;35:1849–55.
487. Dorff GJ, Beimer NF, Rosenthal DR, Rytel MW. *Pseudomonas* septicemia: illustrated evolution of its skin lesions. *Arch Intern Med* 1971;128:591–5.
488. Teplitz C. Pathogenesis of *Pseudomonas vasculitis* and septic lesions. *Arch Pathol* 1965;80:297–307.

489. Roberts R, Tarpay MM, Marks MI, Nitschke R. Erysipelas-like lesions and hyperesthesia as manifestations of *Pseudomonas aeruginosa* sepsis. *JAMA* 1982;248:2156–7.
490. Duncan BW, Adzick NS, deLorimier AA, et al. Necrotizing fasciitis in childhood. *J Pediatr Surg* 1992;27:668–71.
491. McManus AT, Mason AD Jr, McManus WF, Pruitt BA Jr. Twenty-five year review of *Pseudomonas aeruginosa* bacteremia in a burn center. *Eur J Clin Microbiol* 1985;4:219–23.
492. Tredget EE, Shankowsky HA, Joffe AM, et al. Epidemiology of infections with *Pseudomonas aeruginosa* in burn patients: the role of hydrotherapy. *Clin Infect Dis* 1992;15:941–9.
493. Schlech WF III, Simosen N, Sumarah R, Martin RS. Nosocomial outbreak of *Pseudomonas aeruginosa* folliculitis associated with a physiotherapy pool. *Can Med Assoc J* 1986;134:909–13.
494. Fang G, Keys TF, Gentry LO, et al. Prosthetic valve endocarditis resulting from nosocomial bacteremia: a prospective, multicenter study. *Ann Intern Med* 1993;119:560–7.
495. Cohen PS, Maguire JH, Weinstein L. Infective endocarditis caused by gram-negative bacteria: a review of the literature, 1945–1977. *Prog Cardiovasc Dis* 1980;22:205–42.
496. Wise BL, Mathis JL, Jawetz E. Infections of the central nervous system due to *Pseudomonas aeruginosa*. *J Neurosurg* 1969;31:432–4.
497. Bray DA, Calcaterra TC. *Pseudomonas* meningitis complicating head and neck surgery. *Laryngoscope* 1976;86:1386–90.
498. Schein OD, Wasson PJ, Boruchoff SA, Kenyon KR. Microbial keratitis associated with contaminated ocular medications. *Am J Ophthalmol* 1988;105:361–5.
499. Procope JA. Delayed-onset *Pseudomonas* keratitis after radial keratotomy. *J Cataract Refract Surg* 1997;23:1271–2.
500. Sapico FL, Montgomerie JZ. Vertebral osteomyelitis in intravenous drug abusers: report of three cases and review of the literature. *Rev Infect Dis* 1980;2:196–206.
501. Tindel JR, Crowder JG. Septic arthritis due to *Pseudomonas aeruginosa*. *JAMA* 1971;218:559–61.
502. Martone WJ, Tablan OC, Jarvis WR. The epidemiology of nosocomial epidemic *Pseudomonas cepacia* infections. *Eur J Epidemiol* 1987;3:222–32.
503. Goldmann DA, Klinger JD. *Pseudomonas cepacia*: biology, mechanisms of virulence, epidemiology. *J Pediatr* 1986;108:806–12.
504. Widmer AF, Wenzel RP, Trilla A, et al. Outbreak of *Pseudomonas aeruginosa* infections in a surgical intensive care unit: probable transmission via hands of a health care worker. *Clin Infect Dis* 1993;16: 372–6.
505. Döring G, Hörz M, Ortelt J, et al. Molecular epidemiology of *Pseudomonas aeruginosa* in an intensive care unit. *Epidemiol Infect* 1993;110:427–36.
506. Hollyoak V, Allison D, Summers J. *Pseudomonas aeruginosa* wound infection associated with a nursing home whirlpool bath. *CDR Review* 1995;5:R100–2.
507. Grundmann H, Kropec A, Hartung D, Berner R, Daschner F. *Pseudomonas aeruginosa* in a neonatal intensive care unit: reservoirs and ecology of the nosocomial pathogen. *J Infect Dis* 1993;168:943–7.
508. Martino P, Venditti M, Papa G, Orefici G, Serra P. Water supply as a source of *Pseudomonas aeruginosa* in a hospital for hematological malignancies. *Bollettino dell Istituto Sieroterapico Milanese* 1985;64:109–14.
509. Ayliffe GAJ, Babb JR, Collins BJ, Lowbury EJ, Newsom SWB. *Pseudomonas aeruginosa* in hospital sinks. *Lancet* 1974;2:578–81.
510. Kluyver AJ. *Pseudomonas aureofaciens* nov. spec and its pigments. *J Bacteriol* 1956;72:406–11.
511. Romling U, Fiedler B, Bosshammer J, et al. Epidemiology of chronic *Pseudomonas aeruginosa* infections in cystic fibrosis. *J Infect Dis* 1994;170:1616–21.
512. Jones F, Bartlett CL. Infections associated with whirlpools and spas. *Soc Appl Bacteriol Symp Series* 1985;14:61S–6S.
513. Casewell MW, Slater NG, Cooper JE. Operating theater water-baths as a cause of *Pseudomonas* septicemia. *J Hosp Infect* 1981;2:237–47.
514. Rechsteiner J, Landheer JE, de Jong J, van Kregten E, Lindner JG. [Kidney lithotripter as a possible source of hospital infection]. *Nederlands Tijdschrift voor Geneeskunde* 1988;132:1849–59. (Dutch)
515. (308) Taplin D, Mertz PM. Flower vases in hospitals as reservoirs for pathogens. *Lancet* 1973;2: 1279–1281.
516. Kaiser AB. Humidifiers and *Pseudomonas* infections. *N Engl J Med* 1970;283:708.

517. Levin MH, Olson B, Nathan C, et al. *Pseudomonas* in the sinks in an intensive care unit: relation to patients. *J Clin Pathol* 1984;37:424–7.
518. Paszko-Kolva C, Yamamoto H, Shahamat M, Sawyer TK, Morris G, Colwell RR. Isolation of amoebae and *Pseudomonas* and *Legionella* spp. from eyewash stations. *Appl Environ Microbiol* 1991;57:163–7.
519. Struelens MJ, Rost F, Deplano A, et al. *Pseudomonas aeruginosa* and *Enterobacteriaceae* bacteremia after biliary endoscopy: an outbreak investigation using DNA macrorestriction analysis. *Am J Med* 1993;95:489–98.
520. Blanc DS, Parret T, Janin B, Raselli P, Francioli P. Nosocomial infections and pseudoinfections from contaminated bronchoscopes: two-year follow up using molecular markers. *Infect Control Hosp Epidemiol* 1997;18:134–6.
521. Boukadida J, de Montalembert M, Gaillard JL, et al. Outbreak of gut colonization by *Pseudomonas aeruginosa* in immunocompromised children undergoing total digestive decontamination: analysis by pulsed-field electrophoresis. *J Clin Microbiol* 1991;29:2068–71.
522. Grigis A, Goglio A, Parea M, Gnechi F, Minetti B, Barbui T. Nosocomial outbreak of severe *Pseudomonas aeruginosa* infections in haematological patients. *Eur J Epidemiol* 1993;9:390–5.
523. Gupta AK, Shashi S, Mohan M, Lamba IM, Gupta R. Epidemiology of *Pseudomonas aeruginosa* infections in a neonatal intensive care unit. *J Trop Pediatr* 1993;39:32–6.
524. Sader HS, Pignatari AC, Leme IL, et al. Epidemiologic typing of multiply drug-resistant *Pseudomonas aeruginosa* isolated from an outbreak in an intensive care unit. *Diagn Microbiol Infect Dis* 1993;17:13–8.
525. Krecmery V, Trupl J. Nosocomial outbreak of meropenem-resistant *Pseudomonas aeruginosa* infections in a cancer center. *J Hosp Infect* 1994;28:209–18.
526. Jumaa P, Chattopadhyay B. Outbreak of gentamicin, ciprofloxacin-resistant *Pseudomonas aeruginosa* in an intensive care unit, traced to contaminated quivers. *J Hosp Infect* 1994;28:209–18.
527. Carson LA, Favero MS, Bond WW, Petersen NJ. Morphological, biochemical, and growth characteristics of *Pseudomonas cepacia* from distilled water. *Appl Microbiol* 1973;25:476–83.
528. Bassett DC, Stokes KJ, Thomas WR. Wound infection with *Pseudomonas multivorans*: a waterborne contaminant of disinfectant solutions. *Lancet* 1970;1:1188–91.
529. Wishart MM, Riley TV. Infection with *Pseudomonas maltophilia*: hospital outbreak due to contaminated disinfectant. *Med J Aust* 1976;2:710–2.
530. Conly JM, Klass L, Larson L. *Pseudomonas cepacia* colonization and infection in intensive care units. *Can Med Assoc J* 1986;134:363–6.
531. Bosshammer J, Fielder B, Gudowis P, von der Hardt H, Romling U, Tummler B. Comparative hygienic surveillance of contamination with pseudomonads in a cystic fibrosis ward over a 4-year period. *J Hosp Infect* 1995;31:261–74.
532. Hutchinson GR, Parker S, Pryor JA, et al. Home-use nebulizers: a potential primary source of *B. cepacia* and other colistin-resistant, gram-negative bacteria in patients with cystic fibrosis. *J Clin Microbiol* 1996;34:584–7.
533. Pegues DA, Carson LA, Anderson RL, et al. Outbreak of *Pseudomonas cepacia* bacteremia in oncology patients. *Clin Infect Dis* 1993;16:407–11.
534. CDC. Nosocomial *Burkholderia cepacia* infection and colonization with intrinsically contaminated mouthwash — Arizona, 1998. *MMWR* 1998;47:926–8.
535. Berthelot P, Grattard F, Mahul P, et al. Ventilator temperature sensors: an unusual source of *Pseudomonas cepacia* in nosocomial infection. *J Hosp Infect* 1993;25:33–43.
536. Khardori N, Elting L, Wong E, et al. Nosocomial infections due to *Xanthomonas maltophilia* (*Pseudomonas maltophilia*) in patients with cancer. *Rev Infect Dis* 1990;12:997–1003.
537. Oie S, Oomaki M, Yorioka K, et al. Microbial contamination of “sterile water” used in Japanese hospitals. *J Hosp Infect* 1998;38:61–5.
538. Crane LR, Tagle LC, Palutke WA. Outbreak of *Pseudomonas paucimobilis* in an intensive care facility. *JAMA* 1981;246:985–7.
539. Lemaitre D, Elaichouni A, Hundhausen M, et al. Tracheal colonization with *Sphingomonas paucimobilis* in mechanically-ventilated neonates due to contaminated ventilator temperature probes. *J Hosp Infect* 1996;32:199–206.
540. Maki DG, Klein BS, McCormick RD, et al. Nosocomial *Pseudomonas pickettii* bacteremias traced to narcotic tampering. A case for selective drug screening of health care personnel. *JAMA* 1991;265:981–6.
541. Maroye P, Doermann HP, Rogues AM, Gachie JP, Mégraud F. Investigation of an outbreak of *Ralstonia pickettii* in a paediatric hospital by RAPD. *J Hosp Infection* 2000;44:267–72.

542. McNeil MM, Solomon SL, Anderson RL, et al. Nosocomial *Pseudomonas pickettii* colonization associated with a contaminated respiratory therapy solution in a special care nursery. *J Clin Microbiol* 1985;22:903–7.
543. Lamka KG, LeChevallier MW, Seidler RJ. Bacterial contamination of drinking water supplies in a modern rural neighborhood. *Appl Environ Microbiol* 1980;39:734–8.
544. Nakashima AK, McCarthy MA, Martone WJ, Anderson RL. Epidemic septic arthritis caused by *Serratia marcescens* and associated with a benzalkonium chloride antiseptic. *J Clin Microbiol* 1987;25:1014–8.
545. Nakashima AK, Highsmith AK, Martone WJ. Survival of *Serratia marcescens* in benzalkonium chloride and in multiple-dose medication vials: relationship to epidemic septic arthritis. *J Clin Microbiol* 1987;25:1019–21.
546. Bosi C, Davin-Regli A, Charrel R, Rocca B, Monnet D, Bollet C. *Serratia marcescens* nosocomial outbreak due to contamination of hexetidine solution. *J Hosp Infect* 1996;33:217–24.
547. Ehrenkranz NJ, Bolyard EA, Wiener M, Cleary TJ. Antibiotic-sensitive *Serratia marcescens* infections complicating cardiopulmonary operations: contaminated disinfectant as a reservoir. *Lancet* 1980;2:1289–92.
548. Cimolai N, Trombley C, Wensley D, LeBlanc J. Heterogeneous *Serratia marcescens* genotypes from a nosocomial pediatric outbreak. *Chest* 1997;111:194–7.
549. Hartstein AI, Rashad AL, Liebler JM, et al. Multiple intensive care unit outbreaks of *Acinetobacter calcoaceticus* subspecies anitratus respiratory infection and colonization associated with contaminated, reusable ventilator circuits and resuscitation bags. *Am J Med* 1988;85:624–31.
550. Stone JW, Das BC. Investigation of an outbreak of infection with *Acinetobacter calcoaceticus* in a special care baby unit. *J Hosp Infect* 1986;7:42–8.
551. Vandenbroucke-Grauls CMJE, Kerver AJH, Rommes JH, Jansen R, den Dekker C, Verhoef J. Endemic *Acinetobacter anitratus* in a surgical intensive care unit: mechanical ventilators as reservoir. *Eur J Clin Microbiol Infect Dis* 1988;7:485–9.
552. Cefai C, Richards J, Gould FK, McPeake P. An outbreak of *Acinetobacter* respiratory infection resulting from incomplete disinfection of ventilatory equipment. *J Hosp Infect* 1990;15:177–82.
553. Gervich DH, Grout CS. An outbreak of nosocomial *Acinetobacter* infections from humidifiers. *Am J Infect Control* 1985;13:210–5.
554. Castle M, Tenney JH, Weinstein MP, Eickhoff TC. Outbreak of a multiply resistant *Acinetobacter* in a surgical intensive care unit. *Heart Lung* 1978;7:641–4.
555. Smith PW, Massanari RM. Room humidifiers as a source of *Acinetobacter* infections. *JAMA* 1977;237:795–7.
556. Snyderman DR, Maloy MF, Brock SM, Lyons RW, Rubin SJ. Pseudobacteremia: false-positive blood cultures from mist tent contamination. *Am J Epidemiol* 1977;106:154–9.
557. Rosenthal SL. Sources of *Pseudomonas* and *Acinetobacter* species found in human culture materials. *Am J Clin Pathol* 1974;62:807–11.
558. Allen KD, Green HT. Hospital outbreak of multi-resistant *Acinetobacter anitratus*: an airborne mode of spread. *J Hosp Infect* 1987;9:169–75.
559. Crombach WHJ, Dijkshoorn L, van Noort-Klaassen M, Niessen J, van Knippenbert-Gordebeke G. Control of an epidemic spread of multi-resistant *Acinetobacter calcoaceticus* in a hospital. *Intensive Care Med* 1989;15:166–170.
560. Catalano M, Quelle LS, Jeric PE, Di Martino A, Maimone SM. Survival of *Acinetobacter baumannii* on bed rails during an outbreak and during sporadic cases. *J Hosp Infect* 1999;42:27–35.
561. D'Agata EMC, Venkataraman L, DeGirolami P, Samore M. Molecular epidemiology of ceftazidime-resistant gram-negative bacilli on inanimate surfaces and their role in cross-transmission during non-outbreak periods. *J Clin Microbiol* 1999;37:3065–7.
562. Jawad A, Snelling AM, Heritage J, Hawkey PM. Exceptional desiccation tolerance of *Acinetobacter radioresistens*. *J Hosp Infect* 1998;39:235–40.
563. Jawad A, Seifert H, Snelling AM, Heritage J, Hawkey PM. Survival of *Acinetobacter baumannii* on dry surfaces: comparison of outbreak and sporadic isolates. *J Clin Microbiol* 1998;36:1938–41.
564. Getschell-White, SI, Donowitz LG, Groschel DHM. The inanimate environment of an intensive care unit as a potential source of nosocomial bacteria: evidence for long survival of *Acinetobacter calcoaceticus*. *Infect Control Hosp Epidemiol* 1989;10:402–6.
565. Loiwal V, Kumar A, Gupta P, Gomber S, Ramchandran VG. *Enterobacter aerogenes* outbreak in a neonatal intensive care unit. *Pediatr Int* 1999;41:157–61.

566. Matsaniotis NS, Syriopoulou VP, Theodoridou MC, Tzanetou KG, Mostrou GI. *Enterobacter* sepsis in infants and children due to contaminated intravenous fluids. *Infect Control* 1984;5:471-7.
567. Zembrzuska-Sadlowska E. The dangers of infections of the hospitalized patients with the microorganisms present in preparations and in the hospital environment. *Acta Pol Pharm* 1995;52:173-8.
568. Felts SK, Schaffner W, Melly MA, Koenig MG. Sepsis caused by contaminated intravenous fluids. *Ann Intern Med* 1972;77:881-90.
569. Modi N, Damjanovic V, Cooke RW. Outbreak of cephalosporin resistant *Enterobacter cloacae* infection in a neonatal intensive care unit. *Arch Dis Child* 1987;62:148-51.
570. Graham DR, Wu E, Highsmith AK, Ginsburg ML. An outbreak of pseudobacteremia caused by *Enterobacter cloacae* from a phlebotomist's vial of thrombin. *Ann Intern Med* 1981;95:585-8.
571. Andersen BM, Sorlie D, Hotvedt R, et al. Multiply beta-lactam-resistant *Enterobacter cloacae* infections linked to the environmental flora in a unit for cardiothoracic and vascular surgery. *Scand J Infect Dis* 1989;21:181-91.
572. Wisplinghoff H, Perbix W, Seifert H. Risk factors for nosocomial bloodstream infections due to *Acinetobacter baumannii*: a case-control study of adult burn patients. *Clin Infect Dis* 1999;28:59-66.
573. Crowe M, Towner KJ, Humphreys H. Clinical and epidemiological features of an outbreak of *Acinetobacter* infection in an intensive therapy unit. *J Med Microbiol* 1995;43:55-62.
574. National Nosocomial Infections Surveillance (NNIS) Report: Data summary from October 1986-April 1996, issued May 1996. *Am J Infect Control* 1996;24:380-8.
575. Bergogne-Bérézin E, Joly-Guillou ML. Hospital infection with *Acinetobacter* spp.: an increasing problem. *J Hosp Infect* 1991;18 (suppl A):250-5.
576. Fagon JY, Chastre J, Hance AJ, Montravers P, Novara A, Gibert C. Nosocomial pneumonia in ventilated patients: a cohort study evaluating attributable mortality and hospital stay. *Am J Med* 1993;94: 281-8.
577. (143) Seifert H, Strate A, Pulverer G. Nosocomial bacteremia due to *Acinetobacter baumannii*: clinical features, epidemiology, and predictors of mortality. *Medicine* 1995;74:340-9.
578. Cisneros JM, Reyes MJ, Pachón J, et al. Bacteremia due to *Acinetobacter baumannii*: epidemiology, clinical findings, and prognostic features. *Clin Infect Dis* 1996;22:1026-32.
579. Schaberg DR, Culver DH, Gaynes RP. Major trends in the microbial ecology of nosocomial infections. *Am J Med* 1991;91(suppl 3B):72S-5S.
580. Wang CC, Chu ML, Ho LJ, Hwang RC. Analysis of plasmid pattern in pediatric intensive care outbreaks of nosocomial infection due to *Enterobacter cloacae*. *J Hosp Infect* 1991;19:33-40.
581. Acolet D, Ahmet Z, Houang E, Hurley R, Kaufman ME. *Enterobacter cloacae* in a neonatal intensive care unit: account of an outbreak and its relationship to use of third generation cephalosporins. *J Hosp Infect* 1994;28:273-86.
582. Mayhall CG, Lamb VA, Gayle WE Jr, Haynes BW Jr. *Enterobacter cloacae* septicemia in a burn center: epidemiology and control of an outbreak. *J Infect Dis* 1979;139:166-71.
583. John JF Jr, Sharbaugh RJ, Bannister ER. *Enterobacter cloacae*: bacteremia, epidemiology, and antibiotic resistance. *Rev Infect Dis* 1982;4:13-28.
584. McDonald C, Banerjee SN, Jarvis WR, NNIS. Seasonal variation of *Acinetobacter* infections: 1987-1996. *Clin Infect Dis* 1999;29:1133-7.
585. Beck-Sague CM, Jarvis WR, Brook JH, et al. Epidemic bacteremia due to *Acinetobacter baumannii* in five intensive care units. *Am J Epidemiol* 1990;132:723-33.
586. (144) Yu VL. *Serratia marcescens*: historical perspective and clinical review. *N Engl J Med* 1979;300:887-93.
587. Wenger PN, Tokars JI, Brennan P, et al. An outbreak of *Enterobacter hormaechei* infection and colonization in an intensive care nursery. *Clin Infect Dis* 1997;24:1243-4.
588. Buxton AE, Anderson RL, Wedegar D, Atlas E. Nosocomial respiratory tract infection and colonization with *Acinetobacter calcoaceticus*. *Am J Med* 1978;65:507-13.
589. French GL, Casewell MW, Roncoroni AJ, Knight S, Philipps I. A hospital outbreak of antibiotic-resistant *Acinetobacter anitratus*: epidemiology and control. *J Hosp Infect* 1980;1:125-31.
590. Guenter SH, Hendley JO, Wenzel RP. Gram-negative bacilli as nontransient flora on the hands of hospital personnel. *J Clin Microbiol* 1987;25:488-90.
591. Dreyfuss D, Djedaini K, Weber P, et al. Prospective study of nosocomial pneumonia and of patient and circuit colonization during mechanical ventilation with circuit changes every 48 hours versus no change. *Am Rev Respir Dis* 1991;143:738-43.

592. (145) Go SE, Urban C, Burns J, et al. Clinical and molecular epidemiology of *Acinetobacter* infections sensitive only to polymixin B and sublactam. *Lancet* 1994;344:1329–32.
593. Musa EK, Desai N, Casewell MW. The survival of *Acinetobacter calcoaceticus* inoculated on fingertips and on formica. *J Hosp Infect* 1990;15:219–27.
594. Jawad A, Heritage J, Snelling AM, Gascoyne-Binzi DM, Hawkey PM. Influence of relative humidity and suspending menstrua on survival of *Acinetobacter* spp. on dry surfaces. *J Clin Microbiol* 1996;34:2881–7.
595. Mulin B, Talon D, Viel JF, et al. Risk factors for nosocomial colonization with multiresistant *Acinetobacter baumannii*. *Eur J Clin Microbiol Infect Dis* 1995;14:569–76.
596. O'Brien RJ. The epidemiology of nontuberculous mycobacterial disease. *Clin Chest Med* 1989;10:407–18.
597. Böttger EC, Teske A, Kirschner P, et al. Disseminated "*Mycobacterium genavense*" infection in patients with AIDS. *Lancet* 1992;340:76–80.
598. Wallace RJ Jr, Brown BA, Griffith DE. Nosocomial outbreaks/pseudo-outbreaks caused by nontuberculous mycobacteria. *Ann Rev Microbiol* 1998;52:453–90.
599. Chapman JS, Dewlett HJ, Potts WE. Cutaneous reactions to unclassified mycobacterial antigens: a study of children in household contact with patients who excrete unclassified mycobacteria. *Am Rev Respir Dis* 1962;86:547–52.
600. (245) Crow HE, Corpe RF, Smith CE. Is serious pulmonary disease caused by nonphotochromogenic ("atypical") acid-fast mycobacteria communicable? *Dis Chest* 1961;39:372–81.
601. Kuritsky JM, Bullen MG, Broome CV, Silcox VA, Good RC, Wallace, RJ Jr. Sternal wound infections and endocarditis due to organisms of the *Mycobacterium fortuitum* complex. *Ann Intern Med* 1983;98:938–9.
602. Laussucq S, Baltch AL, Smith RP, et al. Nosocomial *Mycobacterium fortuitum* colonization from a contaminated ice machine. *Am Rev Respir Dis* 1988;138:891–4.
603. Panwalker AP, Fuhse E. Nosocomial *Mycobacterium gordonae* pseudo-infection from contaminated ice machines. *Infect Control* 1986;7:67–70.
604. Wallace RJ Jr, Musser JM, Hull SI, et al. Diversity and sources of rapidly growing mycobacteria associated with infections following cardiac surgery. *J Infect Dis* 1989;159:708–16.
605. Burns DN, Wallace RJ Jr, Schultz ME, et al. Nosocomial outbreak of respiratory tract colonization with *Mycobacterium fortuitum*: demonstration of the usefulness of pulsed-field gel electrophoresis in an epidemiologic investigation. *Am Rev Respir Dis* 1991;144:1153–9.
606. Lessing MPA, Walker MM. Fatal pulmonary infection due to *Mycobacterium fortuitum*. *J. Clin Pathol* 1993;46:271–2.
607. (149) Hoy J, Rolston K, Hopfer RL. Pseudoepidemic of *Mycobacterium fortuitum* in bone marrow cultures. *Am J Infect Control* 1987;15:268–71.
608. Lockwood WW, Friedman C, Bus N, Pierson C, Gaynes R. An outbreak of *Mycobacterium terrae* in clinical specimens associated with a hospital potable water supply. *Am Rev Respir Dis* 1989;140:1614–7.
609. Sniadack DH, Ostroff SM, Karlix MA, et al. A nosocomial pseudo-outbreak of *Mycobacterium xenopi* due to a contaminated water supply: lessons in prevention. *Infect Control Hosp Epidemiol* 1993;14:636–41.
610. (148) Cox R, deBorja K, Bach MC. A pseudo-outbreak of *Mycobacterium chelonae* infections related to bronchoscopy. *Infect Control Hosp Epidemiol* 1997;18:136–7.
611. (150) Stine TM, Harris AA, Levin S, Rivera N, Kaplan, RL. A pseudoepidemic due to atypical mycobacteria in a hospital water supply. *JAMA* 1987;258:809–11.
612. (151) Bennett SN, Peterson DE, Johnson DR, Hall WN, Robinson-Dunn B, Dietrich S. Bronchoscopy-associated *Mycobacterium xenopi* pseudo-infections. *Am J Respir Crit Care Med* 1994;150:245–50.
613. Chadha R, Grover M, Sharma A, et al. An outbreak of post-surgical wound infections due to *Mycobacterium abscessus*. *Pediatr Surg Int* 1998;13:406–10.
614. Von Reyn CF, Maslow JN, Barber TW, Falkinham JO III, Arbeit RD. Persistent colonization of potable water as a source of *Mycobacterium avium* infection in AIDS. *Lancet* 1994;343:1137–41.
615. du Moulin GC, Stottmeier KD, Pelletier PA, Tsang AY, Hedley-Whyte J. Concentration of *Mycobacterium avium* by hospital hot water systems. *JAMA* 1988;260:1599–601.
616. Peters M, Müller C, Rüscher-Gerdes S, et al. Isolation of atypical mycobacteria from tap water in hospitals and homes: Is this a possible source of disseminated MAC infection in AIDS patients? *J Infect* 1995;31:39–44.
617. Soto LE, Bobadilla M, Villalobos Y, et al. Post-surgical nasal cellulitis outbreak due to *Mycobacterium chelonae*. *J Hosp Infect* 1991;19:99–106.
618. Wenger JD, Spika JS, Smithwick RW, et al. Outbreak of *Mycobacterium chelonae* infection associated with use of jet injectors. *JAMA* 1990;264:373–6.

619. Safranek TJ, Jarvis WR, Carson LA, et al. *Mycobacterium chelonae* wound infections after plastic surgery employing contaminated gentian violet skin-marking solution. *N Eng J Med* 1987;317:197–201.
620. Gremillion DH, Mursch SB, Lerner CJ. Injection site abscesses caused by *Mycobacterium chelonae*. *Infect Control* 1983;4:25–8.
621. Begg N, O'Mahoney M, Penny P, Richardson AE. *Mycobacterium chelonae* associated with a hospital hydrotherapy pool. *Community Med* 1986;8:348–50.
622. Aubuchon C, Hill JJ Jr, Graham DR. Atypical mycobacterial infection of soft tissue associated with use of a hot tub. A case report. *J Bone Joint Surg* 1986;68-A:766–8.
623. Kirk J, Kaminski GW. *Mycobacterium marinum* infection. *Aust J Dermatol* 1976;17:111–6.
624. Ross BC, Johnson PDR, Oppedisano F, et al. Detection of *Mycobacterium ulcerans* in environmental samples during an outbreak of ulcerative disease. *Appl Environ Microbiol* 1997;63:4135–8.
625. Tokars JI, McNeil MM, Tablan OC, et al. *Mycobacterium gordonae* pseudoinfection associated with a contaminated antimicrobial solution. *J Clin Microbiol* 1990;28:2765–9.
626. Arnow PM, Bakir M, Thompson K, Bova JL. Endemic contamination of clinical specimens by *Mycobacterium gordonae*. *Clin Infect Dis* 2000;31:472–6.
627. Wright EP, Collins CH, Yates MD. *Mycobacterium xenopi* and *Mycobacterium kansasii* in a hospital water supply. *J Hosp Infect* 1985;6:175–8.
628. du Moulin GC, Stottmeier KD. Waterborne mycobacteria: an increasing threat to health. *ASM News* 1986;10:525–9.
629. Engel HWB, Berwald LG. The occurrence of *Mycobacterium kansasii* in tapwater. *Tubercle* 1980;61:21–6.
630. Kubalek I, Mysak J. The prevalence of environmental mycobacteria in drinking water supply systems in a demarcated region in Czech Republic in the period 1984–1989. *Eur J Epidemiol* 1996;12:471–4.
631. Fox C, Smith F, Brogan O, et al. Non-tuberculous mycobacteria in a hospital's piped water supply. *J Hosp Infect* 1992;21:152–4.
632. Aronson T, Holtzman A, Glover N, et al. Comparison of large restriction fragments of *Mycobacterium avium* isolates recovered from AIDS and non-AIDS patients with those of isolates from potable water. *J Clin Microbiol* 1999;37:1008–12.
633. Carson LA, Bland LA, Cusick LB, et al. Prevalence of nontuberculous mycobacteria in water supplies of hemodialysis centers. *Appl Environ Microbiol* 1988;54:3122–5.
634. Carson LA, Petersen NJ, Favero MS, Aguero SM. Growth characteristics of atypical mycobacteria in water and their comparative resistance to disinfectants. *Appl Environ Microbiol* 1978;36:839–46.
635. Taylor RH, Falkinham III JO, Norton CD, LeChevallier MW. Chlorine, chloramine, chlorine dioxide, and ozone susceptibility of *Mycobacterium avium*. *Appl Environ Microbiol* 2000;66:1702–5.
636. Schulze-Röbbecke R, Fischeder R. Mycobacteria in biofilms. *Zbl Hyg* 1989;188:385–90.
637. Schulze-Röbbecke R, Feldmann C, Fischeder R, Janning B, Exner M, Wahl G. Dental units: an environmental study of sources of potentially pathogenic mycobacteria. *Tubercle Lung Dis* 1995;76:318–23.
638. Meisel JL, Perera DR, Meligro C, Rublin CE. Overwhelming watery diarrhea associated with *Cryptosporidium* in an immunosuppressed patient. *Gastroenterology* 1976;70:1156–60.
639. Nime FA, Page DL, Holscher MA, Yardley JH. Acute enterocolitis in a human being infected with the protozoan *Cryptosporidium*. *Gastroenterology* 1976;70:592–8.
640. Goldstein ST, Juranek DD, Ravenholt O, et al. Cryptosporidiosis: an outbreak associated with drinking water despite state-of-the-art treatment. *Ann Intern Med* 1996;124:459–68.
641. Rose JB. Enteric waterborne protozoa: hazard and exposure assessment. In: Craun GF, ed. *Safety of water disinfection: balancing chemical and microbial risks*. Washington, DC: ILSI Press, 1993;115–26.
642. (157) Juranek DD, Addiss D, Bartlett ME, et al. Cryptosporidiosis and public health: workshop report. *J AWWA* 1995;87:69–80.
643. DuPont HL, Chappell CL, Sterling CR, Okhuysen PC, Rose JB, Jakubowski W. The infectivity of *Cryptosporidium parvum* in healthy volunteers. *N Engl J Med* 1995;332:855–9.
644. Okhuysen PC, Chappell CL, Crabb JH, Sterling CR, DuPont HL. Virulence of three distinct *Cryptosporidium parvum* isolates for healthy adults. *J Infect Dis* 1999;180:1275–81.
645. Chappell CL, Okhuysen PC, Sterling CR, Wang C, Jakubowski W, DuPont HL. Infectivity of *Cryptosporidium parvum* in healthy adults with pre-existing anti-*C. parvum* serum immunoglobulin G. *Am J Trop Med* 1999;60:157–64.

646. Meinhardt PL, Casemore DP, Miller KB. Epidemiologic aspects of human cryptosporidiosis and the role of waterborne transmission. *Epidemiol Rev* 1996;18:118–36.
647. Rose JB. Occurrence and significance of *Cryptosporidium* in water. *JAWWA* 1988;80:53–8.
648. Rose JB, Gerba CP, Jakubowski W. Survey of potable water supplies for *Cryptosporidium* and *Giardia*. *Environ Sci Technol* 1991;25:1393–400.
649. LeChevallier MW, Norton WD, Lee RG. *Giardia* and *Cryptosporidium* spp. in filtered drinking water supplies. *Appl Environ Microbiol* 1991;57:2617–21.
650. Mackenzie WR, Hoxie NJ, Proctor ME, et al. A massive outbreak in Milwaukee of *Cryptosporidium* infection transmitted through the public drinking water supply. *N Engl J Med* 1994;331:161–7.
651. Atherton F, Newman CP, Casemore DP. An outbreak of waterborne cryptosporidiosis associated with a public water supply in the UK. *Epidemiol Infect* 1995;115:123–31.
652. Hayes EB, Matte TD, O'Brien TR, et al. Large community outbreak of cryptosporidiosis due to contamination of a filtered public water supply. *N Engl J Med* 1989;320:1372–5.
653. Neill MA, Rice SK, Ahmad NV, Flanigan TP. Cryptosporidiosis: an unrecognized cause of diarrhea in elderly hospitalized patients. *Clin Infect Dis* 1996;22:168–70.
654. Rutala WA, Weber DJ. Water as a reservoir of nosocomial pathogens. *Infect Control Hosp Epidemiol* 1997;18:609–16.
655. Chadwick P. The epidemiological significance of *Pseudomonas aeruginosa* in hospital sinks. *Can J Public Health* 1976;67:323–8.
656. Cordes LG, Wiesenthal AM, Gorman GW, et al. Isolation of *Legionella pneumophila* from hospital shower heads. *Ann Intern Med* 1981;94:195–7.
657. Bollin GE, Plouffe JF, Para MF, Hackman B. Aerosols containing *Legionella pneumophila* generated by shower heads and hot-water faucets. *Appl Environ Microbiol* 1985;50:1128–31.
658. Weber DJ, Rutala WA, Blanchet CN, Jordan M, Gergen MF. Faucet aerators: a source of patient colonization with *Stenotrophomonas maltophilia*. *Am J Infect Control* 1999;27:59–63.
659. Kappstein I, Grundmann H, Hauer T, Niemeyer C. Aerators as a reservoir of *Acinetobacter junii*: an outbreak of bacteraemia in paediatric oncology patients. *J Hosp Infect* 2000;44:27–30.
660. Dennis PJL, Wright AE, Rutter DA, Death JE, Jones BPC. *Legionella pneumophila* in aerosols from shower baths. *J Hyg (Camb)* 1984;93:349–53.
661. (153) American Society of Heating, Refrigerating, and Air-Conditioning Engineers. ASHRAE Guideline 12-2000: minimizing the risk of legionellosis associated with building water systems. Atlanta, GA: ASHRAE, Inc., 2000;1–16.
662. Newsom SWB. Microbiology of hospital toilets. *Lancet* 1972;2:700–3.
663. Gerba CP, Wallis C, Melnick JL. Microbiological hazards of household toilets: droplet production and the fate of residual organisms. *Appl Microbiol* 1975;30:229–37.
664. (152) Hlady WG, Mullen RC, Mintz CS, Shelton BG, Hopkins RS, Daikos GL. Outbreak of Legionnaires' disease linked to a decorative fountain by molecular epidemiology. *Am J Epidemiol* 1993;138:555–62.
665. Rees JC, Allen KD. Holy water — A risk factor for hospital-acquired infection. *J Hosp Infect* 1996;32:51–5.
666. (226) Favero MS, Petersen NJ, Boyer KM, Carson LA, Bond WW. Microbial contamination of renal dialysis systems and associated risks. *ASAIO Trans* 1974;20:175–83.
667. (227) Favero MS, Petersen NJ, Carson LA, Bond WW, Hindman SH. Gram-negative bacteria in hemodialysis systems. *Health Lab Sci* 1975;12:321–34.
668. (228) Favero MS, Petersen NJ. Microbiologic guidelines for hemodialysis systems. *Dialysis Transplant* 1979;6:34–6.
669. Griffiths PA, Babb JR, Bradley CR, Fraise AP. Glutaraldehyde-resistant *Mycobacterium chelonae* from endoscope washer disinfectors. *J Appl Microbiol* 1997;82:519–26.
670. Phillips G, McEwan H, Butler J. Quality of water in washer-disinfectors. *J Hosp Infect* 1995;31:152–4.
671. (259) Cooke RPD, Whymant-Morris A, Umasankar RS, Goddard SV. Bacteria-free water for automatic washer-disinfectors: an impossible dream? *J Hosp Infect* 1998;48:63–5.
672. Humphreys H, Lee JV. Water quality for endoscopy washer-disinfectors. *J Hosp Infect* 1999;42:76–8.
673. Muscarella LF. Are all sterilization processes alike? *AORN J* 1998;67:966–70, 973–6.
674. U.S. Food and Drug Administration. MAUDE database. Available at: www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfMAUDE/search.CFM

675. Alvarado CJ, Stolz SM, Maki DG. Nosocomial infections from contaminated endoscopes: a flawed automated endoscope washer — an investigation using molecular epidemiology. *Am J Med* 1991;91 (Suppl 3b):272–80.
676. Abrutyn E, Goodhart GL, Roos K, Anderson R, Buxton A. *Acinetobacter calcoaceticus* outbreak associated with peritoneal dialysis. *Am J Epidemiol* 1978;107:328–35.
677. Mader JT, Reinartz JA. Peritonitis during peritoneal dialysis — the role of the preheating water bath. *J Chronic Dis* 1978;31:635–41.
678. Kosatsky T, Kleeman J. Superficial and systemic illness related to a hot tub. *Am J Med* 1985;79:10–2.
679. McGuckin MB, Thorpe RJ, Abrutyn E. Hydrotherapy: An outbreak of *Pseudomonas aeruginosa* wound infections related to Hubbard tank treatments. *Arch Phys Med Rehabil* 1981;62:283–5.
680. (243) Koepke GH, Christopher RP. Contamination of whirlpool baths during treatment of infected wounds. *Arch Phys Med Rehabil* 1965;46:261–3.
681. Miller JK, LaForest NT, Hedberg M, Chapman V. Surveillance and control of Hubbard tank bacterial contaminants. *Phys Ther* 1972;50:1482–6.
682. Nelson RM, Reed JR, Kenton DM. Microbiological evaluation of decontamination procedures for hydrotherapy tanks. *Phys Ther* 1972;52:919–23.
683. Page CF. The whirlpool bath and cross-infection. *Arch Phys Med Rehabil* 1954;35:97–8.
684. Newsom SWB. Hospital infection from contaminated ice. *Lancet* 1968;2:620–2.
685. Ravn P, Lundgren JD, Kjaeldgaard P, et al. Nosocomial outbreak of cryptosporidiosis in AIDS patients. *Br Med J* 1991;302:277–80.
686. Bangsberg JM, Uldum S, Jensen JS, Bruun BG. Nosocomial legionellosis in three heart-lung transplant patients: case reports and environmental observations. *Eur J Clin Microbiol Infect Dis* 1995;14:99–104.
687. (246) Stout JE, Yu VL, Muraca P. Isolation of *Legionella pneumophila* from the cold water of hospital ice machines: implications for origin and transmission of the organism. *Infect Control* 1985;6:141–6.
688. Cross DF, Benchimol A, Dimond EG. The faucet aerator — a source of *Pseudomonas* infection. *N Engl J Med* 1966;274:1430–1.
689. *Chryseobacterium (Flavobacterium) meningosepticum* outbreak associated with colonization of water taps in a neonatal intensive care unit. *J Hosp Infect* 2001;47:188–92.
690. Brown DG, Baublis J. Reservoirs of *Pseudomonas* in an intensive care unit for newborn infants: Mechanisms of control. *J Pediatr* 1977;90:453–7.
691. Perryman FA, Flournoy DJ. Prevalence of gentamicin- and amikacin-resistant bacteria in sink drains. *J Clin Microbiol* 1980;12:79–83.
692. Doring G, Horz M, Ortelt J, Grupp H, Wolz C. Molecular epidemiology of *Pseudomonas aeruginosa* in an intensive care unit. *Epidemiol Infect* 1993;110:427–36.
693. Teres D, Schweers P, Bushnell LS, Hedley-Whyte J, Feingold DS. Sources of *Pseudomonas aeruginosa* infection in a respiratory/surgical intensive care unit. *Lancet* 1973;1:415–7.
694. Barbeau J, Tanguay R, Faucher E, et al. Multiparametric analysis of waterline contamination in dental units. *Appl Environ Microbiol* 1996;62:3954–9.
695. Atlas RM, Williams JF, Huntington MK. *Legionella* contamination of dental-unit waters. *Appl Environ Microbiol* 1995;61:1208–13.
696. Fayle SA, Pollard MA. Decontamination of dental unit water systems: a review of current recommendations. *Br Dent J* 1996;181:369–72.
697. Pien FD, Bruce AE. Nosocomial *Ewingella americana* bacteremia in an intensive care unit. *Arch Intern Med* 1986;146:111–2.
698. Stiles GM, Singh L, Imazaki G, Stiles QR. Thermodilution cardiac output studies as a cause of prosthetic valve bacterial endocarditis. *J Thorac Cardiovasc Surg* 1984;88:1035–7.
699. Tyndall RL, Lyle MM, Ironside KS. The presence of free-living amoebae in portable and stationary eye wash stations. *Am Ind Hyg Assoc J* 1987;48:933–4.
700. Bowman EK, Vass AA, Mackowski R, Owen BA, Tyndall RL. Quantitation of free-living amoebae and bacterial populations in eyewash stations relative to flushing frequency. *Am Ind Hyg Assoc J* 1996;57:626–33.
701. Siegman-Igra Y, Shalem A, Berger SA, Livio S, Michaeli D. Should potted plants be removed from hospital wards? *J Hosp Infect* 1986;7:82–5.
702. (309) Kates SG, McGinley KJ, Larson EL, Leyden JJ. Indigenous multiresistant bacteria from flowers in hospital and nonhospital environments. *Am J Infect Control* 1991;19:156–61.

703. Zanetti F, Stampi S, De L, et al. Water characteristics associated with the occurrence of *Legionella pneumophila* in dental units. *Eur J Oral Sci* 2000;108:22–8.
704. Peel MM, Calwell JM, Christopher PJ, Harkness JL, Rouch GJ. *Legionella pneumophila* and water temperatures in Australian hospitals. *Aust NZ J Med* 1985;15:38–41.
705. Groothuis DG, Veenendaal HR, Dijkstra HL. Influence of temperature on the number of *Legionella pneumophila* in hot water systems. *J Appl Bacteriol* 1985;59:529–36.
706. Plouffe JF, Webster LR, Hackman B. Relationship between colonization of a hospital building with *Legionella pneumophila* and hot water temperatures. *Appl Environ Microbiol* 1983;46:769–70.
707. (112) Alary Ma, Joly JR. Factors contributing to the contamination of hospital water distribution systems by Legionellae. *J Infect Dis* 1992;165:565–9.
708. U.K. Health & Safety Executive. The control of legionellosis in hot and cold water systems. Supplement to the control of Legionellosis, including Legionnaires' disease. London: Health & Safety Executive Office, 1998;1–4.
709. (167) Marrie TJ, Haldane D, Bezanson G, Peppard R. Each water outlet is a unique ecologic niche for *Legionella pneumophila*. *Epidemiol Infect* 1992;108:261–70.
710. (154) Snyder MB, Siwicki M, Wireman J, et al. Reduction of *Legionella pneumophila* through heat flushing followed by continuous supplemental chlorination of hospital hot water. *J Infect Dis* 1990;162:127–32.
711. (155) Ezzeddine H, Van Ossel C, Delmee M, Wauters G. *Legionella* spp. in a hospital hot water system: effect of control measures. *J Hosp Infect* 1989;13:121–31.
712. Reichert M. Automatic washers/disinfectors for flexible endoscopes. *Infect Control Hosp Epidemiol* 1991;12:497–9.
713. (45) Joint Commission on Accreditation of Healthcare Organizations. Hospital accreditation standards, 2001: environment of care. Oakbrook Terrace, IL: JCAHO Press, 2001;193–220.
714. (161) Best M, Yu VL, Stout J, Goetz A, Muder RR, Taylor F. *Legionellaceae* in the hospital water supply: epidemiologic link with disease and evaluation of a method for control of nosocomial Legionnaires' disease and Pittsburgh pneumonia. *Lancet* 1983;2:307–10.
715. CDC. Emergency Response Planning and Coordination. Available at: www.cdc.gov/nceh/emergency/emergency.htm
716. McGlown KJ, Fottler MD. The impact of flooding on the delivery of hospital services in the southeastern United States. *Health Care Manage Rev* 1996;21:55–71.
717. Fisher, HL. Emergency evacuation of the Denver Veteran's Administration Medical Center. *Milit Med* 1986;151:154–61.
718. Peters, MS. Hospitals respond to water loss during the midwest floods of 1993: preparedness and improvisation. *J Emerg Med* 1996;14:345–50.
719. (156) Joint Commission on Accreditation of Healthcare Organizations. Comprehensive Accreditation manual for hospitals: the official handbook (CAH00SJ). Oakbrook Terrace, IL: JCAHO Press, 2000.
720. Hargreaves J, Shireley L, Hansen S, et al. Bacterial contamination associated with electronic faucets: a new risk for healthcare facilities. *Infect Control Hosp Epidemiol* 2001;22:202–5.
721. (158) CDC. National surveillance of dialysis-associated diseases in the United States, 1997. Atlanta, GA: Public Health Service, U.S. Department of Health and Human Services, 1998.
722. Stout JE, Best ME, Yu VL. Susceptibility of members of the family *Legionellaceae* to thermal stress: implications for heat eradication methods in water distribution systems. *Appl Environ Microbiol* 1986;52:396–9.
723. Bornstein N, Vieilly C, Nowiki M, Paucod JC, Fleurette J. Epidemiological evidence of legionellosis transmission through domestic hot water supply systems and possibilities of control. *Isr J Med Sci* 1986;13:39–40.
724. (162) Meenhorst PL, Reingold AL, Groothuis DG, et al. Water-related nosocomial pneumonia caused by *Legionella pneumophila* serogroups 1 and 10. *J Infect Dis* 1985;152:356–64.
725. (216) Mandel AS, Sprauer MA, Sniadack DH, Ostroff SM. State regulation in hospital water temperature. *Infect Control Hosp Epidemiol* 1993;14:642–5.
726. (168) Department of Health. The control of *Legionella* in health care premises: a code of practice. London: HMSO, 1991.
727. (169) Helms CM, Massanari RM, Wenzel RP, et al. Legionnaires' disease associated with a hospital water system: a five-year progress report on continuous hyperchlorination. *JAMA* 1988;259:2423–7.

728. (170) Edelstein PH, Whittaker RE, Kreiling RL, Howell, CL. Efficacy of ozone in eradication of *Legionella pneumophila* from hospital plumbing fixtures. *Appl Environ Microbiol* 1982;44:1330–4.
729. (171) Muraca P, Stout JE, Yu, VL. Comparative assessment of chlorine, heat, ozone, and UV light for killing *Legionella pneumophila* within a model plumbing system. *Appl Environ Microbiol* 1987;53:447–53.
730. (172) Domingue EL, Tyndall RL, Mayberry WR, Pancorbo OC. Effects of three oxidizing biocides on *Legionella pneumophila* serogroup 1. *Appl Environ Microbiol* 1988;54:741–7.
731. (173) Landeen LK, Yahya MT, Gerba CP. Efficacy of copper and silver ions and reduced levels of free chlorine in inactivation of *Legionella pneumophila*. *Appl Environ Microbiol* 1989;55:3045–50.
732. (174) Matulonis U, Rosenfeld CS, Shaddock RK. Prevention of *Legionella* infections in bone marrow transplant unit: multifaceted approach to decontamination of a water system. *Infect Control Hosp Epidemiol* 1993;14:571–83.
733. (175) Liu Z, Stout JE, Tedesco L, et al. Controlled evaluation of copper-silver ionization in eradicating *Legionella pneumophila* from a hospital water distribution system. *J Infect Dis* 1994;169:919–22.
734. (176) Margolin AB. Control of microorganisms in source water and drinking water. In: Hurst CJ, Knudsen GR, McInerney MJ, Stetzenback LD, Walter MV, eds. *Manual of environmental microbiology*. Washington, DC: American Society for Microbiology Press, 1997;195–202.
735. (177) Freije MR. *Legionella* control in health care facilities: a guide for minimizing risk. HC Information Resources, Inc. 1996;65–75.
736. (178) Yu-sen E, Lin R, Vidic D, Stout JE, Yu VL. *Legionella* in water distribution systems. *J AWWA* 1998;90:112–21.
737. (179) Biurrun A, Caballero L, Pelaz C, Leon E, Gago A. Treatment of a *Legionella pneumophila*-colonized water distribution system using copper-silver ionization and continuous chlorination. *Infect Control Hosp Epidemiol* 1999;20:426–8.
738. (180) Goetz A, Yu VL. Copper-silver ionization: cautious optimism for *Legionella* disinfection and implications for environmental culturing. *Am J Infect Control* 1997;25:449–51.
739. (181) Stout JE, Lin YS, Goetz AM, Muder RR. Controlling *Legionella* in hospital water systems: experience with the superheat-and-flush method and copper-silver ionization. *Infect Control Hosp Epidemiol* 1998;19:911–4.
740. (182) Walker JT, Mackerness CW, Mallon D, Makin T, Williets T, Keevil CW. Control of *Legionella pneumophila* in a hospital water system by chlorine dioxide. *J Ind Microbiol* 1995;15:384–90.
741. (183) Hambidge A. Reviewing efficacy of alternative water treatment techniques. *Health Estate* 2001;55:23–5.
742. (184) Rohr U, Senger M, Selenka F, Turley R, Wilhelm M. Four years of experience with silver-copper ionization for control of *Legionella* in a German university hospital hot water plumbing system. *Clin Infect Dis* 1999;29:1507–11.
743. (185) Cunliffe DA. Inactivation of *Legionella pneumophila* by monochloramine. *J Appl Bacteriol* 1990;68:453–9.
744. (186) Kirmeyer GJ, Foust GW, Pierson GL, Simmler JJ, LeChevalier MW. Optimizing chloramine treatment. Denver, CO: American Water Works Research Foundation, 1993.
745. (187) Kool JL, Carpenter JC, Fields BS. Effect of monochloramine disinfection of municipal drinking water on risk of nosocomial Legionnaires' disease. *Lancet* 1999;353:272–7.
746. (188) Kool JL, Bergmire-Sweat D, Butler JC, et al. Hospital characteristics associated with colonization of water systems by *Legionella* and risk of nosocomial Legionnaires' disease: a cohort study of 15 hospitals. *Infect Control Hosp Epidemiol* 1999;20:798–805.
747. (213) Yu VL, Beam TR Jr, Lumish RM, et al. Routine culturing for *Legionella* in the hospital environment may be a good idea: a three-hospital prospective study. *Am J Med Sci* 1987;294:97–9.
748. Allegheny County Health Department. Approaches to prevention and control of *Legionella* infection in Allegheny County health care facilities. Pittsburgh, PA: Allegheny County Health Department, 1997;1–13.
749. Goetz AM, Stout JE, Jacobs SL, et al. Nosocomial Legionnaires' disease discovered in community hospitals following cultures of the water system: seek and ye shall find. *Am J Infect Control* 1998;26:8–11.
750. Maryland Department of Health and Mental Hygiene. Report of the Maryland Scientific Working Group to study *Legionella* in the water systems in healthcare institutions. Available at: www.dhmd.state.md.us/html/legionella.htm
751. Yu VL. Nosocomial legionellosis: Current epidemiologic issues. In: Remington JS, Swartz MN, eds. *Current clinical topics in infectious diseases*. New York, NY: McGraw-Hill, 1986;239–53.

752. Vickers RM, Yu VL, Hanna SS. Determinants of *Legionella pneumophila* contamination of water distribution systems: 15-hospital prospective study. *Infect Control* 1987;8:357–63.
753. (214) Tobin JO, Swann RA, Bartlett CLR. Isolation of *Legionella pneumophila* from water systems: methods and preliminary results. *Br Med J* 1981;282:515–7.
754. Marrie TJ, Bezanson G, Fox J, Kuehn R, Haldane D, Birbridge S. Dynamics of *Legionella pneumophila* in the potable water of one floor of a hospital. In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;238–40.
755. Plouffe JF, Para MF, Maher WE, Hackman B, Webster L. Subtypes of *Legionella pneumophila* serogroup 1 associated with different attack rates. *Lancet* 1983;2:649–50.
756. Fraser DW. Sources of legionellosis. In: Thornsberry C, Balows A, Feeley JC, Jakubowski W, eds. *Legionella: Proceedings of the 2nd International Symposium*. Washington, DC: American Society for Microbiology Press, 1994:277–80.
757. Dourmon E, Bibb WF, Rajagopalan P, Desplaces N, McKinney RM. Monoclonal antibody reactivity as a virulence marker for *Legionella pneumophila* serogroup 1 strain. *J Infect Dis* 1992;165:569–73.
758. Brundrett GW. Guides on avoiding Legionnaires' disease. In: *Legionella and building services*. Oxford, UK: Butterworth Heineman, 1992;346–73.
759. (191) Kugler JW, Armitage JO, Helms CM, et al. Nosocomial Legionnaires' disease: occurrence in recipients of bone marrow transplants. *Am J Med* 1983;74:281–8.
760. Lepine LA, Jernigan DB, Butler JC, et al. A recurrent outbreak of nosocomial Legionnaires' disease detected by urinary antigen testing: evidence for long-term colonization of a hospital plumbing system. *Infect Control Hosp Epidemiol* 1998;19:905–10.
761. Barbaree JM. Selecting a subtyping technique for use in investigations of legionellosis epidemics. In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;169–72.
762. Joly JR, McKinney RM, Tobin JO, Bibb WF, Watkins ID, Ramsay D. Development of a standardized subgrouping scheme for *Legionella pneumophila* serogroup 1 using monoclonal antibodies. *J Clin Microbiol* 1986;23:768–71.
763. (209) Schoonmaker D, Helmberger T, Birkhead G. Comparison of ribotyping and restriction enzyme analysis using pulsed-field gel electrophoresis for distinguishing *Legionella pneumophila* isolates obtained during a nosocomial outbreak. *J Clin Microbiol* 1992;30:1491–8.
764. (163) Johnston JM, Latham RH, Meier FA, et al. Nosocomial outbreak of Legionnaires' disease: molecular epidemiology and disease control measures. *Infect Control* 1987;8:53–8.
765. Best MG, Goetz A, Yu VL. Heat eradication measures for control of nosocomial Legionnaires' disease: Implementation, education, and cost analysis. *Infect Control* 1984;12:26–30.
766. (164) Muraca PW, Yu VL, Goetz A. Disinfection of water distribution systems for *Legionella*: a review of application procedures and methodologies. *Infect Control Hosp Epidemiol* 1990;11:79–88.
767. (210) Knirsch CA, Jakob K, Schoonmaker D, et al. An outbreak of *Legionella micdadei* pneumonia in transplant patients: evaluation, molecular epidemiology, and control. *Am J Med* 2000;108:290–5.
768. (211) CDC. Sustained transmission of nosocomial Legionnaires' Disease — Arizona and Ohio. *MMWR* 1997;46:416–21.
769. (218) Patterson WJ, Hay J, Seal DV, McLuckie JC. Colonization of transplant unit water supplies with *Legionella* and protozoa: precautions required to reduce the risk of legionellosis. *J Hosp Infect* 1997;37:7–17.
770. U.S. Department of Labor, Occupational Safety and Health Administration. OSHA technical manual, Section III, Chapter 7. Legionellosis. Available at: www.osha-slc.gov/dts/osta/otm/otm_iii/otm_iii_7.html
771. Rudnick JR, Beck-Sague CM, Anderson RL, Schable B, Miller JM, Jarvis WR. Gram-negative bacteremia in open-heart surgery patients traced to probable tap-water contamination of pressure-monitoring equipment. *Infect Control Hosp Epidemiol* 1996;17:281–5.
772. Miller RP. Cooling towers and evaporative condensers. *Ann Intern Med* 1979;90:667–70.
773. Butler JC, Breiman RF. Legionellosis. In: Evans AS, Brachman PS, eds. *Bacterial infections of humans*, 3rd ed. New York, NY: Plenum Medical, 1998;355–76.
774. Witherell LE, Novick LF, Stone KM, et al. *Legionella* in cooling towers. *J Environ Health* 1986;49:134–9.
775. Cordes LG, Fraser DW, Skaliy P, et al. Legionnaires' disease outbreak at an Atlanta, Georgia country club: evidence for spread from an evaporative condenser. *Am J Epidemiol* 1980;111:425–31.

776. Kaufmann AF, McDade JE, Patton CM, et al. Pontiac fever: isolation of the etiologic agent (*Legionella pneumophila*) and demonstration of its mode of transmission. *Am J Epidemiol* 1981;114:337–47.
777. Morton S, Bartlett CLR, Bibby LF, Hutchinson DM, Dyer JV, Dennis PJ. Outbreak of Legionnaires' disease from a cooling water system in a power station. *Br J Indust Med* 1986;43:630–5.
778. Friedman S, Spitalny K, Barbaree J, Faur Y, McKinney R. Pontiac fever outbreak associated with a cooling tower. *Am J Public Health* 1987;77:568–72.
779. Addiss DG, Davis JP, LaVenture M, Wand PJ, Hutchinson MA, McKinney RM. Community-acquired Legionnaires' disease associated with a cooling tower: evidence for longer-distance transport of *Legionella pneumophila*. *Am J Epidemiol* 1989;130:557–68.
780. Keller DW, Hajjeh R, DeMaria A Jr, et al. Community outbreak of Legionnaires' disease: an investigation confirming the potential for cooling towers to transmit *Legionella* species. *Clin Inf Dis* 1996;22:257–61.
781. Pastoris MC, Ciceroni L, Lo Monaco R, et al. Molecular epidemiology of an outbreak of Legionnaires' disease associated with a cooling tower in Genova-Sestri Ponente, Italy. *Eur J Clin Microbiol Infect Dis* 1997;16:883–92.
782. Brown CM, Nuorti PJ, Breiman RF, et al. A community outbreak of Legionnaires' disease linked to hospital cooling towers: an epidemiological method to calculate dose of exposure. *Inter J Epidemiol* 1999;28:353–9.
783. Broadbent CR. *Legionella* in cooling towers: Practical research, design, treatment, and control guidelines. In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;217–22.
784. (222) Bhopal RS, Barr G. Maintenance of cooling towers following two outbreaks of Legionnaires' disease in a city. *Epidemiol Infect* 1990;104:29–38.
785. CDC. Suggested health and safety guidelines for public spas and hot tubs. Atlanta, GA: Centers for Disease Control, 1985. Publication No. 99–960.
786. (221) World Health Organization. Environmental aspects of the control of Legionellosis, 14th ed. Copenhagen, Denmark: World Health Organization, 1986. Schriftenr Ver Wasser Boden Lufthyg 1993;91:249–52. (German)
787. (223) World Health Organization. Epidemiology, prevention, and control of legionellosis: memorandum from a WHO meeting. *Bull WHO* 1990;68:155–64.
788. Association for the Advancement of Medical Instrumentation. American National Standard Hemodialysis Systems ANSI/AAMI RD5-1981, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 1982.
789. (229) Association for the Advancement of Medical Instrumentation. American National Standard Hemodialysis Systems ANSI/AAMI RD5-1992, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 1993.
790. Association for the Advancement of Medical Instrumentation. Reuse of hemodialyzers ROH-1986, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 1986.
791. (230) Association for the Advancement of Medical Instrumentation. American National Standard Reuse of hemodialyzers ANSI/AAMI RD47-1993, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 1993.
792. (236) Association for the Advancement of Medical Instrumentation. Water treatment equipment for hemodialysis applications. ANSI/AAMI RD62-2001, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 2001.
793. Tokars JI, Miller ER, Alter MJ, Arduino MJ. National surveillance of dialysis associated diseases in the United States, 1997. *Semin Dialysis* 2000;13:75–85.
794. Hindman SH, Carson LA, Petersen NJ, et al. Pyrogenic reactions during hemodialysis caused by extramural endotoxin. *Lancet* 1975;2:732–4.
795. Stamm JE, Engelhard WE, Parson JE. Microbiological study of water softener resins. *Appl Microbiol* 1969;18:376–86.
796. Alter MJ, Favero MS, Miller JK, Coleman BJ, Bland LA. National surveillance of dialysis-associated diseases in the United States, 1988. *ASAIO Trans* 1990;36:107–18.
797. Tokars JI, Alter MJ, Favero MS, Moyer LA, Bland LA. National surveillance of dialysis-associated diseases in the United States, 1990. *ASAIO J* 1993;39:71–80.
798. Tokars JI, Alter MJ, Favero MS, Moyer LA, Bland LA. National surveillance of dialysis-associated diseases in the United States, 1991. *ASAIO J* 1993;39:966–75.

799. Tokars JI, Alter MJ, Favero MS, Moyer LA, Bland LA. National surveillance of dialysis-associated diseases in the United States, 1993. *ASAIO J* 1996;42:219–29.
800. **(231)** Petersen NJ, Boyer KM, Carson LA, Favero MS. Pyrogenic reactions from inadequate disinfection of a dialysis unit distribution system. *Dialysis Transpl* 1978;7:52–7.
801. Gazenfeldt-Gazit E, Elaihou HE. Endotoxin antibodies in patients on maintenance hemodialysis. *Israel J Med Sci* 1969;5:1032–6.
802. Laude-Sharp M, Canoff M, Simard L, Pusineri C, Kazatchkine M, Haeffner-Cavaillon N. Induction of IL-1 during hemodialysis: transmembrane passage of intact endotoxin (LPS). *Kidney Int* 1990;38:1089–94.
803. Arduino MJ, Bland LA, McAllister SK, Favero MS. The effects of endotoxin contaminated dialysate and polysulfone or cellulosic membranes on the release of TNF α during simulated dialysis. *Artif Organs* 1995;19:880–6.
804. Greisman SE, Hornick RB. Comparative pyrogenic reactivity of rabbit and man to bacterial endotoxin. *Proc Soc Exp Biol Med* 1969;131:1154–8.
805. Weary ME, Donohue G, Pearson FC, Story K. Relative potencies of four reference endotoxin standards as measured by the *Limulus* amoebocyte lysate and USP rabbit pyrogen tests. *Appl Environ Microbiol* 1980;40:1148–51.
806. **(239)** Bland LA, Ridgeway MR, Aguerro SM, Carson LA, Favero MS. Potential bacteriologic and endotoxin hazards associated with liquid bicarbonate concentrate. *ASAIO Trans* 1987;33:542–5.
807. **(232)** Dawids SG, Vejlsgaard R. Bacteriological and clinical evaluation of different dialysate delivery systems. *Acta Med Scand* 1976;199:151–5.
808. Favero MS, Alter MJ, Tokars JI, Arduino MJ. Dialysis-associated infections and their control. In: Bennett JV, Brachman PS, eds. *Hospital infections* 4th ed. Philadelphia, PA: Lippincott-Raven, 1998;357–80.
809. **233** Kidd EE. Bacterial contamination of dialyzing fluid of artificial kidney. *Br Med J* 1964;1:880–2.
810. Jones DM, Tobin BM, Harlow GR, et al. Bacteriological studies of the modified kiil dialyzer. *Br Med J* 1970;3:135–7.
811. **(240)** Raji L, Shapiro FL, Michael AF. Endotoxemia in febrile reactions during hemodialysis. *Kidney Int* 1973;4:57–60.
812. Vanholder R, Van Haecke E, Veys N, et al. Endotoxin transfer through dialysis membranes: small versus large-pore membranes. *Nephrol Dial Transplant* 1992;7:333–9.
813. Evans RC, Holmes CJ. In vitro study of the transfer of cytokine-inducing substances across selected high-flux hemodialysis membranes. *Blood Purif* 1991;9:92–101.
814. Lonnemann G, Behme TC, Lenzer B, et al. Permeability of dialyzer membranes to TNF α -inducing substances derived from water bacteria. *Kidney Int* 1992;42:61–8.
815. Ureña P, Herbelin A, Zingraff J, et al. Permeability of cellulosic and non-cellulosic membranes to endotoxin subunits and cytokine production during in-vitro hemodialysis. *Nephrol Dial Transplant*. 1992;7:16–28.
816. **(241)** Bommer J, Becker KP, Urbaschek R. Potential transfer of endotoxin across high-flux polysulfone membranes. *J Am Soc Nephrol* 1996;7:883–8.
817. Yamagami S, Adachi T, Sugimura, T, et al. Detection of endotoxin antibody in long-term dialysis patients. *Int J Artif Organs* 1990;13:205–10.
818. Arduino MJ. CDC investigations of noninfectious outbreaks of adverse events in hemodialysis facilities, 1979–1999. *Semin Dialysis* 2000;13:86–91.
819. Roth V, Jarvis WR. Outbreaks of infection and/or pyrogenic reactions in dialysis patients. *Semin Dialysis* 2000;13:92–100.
820. Gordon SM, Tipple MME, Bland LA, Jarvis WR. Pyrogenic reactions associated with reuse of disposable hollow-fiber hemodialyzers. *JAMA* 1988;260:2077–81.
821. Alter MJ, Tokars JI, Arduino MJ. Nosocomial infections in hemodialysis units — strategies for control. In: Owen WF, Periera BJG, Sayegh MH, eds. *Dialysis and transplantation: a companion to Brenner and Rector's "The Kidney."* Orlando, FL: WB Saunders Company, 1999;337–57.
822. Bernick JJ, Port FK, Favero MS, Brown DG. Bacterial and endotoxin permeability of hemodialysis membranes. *Kidney Int* 1979;16:491–6.
823. Bommer J, Becker KP, Urbaschek R, Ritz E, Urbaschek B. No evidence for endotoxin transfer across high flux polysulfone membranes. *Clin Nephrol* 1987;27:278–82.
824. Schindler R, Lonnemann G, Schaeffer J, et al. The effect of ultrafiltered dialysate on the cellular content of interleukin-1 receptor antagonist in patients on chronic hemodialysis. *Nephron* 1994;68:229–33.

825. Akrum RAE, Frolich M, Gerritsen AF, et al. Improvement of chronic inflammatory state in hemodialysis patients by the use of ultrapure water for dialysate. *J Am Soc Nephrol* 1997;8:226A.
826. Quellhorst E. Methods of Hemodialysis. *Nieren U Hochdruck* 1998;27:35–41.
827. Baz M, Durand C, Ragon A, et al. Using ultrapure water in hemodialysis delays carpal tunnel syndrome. *Int J Artif Organs* 1991;14:681–5.
828. Schwalbe S, Holzhauser M, Schaeffer J, et al. β 2-Microglobulin associated amyloidosis: a vanishing complication of long-term hemodialysis? *Kidney Int* 1997;52:1077–83.
829. (242) Arduino MJ, Favero MS. Microbiologic aspects of hemodialysis: water quality for hemodialysis. AAMI Monograph WQD-1998. Arlington, VA: Association for the Advancement of Medical Instrumentation, 1998.
830. Leyboldt JK, Schmidt B, Gurland, HJ. Measurement of backfiltration rates during hemodialysis with highly permeable membranes. *Blood Purif* 1991;9:74–84.
831. Carson LA, Bland LA, Cusick LB, Collin S, Favero MS, Bolan G. Factors affecting endotoxin levels in fluids associated with hemodialysis procedures. In: Novitsky TJ, Watson SW, eds. Detection of bacterial endotoxins with the Limulus Amoebocyte Lysate Test. New York, NY: Alan R. Liss, 1987;223–4.
832. Anderson RL, Holland BW, Carr JK, Bond WW, Favero MS. Effect of disinfectants on pseudomonads colonized on the interior surface of PVC pipes. *Am J Public Health* 1990;80:17–21.
833. Bland LA, Favero MS. Microbial contamination control strategies for hemodialysis. JCAHO Plant Tech Manage Series 1989;3:30–6.
834. (237) Bland LA. Microbiological and endotoxin assays of hemodialysis fluids. *Adv Renal Replacement Ther* 1995;2:70–9.
835. (238) Arduino MJ, Bland LA, Aguero SM, Carson LA, Ridgeway M, Favero MS. Comparison of microbiologic assay methods for hemodialysis fluids. *J Clin Microbiol* 1991;29:592–4.
836. Association for the Advancement of Medical Instrumentation. American national standard water treatment equipment for hemodialysis applications. ANSI/AAMI RD62-1999. Arlington, VA: Association for the Advancement of Medical Instrumentation, 1999.
837. Arduino MJ. How should dialyzers be reprocessed? *Semin Dialysis* 1998;11:282–4.
838. Jochimsen EM, Frenette C, Delorme M, et al. A cluster of bloodstream infections and pyrogenic reactions among hemodialysis patients traced to dialysis machine waste-handling option units. *Am J Nephrol* 1998;18:485–9.
839. Wang SA, Levine RB, Carson LA, et al. An outbreak of gram-negative bacteremia in hemodialysis patients traced to hemodialysis machine waste drain ports. *Infect Control Hosp Epidemiol* 1999;20:746–51.
840. National Institutes of Health. U.S. Renal Diseases Survey: 1999 Annual Data Report. Bethesda, MD: National Institute of Diabetes, Digestive and Kidney Diseases, Division of Kidney, Urologic, and Hematologic Diseases, 1999.
841. Monsen T, Olofson C, Ronnmark M, Wistrom J. Clonal spread of staphylococci among patients with peritonitis associated with continuous ambulatory peritoneal dialysis. *ASAIO J* 2000;57:613–8.
842. Band JD, Ward JI, Fraser DW, et al. Peritonitis due to a *Mycobacterium chelonae*-like organism associates with intermittent chronic peritoneal dialysis. *J Infect Dis* 1982;145:9–17.
843. Monsen T, Crabtree JH, Siddiqui RA, et al. Dialysis catheter infection related peritonitis: incidence and time dependent risk. *ASAIO J* 1999;45:574–80.
844. Vera G, Lew SQ. *Mycobacterium fortuitum* peritonitis in two patients receiving continuous ambulatory peritoneal dialysis. *Am J Nephrol* 1999;19:586–9.
845. Soriano F, Rodriguez-Tudela JL, Gomez-Garces JL, Velo M. Two possibly related cases of *Mycobacterium fortuitum* peritonitis in continuous ambulatory peritoneal dialysis. *Eur J Clin Microbiol* 1989;8:895–7.
846. Szeto CC, Li PK, Leung CB, Yu AW, Lui SF, Lai NK. *Xanthomonas maltophilia* peritonitis in uremic patients receiving ambulatory peritoneal dialysis. *Am J Kidney Dis* 1997;29:991–5.
847. Panlilio AL, Beck-Sague CM, Siegel JD, et al. Infections and pseudoinfections due to povidone-iodine solution contaminated with *Pseudomonas cepacia*. *Clin Infect Dis* 1992;14:1078–83.
848. Riebel W, Frantz N, Adelstein D, Spanguolo PJ. *Corynebacterium JK*: a cause of nosocomial device-related infection. *Rev Infect Dis* 1986;8:42–9.
849. Radix AE, Bieluch VM, Graeber CW. Peritonitis caused by *Monilia sitophila* in a patient undergoing peritoneal dialysis. *Int J Artif Organs* 1996;19:218–20.
850. Banerjee S, Marwaha RK, Bajwa RP. Fungal peritonitis complicating peritoneal dialysis. *Indian Pediatr* 1995;32:693–7.

851. Bergeson E, Denis R, Cartier P. Peritoneal dialysis: peritonitis and catheter infections. *Annales de Chirurgie* 1996;50:606–12. (French)
852. Troidle L, Kliger AS, Goldie SJ, et al. Continuous peritoneal dialysis-associated peritonitis of nosocomial origin. *Perit Dialysis International* 1996;16:505–10.
853. Smith CA. Reduced incidence of peritonitis by utilizing “flush before fill” in APD. *Adv Perit Dialysis* 1997;13:224–6.
854. Valeri A, Radhakrishnan J, Vernocchi L, Carmichael LD, Stern L. The epidemiology of peritonitis in acute peritoneal dialysis: a comparison between open- and closed drainage systems. *Am J Kidney Dis* 1993;21:300–9.
855. Stamm WE, Colelle JJ, Anderson RL, Dixon RE. Indwelling arterial catheters as a source of nosocomial bacteremia: an outbreak caused by *Flavobacterium* species. *N Engl J Med* 1975;292:1099–102.
856. Schimpff SC. Gram negative bacteremia. *Support Care Cancer* 1993;1:5–18.
857. Graman PS, Quinlan GA, Rank JA. Nosocomial legionellosis traced to contaminated ice. *Infect Control Hosp Epidemiol* 1997;18:637–40.
858. Gahrn-Hansen B, Uldum SA, Schmidt J, Nielsen B, Birkeland SA, Jorgensen KA. [Nosocomial *Legionella pneumophila* infection in a nephrology department]. *Ugeskrift for Laeger* 1995;157:590–4. (German)
859. Wilson IG, Hogg GM, Barr JG. Microbiological quality of ice in hospital and community. *J Hosp Infect* 1997;36:171–80.
860. Spencer RC. The emergence of epidemic, multiple-antibiotic-resistant *Stenotrophomonas (Xanthomonas) maltophilia* and *Burkholderia (Pseudomonas) cepacia*. *J Hosp Infect* 1995;30(suppl):453–64.
861. (248) Cannon RO, Poliner JR, Hirschhorn RB, et al. A multistate outbreak of Norwalk virus gastroenteritis associated with consumption of commercial ice. *J Infect Dis* 1991;164:860–3.
862. (249) Khan AS, Moe CL, Glass RI, et al. Norwalk virus-associated gastroenteritis traced to ice consumption aboard a cruise ship in Hawaii: comparison and application of molecular method-based assays. *J Clin Microbiol* 1994;32:318–22.
863. (244) CDC. Outbreak of viral gastroenteritis — Pennsylvania and Delaware. *MMWR* 1987;36:709–11.
864. Quick R, Paugh K, Addiss D, Kobayashi J, Baron R. Restaurant-associated outbreak of giardiasis. *J Infect Dis* 1992;166:673–6.
865. Hedberg CW, White KE, Johnson JA, et al. An outbreak of *Salmonella enteritidis* infection at a fast food restaurant: implications for foodhandler-associated transmission. *J Infect Dis* 1991;164:1135–40.
866. Burnett IA, Weeks GR, Harris DM. A hospital study of ice-making machines: their bacteriology, design, usage, and upkeep. *J Hosp Infect* 1994;28:305–13.
867. Petersen NJ. Don't culture the ice machines. *Hosp Infect Control* 1982;9:8–9.
868. CDC. Sanitary care and maintenance of ice chests and ice machines. Atlanta, GA: CDC, 1979. Publication No. 00-2384.
869. (247) Manangan LP, Anderson RL, Arduino MJ, Bond WW. Sanitary care and maintenance of ice-storage chests and ice-making machines in healthcare facilities. *Am J Infect Control* 1998;26:111–2.
870. Anonymous. Ice as a source of infection. *CDR Weekly* 1993;3:241.
871. Cardaney CR, Rodeheaver GT, Horowitz JH, Kenney JG, Edlich RF. Influence of hydrotherapy and antiseptic agents on burn wound bacteria contamination. *J Burn Care Rehab* 1985;6:230–2.
872. Gruber RP, Laub DR, Vistnes LM. The effect of hydrotherapy on the clinical course and pH of experimental cutaneous chemical burns. *Plastic Reconstruct Surg* 1975;55:200–4.
873. Mansell RE, Borchardt KA. Disinfecting hydrotherapy equipment. *Arch Phys Med Rehabil* 1974;55:318–20.
874. Hall J, Skevington SM, Maddison PH, Chapman K. A randomized and controlled trial of hydrotherapy in rheumatoid arthritis. *Arthritis Care Res* 1996;9:206–15.
875. Gross A, Cutright DE, Bhaskar SN. Effectiveness of pulsating water jet lavage in treatment of contaminated crush injuries. *Am J Surgery* 1972;124:373–7.
876. Rodeheaver GT, Paltry D, Thacker JG, Edgerton MT, Edlich RF. Wound cleansing by high pressure irrigation. *Surg Gynecol Obstetr* 1975;141:357–62.
877. Saxe A, Goldestein E, Dixon S, Ostrup R. Pulsatile lavage in the management of postoperative wound infections. *Am Surgeon* 1980;46:391–7.
878. Weller K. In search of efficacy and efficiency: an alternative to conventional wound cleansing modalities. *Ostomy/Wound Manage* 1991;37:23–8.
879. Solomon SL. Host factors in whirlpool-associated *Pseudomonas aeruginosa* skin disease. *Infect Control* 1985;6:402–6.

880. Hicks CB, Chulay JD. Bacteremic *Citrobacter freundii* cellulitis associated with tub immersion in a patient with the nephrotic syndrome. *Mil Med* 1988;153:400–1.
881. Mayhall CG, Lamb VA, Gayle WE, Haynes BW. *Enterobacter cloacae* septicemia in a burn center: epidemiology and control of an outbreak. *J Infect Dis* 1979;139:166–71.
882. Marrie TJ, Gass RSR, Yates L. *Legionella pneumophila* in a physiotherapy pool. *Eur J Clin Microbiol* 1987;6:212–3.
883. Havelaar AH, Berwald LG, Groothuis DG, Baas JG. Mycobacteria in semi-public swimming pools and whirlpools. *Ztb Bakteriol Mikrobiol Hyg [B]* 1985;180:505–14.
884. Favero MS. Whirlpool spa-associated infections: are we really in hot water? *Am J Public Health* 1984;74:653–5.
885. Ratnam S, Hogan K, March SB, Butler RW. Whirlpool-associated folliculitis caused by *Pseudomonas aeruginosa*: report of an outbreak and review. *J Clin Microbiol* 1986;23:655–9.
886. Stone HH, Kolb LD. The evolution and spread of gentamicin-resistant *Pseudomonas*. *J Trauma* 1971;11:586–9.
887. Richard P, LeFlock R, Chamoux C, Pannier M, Espaze E, Richet H. *Pseudomonas aeruginosa* outbreak in a burn unit: role of antimicrobials in the emergence of multiply resistant strains. *J Infect Dis* 1994;170:377–83.
888. Berrouane YF, McNutt L-A, Buschelman BJ, et al. Outbreak of severe *Pseudomonas aeruginosa* infections caused by a contaminated drain in a whirlpool bathtub. *Clin Infect Dis* 2000;31:1331–7.
889. (250) Schmidt OW, Cooney MK, Foy HM. Adeno-associated virus in adenovirus type 3 conjunctivitis. *Infect Immun* 1975;11:1362–70.
890. DeJonckheere JF. Hospital hydrotherapy pools treated with ultraviolet light: bad bacteriological quality and presence of thermophilic *Naegleria*. *J Hyg (Lond)* 1982;88:205–14.
891. American Physical Therapy Association. Hydrotherapy/therapeutic pool infection control guidelines. Alexandria, VA: APTA, 1995;112.
892. CDC. Disinfection of hydrotherapy pools and tanks. Atlanta, GA: Centers for Disease Control, Public Health Service, U.S. Department of Health and Human Services, 1974. Publication No. HHS 00-2383.
893. Price D, Ahearn DG. Incidence and persistence of *Pseudomonas aeruginosa* in whirlpools. *J Clin Microbiol* 1988;26:1650–4.
894. (252) White CG. Chemistry of chlorination. In: Handbook of chlorination and alternative disinfectants, 3rd ed. New York, NY: Van Nostrand Reinhold, 1992;184–249.
895. Mayhall CG. Burn patients. In: Pfeiffer J, ed. APIC Text of infection control and epidemiology. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc (APIC), 2000;32.1–32.8.
896. Smith RF, Blasi D, Dayton SL, Chipps DD. Effects of sodium hypochlorite on the microbial flora of burns and normal skin. *J Trauma* 1974;14:938–44.
897. Cardany CR, Rodeheaver GT, Horowitz JH, Kenney JG, Edlich RF. Influence of hydrotherapy and antiseptic agents on burn wound bacterial contamination. *J Burn Care Rehabil* 1985;6:230–2.
898. Steve L, Goodhart P, Alexander J. Hydrotherapy burn treatment: use of chloramine-T against resistant microorganisms. *Arch Phys Med Rehabil* 1979;60:301–3.
899. Golland A. Basic hydrotherapy. *Physiotherapy* 1981;67:258–62.
900. Edlich RF, Becker DG, Phung D, McClelland WA, Day SG. Water treatment of hydrotherapy exercise pools. *J Burn Care Rehabil* 1988;9:9510–5.
901. Penny PT. Hydrotherapy pools of the future — the avoidance of health problems. *J Hosp Infect* 1991;18:535–42.
902. CDC. Swimming pools: safety and disease control through proper design and operation. Atlanta, GA: U.S. Department of Health and Human Services, 1976. Publication No. HHS No. 88–8319.
903. Linneman CC Jr. Nosocomial infections associated with physical therapy, including hydrotherapy. In: Mayhall CG, ed. Hospital epidemiology and infection control, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;931–6.
904. Aspinall ST, Graham R. Two sources of contamination of a hydrotherapy pool by environmental organisms. *J Hosp Infect* 1989;14:285–92.
905. (251) McCandlish R, Renfrew M. Immersion in water during labor and birth: the need for evaluation. *Birth* 1993;20:79–85.
906. Hawkins S. Water vs conventional births: Infection rates compared. *Nursing Times* 1995;91:38–40.

907. Vochem M, Vogt M, Doring G. Sepsis in a newborn due to *Pseudomonas aeruginosa* from a contaminated tub bath. *N Engl J Med* 2001;345:378–9.
908. Eriksson M, Ladfors L, Mattsson LA, Fall O. Warm tub bath during labor: a study of 1385 women with prelabor rupture of the membranes after 34 weeks of gestation. *Acta Obstet Gynaecol Scand* 1996;75:642–4.
909. Rush J, Burlock S, Lambert K, Loosley-Millman M, Hutchinson B, Enkin M. The effects of whirlpool baths in labor: a randomized, controlled trial. *Birth* 1996;23:136–3.
910. Davis BJ. Whirlpool operation and the prevention of infection. *Infect Control* 1985;6:394–7.
911. (253) Muscarella LF. Automatic flexible endoscope reprocessors. *Gastrointest Endosc Clin N Am* 2000;10:245–57.
912. (254) Muscarella LF. Anticipated reliability of liquid chemical sterilants [letter]. *Am J Infect Control* 1998;26:155–6.
913. (255) Muscarella LF. Dejà vu... all over again? The importance of instrument drying [letter]. *Infect Control Hosp Epidemiol* 2000;21:628–9.
914. (256) Gubler JGH, Salfinger M, von Graevenitz A. Pseudoepidemic of nontuberculous mycobacteria due to a contaminated bronchoscope cleaning machine: report of an outbreak and review of the literature. *Chest* 1992;101:1245–9.
915. (257) Fraser VJ, Jones M, Murray PR, Medoff G, Zhang Y, Wallace RJ Jr. Contamination of flexible fiberoptic bronchoscopes with *Mycobacterium chelonae* linked to an automated bronchoscope disinfection machine. *Am Rev Respir Dis* 1992;145:853–5.
916. Maloney S, Welbel S, Daves B, et al. *Mycobacterium abscessus* pseudoinfection traced to an automated endoscope washer: utility of epidemiologic and laboratory investigation. *J Infect Dis* 1994;169:1166–9.
917. Merighi A, Contato E, Scagliarini R, et al. Quality improvement in gastrointestinal endoscopy: microbiologic surveillance of disinfection. *Gastrointest Endosc* 1996;43:457–62.
918. (258) Muscarella LF. Application of environmental sampling to flexible endoscope reprocessing: the importance of monitoring the rinse water. *Infect Control Hosp Epidemiol* 2002;23:285–9.
919. Mitchell DH, Hicks LJ, Chiew R, Montanaro JC, Chen SC. Pseudoepidemic of *Legionella pneumophila* serogroup 6 associated with contaminated bronchoscopes. *J Hosp Infect* 1997;37:19–23.
920. Ido K, Ishino Y, Ota Y, et al. Deficiencies of automatic endoscopic reprocessors: a method to achieve high-grade disinfection of endoscopes. *Gastrointest Endosc* 1996;44:583–6.
921. (260) Allen JJ, Allen MO, Olsen MM, et al. *Pseudomonas* infection of the biliary system resulting from the use of a contaminated endoscope. *Gastroenterology* 1987;92:759–63.
922. Agerton T, Valway S, Gore B, et al. Transmission of a highly drug-resistant strain (Strain W-1) of *Mycobacterium tuberculosis*: community outbreak and nosocomial transmission via a contaminated bronchoscope. *JAMA* 1997;278:1073–7.
923. (261) Michele TM, Cronin WA, Graham NMH, et al. Transmission of *Mycobacterium tuberculosis* by a fiberoptic bronchoscope: identification by DNA fingerprinting. *JAMA* 1997;278:1093–5.
924. Bronowicki J-P, Venard V, Botte C, et al. Patient-to-patient transmission of hepatitis C virus during colonoscopy. *N Engl J Med* 1997;337:237–40.
925. (262) U.S. Food and Drug Administration, CDC. FDA and CDC Public health advisory: infection from endoscopes inadequately reprocessed by an automated endoscope reprocessing system. September 10, 1999. Available at: www.fda.gov/cdrh/safety.html
926. Rey JF. Endoscopic disinfection. A worldwide problem. *J Clin Gastroenterol* 1999;28:291–7.
927. Wang HC, Liaw YS, Yang PC, Kuo SH, Luh KT. A pseudoepidemic of *Mycobacterium chelonae* infection caused by contamination of a fiberoptic bronchoscope suction channel. *Eur Respir J* 1995;8:1259–62.
928. (263) Alvarado CJ, Reichelderfer M. APIC guideline for infection prevention and control in flexible endoscopy. *Am J Infect Control* 2000;28:138–55.
929. Van Klingeren B, Pullen W. Glutaraldehyde resistant mycobacteria from endoscope washers. *J Hosp Infect* 1993;25:147–9.
930. Flournoy DJ, Petrone RL, Voth DW. A pseudo-outbreak of *Methylobacterium mesophilica* isolated from patients undergoing bronchoscopy. *Eur J Clin Microbiol Infect Dis* 1992;11:240–3.
931. Reeves DS, Brown NM. Mycobacterial contamination of fiberoptic bronchoscopes. *J Hosp Infect* 1995;30(suppl):S531–S536.
932. Kelstrup J, Funder-Nielsen T, Theilade J. Microbial aggregate contamination of water lines in dental equipment and its control. *Acta Path Scand* 1977;85:177–83.

933. Challacombe SJ, Fernandes LL. Detecting *Legionella pneumophila* in water systems: a comparison of various dental units. *J Am Dent Assoc* 1995;126:603–8.
934. Singh R, Stine OC, Smith DL, Spitznagel JK Jr, Labib ME, Williams HN. Microbial diversity of biofilms in dental unit water systems. *Appl Environ Microbiol* 2003;69:3412–20.
935. (264) CDC. Statement from the Centers for Disease Control and Prevention (CDC) regarding biofilm and dental unit water quality. Atlanta, GA: U.S. Public Health Service, Department of Health and Human Services, 1999. Available at: www.cdc.gov/nccdphp/oh/ic-fs-biofilm.htm
936. (265) CDC. Recommended infection control practices for dentistry, 1993. *MMWR* 1993;42(No. RR-8):1–12.
937. (268) Bagga BS, Murphy RA, Anderson AW, Punwani I. Contamination of dental unit cooling water with oral microorganisms and its prevention. *J Am Dent Assoc* 1984;109:712–6.
938. Scheid RC, Rosen S, Beck FM. Reduction of CFUs in high-speed handpiece water lines over time. *Clin Prev Dent* 1990;12:9–12.
939. Williams JF, Johnston AM, Johnson B, Huntington MK, Mackenzie CD. Microbial contamination of dental unit waterlines: prevalence, intensity, and microbiological characteristics. *J Am Dent Assoc* 1993;124:59–65.
940. Santiago JI, Huntington MK, Johnston AM, Quinn RS, Williams JF. Microbial contamination of dental unit waterlines: short- and long-term effects of flushing. *Gen Dent* 1994;42:528–35.
941. Williams HN, Johnson A, Kelley JI, et al. Bacterial contamination of the water supply in newly installed dental units. *Quintessence Int* 1995;26:331–7.
942. CDC. Guideline for infection control in dental health-care settings. *MMWR* 2003;52:in press.
943. (266) Office of Safety and Asepsis Procedures Research Foundation. Position paper on dental unit waterlines. Annapolis, MD: OSAPRF, 2000. Available at: www.osap.org/issues/pages/water/duwl.htm
944. (267) U.S. Environmental Protection Agency. National primary drinking water regulations, 40 CFR 1, Part 141, Subpart G;1999. At: www.epa.gov/safewater/mcl.html
945. (344) Eaton AD, Clesceri LS, Greenberg AE, eds. Standard methods for the examination of water and wastewater, 20th ed. Washington, DC: American Public Health Association, 1998;9–1 through 9–41.
946. (269) Shearer BG. Biofilm and the dental office. *J Am Dent Assoc* 1996;127:181–9.
947. Maki DG, Alvarado CJ, Hassemer CA, Zilz MA. Relation of the inanimate hospital environment to endemic nosocomial infection. *N Engl J Med* 1982;307:1562–6.
948. Danforth D, Nicolle LE, Hume K, Alfieri N, Sims H. Nosocomial infections on nursing units with floors cleaned with a disinfectant compared with detergent. *J Hosp Infect* 1987;10:229–35.
949. Spaulding EH. Role of chemical disinfection in the prevention of nosocomial infections. In: Brachman PS, Eickhoff TC, eds. *Proceedings of the International Conference on Nosocomial Infections, 1970*. Chicago, IL: American Hospital Association, 1971;247–54.
950. Spaulding EH. Chemical disinfection and antisepsis in the hospital. *J Hosp Res* 1972;9:5–31.
951. (273) Favero MS, Bond WW. Chemical disinfection of medical and surgical materials. In: Block SS, ed. *Disinfection, sterilization, and preservation, 5th ed.* Philadelphia, PA: Lippincott Williams & Wilkins, 2001;881–917.
952. (274) Rutala WA. APIC guideline for selection and use of disinfectants. *Am J Infect Control* 1996;24:313–42.
953. Agolini G, Russo A, Clementi M. Effect of phenolic and chlorine disinfectants on hepatitis C virus binding and infectivity. *Am J Infect Control* 1999;27:236–9.
954. (279) Favero MS, Bond WW. Sterilization, disinfection, and antisepsis in the hospital. In: Balows A, Hausler WJ Jr, Herrmann KL, Isenberg HD, Shadomy HJ, eds. *Manual of clinical microbiology, 5th ed.* Washington, DC: American Society for Microbiology, 1991;183–200.
955. Nyström B. Bioburden of non-disposable surgical instruments and operating room textiles. In: Gaughran ERL, Morrissey RF, eds. *Sterilization of medical products, Vol II*. Montreal, Québec: Multiscience Publications Ltd., 1981;156–63.
956. Nyström B. Disinfection of surgical instruments. *J Hosp Infect* 1981;2:3636–8.
957. Rutala WA, Weber DJ. FDA labeling requirements for disinfection of endoscopes: a counterpoint. *Infect Control Hosp Epidemiol* 1995;16:231–5.
958. Parker HH IV, Johnson RD. Effectiveness of ethylene oxide for sterilization of dental handpieces. *J Dent* 1995;1:1–3.

959. Alfa MJ, DeGagne P, Olson N, Puchalski T. Comparison of ion plasma, vaporized hydrogen peroxide, and 100% ethylene oxide sterilizers to the 12/88 ethylene oxide gas sterilizer. *Infect Control Hosp Epidemiol* 1996;17:92–100.
960. Rutala WA, Gergen MF, Jones JF, Weber DJ. Levels of microbial contamination on surgical instruments. *Am J Infect Control* 1998;26:143–5.
961. (275) Stingeni L, Lapomarda V, Lisi P. Occupational hand dermatitis in hospital environments. *Contact Dermatitis* 1995;33:172–6.
962. (276) Ashdown BC, Stricof DD, May ML, Sherman SJ, Carmody RF. Hydrogen peroxide poisoning causing brain infarction: neuroimaging findings. *AM J Roentgenol* 1998;170:1653–5.
963. (277) Busch A, Werner E. [Animal tolerance to peracetic acid. 1. Experimental results following the application of peracetic acid solutions on the skin of pigs]. *Monatshefte für Veterinaermedizin* 1974;29:494–8. (German)
964. (278) U.S. Food and Drug Administration. Medical devices: adequate directions for use. 21 CFR Part 801.5, 807.87.e.
965. U.S. Food and Drug Administration (FDA) and U.S. Environmental Protection Agency (EPA). Memorandum of understanding between the Food and Drug Administration, Public Health Service, Department of Health and Human Services, and the Environmental Protection Agency: Notice regarding matters of mutual responsibility — regulation of liquid chemical germicides intended for use on medical devices, 1993. Available from FDA, Center for Devices and Radiological Health (CDRH), Office of Health and Industry Programs, Division of Small Manufacturers Assistance, Rockville MD 20850, or EPA, Registration Division, Antimicrobial Program Branch, 401 M St., SW, Washington DC 20460.
966. U.S. Food and Drug Administration (FDA). Interim measures for the registration of antimicrobial products/liquid chemical germicides with medical device use claims under the memorandum of understanding between EPA and FDA, 1994. CDRH Facts on Demand, Shelf #851, p. 14; June 30, 1994. Available from FDA, CDRH, Office of Health and Industry Programs, Division of Small Manufacturers Assistance, Rockville MD 20850.
967. (293) U.S. Department of Labor, Occupational Safety and Health Administration. Occupational Exposure to Bloodborne Pathogens: final rule (29 CFR 1910.1030). *Federal Register* 1991;56:64004–182.
968. Collins BJ. The hospital environment: how clean should a hospital be? *J Hosp Infect* 1988;11 (Suppl A): 53–6.
969. Van den Berg RWA, Claahsen HL, Niessen M, Muytjens HL, Liem K, Voss A. *Enterobacter cloacae* outbreak in the NICU related to disinfected thermometers. *J Hosp Infect* 2000;45:29–34.
970. Spaulding EH. Alcohol as a surgical disinfectant. *AORN J* 1964;2:67–71.
971. (282) Ayliffe GAJ, Collins BJ, Lowbury EJJ, Babb JR, Lilly HA. Ward floors and other surfaces as reservoirs of hospital infection. *J Hyg (Camb)* 1967;65:515–37.
972. (283) Dancer SJ. Mopping up hospital infection. *J Hosp Infect* 1999;43:85–100.
973. Gable TS. Bactericidal effectiveness of floor cleaning methods in a hospital environment. *Hospitals JAHA* 1966;40:107–11.
974. (271) U.S. Environmental Protection Agency. Federal Insecticide, Fungicide, and Rodenticide Act. 7 USC 6 § 136 et seq.; 1972. Available at: www4.law.cornell.edu/uscode/7/ch6schII.html
975. Petersen NJ, Marshall JH, Collins DE. Why wash walls in hospital isolation rooms? *Health Lab Sci* 1973;10:23–7.
976. (285) Mallison GF. Decontamination, disinfection, and sterilization. *Nurs Clin North Am* 1980;15:757–67.
977. Ayliffe GAJ, Collins BJ, Lowbury EJJ. Cleaning and disinfection of hospital floors. *Br Med J* 1966;2:442–5.
978. Vesley D, Pryor AK, Walter WG, Shaffer JG. A cooperative microbiological evaluation of floor-cleaning procedures in hospital patient rooms. *Health Lab Sci* 1970;7:256–64.
979. Daschner J, Rabbenstein G, Langmaack H. [Flächenkontamination zur Verhütung und Bekämpfung von Drakenhausinfektionen.] *Deutsche Medizinische Wochenschrift* 1980;10:325–9. (German)
980. Dharan S, Mourouga P, Copin P, Bessmer G, Tschanz B, Pittet D. Routine disinfection of patients' environmental surfaces: Myth or reality? *J Hosp Infect* 1999;42:113–7.
981. Palmer PH, Yeoman DM. A study to assess the value of disinfectants when washing ward floors. *Med J Australia* 1972;2:1237–9.
982. (284) Schmidt EA, Coleman DL, Mallison GF. Improved system for floor cleaning in health care facilities. *Appl Environ Microbiol* 1984;47:942–6.

983. (272) Mallison GF. Hospital disinfectants for housekeeping: floors and tables. *Infect Control* 1984;5:537.
984. Vesley D, Klapes NA, Benzow K, Le CT. Microbiological evaluation of wet and dry floor sanitization systems in hospital patient rooms. *Appl Environ Microbiol* 1987;53:1042–5.
985. Werry C, Lawrence JM, Sanderson PJ. Contamination of detergent cleaning solutions during hospital cleaning. *J Hosp Infect* 1988;11:44–9.
986. (280) Chou T. Environmental services. In: APIC Text of Infection Control and Epidemiology. Pfeiffer J, ed. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc.;2000;73.1–73.8.
987. (281) Rutala WA, Weber D. General information on cleaning, disinfection, and sterilization. In: Pfeiffer J, ed. APIC Text of infection control and epidemiology. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc (APIC);2000:55.1–55.6.
988. (286) Walter CW, Kundsinn RB. The floor as a reservoir of hospital infections. *Surg Gynec Obstet* 1960;111:412–22.
989. (287) Scott E, Bloomfield SF. The survival and transfer of microbial contamination via cloths, hands and utensils. *J Appl Bacteriol* 1990;68:271–8.
990. (288) Scott E, Bloomfield SF. Investigations of the effectiveness of detergent washing, drying and chemical disinfection on contamination of cleaning cloths. *J Appl Bacteriol* 1990;68:279–83.
991. Givan KF, Black BL, Williams PF. Multiplication of *Pseudomonas* species in phenolic germicidal detergent solution. *Can J Pub Health* 1971;62:72.
992. Thomas MEM, Piper E, Maurer IM. Contamination of an operating theater by gram-negative bacteria: examination of water supplies, cleaning methods, and wound infections. *J Hyg (Camb)* 1972;70:63–73.
993. Medcraft JW, Hawkins JM, Fletcher BN, Dadswell JV. Potential hazard from spray cleaning of floors in hospital wards. *J Hosp Infect* 1987;9:151–7.
994. (289) Brown DG, Schaltzle K, Gable T. The hospital vacuum cleaner: mechanism for redistributing microbial contaminants. *J Environ Health* 1980;42:192–6.
995. (290) Wysowski DK, Flynt JW, Goldfield M, et al. Epidemic hyperbilirubinemia and use of a phenolic disinfectant detergent. *Pediatrics* 1978;61:165–70.
996. (291) Doan HM, Keith L, Shennan AT. Phenol and neonatal jaundice. *Pediatrics* 1979;64:324–5.
997. (292) American Academy of Pediatrics, American College of Obstetricians and Gynecologists. Infection control. In: Guidelines for perinatal care, 4th ed. Evanston, IL: AAP, ACOG, 1997;269–74.
998. (294) Spire B, Montagnier L, Barré-Sinoussi F, Chermann JC. Inactivation of lymphadenopathy associated virus by chemical disinfectants. *Lancet* 1984;2:899–901.
999. (295) Martin LS, McDougal JS, Loskoski SL. Disinfection and inactivation of the human T lymphotropic virus type-III/lymphadenopathy-associated virus. *J Infect Dis* 1985;152:400–3.
1000. (296) Hanson PJ, Gor D, Jeffries DJ, Collins JV. Chemical inactivation of HIV on surfaces. *Br Med J* 1989;298:862–4.
1001. (297) Bloomfield SF, Smith-Burchnell CA, Dalglish AG. Evaluation of hypochlorite-releasing disinfectants against the human immunodeficiency virus (HIV). *J Hosp Infect* 1990;15:273–8.
1002. (298) Druce JD, Jardine D, Locarnini SA, Birch CJ. Susceptibility of HIV to inactivation by disinfectants and ultraviolet light. *J Hosp Infect* 1995;30:167–80.
1003. (299) Van Bueren J, Simpson RA, Salman H, Farrelly HD, Cookson BD. Inactivation of HIV-1 by chemical disinfectants: sodium hypochlorite. *Epidemiol Infect* 1995;115:567–79.
1004. (300) Prince DL, Prince HN, Thraehart O, et al. Methodological approaches to disinfection of human hepatitis viruses. *J Clin Microbiol* 1993;31:3296–304.
1005. Tabor E, Gerety RJ. A survey of formalin inactivation of hepatitis A virus, hepatitis B virus, and a non-A, non-B hepatitis agent. In: Second International Max von Pettenkofer Symposium on Viral Hepatitis. Munich, Germany: October 1982.
1006. Thraehart O, Kuwert EK, Scheiermann N, et al. Comparison of the morphological alteration and disintegration test (MADT) and the chimpanzee infectivity test for determination of hepatitis B virucidal activity of chemical disinfectants. *Zentralbl Bakteriol Mikrobiol Hyg (B)* 1982;176:472–84.
1007. (303) U.S. Department of Labor, Occupational Safety and Health Administration. EPA-registered disinfectants for HIV/HBV. [Memorandum 2/28/97]. Available at: www.osha-slc.gov/OshDoc/Interp_data/I19970228C.html
1008. U.S. Environmental Protection Agency. Lists A, B, C, D, E, and F: EPA registered disinfectants, sanitizers and sterilants. Available at: www.epa.gov/oppad001/chemregindex.htm

1009. U.S. Environmental Protection Agency. Protocols for testing the efficacy of disinfectants against hepatitis B. *Federal Register* 2000;65:51828–30.
1010. (301) CDC. Recommendations for prevention of HIV transmission in health-care settings. *MMWR* 1987;36(No.2S):1S–18S.
1011. (304) Weber DJ, Barbee SL, Sobsey MD, Rutala WA. The effect of blood on the antiviral activity of sodium hypochlorite, a phenolic, and a quaternary ammonium compound. *Infect Control Hosp Epidemiol* 1999;20:821–7.
1012. (302) Sattar SA, Springthorpe VS. Survival and disinfectant inactivation of the human immunodeficiency virus: a critical review. *Rev Infect Dis* 1991;13:430–47.
1013. (400) CDC, National Institutes of Health. Biosafety in microbiological and biomedical laboratories, 4th ed. Washington, DC: U.S. Government Printing Office, 1999.
1014. Lee R. The advantages of carpets in mental hospitals. *Ment Hosp* 1965;16:324–5.
1015. Simmons D, Reizenstein J, Grant M. Considering carpets in hospital use. *Dimensions* 1982;June:18–21.
1016. Willmott M. The effect of a vinyl floor surface and a carpeted floor surface upon walking in elderly hospital in-patients. *Age Ageing* 1986;15:119–20.
1017. Shaffer J, Key I. The microbiological effects of carpeting on the hospital environment. *Hospitals JAHA* 1966;40:126–39.
1018. Walter W, Stober A. Quantitative and qualitative microbial studies of hospital carpets. *J Environ Health* 1967;30:293–300.
1019. Anderson RL. Biological evaluation of carpeting. *Appl Microbiol* 1969;18:180–7.
1020. Lanese RR, Keller MD, Macpherson CR, Covey RC. A study of microflora on tiled and carpeted surfaces in a hospital nursery. *Am J Public Health* 1973;63:174–8.
1021. Bonde GJ. Bacterial flora of synthetic carpets in hospitals. *Health Lab Sci* 1973;10:308–18.
1022. Rylander R, Myrback K, Verner-Carlson B, Ohrstrom M. Bacteriological investigation of wall-to-wall carpeting. *Am J Public Health* 1974;64:163–8.
1023. (305) Suzuki A, Namba Y, Matsuura M, Horisawa A. Bacterial contamination of floors and other surfaces in operating rooms: a five-year survey. *J Hyg (Camb)* 1984;93:559–66.
1024. Skoutelis AT, Westenfelder GO, Beckerdite M, Phair JP. Hospital carpeting and epidemiology of *Clostridium difficile*. *Am J Infect Control* 1993;22:212–7.
1025. Anderson RL, Mackel DC, Stoler BS, Mallison GF. Carpeting in hospitals: an epidemiological evaluation. *J Clin Microbiol* 1982;15:408–15.
1026. (160) Vesper S, Dearborn DG, Yike I, et al. Evaluation of *Stachybotrys chartarum* in the house of an infant with pulmonary hemorrhage: quantitative assessment before, during, and after remediation. *J Urban Health* 2000;77:68–85.
1027. Bakker PGH, Faogali JL. The effect of carpet on the number of microbes in the hospital environment. *N Zeal Med J* 1977;85:88–92.
1028. (306) Richet H, McNeil M, Pewters W, et al. *Aspergillus flavus* in a bone marrow transplant unit (BMTU): Pseudofungemia traced to hallway carpeting [abstract]. In: Abstracts of the 89th Annual Meeting of the American Society for Microbiology, 1989.
1029. CDC. Respiratory illness associated with carpet cleaning at a hospital clinic — Virginia. *MMWR* 1983;32:378,383–4.
1030. Maley MP. Bacterial threats to new hospitals. *Lancet* 1997;350:223–4.
1031. U.S. Environmental Protection Agency. Office of Toxic Substances. Efficacy data requirements. Supplemental recommendations DIS/TSS-2;1/25/79.
1032. (307) U.S. Department of Labor, Occupational Safety and Health Administration. OSHA Standards Interpretation and Compliance Letters;6/10/94: Decontamination of a plush carpet surface after a spill. Available at: www.osha-slc.gov/OshDoc/Interp_data/I19940610.html
1033. Richards K. New York City: special suites — Memorial Sloan Kettering. *Interiors* 1998;157:56–9.
1034. Noskin BA, Bednarz P, Suriano T, Reiner S, Peterson LR. Persistent contamination of fabric-covered furniture by vancomycin-resistant enterococci: implications for upholstery selection in hospitals. *Am J Infect Control* 2000;28:311–3.
1035. Sanderson PJ, Alshafi KM. Environmental contamination by organisms causing urinary tract infection. *J Hosp Infect* 1995;29: 301–3.
1036. Babe KS Jr, Arlian LG, Confer PD, Kim R. House dust mite (*Dermatophagoides farinae* and *Dermatophagoides pteronyssinus*) prevalence in the rooms and hallways of a tertiary care hospital. *J Allergy Clin Immunol* 1995;95:801–5.

1037. Custovic A, Fletcher A, Pickering CAC, et al. Domestic allergens in public places III: house dust mite, cat, dog, and cockroach allergens in British hospitals. *Clin Exper Allergy* 1998;28:53–9.
1038. Ansorg R, Thomssen R, Stubbe P. *Erwinia* species causing fatal septicemia in a newborn. *Med Microbiol Immunol (Berl)* 1974;159:161–70.
1039. Trust TJ, Bartlett KH. Isolation of *Pseudomonas aeruginosa* and other bacterial species from ornamental aquarium plants. *Appl Environ Microbiol* 1976;31:992–4.
1040. (310) Bartzokas CA, Holley MP, Sharp CA. Bacteria in flower vase water: incidence and significance in general ward practice. *Br J Surg* 1975;62:295–7.
1041. Watson AG, Koons CE. *Pseudomonas* on the chrysanthemums. *Lancet* 1973;2:91.
1042. (311) Siegman-Igra Y, Shalem A, Berger SA, Livio S, Michaeli D. Should potted plants be removed from hospital wards? *J Hosp Infect* 1986;7:82–5.
1043. Rosenzweig AL. Contaminated flower vases. *Lancet* 1973;2:568–9.
1044. Johansen KS, Laursen H, Wilhjelmsen BJ. Flower vases as reservoirs of pathogens. *Lancet* 1974;1:359.
1045. Rogues AM, Quesnel C, Revel P, Saric J, Gachie JP. Potted plants as a potential reservoir of *Fusarium* species. *J Hosp Infect* 1997;35:163–4.
1046. (312) Lass-Flörl C, Rath P, Niederwieser D, et al. *Aspergillus terreus* infections in haematological malignancies: molecular epidemiology suggests association with in-hospital plants. *J Hosp Infect* 2000;46:31–5.
1047. Levine OS, Levine MM. Houseflies (*Musca domestica*) as mechanical vectors of shigellosis. *Rev Infect Dis* 1991;13:688–96.
1048. Šrámová H, Daniel M, Absolonová V, Dědičová D, Jedličková Z, Lhotová H, Petráš P, Subertová V. Epidemiological role of arthropods detectable in health facilities. *J Hosp Infect* 1992;20:281–92.
1049. Tan SW, Yap KL, Lee HL. Mechanical transport of rotavirus by the legs and wings of *Musca domestica* (Diptera: *Muscidae*). *J Med Entomol* 1997;34:527–31.
1050. (313) Burgess NR. Hospital design and cockroach control. *Trans R Soc Trop Med Hyg* 1984;78:293–4.
1051. Ash N, Greenberg B. Vector potential for the German cockroach (Diptera: *Blattellidae*) in dissemination of *Salmonella enteritidis* serotype typhimurium. *J Med Entomol* 1980;17:417–23.
1052. Fotedar R, Banerjee U. Nosocomial fungal infections — study of the possible role of cockroaches (*Blattella germanica*) as vectors. *Acta Trop* 1992;50:339–43.
1053. Rosef O, Kapperud G. House flies (*Musca domestica*) as possible vectors of *Campylobacter fetus* subsp. jejuni. *Appl Environ Microbiol* 1983;45:381–3.
1054. Forsey T, Darougar S. Transmission of chlamydiae by the housefly. *Br J Ophthalmol* 1981;65:147–50.
1055. Grübel P, Hoffman JS, Chong FK, Burstein NA, Mepani C, Cave DR. Vector potential of houseflies (*Musca domestica*) for *Helicobacter pylori*. *J Clin Microbiol* 1997;35:1300–3.
1056. Oothuman P, Jeffery J, Aziz, AHA, Bakar EA, Jegathesan M. Bacterial pathogens isolated from cockroaches trapped from pediatric wards in peninsular Malaysia. *Trans R Soc Trop Med Hyg* 1989;83:133–5.
1057. Beatson SH. Pharaoh's ants as pathogen vectors in hospitals. *Lancet* 1972;1:425–7.
1058. LeGuyader A, Rivault C, Chaperon J. Microbial organisms carried by brown-banded cockroaches in relation to their spatial distribution in a hospital. *Epidemiol Infect* 1989;102:485–92.
1059. Fotedar R, Banerjee U, Shrinivas, Verma A. Cockroaches (*Blattella germanica*) as carriers of microorganisms of medical importance in hospitals. *Epidemiol Infect* 1991;107:181–7.
1060. Fotedar R, Banerjee U, Singh S, Shrinivas, Verma AK. The housefly (*Musca domestica*) as a carrier of pathogenic microorganisms in a hospital environment. *J Hosp Infect* 1992;20:209–15.
1061. Fotedar R, Shrinivas, Banerjee U, Samantray JC, Nayar E, Verma A. Nosocomial infections: cockroaches as possible vectors of drug-resistant *Klebsiella*. *J Hosp Infect* 1991;18:155–9.
1062. Devi SJN, Murray CJ. Cockroaches (*Blatta* and *Periplaneta* species) as reservoirs of drug-resistant salmonellas. *Epidemiol Infect* 1991;107:357–64.
1063. Cotton MF, Wasserman E, Pieper CH, et al. Invasive disease due to extended spectrum beta-lactamase-producing *Klebsiella pneumoniae* in a neonatal unit: the possible role of cockroaches. *J Hosp Infect* 2000;44:13–7.
1064. Baker LF. Pests in hospitals. *J Hosp Infect* 1981;2:5–9.
1065. (316) Allen BW. Excretion of viable tubercle bacilli by *Blatta orientalis* (the oriental cockroach) following ingestion of heat-fixed sputum smears: a laboratory investigation. *Trans R Soc Trop Med Hyg* 1987;81:98–9.

1066. (317) Laszlo A. Technical guide: sputum examination for tuberculosis by direct microscopy in low income countries, 5th ed. Paris, France: International Union Against Tuberculosis and Lung Disease, 2000. Available at: www.iatld.org/html/body_guides.htm
1067. Cohen D, Green M, Block C, et al. Reduction of transmission of shigellosis by control of houseflies (*Musca domestica*). *Lancet* 1991;337:993–7.
1068. Daniel M, Šrámová H, Zálabská E. *Lucilia sericata* (Diptera: Calliphoridae) causing hospital-acquired myiasis of a traumatic wound. *J Hosp Infect* 1994;28:149–52.
1069. Jacobson JA, Kolts RL, Conti M, Burke JP. Hospital-acquired myiasis. *Infect Control* 1980;1:319–20.
1070. Mielke U. Nosocomial myiasis. *J Hosp Infect* 1997;37:1–5.
1071. Sherman RA. Wound myiasis in urban and suburban United States. *Arch Intern Med* 2000;160:2004–14.
1072. (314) Lukin LG. Human cutaneous myiasis in Brisbane: a prospective study. *Med J Aust* 1989;150:237–40.
1073. Watkins M, Wyatt T. A ticklish problem: pest infestation in hospitals. *Prof Nurse* 1989;4:389–92.
1074. Schoninger S. Pest control and extermination in health care facilities. *Prof Sanit Manage* 1978;9:24–7.
1075. (315) Bruesch J. Institutional pest management: current trends. *Exec Housekeep Today* 1994;15:6–12.
1076. Tenover FC. VRSA, VISA, GISA: the dilemma behind the name game. *Clin Microbiol Newsletter* 2000;22:49–53.
1077. CDC. National Nosocomial Infections Surveillance System (NNIS): Semiannual report. December 1999. Available at: www.cdc.gov/ncidod/hip/SURVEILL/NNIS.HTM
1078. Hartstein AI, Mulligan ME. Methicillin-resistant *Staphylococcus aureus*. In: Mayhall CG, ed. Hospital epidemiology and infection control, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;347–64.
1079. Walsh TJ, Vlahov D, Hansen SL, et al. Prospective microbiologic surveillance in control of nosocomial methicillin-resistant *Staphylococcus aureus*. *Infect Control* 1987;8:7–14.
1080. Walsh TJ, Auger F, Tatem BA, Hansen SL, Standford HJ. Novobiocin and rifampin in combination against methicillin-resistant *Staphylococcus aureus*: an in vitro comparison with vancomycin plus rifampin. *J Antimicrob Chemother* 1986;17:75–82.
1081. McNeil MM, Solomon SL. The epidemiology of MRSA. *Antimicrobiol Newsl* 1985;2:49–56.
1082. Oie S, Kamiya A. Survival of methicillin-resistant *Staphylococcus aureus* (MRSA) on naturally contaminated dry mops. *J Hosp Infect* 1996;34:145–9.
1083. Arnow PM, Allyn PA, Nichols EM, Hill DL, Pezzlo M, Bartlett RH. Control of methicillin-resistant *Staphylococcus aureus* in a burn unit: role of nurse staffing. *J Trauma* 1982;22:954–9.
1084. (329) Karanfil LV, Murphy M, Josephson A, et al. A cluster of vancomycin-resistant *Enterococcus faecium* in an intensive care unit. *Infect Control Hosp Epidemiol* 1992;13:195–200.
1085. Handwerker S, Raucher B, Altarac D, et al. Nosocomial outbreak due to *Enterococcus faecium* highly resistant to vancomycin, penicillin, and gentamicin. *Clin Infect Dis* 1993;16:750–5.
1086. Boyle JF, Soumakis SA, Rendo A, et al. Epidemiologic analysis and genotypic characterization of a nosocomial outbreak of vancomycin-resistant enterococci. *J Clin Microbiol* 1993;31:1280–5.
1087. (330) Boyce JM, Opal SM, Chow JW, et al. Outbreak of multidrug-resistant *Enterococcus faecium* with transferable vanB class vancomycin resistance. *J Clin Microbiol* 1994;32:1148–53.
1088. (331) Rhinehart E, Smith NE, Wennerstein C, et al. Rapid dissemination of beta-lactamase-producing, aminoglycoside-resistant *Enterococcus faecalis* among patients and staff on an infant-toddler surgical ward. *N Engl J Med* 1990;323:1814–8.
1089. Crossley K, Landesman B, Zaske D. An outbreak of infections caused by strains of *Staphylococcus aureus* resistant to methicillin and aminoglycosides. II. epidemiologic studies. *J Infect Dis* 1979;139:280–7.
1090. Peacock JE Jr, Marsik FJ, Wenzel RP. Methicillin-resistant *Staphylococcus aureus*: introduction and spread within a hospital. *Ann Intern Med* 1980;93:526–32.
1091. Walsh TJ, Hansen SL, Tatem BA, Auger F, Standford HJ. Activity of novobiocin against methicillin-resistant *Staphylococcus aureus*. *J Antimicrob Chemother* 1985;15:435–40.
1092. (332) Livornese LL Jr, Sias S, Samel C, et al. Hospital-acquired infection with vancomycin-resistant *Enterococcus faecium* transmitted by electronic thermometers. *Ann Intern Med* 1992;117:112–6.
1093. Gould FK, Freeman R. Nosocomial infection with microsphere beds. *Lancet* 1993;342:241–2.

1094. Morris JG, Shay DK, Hebden JN, et al. *Enterococci* resistant to multiple antimicrobial agents, including vancomycin: establishment of endemicity in a university medical center. *Ann Intern Med* 1995;123:250–9.
1095. Edmond MB, Ober JS, Weinbaum DL, et al. Vancomycin-resistant *Enterococcus faecium* bacteremia: risk factors for infection. *Clin Infect Dis* 1995;20:1126–33.
1096. (333) Zervos MJ, Kauffman CA, Therasse PM, Bergman AG, Mikesell TS, Schaberg DR. Nosocomial infection by gentamicin-resistant *Streptococcus faecalis*: an epidemiologic study. *Ann Intern Med* 1987;106:687–91.
1097. Zervos MJ, Dembinski S, Mikesell T, Schaberg DR. High-level resistance to gentamicin in *Streptococcus faecalis*: risk factors and evidence for exogenous acquisition of infection. *J Infect Dis* 1986;153: 1075–83.
1098. Bonilla HF, Zervos MA, Lyons MJ, et al. Colonization with vancomycin-resistant *Enterococcus faecium*: comparison of a long-term-care unit with an acute-care hospital. *Infect Control Hosp Epidemiol* 1997;18:333–9.
1099. Bonilla HF, Zervos MJ, Kauffman CA. Long-term survival of vancomycin-resistant *Enterococcus faecium* on a contaminated surface. *Infect Control Hosp Epidemiol* 1996;17:770–1.
1100. Boyce JM, Bermel LA, Zervos MJ, et al. Controlling vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 1995;16:634–7.
1101. Boyce JM, Potter-Bynoe G, Chenevert C, King T. Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. *Infect Control Hosp Epidemiol* 1997;18:622–7.
1102. (328) Layton MC, Perez M, Heald P, Patterson JE. An outbreak of mupirocin-resistant *Staphylococcus aureus* on a dermatology ward associated with an environmental reservoir. *Infect Control Hosp Epidemiol* 1993;14:369–75.
1103. Collins SM, Hacek DM, Degen LA, Wright MP, Noskin GA, Peterson LR. Contamination of the clinical microbiology laboratory with vancomycin-resistant enterococci and multidrug-resistant *Enterobacteriaceae*: implications for hospital and laboratory workers. *J Clin Microbiol* 2001;39:3772–4.
1104. Bonten MJM, Hayden MK, Nathan C, et al. Epidemiology of colonisation of patients and environment with vancomycin-resistant enterococci. *Lancet* 1996;348:1615–9.
1105. Wendt C, Wiesenthal B, Dietz E, Rüden H. Survival of vancomycin-resistant and vancomycin-susceptible enterococci on dry surfaces. *J Clin Microbiol* 1998;36:3734–46.
1106. (323) Bradley CR, Fraise AP. Heat and chemical resistance of enterococci. *J Hosp Infect* 1996;34:191–6.
1107. (324) Anderson RL, Carr JH, Bond WW, Favero MS. Susceptibility of vancomycin-resistant enterococci to environmental disinfectants. *Infect Control Hosp Epidemiol* 1997;18:195–9.
1108. (325) Saurina G, Landman D, Quale JM. Activity of disinfectants against vancomycin-resistant *Enterococcus faecium*. *Infect Control Hosp Epidemiol* 1997;18:345–7.
1109. (326) Rutala WA, Stiegel MM, Sarubbi FA, Weber DJ. Susceptibility of antibiotic-susceptible and antibiotic-resistant hospital bacteria to disinfectants. *Infect Control Hosp Epidemiol* 1997;18:417–21.
1110. (327) Schulster LM, Anderson RL. Susceptibility of glycopeptide-intermediate resistant *Staphylococcus aureus* (GISA) to surface disinfectants, hand washing chemicals, and a skin antiseptic [abstract Y-3]. In: Abstracts of the 98th General Meeting, American Society for Microbiology. 1998.
1111. Armstrong-Evans M, Litt M, McArthur MA, et al. Control of transmission of vancomycin-resistant *Enterococcus faecium* in a long-term-care facility. *Infect Control Hosp Epidemiol* 1999;20:312–17.
1112. Global Consensus Conference: Final Recommendations. Global Consensus Conference on Infection Control Issues Related to Antimicrobial Resistance. *Am J Infect Control* 1999;27:503–13.
1113. Freeman R, Kearns AM, Lightfoot NF. Heat resistance of nosocomial enterococci. *Lancet* 1994;345:64–5.
1114. CDC. *Staphylococcus aureus* resistant to vancomycin — United States. *MMWR* 2002;51:565–7.
1115. CDC. Vancomycin resistant *Staphylococcus aureus* — Pennsylvania, 2002. *MMWR* 2002;51:902.
1116. (320) Weber DJ, Rutala WA. Role of environmental contamination in the transmission of vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 1997;18:306–9.
1117. (321) Lai KK, Kelley AL, Melvin ZS, Belliveau PP, Fontecchio SA. Failure to eradicate vancomycin-resistant enterococci in a university hospital and the cost of barrier precautions. *Infect Control Hosp Epidemiol* 1998;19:647–2.

1118. (322) Byers KE, Durbin LJ, Simonton BM, Anglim AM, Adal KA, Farr BM. Disinfection of hospital rooms contaminated with vancomycin-resistant *Enterococcus faecium*. *Infect Control Hosp Epidemiol* 1998;19:261–4.
1119. Siegel DL, Edelstein PH, Nachamkin I. Inappropriate testing for diarrheal diseases in the hospital. *JAMA* 1990;263:979–82.
1120. Yannelli B, Gurevich I, Schoch PE, Cunha BA. Yield of stool cultures, ova and parasite tests, and *Clostridium difficile* determination in nosocomial diarrhea. *Am J Infect Control* 1988;16:246–9.
1121. Gerding DN, Olson MM, Peterson LR, et al. *Clostridium difficile*-associated diarrhea and colitis in adults: a prospective case-controlled epidemiologic study. *Arch Intern Med* 1986;146:95–100.
1122. Svenungsson B, Burman LG, Jalakas-Pörnnull K, Lagergren Å, Struwe J, Åkerlund T. Epidemiology and molecular characterization of *Clostridium difficile* strains from patients with diarrhea: low disease incidence and evidence of limited cross-infection in a Swedish teaching hospital. *J Clin Microbiol* 2003;41:4031–7.
1123. Barlett JG. Antibiotic-associated colitis. *Dis Mon* 1984;30:1–55.
1124. Pierce PF Jr, Wilson R, Silva J Jr, et al. Antibiotic-associated pseudomembranous colitis: an epidemiologic investigation of a cluster of cases. *J Infect Dis* 1982;145:269–74.
1125. Aronsson B, Möllby, Nord C-E. Antimicrobial agents and *Clostridium difficile* in acute enteric disease: epidemiologic data from Sweden, 1980–1982. *J Infect Dis* 1985;151:476–81.
1126. Thibault A, Miller MA, Gaese C. Risk factors for the development of *Clostridium difficile*-associated diarrhea during a hospital outbreak. *Infect Control Hosp Epidemiol* 1991;12:345–8.
1127. McFarland LV, Surawicz CM, Stamm WE. Risk factors for *Clostridium difficile* carriage and *Clostridium difficile*-associated diarrhea in a cohort of hospitalized patients. *J Infect Dis* 1990;162:678–84.
1128. Zadik PM, Moore AP. Antimicrobial associations of an outbreak of diarrhoea due to *Clostridium difficile*. *J Hosp Infect* 1998;39:189–93.
1129. Johnson S, Homann SR, Bettin KM, et al. Treatment of asymptomatic *Clostridium difficile* carriers (fecal excretors) with vancomycin or metronidazole: a randomized, placebo controlled trial. *Ann Intern Med* 1992;117:297–302.
1130. (319) Gerding DN, Johnson S, Peterson LR, Mulligan ME, Silva J Jr. *Clostridium difficile*-associated diarrhea and colitis. *Infect Control Hosp Epidemiol* 1995;16:459–77.
1131. Titov L, Lebedkova N, Shabanov A, Tang YJ, Cohen SH, Silva J Jr. Isolation and molecular characterization of *Clostridium difficile* strains from patients and the hospital environment in Belarus. *J Clin Microbiol* 2000;38:1200–2.
1132. Mulligan ME, Rolfe RD, Finegold SM, George WL. Contamination of a hospital environment by *Clostridium difficile*. *Curr Microbiol* 1979;3:173–5.
1133. Fekety R, Kim KH, Brown D, Batts DH, Cudmore M, Silva J Jr. Epidemiology of antibiotic-associated colitis: isolation of *Clostridium difficile* from the hospital environment. *Am J Med* 1981;70:906–8.
1134. Malamou-Ladas H, Farrell SO, Nash JO, Tabaqchali S. Isolation of *Clostridium difficile* from patients and the environment of hospital wards. *J Clin Pathol* 1983;6:88–92.
1135. Kaatz GW, Gitlin SD, Schaberg DR, et al. Acquisition of *Clostridium difficile* from the hospital environment. *Am J Epidemiol* 1988;127:1289–94.
1136. Cohen SH, Tang YJ, Muenzer J, Gumerlock PH, Silva J Jr. Isolation of various genotypes of *Clostridium difficile* from patients and the environment in an oncology ward. *J Infect Dis* 1997;889–93.
1137. Savage AM. Nosocomial spread of *Clostridium difficile*. *Infect Control* 1983;4:31–3.
1138. Brooks SE, Veal RO, Kramer M, Dore L, Schupf N, Adachi M. Reduction in the incidence of *Clostridium difficile*-associated diarrhea in an acute care hospital and a skilled nursing facility following replacement of electronic thermometers with single-use disposables. *Infect Control Hosp Epidemiol* 1992;13:98–103.
1139. Johnson S, Gerding DN, Olson MM, et al. Prospective, controlled study of vinyl glove use to interrupt *Clostridium difficile* nosocomial transmission. *Am J Med* 1990;88:137–40.
1140. McFarland LV, Mulligan ME, Kwok RYY, Stamm WE. Nosocomial acquisition of *Clostridium difficile* infection. *New Engl J Med* 1989;320:204–10.
1141. Mayfield JL, Leet T, Miller J, Mundy LM. Environmental control to reduce transmission of *Clostridium difficile*. *Clin Inf Dis* 2000;31:995–1000.

1142. Wilcox MH, Fawley WN, Wigglesworth N, Parnell P, Verity P, Freeman J. Comparison of the effect of detergent versus hypochlorite cleaning on environmental contamination and incidence of *Clostridium difficile* infection. *J Hosp Infect* 2003;54:109–14.
1143. (334) Worsley MA. Infection control and prevention of *Clostridium difficile* infection. *J Antimicrobial Chemother* 1998;41 (Suppl. C):59–66.
1144. von Rheinbaben F, Schünemann S, Groß T, Wolff MH. Transmission of viruses via contact in a household setting: experiments using bacteriophage ϕ X174 as a model virus. *J Hosp Infect* 2000;46:61–6.
1145. Hall CB, Douglas G Jr, Gelman JM. Possible transmission by fomites of respiratory syncytial virus. *J Infect Dis* 1980;141:98–102.
1146. Brady MT, Evans J, Cuartas J. Survival and disinfection of parainfluenza viruses on environmental surfaces. *Am J Infect Control* 1990;18:18–23.
1147. Hendley JO, Wenzel RP, Gwaltney JM Jr. Transmission of rhinovirus colds by self-inoculation. *N Engl J Med* 1973;288:1361–4.
1148. Butz AM, Fosarelli P, Dick J, Cusack T, Yolken R. Prevalence of rotavirus on high-risk fomites in day-care facilities. *Pediatrics* 1993;92:202–5.
1149. Wilde J, Van R, Pickering LK, Eiden J, Yolken R. Detection of rotaviruses in the day care environment — detection by reverse transcriptase polymerase chain reaction. *J Infect Dis* 1992;166:507–11.
1150. Chapin M, Yatabe J, Cherry JD. An outbreak of rotavirus gastroenteritis on a pediatric unit. *Am J Infect Control* 1983;11:88–91.
1151. Appleton H, Higgins PG. Viruses and gastroenteritis in infants. *Lancet* 1975;i:1297.
1152. Abad FX, Villena C, Guix S, Caballero S, Pintó RM, Bosch A. Potential role of fomites in the vehicular transmission of human astroviruses. *Appl Environ Microbiol* 2001;67:3904–7.
1153. Chadwick PR, Beards G, Brown D, et al. Management of hospital outbreaks of gastro-enteritis due to small round structured viruses. Report of the Public Health Laboratory Service, Viral Gastroenteritis Working Group. *J Hosp Infect* 2000;45:1–10.
1154. Spender QW, Lewis D, Price EH. Norwalk-like viruses: study of an outbreak. *Arch Dis Child* 1986;61:142–7.
1155. Storr J, Rice S, Phillips AD, Price E, Walker Smith JA. Clinical associations of Norwalk-like virus in the stools of children. *J Pediatr Gastroenterol Nutr* 1986;5:576–80.
1156. Russo PL, Spelman DW, Harrington GA, et al. Hospital outbreak of Norwalk-like virus. *Infect Control Hosp Epidemiol* 1997;17:1374–8.
1157. Springthorpe VS, Grenier JL, Lloyd-Evans N, Sattar SA. Chemical disinfection of human rotaviruses: efficacy of commercially-available products in suspension tests. *J Hyg (Camb)* 1986;97:139–61.
1158. (335) Lloyd-Evans N, Springthorpe VS, Sattar SA. Chemical disinfection of human rotavirus-contaminated inanimate surfaces. *J Hyg (Camb)* 1986;97:163–73.
1159. CDC. Interim recommendations for cleaning and disinfection of the SARS patient environment. At: www.cdc.gov/ncidod/sars/cleaningpatientenviro.htm
1160. Brown P, Gajdusek DC. The human spongiform encephalopathies: Kuru, Creutzfeldt-Jakob disease, and Gerstmann-Sträussler-Scheinker syndrome. *Curr Top Microbiol Immunol* 1991;172:1–20.
1161. Will RG. Epidemiology of Creutzfeldt-Jakob disease. *Br Med Bull* 1993;49:960–70.
1162. Holman RC, Khan AS, Belay ED, Schonberger LB. Creutzfeldt-Jakob disease in the United States, 1979–1994: using national mortality data to assess the possible occurrence of variant cases. *Emerg Infect Dis* 1996;2:333–7.
1163. Will RG, Ironside JW, Zeidler M, et al. A new variant of Creutzfeldt-Jakob disease in the U.K. *Lancet* 1996;347:921–5.
1164. Lasmézas CI, Deslys JP, Demaimay R, et al. BSE transmission to macaques. *Nature* 1996;381:743–4.
1165. Collinge J, Sidle KCL, Heads J, Ironside J, Hill AF. Molecular analysis of prion strain variation and the aetiology of “new variant” CJD. *Nature* 1996;383:685–90.
1166. Bruce ME, Will RG, Ironside JW, et al. Transmission to mice indicates that “new variant” CJD is caused by the BSE agent. *Nature* 1997;389:498–501.
1167. Prusiner SB. Biology and genetics of prion diseases. *Ann Rev Microbiol* 1994;48:655–86.
1168. Prusiner SB. Human prion diseases. In: Zuckerman AJ, Banatvala JE, Pattison JR, eds. Principles and practice of clinical virology, 3rd ed. Chichester, UK: John Wiley & Sons, 1995;703–29.
1169. Prusiner SB. Prions. *Proc Natl Acad Sci USA* 1998;95:13363–83.

1170. (337) Kimberlin RH, Walker CA, Millson GC, et al. Disinfection studies with two strains of mouse-passaged scrapie agent. Guidelines for Creutzfeldt-Jakob and related agents. *J Neurol Sci* 1983;59:349–55.
1171. Sklaviadis TK, Manuelidis L, Manuelidis EE. Physical properties of the Creutzfeldt-Jakob disease agent. *J Virol* 1989;63:1212–22.
1172. Brown P, Gajdusek DC, Gibbs CJ Jr, Asher DM. Potential epidemic of Creutzfeldt-Jakob disease from human growth hormone therapy. *N Engl J Med* 1985;12:728–33.
1173. Brown P, Preece MA, Will RG. “Friendly fire” in medicine: hormones, homografts and Creutzfeldt-Jakob disease. *Lancet* 1992;340:24–7. Brown P, Preece MA, Will RG. “Friendly fire” in medicine: hormones, homografts and Creutzfeldt-Jakob disease. *Lancet* 1992;340:24–7.
1174. Frasier D, Foley TP Jr. Creutzfeldt-Jakob disease in recipients of pituitary hormones. *J Clin Endocrinol Metabol* 1994;78:1277–9.
1175. Centers for Disease Control. Epidemiologic notes and reports: rapidly progressive dementia in a patient who received a cadaveric dura mater graft. *MMWR* 1987;36:49–50, 55.
1176. Centers for Disease Control. Epidemiologic notes and reports update: Creutzfeldt-Jakob disease in a patient receiving cadaveric dura mater graft. *MMWR* 1987;36:324–5.
1177. Centers for Disease Control. Epidemiologic notes and reports update: Creutzfeldt-Jakob disease in a second patient who received a cadaveric dura mater graft. *MMWR* 1989;38:37–8, 43.
1178. CDC. Creutzfeldt-Jakob disease in patients who received a cadaveric dura mater graft — Spain, 1985–1992. *MMWR* 1993;42:560–3.
1179. Martinez-Lage JF, Poza M, Sola J, et al. Accidental transmission of Creutzfeldt-Jakob disease by dural cadaveric grafts. *J Neurol Neurosurg Psychiatry* 1994;57:1091–4.
1180. CDC. Creutzfeldt-Jakob disease associated with cadaveric dura mater grafts — Japan, January 1979–May 1997. *MMWR* 1997;46:1066–9.
1181. Lang CLG, Heckmann JG, Neundörfer B. Creutzfeldt-Jakob disease via dural and corneal transplants. *J Neurol Sci* 1998;160:128–39.
1182. Nevin S, McMenemey WH, Behrman S, Jones DP. Subacute spongiform encephalopathy — a subacute form of encephalopathy attributable to vascular dysfunction (spongiform cerebral atrophy). *Brain* 1960;83:519–69.
1183. Bernoulli C, Siegfried J, Baumgartner G, et al. Danger of accidental person-to-person transmission of Creutzfeldt-Jakob disease by surgery. *Lancet* 1977;1:478–9.
1184. Will RG, Matthews WB. Evidence for case-to-case transmission of Creutzfeldt-Jakob disease. *J Neurol Neurosurg Psychiatry* 1982;45:235–8.
1185. El Hachimi KH, Chaunu M-P, Cervenakova L, Brown P, Foncin J-F. Putative neurosurgical transmission of Creutzfeldt-Jakob disease with analysis of donor and recipient: agent strains. *Comp Rendus Acad Sci Iii: Science de la vie* 1997;320:319–28.
1186. Brown P, Gibbs CJ, Amyx HL, et al. Chemical disinfection of Creutzfeldt-Jakob disease virus. *N Engl J Med* 1982;306:1279–82.
1187. Brown P, Rohwer RG, Gajdusek DC. Newer data on the inactivation of scrapie virus or Creutzfeldt-Jakob disease virus in brain tissue. *J Infect Dis* 1986;153:1145–8.
1188. (338) Rosenberg RN, White CL, Brown P, Gajdusek DC, Volpe JJ, Dyck PJ. Precautions in handling tissues, fluids, and other contaminated materials from patients with documented or suspected Creutzfeldt-Jakob disease. *Ann Neurol* 1986;19:75–7.
1189. Taylor DM. Resistance of the ME7 scrapie agent to peracetic acid. *Vet Microbiol* 1991;27:19–24.
1190. Taguchi F, Tamai Y, Uchida K, et al. Proposal for a procedure for complete inactivation of the Creutzfeldt-Jakob disease agent. *Arch Virol* 1991;119:297–301.
1191. (339) Taylor D. Inactivation of the unconventional agents of scrapie, bovine spongiform encephalopathy, and Creutzfeldt-Jakob disease. *J Hosp Infect* 1991;18 (Suppl A):141–6.
1192. Favero MS. Current issues in hospital hygiene and sterilization technology. *J Infect Control (Asia Pacific Edition)* 1998;1:8–10.
1193. Ricketts MN, Cashman NR, Stratton EE, El Saadany S. Is Creutzfeldt-Jakob disease transmitted in blood? *Emerg Infect Dis* 1997;3:155–63.
1194. Will RG, Kimberlin RH. Creutzfeldt-Jakob disease and the risk from blood or blood products. *Vox Sang* 1998;75:178–80.
1195. Evatt B, Austin H, Barnhart E, et al. Surveillance for Creutzfeldt-Jakob disease among persons with hemophilia. *Transfusion* 1998;38:817–20.

1196. Patry D, Curry B, Easton D, Mastrianni JA, Hogan DB. Creutzfeldt-Jakob disease (CJD) after blood product transfusion from a donor with CJD. *Neurology* 1998;50:1872-3.
1197. (340) Budka H, Aguzzi A, Brown P, et al. Tissue handling in suspected Creutzfeldt-Jakob disease (CJD) and other human spongiform encephalopathies (prion diseases). *Brain Pathol* 1995;5:319-22.
1198. (341) Ironside JW, Bell JE. The "high-risk" neuropathological autopsy in AIDS and Creutzfeldt-Jakob disease: principles and practice. *Neuropathol Appl Neurobiol* 1996;22:388-93.
1199. (336) Rutala WA, Weber DJ. Creutzfeldt-Jakob disease: recommendations for disinfection and sterilization. *Clin Infect Dis* 2001;32:1348-56.
1200. Joint Commission for the Accreditation of Healthcare Organizations. Exposure to Creutzfeldt-Jakob Disease. Sentinel Alert, Issue 20; June 2001. Available at: www.jcaho.org/edu_pub/sealert/sea20.html
1201. (342) World Health Organization. WHO Infection control guideline for transmissible spongiform encephalopathies: report of a WHO consultation. Geneva, Switzerland: WHO, 1999. Available at: www.who.int/emc-documents/tse/whocdscsraph2003c.html
1202. Litsky BY. Results of bacteriological surveys highlight problem areas in hospitals. *Hospital Management* 1966;101:82-8.
1203. Eickhoff TC. Microbiologic sampling. *Hospitals* 1970;44:86-7.
1204. American Hospital Association Committee on Infections Within the Hospitals. Statement on microbiologic sampling in the hospital 1974;48:125-6.
1205. Rafferty KM, Pancoast SJ. Brief report: bacteriological sampling of telephones and other hospital staff-hand contact objects. *Infect Control* 1984;5:533-5.
1206. Haley RW, Shachtman RS. The emergence of infection control programs in U.S. hospitals: an assessment, 1976. *Am J Epidemiol* 1980;111:574-91.
1207. Mallison GF, Haley RW. Microbiologic sampling of the inanimate environment in U.S. hospitals, 1976-1977. *Am J Med* 1981;70:941-6.
1208. Gröschel DHM. Air sampling in hospitals. *Ann NY Acad Sci* 1980;353:230-40.
1209. (208, Appendix; 6) Barbaree JM, Gorman GW, Martin WT, Fields BS, Morrill WE. Protocol for sampling environmental sites for legionellae. *Appl Environ Microbiol* 1987;53:1454-8.
1210. Eickhoff TC. Microbiologic sampling of the hospital environment. *Health Lab Sci* 1974;11:73-5.
1211. Isenberg HD. Significance of environmental microbiology in nosocomial infections and the care of hospitalized patients. In: Lorian V, ed. Significance of medical microbiology in the care of patients. Baltimore, MD: Williams & Wilkins, 1977;220-34.
1212. McGowan JE Jr, Weinstein RA. The role of the laboratory in control of nosocomial infection. In: Bennett JV, Brachman PS, eds. *Hospital infections*, 4th ed. Philadelphia, PA: Lippincott Raven, 1998;143-64.
1213. Turner AG, Wilkins JR, Craddock JG. Bacterial aerosolization from an ultrasonic cleaner. *J Clin Microbiol* 1975;1:289-93.
1214. (343) Bond WW, Schulster LM. Microbiological culturing of environmental and medical-device surfaces. In: Isenberg HD, Miller JM, Bell M, eds. *Clinical microbiology procedures handbook*, section 11. Washington, DC: American Society for Microbiology Press, 2004 (in press).
1215. Cole EC, Cook CE. Characterization of infectious aerosols in health care facilities: an aid to effective engineering controls and preventive strategies. *Am J Infect Control* 1998;26:452-64.
1216. Nevalainen A, Willeke K, Liebhaber F, Pastuszka J, Burge H, Henningson E. Bioaerosol sampling. In: Willeke K, Baron PA, eds. *Aerosol management*. New York, NY: Van Nostrand Reinhold, 1993;471-92.
1217. Cox CS. *The aerobiological pathway of microorganisms*. Chichester UK: John Wiley & Sons, 1987.
1218. (349) Wolf HW, Skaliy P, Hall LB, et al. Sampling microbiological aerosols. Public Health Service publication No. 686. Government Printing Office, Washington, DC: 1964.
1219. Zeterberg JM. A review of respiratory virology and the spread of virulent and possible antigenic viruses via air conditioning systems. *Ann Allergy* 1973;31:228-34.
1220. Randall CW, Ledbetter JO. Bacterial air pollution from activated sludge units. *Am Ind Hyg Assoc J* 1966;Nov/Dec:506-19.
1221. Salem H, Gardner DE. Health aspects of bioaerosols. In: Lighthart B, Mohr AJ, eds. *Atmospheric microbial aerosols, theory and applications*. New York, NY: Chapman and Hall, 1985;304-30.
1222. Sattar SA, Ijaz MK. Spread of viral infections by aerosols. *Crit Rev Environ Control* 1987;17:89-131.
1223. (345) Buttner MP, Willeke K, Grinshpun SA. Sampling and analysis of airborne microorganisms. In: Hurst CJ, Knudsen GR, McInerney MJ, Stetzenbach LD, Walter MV, eds. *Manual of environmental microbiology*. Washington, DC: American Society for Microbiology Press, 1997;629-40.

1224. (346) Jensen PA, Schafer MP. Sampling and characterization of bioaerosols. In: NIOSH Manual of Analytical Methods; Cincinnati OH; CDC; 1998: p. 82–112. Available at: www.cdc.gov/niosh/nmam/pdfs/chapter-j.pdf
1225. Jolley AE. The value of surveillance cultures on neonatal intensive care units. *J Hosp Infect* 1993;25:153–9.
1226. Hardy KA, McGowan KL, Fisher MC, Schidlow DV. *Pseudomonas cepacia* in the hospital setting: lack of transmission between cystic fibrosis patients. *J Pediatr* 1986;109:51–4.
1227. Hambræus A, Lagerqvist-Widh A, Zettersten U, Engberg S, Sedin G, Sjöberg L. Spread of *Klebsiella* in a neonatal ward. *Scand J Infect Dis* 1991;23:189–94.
1228. Humphreys H, Peckham D, Patel P, Knox A. Airborne dissemination of *Burkholderia (Pseudomonas) cepacia* from adult patients with cystic fibrosis. *Thorax* 1994;49:1157–9.
1229. Pankhurst CL, Harrison VE, Philpott-Howard J. Evaluation of contamination of the dentist and dental surgery environment with *Burkholderia (Pseudomonas) cepacia* during treatment of children with cystic fibrosis. *Int J Paediatr Dent* 1995;5:243–7.
1230. Weber DO, Gooch JJ, Wood WR, Britt EM, Kraft RO. Influence of operating room surface contamination on surgical wounds: a prospective study. *Arch Surg* 1976;111:484–8.
1231. Pfeiffer EH, Wittig JR, Dunkelberg H, Werner HP. Hygienic and bacteriological comparative studies in 50 hospitals. V. Bacterial contamination of hospital surfaces. *Zentralbl Bakteriol [B]* 1978;167:11–21. (German)
1232. Sattar SA, Lloyd-Evans N, Springthorpe VS. Institutional outbreaks of rotavirus diarrhea: potential role of fomites and environmental surfaces as vehicles for virus transmission. *J Hyg (Camb)* 1986;96:277–89.
1233. Smith SM, Eng RH, Padberg FT Jr. Survival of nosocomial pathogenic bacteria at ambient temperature. *J Med* 1996;27:293–302.
1234. Craythorn JM, Barbour AG, Matsen JM, Britt MR, Garibaldi RA. Membrane filter contact technique for bacteriological sampling of moist surfaces. *J Clin Microbiol* 1980;12:250–5.
1235. Scott E, Bloomfield SF, Barlow CG. A comparison of contact plate and calcium alginate swab techniques for quantitative assessment of bacteriological contamination of environmental surfaces. *J Appl Bacteriol* 1984;56:317–20.
1236. Poletti L, Pasquarella C, Pitzurra M, Savino A. Comparative efficiency of nitrocellulose membranes versus RODAC plates in microbial sampling on surfaces. *J Hosp Infect* 1999;41:195–201.
1237. Russell AD. Factors influencing the efficacy of antimicrobial agents. In: Russell AD, Hugo WB, Ayliffe GAJ, eds. Principles and practices of disinfection, preservation and sterilization. Oxford, UK: Blackwell Science, 1999;95–123.
1238. (347) International Organization for Standardization (ISO). Sterilization of medical devices — microbiological methods, Part 1. ISO Standard 11737-1. Paramus, NJ: International Organization for Standardization, 1995.
1239. Favero MS, Gabis DA, Vesley D. Environmental monitoring procedures. In: Speck ML, ed. Compendium of methods for the microbiological examination of foods, 2nd ed. Washington, DC: American Public Health Association; 1984;47–61.
1240. Favero MS, Bond WW, Petersen NJ, Berquist KR, Maynard JE. Detection methods for study of the stability of hepatitis B antigen on surfaces. *J Infect Dis* 1974;129:210–2.
1241. Favero MS, McDade JJ, Robertsen JA, Hoffmann RK, Edwards RW. Microbiological sampling of surfaces. *J Appl Bacteriol* 1968;31:336–43.
1242. Petersen NJ, Collins DE, Marshall JH. Evaluation of skin cleansing procedures using the wipe-rinse technique. *Health Lab Sci* 1974;11:182–97.
1243. Schalkowsky S, Hall LB, Kline RC. Potential effects of recent findings on spacecraft sterilization requirements. *Space Life Sci* 1969;1:520–30.
1244. Hall LB, Lyle RG. Foundations of planetary quarantine. *Environ Biol Med* 1971;1:5–8.
1245. Rutala WA, Weber DJ. Uses of inorganic hypochlorite (bleach) in health-care facilities. *Clin Microbiol Rev* 1997;10:597–610.
1246. Mallison GF. Central services and linens and laundry. In: Bennett JV, Brachman PS, eds. Hospital infections. Boston, MA: Little, Brown, & Co, 1986;251–6.
1247. (365) Blaser MJ, Smith PE, Cody HJ, Wang W-LL, LaForce FM. Killing of fabric-associated bacteria in hospital laundry by low-temperature washing. *J Infect Dis* 1984;149:48–57.
1248. Centers for Disease Control. Outbreak of viral hepatitis in the staff of a pediatric ward — California. *MMWR* 1977;28:77–9.

1249. Shah PC, Krajden S, Kane J, Summerbell RC. Tinea corporis caused by *Microsporum canis*: report of a nosocomial outbreak. *Eur J Epidemiol* 1988;4:33–7.
1250. (353) Barrie D, Hoffman PN, Wilson JA, Kramer JM. Contamination of hospital linen by *Bacillus cereus*. *Epidemiol Infect* 1994;113:297–306.
1251. Standaert SM, Hutcheson RH, Schaffner W. Nosocomial transmission of *Salmonella gastroenteritis* to laundry workers in a nursing home. *Infect Control Hosp Epidemiol* 1994;15:22–6.
1252. Pasternak J, Richtmann R, Ganme APP, et al. Scabies epidemic: price and prejudice. *Infect Control Hosp Epidemiol* 1994;15:540–2.
1253. (357) Association for the Advancement of Medical Instrumentation. Processing of reusable surgical textiles for use in health care facilities: ANSI/AAMI recommended practice ST65. Arlington, VA: Association for the Advancement of Medical Instrumentation, 2000;16.
1254. Association of Operating Room Nurses. Recommended practices for surgical attire: AORN standards and recommended practices. *AORN J* 1995;62:141–2.
1255. Loh W, Ng VV, Holton J. Bacterial flora on the white coats of medical students. *J Hosp Infect* 2000;45:65–8.
1256. Belkin NL. Use of scrubs and related apparel in healthcare facilities. *Am J Infect Control* 1997;25:401–4.
1257. Belkin NL. Home laundering of soiled surgical scrubs: surgical site infections and the home environment. *Am J Infect Control* 2000;29:58–64.
1258. (355) Joint Committee on Healthcare Laundry Guidelines. Guidelines for healthcare linen service. Hallendale, FL: Textile Rental Service Association of America, 1999.
1259. (356) Greene VW. Microbiological contamination control in hospitals: part 6 — roles of central service and the laundry. *Hospitals JAHA* 1970;44:98–103.
1260. (350) Wagner RA. Partitioned laundry improves bacteria control. *Hospitals JAHA* 1966;40:148–51.
1261. (351) Hambraeus A, Malmborg AS. Is a bed centre in a hospital a hygienic hazard? *J Hyg (Camb)* 1982;88:143–7.
1262. (352) McDonald LL, Pugliese G. Textile processing service. In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;1031–4.
1263. (354) Legnani PP, Leoni E. Factors affecting the bacteriological contamination of commercial washing machines. *Zentralbl Hyg Umweltmed* 1997;200:319–33.
1264. Maki DG, Alvarado C, Hassemer C. Double-bagging of items from isolation rooms is unnecessary as an infection control measure: a comparative study of surface contamination with single- and double-bagging. *Infect Control* 1986;7:535–7.
1265. Garner JS, Simmons BP. CDC guideline for isolation precautions in hospitals. *Infect Control* 1983;4:245–325 and *Am J Infect Control* 1984;12:103–63.
1266. Weinstein SA, Gantz NM, Pelletier C, Hibert D. Bacterial surface contamination of patients' linen: isolation precautions versus standard care. *Am J Infect Control* 1989;17:264–7.
1267. (358) Hughes HG. Chutes in hospitals. *Can Hosp* 1964;41:56–7, 87.
1268. (359) Michaelsen GS. Designing linen chutes to reduce spread of infectious organisms. *Hospitals JAHA* 1965;39 (3):116–9.
1269. (360) Hoch KW. Laundry chute cleaning recommendations [letter]. *Infect Control* 1982;3:360.
1270. (361) Whyte W, Baird G, Annand R. Bacterial contamination on the surface of hospital linen chutes. *J Hyg (Camb)* 1969;67:427–35.
1271. (363) Walter WG, Schillinger JE. Bacterial survival in laundered fabrics. *Appl Microbiol* 1975;29:368–73.
1272. (362) Taylor LJ. Segregation, collection, and disposal of hospital laundry and waste. *J Hosp Infect* 1988;11 (Suppl. A):57–63.
1273. Barrie D. How hospital linen and laundry services are provided. *J Hosp Infect* 1994;27:219–35.
1274. Riggs CH, Sherrill JC. Textile laundering technology. Hallendale FL: Textile Rental Service Association; 1999;92–7.
1275. Mouton RP, Bekkers JH. Bacteriological results of routine procedures in a hospital laundry. *Folia Med Neerl* 1967;10:71–6.
1276. Nicholes PS. Bacteria in laundered fabrics. *Am J Public Health* 1970;60:2175–80.
1277. Arnold L. A sanitary study of commercial laundry practices. *Am J Public Health* 1938;28:839–44.

1278. (364) Belkin NL. Aseptics and aesthetics of chlorine bleach: can its use in laundering be safely abandoned? *Am J Infect Control* 1998;26:149–51.
1279. Jordan WE, Jones DV. Antiviral effectiveness of chlorine bleach in household laundry use. *Am J Dis Child* 1969;117:313–6.
1280. Hittman Associates, Inc. Energy efficient water use in hospitals [Final summary report (H-W8000-78-756FR)]. Prepared for the University of California, Lawrence Berkeley Laboratory, 1979. Contract No. P.O. 4627702.
1281. (366) Jaska JM, Fredell DL. Impact of detergent systems on bacterial survival on laundered fabrics. *Appl Environ Microbiol* 1980;39:743–8.
1282. (367) Battles DR, Vesley D. Wash water temperature and sanitation in the hospital laundry. *J Environ Health* 1981;43:244–50.
1283. (368) Christian RR, Manchester JT, Mellor MT. Bacteriological quality of fabrics washed at lower-than-standard temperatures in a hospital laundry facility. *Appl Environ Microbiol* 1983;45:591–7.
1284. (369) Smith JA, Neil KR, Davidson CG, Davidson RW. Effect of water temperature on bacterial killing in laundry. *Infect Control* 1987;8:204–9.
1285. (370) Tompkins DS, Johnson P, Fittall BR. Low-temperature washing of patients' clothing: effects of detergent with disinfectant and a tunnel drier on bacterial survival. *J Hosp Infect* 1988;12:51–8.
1286. (371) Ayliffe GAJ, Collins BJ, Taylor LJ. Laundering. In: Wright PSG, ed. *Hospital-acquired Infection: principles and Prevention*. Bristol, UK: 1982;101–6.
1287. Koller W, Wewalka G. A new method for microbiological evaluation of disinfecting laundering processes. *Zbl Bakt Hyg I Abt Orig B* 1982;176:463–71.
1288. (372) Meyer CL, Eitzen HE, Schreiner RL, Gfell MA, Moye L, Kleiman MB. Should linen in newborn intensive care units be autoclaved? *Pediatrics* 1981;67:362–4.
1289. (373) Wagg RE. Disinfection of textiles in laundering and dry cleaning. *Chem Ind* 1965;44:1830–4.
1290. (374) Bates CJ, Wilcox MH, Smith TL, Spencer RC. The efficacy of a hospital dry cleaning cycle in disinfecting material contaminated with bacteria and viruses. *J Hosp Infect* 1993;23:255–62.
1291. (375) Oehnel E. Drycleaning in the hospital laundry. *Can Hosp* 1971;September:66–7.
1292. DiGacomo JC, Odom JW, Ritoto PC, Swan KC. Cost containment in the operating room: use of reusables versus disposable clothing. *Am Surg* 1992;58:654–6.
1293. American Society for Testing Materials. Standard test method for resistance of materials used in protective clothing to penetration by synthetic blood. ASTM, 1998;F1670–98.
1294. American Society for Testing Materials. Standard test method for resistance of materials used in protective clothing to penetration by bloodborne pathogens using phi-X174 bacteriophage penetration as a test system. ASTM 1997;F1671–976.
1295. Belkin NL. Are “impervious” surgical gowns really liquid-proof? *Bull Am Col Surgeons* 1999;84:19–36.
1296. Belkin NL. OR gowns — even a “pass” can fail. *AORN J* 1999;70:302–4.
1297. Laufman H, Belkin NL, Meyer KK. A critical review of a century's progress in surgical apparel: how far have we come? *J Am Col Surgeons* 2000;191:554–68.
1298. Leonas KK, Jinkins RS. The relationship of selected fabric characteristics and the barrier effectiveness of surgical gown fabrics. *Am J Infect Control* 1997;25:16–23.
1299. Meyer KK, Beck WC. Gown-glove interface: a possible solution to the danger zone. *Infect Control Hosp Epidemiol* 1995;16:488–90.
1300. McCullough EA. Methods for determining the barrier efficacy of surgical gowns. *Am J Infect Control* 1993;21:368–74.
1301. Pissiotis CA, Komborozos V, Papoutsi C, Skrekas G. Factors that influence the effectiveness of surgical gowns in the operating theater. *Eur J Surg* 1997;163:597–604.
1302. Association of Operating Room Nurses (AORN). Recommended practices for use and selection of barrier materials for surgical gowns and drapes. Association of Operating Room Nurses. *AORN J* 1996;63:650, 653–4.
1303. Belkin NL, Koch FT. OR barrier materials — Necessity or extravagance? *AORN J* 1998;67:443–5.
1304. Rutala WA, Weber DJ. A review of single-use and reusable gowns and drapes in health care. *Infect Control Hosp Epidemiol* 2001;22:248–57.
1305. Murphy L. Cost/benefit study of reusable and disposable OR draping materials. *J Healthc Mater Manage* 1993;11:44–8.

1306. (376) U.S. Environmental Protection Agency. Consumer products treated with pesticides. Office of Pesticide Programs. Available at: www.epa.gov/opp00001/citizens/treatart.htm
1307. Kalyon BD, Olgun U. Antibacterial efficacy of triclosan-incorporated polymers. *Am J Infect Control* 2001;29:124–5.
1308. U.S. Environmental Protection Agency. Clarification of treated articles exemption. Availability of draft PR notice. *Federal Register* 1998;63:19256–8.
1309. Mayer CE. FTC Challenges antibacterial product. Washington, DC: Washington Post, September 17, 1999;A09.
1310. (377) Fujita K, Lilly HA, Kidson A, Ayliffe GAJ. Gentamicin-resistant *Pseudomonas aeruginosa* infection from mattresses in a burns unit. *Br Med J* 1981;283:219–20.
1311. (378) Grubb DJ, Watson KC. *Pseudomonas* septicaemia from plastic mattresses [letter]. *Lancet* 1982;1:518.
1312. (379) Sherertz RJ, Sullivan ML. An outbreak of infections with *Acinetobacter calcoaceticus* in burn patients: contamination of patients' mattresses. *J Infect Dis* 1985;151:252–8.
1313. (380) Ndawula EM, Brown L. Mattresses as reservoirs of epidemic methicillin-resistant *Staphylococcus aureus* [letter]. *Lancet* 1991;337:488.
1314. (381) O'Donoghue MAT, Allen KD. Costs of an outbreak of wound infections in an orthopaedic ward. *J Hosp Infect* 1992;22:73–9.
1315. (382) Weernink A, Severin WPJ, Thernberg T, Dijkshoorn L. Pillows, an unexpected source of *Acinetobacter*. *J Hosp Infect* 1995;29:189–99.
1316. Newsome TW, Johns LA, Pruitt BA Jr. Use of an air-fluidized bed in the care of patients with extensive burns. *Am J Surg* 1972;124:52–6.
1317. (383) Scheidt A, Drusin LM. Bacteriologic contamination in an air-fluidized bed. *J Trauma* 1983;23:241–2.
1318. (384) Freeman R, Gould FK, Ryan DW, Chamberlain J, Sisson PR. Nosocomial infection due to enterococci attributed to a fluidized microsphere bed: the value of pyrolysis mass spectrometry. *J Hosp Infect* 1994;27:187–93.
1319. Sharbaugh RJ, Hargest TS. Bactericidal effect of the air-fluidized bed. *Am Surgeon* 1971;37:583–6.
1320. Sharbaugh RJ, Hargest TS, Wright FA. Further studies on the bactericidal effect of the air-fluidized bed. *Am Surgeon* 1973;39:253–6.
1321. Winters WD. A new perspective of microbial survival and dissemination in a prospectively contaminated air-fluidized bed model. *Am J Infect Control* 1990;18:307–15.
1322. (385) Clancy MJ. Nosocomial infection and microsphere beds [letter]. *Lancet* 1993;342:680–1.
1323. (386) Clancy MJ. Nosocomial infection due to enterococci attributed to a fluidized microsphere bed [letter]. *J Hosp Infect* 1994;28:324–5.
1324. Vesley D, Hankinson SE, Lauer JL. Microbial survival and dissemination associated with an air-fluidized therapy unit. *Am J Infect Control* 1986;14:35–40.
1325. Bolyard EA, Townsend TR, Horan T. Airborne contamination associated with in-use air-fluidized beds: a descriptive study. *Am J Infect Control* 1987;15:75–8.
1326. (387) Jacobsen E, Gurevich I, Cunha BA. Air-fluidized beds and negative-pressure isolation rooms [letter]. *Am J Infect Control* 1993;21:217–8.
1327. Cooper JE. Pets in hospitals. *Br Med J* 1976;1:698–700.
1328. Egerton JR. Pets and zoonoses. *Med J Aust* 1982;2:311.
1329. Yamauchi T. Pet programs in hospitals. *Pediatr Infect Dis J* 1993;12:707.
1330. Khan MA, Farrag N. Animal-assisted activity and infection control implications in a healthcare setting. *J Hosp Infect* 2000;46:4–11.
1331. Weber DJ, Rutala WA. Epidemiology and prevention of nosocomial infections associated with animals in the hospital. In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;1399–421.
1332. Acha PN, Szyfres B. Zoonoses and communicable diseases common to man and animals, 2nd ed. Washington, DC: Pan American Health Organization, 1987. Scientific publication No. 503.
1333. Elliot DL, Tolle SW, Goldberg L, Miller JB. Pet-associated illness. *N Engl J Med* 1985;313:985–95.
1334. Marx MB. Parasites, pets, and people. *Primary Care* 1991;18:153–65.
1335. Goldstein EJ. Household pets and human infections. *Infect Dis Clin North Am* 1991;5:117–30.
1336. Chomel BB. Zoonoses of house pets other than dogs, cats, and birds. *Pediatr Infect Dis J* 1992;11:479–87.

1337. Gnann JW Jr, Bressler GS, Bodet CA III, Avent CK. Human blastomycosis after a dog bite. *Ann Intern Med* 1983;98:48–9.
1338. Garcia VF. Animal bites and *Pasteurella* infections. *Pediatr Rev* 1997;18:127–30.
1339. Crowder HR, Dorn CR, Smith RE. Group A *Streptococcus* in pets and group A streptococcal disease in man. *Int J Zoonoses* 1978;5:45–54.
1340. (397) CDC. Reptile-associated salmonellosis — selected states, 1996–1998. *MMWR* 1999;48:1009–13.
1341. Devriese LA, Ieven M, Goossens H, et al. Presence of vancomycin-resistant enterococci in farm and pet animals. *Antimicrob Agent Chemother* 1996;40:2285–7.
1342. Scott GM, Thomson R, Malone-Lee J, Ridgway GL. Cross-infection between animals and man: Possible feline transmission of *Staphylococcus aureus* infection in humans? *J Hosp Infect* 1988;12:29–34.
1343. Weinberg A. Ecology and epidemiology of zoonotic pathogens. *Infect Dis Clin North Am* 1991;5:1–6.
1344. Yu V, Meissner C. Zoonoses. In: Schaechter M, Medoff G, Schlessinger D, eds. *Mechanisms of microbial diseases*. Baltimore, MD: Williams & Wilkins, 1989;749–64.
1345. Ryan KJ. Some bacteria causing zoonotic diseases. In: Sherris JC, ed. *Medical microbiology*, 2nd ed. New York, NY: Elsevier, 1990;489–98.
1346. Chang HJ, Miller HL, Watkins N, et al. An epidemic of *Malassezia pachydermatis* in an intensive care nursery associated with colonization of healthcare workers' pet dog. *N Engl J Med* 1998;338:706–11.
1347. Drusin LM, Ross BG, Rhodes KH, Krause AN, Scott RA. Nosocomial ringworm in a neonatal intensive care unit: a nurse and her cat. *Infect Control Hosp Epidemiol* 2000;21:605–7.
1348. Richet HM, Craven PC, Brown JM, et al. A cluster of *Rhodococcus (Gordona) bronchialis* sternal-wound infections after coronary-artery bypass surgery. *N Engl J Med* 1991;324:104–9.
1349. (394) Saylor K. Pet visitation program. *J Gerontol Nurs* 1998;24:36–8.
1350. Corson SA, O'Leary Corson E. Pets as mediators of therapy. *Curr Psychiatr Ther* 1978;18:195–205.
1351. Fick KM. The influence of an animal on social interactions of nursing home residents in a group setting. *Am J Occup Ther* 1993;47:529–34.
1352. Gunby P. Patient progressing well? He must have a pet. *JAMA* 1979;241:438.
1353. Culliton BJ. Take two pets and call me in the morning. *Science* 1987;237:1560–1.
1354. Wilkes CN, Shalko TK, Trahan M. Pet Rx: Implications for good health. *Health Educ* 1989;20:6–9.
1355. Doyle K, Kukowski T. Utilization of pets in a hospice program. *Health Educ* 1989;20:10–1.
1356. Teeter LM. Pet therapy program. *J Amer Vet Med Assoc* 1997;210:1435–8.
1357. Gammonley J, Yates J. Pet projects: animal assisted therapy in nursing homes. *J Gerontol Nurs* 1991;17:12–5.
1358. (395) Draper RJ, Gerber GJ, Layng EM. Defining the role of pet animals in psychotherapy. *Psychiat J Univ Ottawa* 1990;15:169–72.
1359. Allen DT. Effects of dogs on human health. *J Amer Vet Med Assoc* 1997;210:1136–9.
1360. (391) Delta Society. *Standards of practice for animal-assisted activities and animal-assisted therapy*. Renton, WA: Delta Society, 1996.
1361. (392) Fox JG. Transmissible drug resistance in *Shigella* and *Salmonella* isolated from pet monkeys and their owners. *J Med Primatol* 1975;4:165–71.
1362. (393) Ostrowski SR, Leslie MJ, Parrott T, Abelt S, Piercy PE. B-virus from pet macaque monkeys: an emerging threat in the United States? *Emerg Infect Dis* 1998;4:117–21.
1363. CDC. How to prevent transmission of intestinal roundworms from pets to people. Available at: www.cdc.gov/ncidod/diseases/roundworm/roundworm.htm
1364. (146) Boyce JM, Pittet D. Guideline for hand hygiene in health-care settings: recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force. *Infect Control Hosp Epidemiol* 2002;23 (Suppl):S1–S40.
1365. (388) American Academy of Allergy, Asthma, and Immunology. Tips to remember: indoor allergens. Available at: www.aaaai.org/patients/publicedmat/tips/indoorallergens.stm
1366. (389) Duncan SL, APIC Guideline Committee. APIC State-of-the-art report: the implications of service animals in healthcare settings. *Am J Infect Control* 2000;28:170–80.
1367. (390) Murray AB, Ferguson A, Morrison BJ. The frequency and severity of cat allergy vs. dog allergy in atopic children. *J Allergy Clin Immunol* 1983;72:145–9.
1368. Hodson T, Custovic A, Simpson A, Chapman M, Woodcock A, Green R. Washing the dog reduces dog allergen levels, but the dog needs to be washed twice a week. *J Allergy Clin Immunol* 1999;103:581–5.
1369. Brickel CN. The therapeutic roles of cat mascots with a hospital based population: a staff survey. *Gerontologist* 1979;19:368–72.

1370. Thomas W, Stermer M. Eden alternative principles hold promise for the future of long-term care. *Balance* 1999;3:14–7.
1371. Tavormina CE. Embracing the Eden alternative in long-term care environments. *Geriatr Nurs* 1999;20:158–61.
1372. Brook I, Fish CH, Schantz PM, Cotton DD. Toxocariasis in an institution for the mentally retarded. *Infect Control* 1981;2:317–9.
1373. Huminer D, Symon R, Groskopf I, et al. Seroepidemiological study of toxocariasis and strongyloidiasis in adult mentally retarded institutionalized subjects. *Am J Trop Med Hyg* 1992;46:278–81.
1374. Huminer D, Pitlik SD, Block C, Kaufman L, Amit S, Rosenfeld JB. Aquarium-borne *Mycobacterium marinum* skin infection: report of a case and review of the literature. *Arch Dermatol* 1986;122:698–703.
1375. Lewis FMT, Marsh BJ, von Reyn CF. Fish tank exposure and cutaneous infections due to *Mycobacterium marinum*: tuberculin skin testing, treatment, and prevention. *Clin Infect Dis* 2003;37:390–7.
1376. (398) U.S. Department of Justice. Americans with Disabilities Act. Public Law 101-336 (28 CFR 36.101 et seq.). Title III, Public Accommodations Operated by Private Entities, Sect. 302, Prohibition of Discrimination by Public Accommodations;42 USC 12101 et seq. July 26, 1990.
1377. U.S. Department of Justice, Civil Rights Division, Disability Rights Section. Commonly asked questions about service animals in places of business, 1996.
1378. Schachter J, Sung M, Meyer KF. Potential danger of Q fever in a university hospital environment. *J Infect Dis* 1971;123:301–4.
1379. Konkle DM, Nelson KN, Lunn DP. Nosocomial transmission of *Cryptosporidium* in a veterinary hospital. *J Vet Intern Med* 1997;11:340–3.
1380. House JK, Mainar-Jaime RC, Smith BP, House AM, Kamiya DY. Risk factors for nosocomial *Salmonella* infection among hospitalized horses. *J Am Vet Med Assoc* 1999;214:1511–6.
1381. Weese JS, Staempfli HR, Prescott JF. Isolation of environmental *Clostridium difficile* from a veterinary teaching hospital. *J Vet Diagn Invest* 2000;12:449–52.
1382. Boerlin P, Eugster S, Gaschen F, Straub R, Schawalder P. Transmission of opportunistic pathogens in a veterinary teaching hospital. *Vet Microbiol* 2001;82:347–9.
1383. Schott HC II, Ewart SL, Walker RD, et al. An outbreak of salmonellosis among horses at a veterinary teaching hospital. *J Am Vet Med Assoc* 2001;218:1152–9,1170.
1384. Kim LM, Morley PS, Traub-Dargatz JL, Salman MD, Gentry-Weeks C. Factors associated with *Salmonella* shedding among equine colic patients at a veterinary teaching hospital. *J Am Vet Med Assoc* 2001;218:740–8.
1385. Seguin JC, Walker RD, Caron JP, et al. Methicillin-resistant *Staphylococcus aureus* outbreak in a veterinary teaching hospital: potential human-to-animal transmission. *J Clin Microbiol* 1999;37:1459–63.
1386. Shen DT, Crawford TB, Gorham JR, et al. Inactivation of equine infectious anemia virus by chemical disinfectants. *Am J Vet Res* 1977;38:1217–9.
1387. Scott FW. Virucidal disinfectants and feline viruses. *Am J Vet Res* 1980;41:410–4.
1388. Brown TT. Laboratory evaluation of selected disinfectants as virucidal agents against porcine parvovirus, pseudorabies virus, and transmissible gastroenteritis virus. *Am J Vet Res* 1981;42:1033–6.
1389. Saknimit M, Inatsuki I, Sugiyama Y, et al. Virucidal efficacy of physico-chemical treatments against coronaviruses and parvoviruses of laboratory animals. *Exp Anim* 1988;37:341–5.
1390. Bruins G, Dyer JA. Environmental considerations of disinfectants used in agriculture. *Rev Sci Tech* 1995;14:81–94.
1391. Quinn PJ, Markey BK. Disinfection and disease prevention in veterinary medicine. In: Block SS, ed. *Disinfection, sterilization, and preservation*, 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2001;1069–103.
1392. Fox JG, Lipman NS. Infections transmitted by large and small laboratory animals. *Infect Dis Clin North Am* 1991;5:131–63.
1393. (401) U.S. Department of Labor, Occupational Safety and Health Administration. Personal Protective Equipment for General Industry, Final Rule (29 CFR 1910 §1910.132, 1910.138). *Federal Register* 1994;59:16334–64.
1394. U.S. Department of Agriculture. Public Law 89-544 (The Animal Welfare Act of 1966). 7 USC § 2131-2156.

1395. **(399)** U.S. Department of Agriculture. Public Law 99-198 Food Security Act of 1985, Subtitle F - Animal Welfare. 7 USC § 2131.
1396. Althaus H, Sauerwald M, Schrammeck E. Waste from hospitals and sanatoria. *Zbl Bakteriol Hyg I Abt Orig B* 1983;178:1–29.
1397. Kalnowski G, Wiegand H, Henning R. The microbial contamination of hospital waste. *Zbl Bakteriol Hyg I Abt Orig B* 1983;178:364–79.
1398. Mose JR, Reinthaler F. Microbial contamination of hospital waste and household refuse. *Zbl Bakteriol Hyg I Abt Orig B* 1985;181:98–110.
1399. Collins CH, Kennedy DA. The microbiological hazards of municipal and clinical wastes. *J Appl Bacteriol* 1992;73:1–6.
1400. Rutala WA, Odette RL, Samsa GP. Management of infectious waste by U.S. hospitals. *JAMA* 1989;262:1635–40.
1401. Agency for Toxic Substances and Disease Registry. The public health implications of medical waste: a report to Congress. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, 1990.
1402. Hedrick ER. Infectious waste management — will science prevail? *Infect Control Hosp Epidemiol* 1988;9:488–90.
1403. Keene J. Medical waste management: public pressure vs. sound science. *Hazard Mat Control* 1989;Sept/Oct:29–36.
1404. Keene J. Medical waste: a minimal hazard. *Infect Control Hosp Epidemiol* 1991;12:682–5.
1405. Rutala WA, Weber DJ. Mismatch between science and policy. *N Engl J Med* 1991;325:578–82.
1406. U.S. Environmental Protection Agency. EPA guide for infectious waste management. Washington, DC: U.S. Government Printing Office, 1986. EPA Publication No. 530SW86014.
1407. **(402)** U.S. Department of Transportation. Hazardous materials regulations. 49 CFR Parts 171–180, Division 6.2; and Hazardous Materials: Revision to Standards for Infectious Substances and Genetically-Modified Microorganisms: Proposed Rule. *Federal Register* 1998;63:46843–59.
1408. **(403)** U.S. Postal Service. C 023.8.0 Infectious substances (Hazard Class 6, Division 6.2) et seq. At: <http://pe.usps.gov/text/dmm/c023.htm>
1409. **(404)** Greene R, Miele DJ, Slavik NS. Technical assistance manual: state regulatory oversight of medical waste treatment technologies, 2nd ed: a report of the State and Territorial Association on Alternative Treatment Technologies, 1994.
1410. U.S. Environmental Protection Agency (EPA). 40 CFR Part 60. Standards of performance for new stationary sources and emission guidelines for existing sources: hospital/medical/infectious waste incinerators; Final Rule. *Federal Register* 1997;62:48347–91.
1411. CDC/National Institutes of Health. Biosafety in microbiological and biomedical laboratories. Washington, DC: U.S. Government Printing Office, 1985. DHHS Publication No. (CDC) 93–8395.
1412. **(409)** U.S. Department of Health and Human Services, Office of Inspector General, CDC. Possession, use, and transfer of select agents and toxins, interim final rule (42 CFR Part 73). *Federal Register*, December 13, 2002;67(240):76885–905.
1413. U.S. Department of Agriculture, Animal and Plant Health Inspection Service. Agricultural Bioterrorism Protection Act of 2002; Possession, use, and transfer of biological agents and toxins: interim final rule (9 CFR Part 121). *Federal Register*. December 13, 2002;67(240):76907–38.
1414. **(410)** CDC. Recommendations on infective waste. Atlanta, GA: Office of Biosafety and Hospital Infections Program, 1988;1–6.
1415. **(405)** CDC. National Institute for Occupational Safety and Health. NIOSH Alert: Preventing needlestick injuries in health care settings. Cincinnati, OH: DHHS, 1999. DHHS-NIOSH Publication No. 2000–108.
1416. Rutala WA, Stiegel MM, Sarubbi FA. Decontamination of laboratory microbiological waste by steam sterilization. *Appl Environ Microbiol* 1982;43:1311–6.
1417. Lauer JL, Battles DR, Vesley D. Decontaminating infectious laboratory waste by autoclaving. *Appl Environ Microbiol* 1982;44:690–4.
1418. Palenik CJ, Cumberlander ND. Effects of steam sterilization on the contents of sharps containers. *Am J Infect Control* 1993;21:28–33.
1419. **(406)** Weber AM, Boudreau Y, Mortimer VD. Stericycle, Inc., Morton, WA. HETA 98-0027-2709. NIOSH, CDC, Cincinnati, OH, 1998.

1420. (407) Johnson KR, Braden CR, Cairns KL, et al. Transmission of *Mycobacterium tuberculosis* from medical waste. *JAMA* 2000;284:1683–8.
1421. (408) Emery R, Sprau D, Lao YJ, Pryor W. Release of bacterial aerosols during infectious waste compaction: An initial hazard evaluation for health care workers. *Am Ind Hyg Assoc J* 1992;53:339–45.
1422. National Committee for Clinical Laboratory Standards (NCCLS). Protection of laboratory workers from instrument biohazards and infectious disease transmitted by blood, body fluids, and tissue. Approved guideline. 1997, NCCLS Document M29-A (ISBN1-56238-339-6).
1423. Snyder JW, Check W. Bioterrorism threats to our future: the role of the clinical microbiology laboratory in detection, identification, and confirmation of biological agents. Report of the October 27–29, 2000 Colloquium, American Academy of Microbiology, American College of Microbiology. Available at: www.asm.org
1424. CDC. List of select agents and biological toxins. At: www.cdc.gov/od/sap/docs/salist.pdf
1425. Bond WW. Survival of hepatitis B virus in the environment. *JAMA* 1984;252:397–8.
1426. Slade JE, Pike EB, Eglin RP, Colbourne JS, Kurtz JB. The survival of human immunodeficiency virus in water, sewage, and sea water. *Water Sci Technol* 1989;21:55–9.
1427. Geertsma RE, Van Asten JAAM. Sterilization of prions. *Zentr Steril* 1995;3:385–94.
1428. (52) Johnson MW, Mitch WE, Heller AH, Spector R. The impact of an educational program on gentamicin use in a teaching hospital. *Am J Med* 1982;73:9–14.
1429. (53) Soumerai SB, Salem-Schatz S, Avorn J, Casteris CS, Ross-Degnan D, Popovsky MA. A controlled trial of educational outreach to improve blood transfusion practice. *JAMA* 1993;270:961–6.
1430. (54) Eisenberg JM. An education program to modify laboratory use by house staff. *J Med Educ* 1977;52:578–81.
1431. (55) Rello J, Quintana E, Ausina V, Puzo V, Puzo C, Net A, Prats G. Risk factors for *Staphylococcus aureus* nosocomial pneumonia in critically ill patients. *Am Rev Respir Dis* 1990;142:1320–4.
1432. (56) McWhinney PHM, Kibbler CC, Hamon MD, et al. Progress in the diagnosis and management of aspergillosis in bone marrow transplantation: 13 years' experience. *Clin Infect Dis* 1993;17:397–404.
1433. (74) Aisner J, Murillo J, Schimpff SC, Steere AC. Invasive aspergillosis in acute leukemia: Correlation with nose cultures and antibiotic use. *Ann Intern Med* 1979;90:4–9.
1434. (102) Rhome FS. Endemic nosocomial filamentous fungal disease: a proposed structure for conceptualizing and studying the environmental hazard. *Infect Control* 1986;7S:124–5.
1435. (Table 1) Mutchler JE. Principles of ventilation. In: NIOSH. The industrial environment — its evaluation and control. Washington, DC: U.S. Department of Health, Education, and Welfare, Public Health Service, NIOSH, 1973. Publication #74-117. Available at: www.cdc.gov/niosh/74-117.html
1436. Breiman RF, Cozen W, Fields BS, et al. Role of air sampling in investigation of an outbreak of Legionnaires' disease associated with exposure to aerosols from an evaporative condenser. *J Infect Dis* 1990;161:1257–61.
1437. (Appendix; 9) CDC. Procedures for the recovery of *Legionella* from the environment. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, CDC, 1992;1–13.
1438. Costerton JW, Lewandowski Z, Caldwell DE, Korber DR, Lappin-Scott HM. Microbial biofilms. *Ann Rev Microbiol* 1995;49:711–45.
1439. LeChevallier MW, Babcock TM, Lee RG. Examination and characterization of distribution system biofilms. *Appl Environ Microbiol* 1987;53:2714–24.
1440. Nagy LA, Olson BH. Occurrence and significance of bacteria, fungi, and yeasts associated with distribution pipe surfaces. *Proceeds of the Water Quality Technology Conference*. Portland, OR: 1985;213–38.
1441. Maki DG, Martin WT. Nationwide epidemic of septicemia caused by contaminated infusion products. IV growth of microbial pathogens in fluids for intravenous infusion. *J Infect Dis* 1975;131:267–72.
1442. Costerton JW, Stewart PS, Greenberg EP. Bacterial biofilms: a common cause of persistent infections. *Science* 1999;284:1318–22.
1443. Costerton JW, Khoury AE, Ward KH, Anwar H. Practical measures to control device-related bacterial infections. *Int J Artif Organs* 1993;16:765–70.
1444. Nickel JC, Costerton JW, McLean RJC, Olson M. Bacterial biofilms: influence on the pathogenesis, diagnosis, and treatment of urinary tract infections. *J Antimicrobial Chemother* 1994;33 (Suppl. A):31–41.
1445. LeChevallier MW, Cawthon CD, Lee RG. Inactivation of biofilm bacteria. *Appl Environ Microbiol* 1988;54:2492–9.

1446. Anwar J, Strap JL, Costerton JW. Establishment of aging biofilms: possible mechanism of bacterial resistance to antimicrobial therapy. *Antimicrobiol Agents Chemotherapy* 1992;36:1347–51.
1447. Stewart PS. Biofilm accumulation model that predicts antibiotic resistance to *Pseudomonas aeruginosa* biofilms. *Antimicrobiol Agents Chemotherapy* 1994;38:1052–8.
1448. Chen X, Stewart PS. Chlorine penetration into artificial biofilm is limited by a reaction-diffusion interaction. *Environ Sci Technol* 1996;30:2078–83.
1449. Huang C-T, Yu FP, McFeters GA, Stewart PS. Non-uniform spatial patterns of respiratory activity with biofilms during disinfection. *Appl Environ Microbiol* 1995;61:2252–6.
1450. Donlan RM, Pipes WO. Selected drinking water characteristics and attached microbial population density. *JAWWA* 1988;80:70–6.
1451. Reasoner DJ, Geldreich EE. A new medium for the enumeration and subculture of bacteria from potable water. *Appl Environ Microbiol* 1985;49:1–7.
1452. Pass T, Wright R, Sharp B, Harding GB. Culture of dialysis fluids on nutrient-rich media for short periods at elevated temperatures underestimates microbial contamination. *Blood Purif* 1996;14:136–45.
1453. Arduino MJ, Bland LA, Agüero SM, et al. Effects of incubation time and temperature on microbiologic sampling procedures for hemodialysis fluids. *J Clin Microbiol* 1991;29:1462–5.
1454. (234) Klein E, Pass T, Harding GB, Wright R, Million C. Microbial and endotoxin contamination in water and dialysate in the central United States. *Artif Organs* 1990;14:85–94.
1455. (235) Man N-K, Degremont A, Darbord J-C, Collet M, Vaillant P. Evidence of bacterial biofilm in tubing from hydraulic pathway of hemodialysis system. *Artif Organs* 1998;22:596–600.
1456. Pearson FC, Weary ME, Sargent HE, et al. Comparison of several control standard endotoxins to the National Reference Standard Endotoxin — an HIMA collaborative study. *Appl Environ Microbiol* 1985;50:91–3.
1457. (Appendix; 1) Arnow PM, Weil D, Para MF. Prevalence of significance of *Legionella pneumophila* contamination of residential hot-tap water systems. *J Infect Dis* 1985;152:145–51.
1458. (Appendix; 2) Shelton BG, Morris GK, Gorman GW. Reducing risks associated with *Legionella* bacteria in building water systems. In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;279–81.
1459. (Appendix; 3) Joly JR. Monitoring for the presence of *Legionella*: where, when, and how? In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;211–6.
1460. (Appendix; 7) Brenner DJ, Feeley JC, Weaver RE. Family VII. *Legionellaceae*. In: Krieg NR, Holt JG, eds. *Bergey's manual of systemic bacteriology*, volume 1. Baltimore, MD: Williams & Wilkins, 1984;279–88.
1461. (Appendix; 8) Katz SM, Hammel JM. The effect of drying, heat, and pH on the survival of *Legionella pneumophila*. *Ann Clin Lab Sci* 1987;17:150–6.
1462. (Box 2) Alary MA, Joly JR. Comparison of culture methods and an immunofluorescence assay for the detection of *Legionella pneumophila* in domestic hot water devices. *Curr Microbiol* 1992;25:19–25.
1463. (Box 2) Vickers RM, Stout JE, Yu VL. Failure of a diagnostic monoclonal immunofluorescent reagent to detect *Legionella pneumophila* in environmental samples. *Appl Environ Microbiol* 1990;56:2912–4.
1464. (Box 2) Flournoy DJ, Belobraydic KA, Silberg SL, Lawrence CH, Guthrie PJ. False positive *Legionella pneumophila* direct immunofluorescence monoclonal antibody test caused by *Bacillus cereus* spores. *Diag Microbiol Infect Dis* 1988;9:123–5.
1465. (Box 2) Bej AK, Majbubani MH, Atlas RM. Detection of viable *Legionella pneumophila* in water by polymerase chain reaction and gene probe methods. *Appl Environ Microbiol* 1991;57:597–600.
1466. Schulze-Röbbecke R, Jung KD, Pullman H, Hundgeburth J. Control of *Legionella pneumophila* in a hospital hot water system. *Zbl Hyg* 1990;190:84–100.
1467. Colbourne JS, Pratt DJ, Smith MG, Fisher-Hoch SP, Harper D. Water fittings as sources of *Legionella pneumophila* in a hospital plumbing system. *Lancet* 1984;1:210–3.
1468. U.S. Environmental Protection Agency. National interim primary drinking water regulations: control of trihalomethanes in drinking water: final rules. *Federal Register* 1979;44:68624–705.
1469. U.S. Environmental Protection Agency. National interim primary drinking water regulations: Trihalomethanes. *Federal Register* 1983;48:8406–14.

Part IV. Appendices

Appendix A. Glossary of Terms

Acceptable indoor air quality: air in which there are no known contaminants at harmful concentrations as determined by knowledgeable authorities and with which a substantial majority ($\geq 80\%$) of the people exposed do not express dissatisfaction.

ACGIH: American Conference of Governmental Industrial Hygienists.

Action level: the concentration of a contaminant at which steps should be taken to interrupt the trend toward higher, unacceptable levels.

Aerosol: particles of respirable size generated by both humans and environmental sources and that have the capability of remaining viable and airborne for extended periods in the indoor environment.

AIA: American Institute of Architects, a professional group responsible for publishing the *Guidelines for Design and Construction of Hospitals and Healthcare Facilities*, a consensus document for design and construction of health-care facilities endorsed by the U.S. Department of Health and Human Services, health-care professionals, and professional organizations.

Air changes per hour (ACH): the ratio of the volume of air flowing through a space in a certain period of time (the airflow rate) to the volume of that space (the room volume). This ratio is expressed as the number of air changes per hour (ACH).

Air mixing: the degree to which air supplied to a room mixes with the air already in the room, usually expressed as a mixing factor. This factor varies from 1 (for perfect mixing) to 10 (for poor mixing). It is used as a multiplier to determine the actual airflow required (i.e., the recommended ACH multiplied by the mixing factor equals the actual ACH required).

Airborne transmission: a means of spreading infection when airborne droplet nuclei (small particle residue of evaporated droplets $\leq 5 \mu\text{m}$ in size containing microorganisms that remain suspended in air for long periods of time) are inhaled by the susceptible host.

Air-cleaning system: a device or combination of devices applied to reduce the concentration of airborne contaminants (e.g., microorganisms, dusts, fumes, aerosols, other particulate matter, and gases).

Air conditioning: the process of treating air to meet the requirements of a conditioned space by controlling its temperature, humidity, cleanliness, and distribution.

Allogeneic: non-twin, non-self. The term refers to transplanted tissue from a donor closely matched to a recipient but not related to that person.

Ambient air: the air surrounding an object.

Anemometer: a flow meter which measures the wind force and velocity of air. An anemometer is often used as a means of determining the volume of air being drawn into an air sampler.

Anteroom: a small room leading from a corridor into an isolation room. This room can act as an airlock, preventing the escape of contaminants from the isolation room into the corridor.

ASHE: American Society for Healthcare Engineering, an association affiliated with the American Hospital Association.

ASHRAE: American Society of Heating, Refrigerating, and Air-Conditioning Engineers Inc.

Autologous: self. The term refers to transplanted tissue whose source is the same as the recipient, or an identical twin.

Automated cyler: a machine used during peritoneal dialysis which pumps fluid into and out of the patient while he/she sleeps.

Biochemical oxygen demand (BOD): a measure of the amount of oxygen removed from aquatic environments by aerobic microorganisms for their metabolic requirements. Measurement of BOD is used to determine the level of organic pollution of a stream or lake. The greater the BOD, the greater

the degree of water pollution. The term is also referred to as Biological Oxygen Demand (BOD).

Biological oxygen demand (BOD): an indirect measure of the concentration of biologically degradable material present in organic wastes (pertaining to water quality). It usually reflects the amount of oxygen consumed in five days by biological processes breaking down organic waste (BOD5).

Biosafety level: a combination of microbiological practices, laboratory facilities, and safety equipment determined to be sufficient to reduce or prevent occupational exposures of laboratory personnel to the microbiological agents they work with. There are four biosafety levels based on the hazards associated with the various microbiological agents.

BOD5: the amount of dissolved oxygen consumed in five days by biological processes breaking down organic matter.

Bonneting: a floor cleaning method for either carpeted or hard surface floors that uses a circular motion of a large fibrous disc to lift and remove soil and dust from the surface.

Capped spur: a pipe leading from the water recirculating system to an outlet that has been closed off ("capped"). A capped spur cannot be flushed, and it might not be noticed unless the surrounding wall is removed.

CFU/m³: colony forming units per cubic meter (of air).

Chlamydo spores: thick-walled, typically spherical or ovoid resting spores asexually produced by certain types of fungi from cells of the somatic hyphae.

Chloramines: compounds containing nitrogen, hydrogen, and chlorine. These are formed by the reaction between hypochlorous acid (HOCl) and ammonia (NH₃) and/or organic amines in water. The formation of chloramines in drinking water treatment extends the disinfecting power of chlorine. The term is also referred to as Combined Available Chlorine.

Cleaning: the removal of visible soil and organic contamination from a device or surface, using either the physical action of scrubbing with a surfactant or detergent and water, or an energy-based process (e.g., ultrasonic cleaners) with appropriate chemical agents.

Coagulation-flocculation: coagulation is the clumping of particles that results in the settling of impurities. It may be induced by coagulants (e.g., lime, alum, and iron salts). Flocculation in water and wastewater treatment is the agglomeration or clustering of colloidal and finely-divided suspended matter after coagulation by gentle stirring by either mechanical or hydraulic means, such that they can be separated from water or sewage.

Commissioning (a room): testing a system or device to ensure that it meets the pre-use specifications as indicated by the manufacturer or predetermined standard, or air sampling in a room to establish a pre-occupancy baseline standard of microbial or particulate contamination. The term is also referred to as benchmarking at 77°F (25°C).

Completely packaged: functionally packaged, as for laundry.

Conidia: asexual spores of fungi borne externally.

Conidiophores: specialized hyphae that bear conidia in fungi.

Conditioned space: that part of a building that is heated or cooled, or both, for the comfort of the occupants.

Contaminant: an unwanted airborne constituent that may reduce the acceptability of air.

Convection: the transfer of heat or other atmospheric properties within the atmosphere or in the airspace of an enclosure by the circulation of currents from one region to another, especially by such motion directed upward.

Cooling tower: a structure engineered to receive accumulated heat from ventilation systems and equipment and transfer this heat to water, which then releases the stored heat to the atmosphere through evaporative cooling.

Critical item (medical instrument): a medical instrument or device that contacts normally sterile areas of the body or enters the vascular system. There is a high risk of infection from such devices if they are microbiologically contaminated prior to use. These devices must be sterilized before use.

Dead legs: areas in the water system where water stagnates. A dead leg is a pipe or spur, leading from the water recirculating system to an outlet that is used infrequently, resulting in inadequate flow of

water from the recirculating system to the outlet. This inadequate flow reduces the perfusion of heat or chlorine into this part of the water distribution system, thereby adversely affecting the disinfection of the water system in that area.

Deionization: removal of ions from water by exchange with other ions associated with fixed charges on a resin bed. Cations are usually removed and H^+ ions are exchanged; OH^- ions are exchanged for anions.

Detritus: particulate matter produced by or remaining after the wearing away or disintegration of a substance or tissue.

Dew point: the temperature at which a gas or vapor condenses to form a liquid; the point at which moisture begins to condense out of the air. At dew point, air is cooled to the point where it is at 100% relative humidity or saturation.

Dialysate: the aqueous electrolyte solution, usually containing dextrose, used to make a concentration gradient between the solution and blood in the hemodialyzer (dialyzer).

Dialyzer: a device that consists of two compartments (blood and dialysate) separated by a semipermeable membrane. A dialyzer is usually referred to as an artificial kidney.

Diffuser: the grille plate that disperses the air stream coming into the conditioned air space.

Direct transmission: involves direct body surface-to-body surface contact and physical transfer of microorganisms between a susceptible host and an infected/colonized person, or exposure to cloud of infectious particles within 3 feet of the source; the aerosolized particles are $>5 \mu m$ in size.

Disability: as defined by the Americans with Disabilities Act, a disability is any physical or mental impairment that substantially limits one or more major life activities, including but not limited to walking, talking, seeing, breathing, hearing, or caring for oneself.

Disinfection: a generally less lethal process of microbial inactivation (compared to sterilization) that eliminates virtually all recognized pathogenic microorganisms but not necessarily all microbial forms (e.g., bacterial spores).

Drain pans: pans that collect water within the HVAC system and remove it from the system. Condensation results when air and steam come together.

Drift: circulating water lost from the cooling tower in the form as liquid droplets entrained in the exhaust air stream (i.e., exhaust aerosols from a cooling tower).

Drift eliminators: an assembly of baffles or labyrinth passages through which the air passes prior to its exit from the cooling tower. The purpose of a drift eliminator is to remove entrained water droplets from the exhaust air.

Droplets: particles of moisture, such as are generated when a person coughs or sneezes, or when water is converted to a fine mist by a device such as an aerator or shower head. These particles may contain infectious microorganisms. Intermediate in size between drops and droplet nuclei, these particles tend to quickly settle out from the air so that any risk of disease transmission is generally limited to persons in close proximity to the droplet source.

Droplet nuclei: sufficiently small particles ($1-5 \mu m$ in diameter) that can remain airborne indefinitely and cause infection when a susceptible person is exposed at or beyond 3 feet of the source of these particles.

Dual duct system: an HVAC system that consists of parallel ducts that produce a cold air stream in one and a hot air stream in the other.

Dust: an air suspension of particles (aerosol) of any solid material, usually with particle sizes $\leq 100 \mu m$ in diameter.

Dust-spot test: a procedure that uses atmospheric air or a defined dust to measure a filter's ability to remove particles. A photometer is used to measure air samples on either side of the filter, and the difference is expressed as a percentage of particles removed.

Effective leakage area: the area through which air can enter or leave the room. This does not include supply, return, or exhaust ducts. The smaller the effective leakage area, the better isolated the room.

Endotoxin: the lipopolysaccharides of gram-negative bacteria, the toxic character of which resides in the lipid portion. Endotoxins generally produce pyrogenic reactions in persons exposed to these

bacterial components.

Enveloped virus: a virus whose outer surface is derived from a membrane of the host cell (either nuclear or the cell's outer membrane) during the budding phase of the maturation process. This membrane-derived material contains lipid, a component that makes these viruses sensitive to the action of chemical germicides.

Evaporative condenser: a wet-type, heat-rejection unit that produces large volumes of aerosols during the process of removing heat from conditioned space air.

Exhaust air: air removed from a space and not reused therein.

Exposure: the condition of being subjected to something (e.g., infectious agents) that could have a harmful effect.

Fastidious: having complex nutritional requirements for growth, as in microorganisms.

Fill: that portion of a cooling tower which makes up its primary heat transfer surface. Fill is alternatively known as "packing."

Finished water: treated, or potable water.

Fixed room-air HEPA recirculation systems: nonmobile devices or systems that remove airborne contaminants by recirculating air through a HEPA filter. These may be built into the room and permanently ducted or may be mounted to the wall or ceiling within the room. In either situation, they are fixed in place and are not easily movable.

Fomite: an inanimate object that may be contaminated with microorganisms and serves in their transmission.

Free and available chlorine: the term applied to the three forms of chlorine that may be found in solution (i.e., chlorine [Cl₂], hypochlorite [OCl⁻], and hypochlorous acid [HOCl]).

Germicide: a chemical that destroys microorganisms. Germicides may be used to inactivate microorganisms in or on living tissue (antiseptics) or on environmental surfaces (disinfectants).

Health-care-associated: an outcome, usually an infection, that occurs in any health-care facility as a result of medical care. The term "health-care-associated" replaces "nosocomial," the latter term being limited to adverse infectious outcomes occurring only in hospitals.

Hemodiafiltration: a form of renal replacement therapy in which waste solutes in the patient's blood are removed by both diffusion and convection through a high-flux membrane.

Hemodialysis: a treatment for renal replacement therapy in which waste solutes in the patient's blood are removed by diffusion and/or convection through the semipermeable membrane of an artificial kidney or dialyzer.

Hemofiltration: cleansing of waste products or other toxins from the blood by convection across a semipermeable, high-flux membrane where fluid balance is maintained by infusion of sterile, pyrogen-free substitution fluid pre- or post-hemodialyzer.

HEPA filter: High Efficiency Particulate Air filters capable of removing 99.97% of particles 0.3 μm in diameter and may assist in controlling the transmission of airborne disease agents. These filters may be used in ventilation systems to remove particles from the air or in personal respirators to filter air before it is inhaled by the person wearing the respirator. The use of HEPA filters in ventilation systems requires expertise in installation and maintenance. To test this type of filter, 0.3 μm particles of dioctylphthalate (DOP) are drawn through the filter. Efficiency is calculated by comparing the downstream and upstream particle counts. The optimal HEPA filter allows only three particles to pass through for every 10,000 particles that are fed to the filter.

Heterotrophic (heterotroph): that which requires some nutrient components from exogenous sources. Heterotrophic bacteria cannot synthesize all of their metabolites and therefore require certain nutrients from other sources.

High-efficiency filter: a filter with a particle-removal efficiency of 90%–95%.

High flux: a type of dialyzer or hemodialysis treatment in which large molecules (>8,000 daltons [e.g., β₂ microglobulin]) are removed from blood.

High-level disinfection: a disinfection process that inactivates vegetative bacteria, mycobacteria, fungi, and viruses, but not necessarily high numbers of bacterial spores.

Housekeeping surfaces: environmental surfaces (e.g., floors, walls, ceilings, and tabletops) that are not involved in direct delivery of patient care in health-care facilities.

Hoyer lift: an apparatus that facilitates the repositioning of the non-ambulatory patient from bed to wheelchair or gurney and subsequently to therapy equipment (immersion tanks).

Hubbard tank: a tank used in hydrotherapy that may accommodate whole-body immersion (e.g., as may be indicated for burn therapy). Use of a Hubbard tank has been replaced largely by bedside post-lavage therapy for wound care management.

HVAC: Heating, Ventilation, Air Conditioning.

Iatrogenic: induced in a patient by a physician's activity, manner, or therapy. The term is used especially in reference to an infectious complication or other adverse outcome of medical treatment.

Impactor: an air-sampling device in which particles and microorganisms are directed onto a solid surface and retained there for assay.

Impingement: an air-sampling method during which particles and microorganisms are directed into a liquid and retained there for assay.

Indirect transmission: involves contact of a susceptible host with a contaminated intermediate object, usually inanimate (a fomite).

Induction unit: the terminal unit of an in-room ventilation system. Induction units take centrally conditioned air and further moderate its temperature. Induction units are not appropriate for areas with high exhaust requirements (e.g., research laboratories).

Intermediate-level disinfection: a disinfection process that inactivates vegetative bacteria, most fungi, mycobacteria, and most viruses (particularly the enveloped viruses), but does not inactivate bacterial spores.

Isoform: a possible configuration (tertiary structure) of a protein molecule. With respect to prion proteins, the molecules with large amounts of α -conformation are the normal isoform of that particular protein, whereas those prions with large amounts of β -sheet conformation are the proteins associated with the development of spongiform encephalopathy (e.g., Creutzfeldt-Jakob disease [CJD]).

Laminar flow: HEPA-filtered air that is blown into a room at a rate of 90 ± 10 feet/min in a unidirectional pattern with 100 ACH–400 ACH.

Large enveloped virus: viruses whose particle diameter is >50 nm and whose outer surface is covered by a lipid-containing structure derived from the membranes of the host cells. Examples of large enveloped viruses include influenza viruses, herpes simplex viruses, and poxviruses.

Laser plume: the transfer of electromagnetic energy into tissues which results in a release of particles, gases, and tissue debris.

Lipid-containing viruses: viruses whose particle contains lipid components. The term is generally synonymous with enveloped viruses whose outer surface is derived from host cell membranes. Lipid-containing viruses are sensitive to the inactivating effects of liquid chemical germicides.

Lithotriptors: instruments used for crushing calculi (i.e., calcified stones, and sand) in the bladder or kidneys.

Low efficiency filter: the prefilter with a particle-removal efficiency of approximately 30% through which incoming air first passes. See also Prefilter.

Low-level disinfection: a disinfection process that will inactivate most vegetative bacteria, some fungi, and some viruses, but cannot be relied upon to inactivate resistant microorganisms (e.g., mycobacteria or bacterial spores).

Makeup air: outdoor air supplied to the ventilation system to replace exhaust air.

Makeup water: a cold water supply source for a cooling tower.

Manometer: a device that measures the pressure of liquids and gases. A manometer is used to verify air filter performance by measuring pressure differentials on either side of the filter.

Membrane filtration: an assay method suitable for recovery and enumeration of microorganisms from liquid samples. This method is used when sample volume is large and anticipated microbial contamination levels are low.

Mesophilic: that which favors a moderate temperature. For mesophilic bacteria, a temperature range of

68°F–131°F (20°C–55°C) is favorable for their growth and proliferation.

Mixing box: the site where the cold and hot air streams mix in the HVAC system, usually situated close to the air outlet for the room.

Mixing faucet: a faucet that mixes hot and cold water to produce water at a desired temperature.

MMAD: Mass Median Aerodynamic Diameter. This is the unit used by ACGIH to describe the size of particles when particulate air sampling is conducted.

Moniliaceous: hyaline or brightly colored. This is a laboratory term for the distinctive characteristics of certain opportunistic fungi in culture (e.g., *Aspergillus* spp. and *Fusarium* spp.).

Monochloramine: the result of the reaction between chlorine and ammonia that contains only one chlorine atom. Monochloramine is used by municipal water systems as a water treatment.

Natural ventilation: the movement of outdoor air into a space through intentionally provided openings (i.e., windows, doors, or nonpowered ventilators).

Negative pressure: air pressure differential between two adjacent airspaces such that air flow is directed into the room relative to the corridor ventilation (i.e., room air is prevented from flowing out of the room and into adjacent areas).

Neutropenia: a medical condition in which the patient's concentration of neutrophils is substantially less than that in the normal range. Severe neutropenia occurs when the concentration is <1,000 polymorphonuclear cells/ μ L for 2 weeks or <100 polymorphonuclear cells /mL for 1 week, particularly for hematopoietic stem cell transplant (HSCT) recipients.

Noncritical devices: medical devices or surfaces that come into contact with only intact skin. The risk of infection from use of these devices is low.

Non-enveloped virus: a virus whose particle is not covered by a structure derived from a membrane of the host cell. Non-enveloped viruses have little or no lipid compounds in their biochemical composition, a characteristic that is significant to their inherent resistance to the action of chemical germicides.

Nosocomial: an occurrence, usually an infection, that is acquired in a hospital as a result of medical care.

NTM: nontuberculous mycobacteria. These organisms are also known as atypical mycobacteria, or as "Mycobacteria other than tuberculosis" (MOTT). This descriptive term refers to any of the fast- or slow-growing *Mycobacterium* spp. found in primarily in natural or man-made waters, but it excludes *Mycobacterium tuberculosis* and its variants.

Nuisance dust: generally innocuous dust, not recognized as the direct cause of serious pathological conditions.

Oocysts: a cyst in which sporozoites are formed; a reproductive aspect of the life cycle of a number of parasitic agents (e.g., *Cryptosporidium* spp., and *Cyclospora* spp.).

Outdoor air: air taken from the external atmosphere and, therefore, not previously circulated through the ventilation system.

Parallel streamlines: a unidirectional airflow pattern achieved in a laminar flow setting, characterized by little or no mixing of air.

Particulate matter (particles): a state of matter in which solid or liquid substances exist in the form of aggregated molecules or particles. Airborne particulate matter is typically in the size range of 0.01–100 μ m diameter.

Pasteurization: a disinfecting method for liquids during which the liquids are heated to 140°F (60°C) for a short time (\geq 30 mins.) to significantly reduce the numbers of pathogenic or spoilage microorganisms.

Plinth: a treatment table or a piece of equipment used to reposition the patient for treatment.

Portable room-air HEPA recirculation units: free-standing portable devices that remove airborne contaminants by recirculating air through a HEPA filter.

Positive pressure: air pressure differential between two adjacent air spaces such that air flow is directed from the room relative to the corridor ventilation (i.e., air from corridors and adjacent areas is prevented from entering the room).

Potable (drinking) water: water that is fit to drink. The microbiological quality of this water as defined by EPA microbiological standards from the Surface Water Treatment Rule: a) *Giardia lamblia*: 99.9% killed/inactivated; b) viruses: 99.9% inactivated; c) *Legionella* spp.: no limit, but if *Giardia* and viruses are inactivated, *Legionella* will also be controlled; d) heterotrophic plate count [HPC]: ≤ 500 CFU/mL; and e) $>5\%$ of water samples total coliform-positive in a month.

PPE: Personal Protective Equipment.

ppm: parts per million. The term is a measure of concentration in solution. Chlorine bleaches (undiluted) that are available in the U.S. (5.25%–6.15% sodium hypochlorite) contain approximately 50,000–61,500 parts per million of free and available chlorine.

Prefilter: the first filter for incoming fresh air in a HVAC system. This filter is approximately 30% efficient in removing particles from the air. See also Low-Efficiency Filter.

Prion: a class of agent associated with the transmission of diseases known as transmissible spongiform encephalopathies (TSEs). Prions are considered to consist of protein only, and the abnormal isoform of this protein is thought to be the agent that causes diseases such as Creutzfeldt-Jakob disease (CJD), kuru, scrapie, bovine spongiform encephalopathy (BSE), and the human version of BSE which is variant CJD (vCJD).

Product water: water produced by a water treatment system or individual component of that system.

Protective environment: a special care area, usually in a hospital, designed to prevent transmission of opportunistic airborne pathogens to severely immunosuppressed patients.

Pseudoepidemic (pseudo-outbreak): a cluster of positive microbiologic cultures in the absence of clinical disease. A pseudoepidemic usually results from contamination of the laboratory apparatus and process used to recover microorganisms.

Pyrogenic: an endotoxin burden such that a patient would receive ≥ 5 endotoxin units (EU) per kilogram of body weight per hour, thereby causing a febrile response. In dialysis this usually refers to water or dialysate having endotoxin concentrations of ≥ 5 EU/mL.

Rank order: a strategy for assessing overall indoor air quality and filter performance by comparing airborne particle counts from lowest to highest (i.e., from the best filtered air spaces to those with the least filtration).

RAPD: a method of genotyping microorganisms by randomly amplified polymorphic DNA. This is one version of the polymerase chain reaction method.

Recirculated air: air removed from the conditioned space and intended for reuse as supply air.

Relative humidity: the ratio of the amount of water vapor in the atmosphere to the amount necessary for saturation at the same temperature. Relative humidity is expressed in terms of percent and measures the percentage of saturation. At 100% relative humidity, the air is saturated. The relative humidity decreases when the temperature is increased without changing the amount of moisture in the air.

Reprocessing (of medical instruments): the procedures or steps taken to make a medical instrument safe for use on the next patient. Reprocessing encompasses both cleaning and the final or terminal step (i.e., sterilization or disinfection) which is determined by the intended use of the instrument according to the Spaulding classification.

Residuals: the presence and concentration of a chemical in media (e.g., water) or on a surface after the chemical has been added.

Reservoir: a nonclinical source of infection.

Respirable particles: those particles that penetrate into and are deposited in the nonciliated portion of the lung. Particles $>10 \mu\text{m}$ in diameter are not respirable.

Return air: air removed from a space to be then recirculated.

Reverse osmosis (RO): an advanced method of water or wastewater treatment that relies on a semi-permeable membrane to separate waters from pollutants. An external force is used to reverse the normal osmotic process resulting in the solvent moving from a solution of higher concentration to one of lower concentration.

Riser: water piping that connects the circulating water supply line, from the level of the base of the tower or supply header, to the tower's distribution system.

RODAC: Replicate Organism Direct Agar Contact. This term refers to a nutrient agar plate whose convex agar surface is directly pressed onto an environmental surface for the purpose of microbiologic sampling of that surface.

Room-air HEPA recirculation systems and units: devices (either fixed or portable) that remove airborne contaminants by recirculating air through a HEPA filter.

Routine sampling: environmental sampling conducted without a specific, intended purpose and with no action plan dependent on the results obtained.

Sanitizer: an agent that reduces microbial contamination to safe levels as judged by public health standards or requirements.

Saprophytic: a naturally-occurring microbial contaminant.

Sedimentation: the act or process of depositing sediment from suspension in water. The term also refers to the process whereby solids settle out of wastewater by gravity during treatment.

Semicritical devices: medical devices that come into contact with mucous membranes or non-intact skin.

Service animal: any animal individually trained to do work or perform tasks for the benefit of a person with a disability.

Shedding: the generation and dispersion of particles and spores by sources within the patient area, through activities such as patient movement and airflow over surfaces.

Single-pass ventilation: ventilation in which 100% of the air supplied to an area is exhausted to the outside.

Small, non-enveloped viruses: viruses whose particle diameter is <50 nm and whose outer surface is the protein of the particle itself and not that of host cell membrane components. Examples of small, non-enveloped viruses are polioviruses and hepatitis A virus.

Spaulding Classification: the categorization of inanimate medical device surfaces in the medical environment as proposed in 1972 by Dr. Earle Spaulding. Surfaces are divided into three general categories, based on the theoretical risk of infection if the surfaces are contaminated at time of use. The categories are “critical,” “semicritical,” and “noncritical.”

Specific humidity: the mass of water vapor per unit mass of moist air. It is expressed as grains of water per pound of dry air, or pounds of water per pound of dry air. The specific humidity changes as moisture is added or removed. However, temperature changes do not change the specific humidity unless the air is cooled below the dew point.

Splatter: visible drops of liquid or body fluid that are expelled forcibly into the air and settle out quickly, as distinguished from particles of an aerosol which remain airborne indefinitely.

Steady state: the usual state of an area.

Sterilization: the use of a physical or chemical procedure to destroy all microbial life, including large numbers of highly-resistant bacterial endospores.

Stop valve: a valve that regulates the flow of fluid through a pipe. The term may also refer to a faucet.

Substitution fluid: fluid that is used for fluid management of patients receiving hemodiafiltration. This fluid can be prepared on-line at the machine through a series of ultrafilters or with the use of sterile peritoneal dialysis fluid.

Supply air: air that is delivered to the conditioned space and used for ventilation, heating, cooling, humidification, or dehumidification.

Tensile strength: the resistance of a material to a force tending to tear it apart, measured as the maximum tension the material can withstand without tearing.

Therapy animal: an animal (usually a personal pet) that, with their owners or handlers, provide supervised, goal-directed intervention to clients in hospitals, nursing homes, special-population schools, and other treatment sites.

Thermophilic: capable of growing in environments warmer than body temperature.

Thermotolerant: capable of withstanding high temperature conditions.

TLV®: an exposure level under which most people can work consistently for 8 hours a day, day after day, without adverse effects. The term is used by the ACGIH to designate degree of exposure to

contaminants. TLV® can be expressed as approximate milligrams of particulate per cubic meter of air (mg/m^3). TLVs® are listed as either an 8-hour TWA (time weighted average) or a 15-minute STEL (short term exposure limit).

TLV-TWA: Threshold Limit Value-Time Weighted Average. The term refers to the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed repeatedly, day after day, without adverse effects. The TLV-TWA for “particulates (insoluble) not otherwise classified” (PNOC) - (sometimes referred to as nuisance dust) - are those particulates containing no asbestos and <1% crystalline silica. A TLV-TWA of $10 \text{ mg}/\text{m}^3$ for inhalable particulates and a TLV-TWA of $3 \text{ mg}/\text{m}^3$ for respirable particulates (particulates $\leq 5 \mu\text{m}$ in aerodynamic diameter) have been established.

Total suspended particulate matter: the mass of particles suspended in a unit of volume of air when collected by a high-volume air sampler.

Transient: a change in the condition of the steady state that takes a very short time compared with the steady state. Opening a door, and shaking bed linens are examples of transient activities.

TWA: average exposure for an individual over a given working period, as determined by sampling at given times during the period. TWA is usually presented as the average concentration over an 8-hour workday for a 40-hour workweek.

Ultraclean air: air in laminar flow ventilation that has also passed through a bank of HEPA filters.

Ultrafilter: a membrane filter with a pore size in the range of $0.001\text{--}0.05 \mu\text{m}$, the performance of which is usually rated in terms of a nominal molecular weight cut-off (defined as the smallest molecular weight species for which the filter membrane has more than 90% rejection).

Ultrafiltered dialysate: the process by which dialysate is passed through a filter having a molecular weight cut-off of approximately 1 kilodalton for the purpose of removing bacteria and endotoxin from the bath.

Ultraviolet germicidal irradiation (UVGI): the use of ultraviolet radiation to kill or inactivate microorganisms.

Ultraviolet germicidal irradiation lamps: lamps that kill or inactivate microorganisms by emitting ultraviolet germicidal radiation, predominantly at a wavelength of 254 nm. UVGI lamps can be used in ceiling or wall fixtures or within air ducts of ventilation systems.

Vapor pressure: the pressure exerted by free molecules at the surface of a solid or liquid. Vapor pressure is a function of temperature, increasing as the temperature rises.

Vegetative bacteria: bacteria that are actively growing and metabolizing, as opposed to a bacterial state of quiescence that is achieved when certain bacteria (gram-positive bacilli) convert to spores when the environment can no longer support active growth.

Vehicle: any object, person, surface, fomite, or media that may carry and transfer infectious microorganisms from one site to another.

Ventilation: the process of supplying and removing air by natural or mechanical means to and from any space. Such air may or may not be conditioned.

Ventilation air: that portion of the supply air consisting of outdoor air plus any recirculated air that has been treated for the purpose of maintaining acceptable indoor air quality.

Ventilation, dilution: an engineering control technique to dilute and remove airborne contaminants by the flow of air into and out of an area. Air that contains droplet nuclei is removed and replaced by contaminant-free air. If the flow is sufficient, droplet nuclei become dispersed, and their concentration in the air is diminished.

Ventilation, local exhaust: ventilation used to capture and removed airborne contaminants by enclosing the contaminant source (the patient) or by placing an exhaust hood close to the contaminant source.

v/v: volume to volume. This term is an expression of concentration of a percentage solution when the principle component is added as a liquid to the diluent.

w/v: weight to volume. This term is an expression of concentration of a percentage solution when the principle component is added as a solid to the diluent.

Weight-arrestance: a measure of filter efficiency, used primarily when describing the performance of low- and medium-efficiency filters. The measurement of weight-arrestance is performed by feeding a standardized synthetic dust to the filter and weighing the fraction of the dust removed.

Appendix B. Air

1. Airborne Contaminant Removal

Table B.1. Air changes/hour (ACH) and time required for airborne-contaminant removal efficiencies of 99% and 99.9%*

ACH+ § ¶	Time (mins.) required for removal:	
	99% efficiency	99.9% efficiency
2	138	207
4	69	104
6	46	69
8	35	52
10	28	41
12	23	35
15	18	28
20	14	21
50	6	8

* This table is revised from Table S3-1 in reference 4 and has been adapted from the formula for the rate of purging airborne contaminants presented in reference 1435.

+ Shaded entries denote frequently cited ACH for patient-care areas.

§ Values were derived from the formula:

$$t_2 - t_1 = -[\ln(C_2 / C_1) / (Q / V)] \times 60, \text{ with } t_1 = 0 \text{ and where}$$

t_1 = initial timepoint in minutes

t_2 = final timepoint in minutes

C_1 = initial concentration of contaminant

C_2 = final concentration of contaminant

$C_2 / C_1 = 1 - (\text{removal efficiency} / 100)$

Q = air flow rate in cubic feet/hour

V = room volume in cubic feet

$Q / V = \text{ACH}$

¶ Values apply to an empty room with no aerosol-generating source. With a person present and generating aerosol, this table would not apply. Other equations are available that include a constant generating source. However, certain diseases (e.g., infectious tuberculosis) are not likely to be aerosolized at a constant rate. The times given assume perfect mixing of the air within the space (i.e., mixing factor = 1). However, perfect mixing usually does not occur. Removal times will be longer in rooms or areas with imperfect mixing or air stagnation.²¹³ Caution should be exercised in using this table in such situations. For booths or other local ventilation enclosures, manufacturers' instructions should be consulted.

2. Air Sampling for Aerosols Containing Legionellae

Air sampling is an insensitive means of detecting *Legionella pneumophila*, and is of limited practical value in environmental sampling for this pathogen. In certain instances, however, it can be used to a) demonstrate the presence of legionellae in aerosol droplets associated with suspected bacterial

reservoirs; b) define the role of certain devices [e.g., showers, faucets, decorative fountains, or evaporate condensers] in disease transmission; and c) quantitate and determine the size of the droplets containing legionellae.¹⁴³⁶ Stringent controls and calibration are necessary when sampling is used to determine particle size and numbers of viable bacteria.¹⁴³⁷ Samplers should be placed in locations where human exposure to aerosols is anticipated, and investigators should wear a NIOSH-approved respirator (e.g., N95 respirator) if sampling involves exposure to potentially infectious aerosols.

Methods used to sample air for legionellae include impingement in liquid, impaction on solid medium, and sedimentation using settle plates.¹⁴³⁶ The Chemical Corps.-type all-glass impingers (AGI) with the stem 30 mm from the bottom of the flask have been used successfully to sample for legionellae.¹⁴³⁶ Because of the velocity at which air samples are collected, clumps tend to become fragmented, leading to a more accurate count of bacteria present in the air. The disadvantages of this method are a) the velocity of collection tends to destroy some vegetative cells; b) the method does not differentiate particle sizes; and c) AGIs are easily broken in the field. Yeast extract broth (0.25%) is the recommended liquid medium for AGI sampling of legionellae;¹⁴³⁷ standard methods for water samples can be used to culture these samples.

Andersen samplers are viable particle samplers in which particles pass through jet orifices of decreasing size in cascade fashion until they impact on an agar surface.¹²¹⁸ The agar plates are then removed and incubated. The stage distribution of the legionellae should indicate the extent to which the bacteria would have penetrated the respiratory system. The advantages of this sampling method are a) the equipment is more durable during use; b) the sampler can determine the number and size of droplets containing legionellae; c) the agar plates can be placed directly in an incubator with no further manipulations; and d) both selective and nonselective BCYE agar can be used. If the samples must be shipped to a laboratory, they should be packed and shipped without refrigeration as soon as possible.

3. Calculation of Air Sampling Results

Assuming that each colony on the agar plate is the growth from a single bacteria-carrying particle, the contamination of the air being sampled is determined from the number of colonies counted. The airborne microorganisms may be reported in terms of the number per cubic foot of air sampled. The following formulas can be applied to convert colony counts to organisms per cubic foot of air sampled.¹²¹⁸

For solid agar impactor samplers:

$$C / (R \times P) = N \quad \text{where}$$

N = number of organisms collected per cubic foot of air sampled
 C = total plate count
 R = airflow rate in cubic feet per minute
 P = duration of sampling period in minutes

For liquid impingers:

$$(C \times V) / (Q \times P \times R) = N \quad \text{where}$$

C = total number of colonies from all aliquots plated
 V = final volume in mL of collecting media
 Q = total number of mL plated
 P, R, and N are defined as above

Area designation	Air movement relationship to adjacent area ²	Minimum air changes of outdoor air per hour ³	Minimum total air changes per hour ^{4, 5}	All air exhausted directly to outdoors ⁶	Recirculated by means of room units ⁷	Relative humidity ⁸ (%)	Design temperature ⁹ (degrees F [C])
<u>Ancillary</u>							
Radiology¹⁹							
X-ray (surgical/critical care and catheterization)	Out	3	15	–	No	30-60	70–75 (21–24)
X-ray (diagnostic & treatment)	–	–	6	–	–	–	75 (24)
Darkroom	In	–	10	Yes	No	–	–
Laboratory							
General ¹⁹	–	–	6	–	–	–	75 (24)
Biochemistry ¹⁹	Out	–	6	–	No	–	75 (24)
Cytology	In	–	6	Yes	No	–	75 (24)
Glass washing	In	–	10	Yes	–	–	–
Histology	In	–	6	Yes	No	–	75 (24)
Microbiology ¹⁹	In	–	6	Yes	No	–	75 (24)
Nuclear medicine	In	–	6	Yes	No	–	75 (24)
Pathology	In	–	6	Yes	No	–	75 (24)
Serology	Out	–	6	–	No	–	75 (24)
Sterilizing	In	–	10	Yes	–	–	–
Autopsy room ¹¹	–	–	12	Yes	No	–	–
Nonrefrigerated body-holding room	In	–	10	Yes	–	–	70 (21)
Pharmacy	Out	–	4	–	–	–	–
<u>Diagnostic and treatment</u>							
Examination room	–	–	6	–	–	–	75 (24)
Medication room	Out	–	4	–	–	–	–
Treatment room	–	–	6	–	–	–	75 (24)
Physical therapy and hydrotherapy	In	–	6	–	–	–	75 (24)
Soiled workroom or soiled holding	In	–	10	Yes	No	–	–
Clean workroom or clean holding	Out	–	4	–	–	–	–
<u>Sterilizing and supply</u>							
ETO-sterilizer room	In	–	10	Yes	No	30-60	75 (24)
Sterilizer equipment room	In	–	10	Yes	–	–	–
Central medical and surgical supply							
Soiled or decontamination room	In	–	6	Yes	No	–	68–73 (20–23)
Clean workroom	Out	–	4	–	No	30-60	75 (24)
Sterile storage	Out	–	4	–	–	(Max.) 70	–

Area designation	Air movement relationship to adjacent area ²	Minimum air changes of outdoor air per hour ³	Minimum total air changes per hour ^{4, 5}	All air exhausted directly to outdoors ⁶	Recirculated by means of room units ⁷	Relative humidity ⁸ (%)	Design temperature ⁹ (degrees F [C])
Service							
Food preparation center ²⁰	–	–	10	–	No	–	–
Ware washing	In	–	10	Yes	No	–	–
Dietary day storage	In	–	2	–	–	–	–
Laundry, general	–	–	10	Yes	–	–	–
Soiled linen (sorting and storage)	In	–	10	Yes	No	–	–
Clean linen storage	Out	–	2	–	–	–	–
Soiled linen and trash chute room	In	–	10	Yes	No	–	–
Bedpan room	In	–	10	Yes	–	–	–
Bathroom	In	–	10	–	–	–	75 (24)
Janitor's closet	In	–	10	Yes	No	–	–

Notes:

1. The ventilation rates in this table cover ventilation for comfort, as well as for asepsis and odor control in areas of acute care hospitals that directly affect patient care and are determined based on health-care facilities being predominantly “No Smoking” facilities. Where smoking may be allowed, ventilation rates will need adjustment. Areas where specific ventilation rates are not given in the table shall be ventilated in accordance with ASHRAE Standard 62, *Ventilation for Acceptable Indoor Air Quality*, and ASHRAE *Handbook - HVAC Applications*. Specialized patient care areas, including organ transplant units, burn units, specialty procedure rooms, etc., shall have additional ventilation provisions for air quality control as may be appropriate. OSHA standards and/or NIOSH criteria require special ventilation requirements for employee health and safety within health-care facilities.
2. Design of the ventilation system shall provide air movement which is generally from clean to less clean areas. If any form of variable air volume or load shedding system is used for energy conservation, it must not compromise the corridor-to-room pressure balancing relationships or the minimum air changes required by the table.
3. To satisfy exhaust needs, replacement air from the outside is necessary. Table B2 does not attempt to describe specific amounts of outside air to be supplied to individual spaces except for certain areas such as those listed. Distribution of the outside air, added to the system to balance required exhaust, shall be as required by good engineering practice. Minimum outside air quantities shall remain constant while the system is in operation.
4. Number of air changes may be reduced when the room is unoccupied if provisions are made to ensure that the number of air changes indicated is reestablished any time the space is being utilized. Adjustments shall include provisions so that the direction of air movement shall remain the same when the number of air changes is reduced. Areas not indicated as having continuous directional control may have ventilation systems shut down when space is unoccupied and ventilation is not otherwise needed, if the maximum infiltration or exfiltration permitted in Note 2 is not exceeded and if adjacent pressure balancing relationships are not compromised. Air quantity calculations must account for filter loading such that the indicated air change rates are provided up until the time of filter change-out.
5. Air change requirements indicated are minimum values. Higher values should be used when required to maintain indicated room conditions (temperature and humidity), based on the cooling load of the space (lights, equipment, people, exterior walls and windows, etc.).

6. Air from areas with contamination and/or odor problems shall be exhausted to the outside and not recirculated to other areas. Note that individual circumstances may require special consideration for air exhaust to the outside, (e.g., in intensive care units in which patients with pulmonary infection are treated) and rooms for burn patients.
7. Recirculating room HVAC units refer to those local units that are used primarily for heating and cooling of air, and not disinfection of air. Because of cleaning difficulty and potential for buildup of contamination, recirculating room units shall not be used in areas marked “No.” However, for airborne infection control, air may be recirculated within individual isolation rooms if HEPA filters are used. Isolation and intensive care unit rooms may be ventilated by reheat induction units in which only the primary air supplied from a central system passes through the reheat unit. Gravity-type heating or cooling units such as radiators or convectors shall not be used in operating rooms and other special care areas. See this table’s Appendix I for a description of recirculation units to be used in isolation rooms (A7).
8. The ranges listed are the minimum and maximum limits where control is specifically needed. The maximum and minimum limits are not intended to be independent of a space’s associated temperature. The humidity is expected to be at the higher end of the range when the temperature is also at the higher end, and vice versa.
9. Where temperature ranges are indicated, the systems shall be capable of maintaining the rooms at any point within the range during normal operation. A single figure indicates a heating or cooling capacity of at least the indicated temperature. This is usually applicable when patients may be undressed and require a warmer environment. Nothing in these guidelines shall be construed as precluding the use of temperatures lower than those noted when the patients' comfort and medical conditions make lower temperatures desirable. Unoccupied areas such as storage rooms shall have temperatures appropriate for the function intended.
10. National Institute for Occupational Safety and Health (NIOSH) criteria documents regarding “Occupational Exposure to Waste Anesthetic Gases and Vapors,” and “Control of Occupational Exposure to Nitrous Oxide” indicate a need for both local exhaust (scavenging) systems and general ventilation of the areas in which the respective gases are utilized.
11. Differential pressure shall be a minimum of 0.01" water gauge (2.5 Pa). If alarms are installed, allowances shall be made to prevent nuisance alarms of monitoring devices.
12. Some surgeons may require room temperatures which are outside of the indicated range. All operating room design conditions shall be developed in consultation with surgeons, anesthesiologists, and nursing staff.
13. The term “trauma room” as used here is the operating room space in the emergency department or other trauma reception area that is used for emergency surgery. The “first aid room” and/or “emergency room” used for initial treatment of accident victims may be ventilated as noted for the “treatment room.” Treatment rooms used for bronchoscopy shall be treated as Bronchoscopy rooms. Treatment rooms used for cryosurgery procedures with nitrous oxide shall contain provisions for exhausting waste gases.
14. In a ventilation system that recirculates air, HEPA filters can be used in lieu of exhausting the air from these spaces to the outside. In this application, the return air shall be passed through the HEPA filters before it is introduced into any other spaces.
15. If it is not practical to exhaust the air from the airborne infection isolation room to the outside, the air may be returned through HEPA filters to the air-handling system exclusively serving the isolation room.
16. Total air changes per room for patient rooms, labor/delivery/recovery rooms, and labor/delivery/recovery/postpartum rooms may be reduced to 4 when supplemental heating and/or cooling systems (radiant heating and cooling, baseboard heating, etc.) are used.
17. The protective environment airflow design specifications protect the patient from common environmental airborne infectious microbes (i.e., *Aspergillus* spores). These special ventilation areas shall be designed to provide directed airflow from the cleanest patient care area to less clean areas. These rooms shall be protected with HEPA filters at 99.97 percent efficiency for a 0.3 μm sized particle in the supply airstream. These interrupting filters protect patient rooms from maintenance-derived release of environmental microbes from the ventilation system components. Recirculation HEPA filters can be used to increase the equivalent room air exchanges. Constant volume airflow is required for consistent ventilation for the protected environment. If the facility determines that airborne infection isolation is necessary for protective environment patients, an anteroom should be

provided. Rooms with reversible airflow provisions for the purpose of switching between protective environment and airborne infection isolation functions are not acceptable.

18. The infectious disease isolation room described in these guidelines is to be used for isolating the airborne spread of infectious diseases, such as measles, varicella, or tuberculosis. The design of airborne infection isolation (AII) rooms should include the provision for normal patient care during periods not requiring isolation precautions. Supplemental recirculating devices may be used in the patient room to increase the equivalent room air exchanges; however, such recirculating devices do not provide the outside air requirements. Air may be recirculated within individual isolation rooms if HEPA filters are used. Rooms with reversible airflow provisions for the purpose of switching between protective environment and AII functions are not acceptable.

19. When required, appropriate hoods and exhaust devices for the removal of noxious gases or chemical vapors shall be provided (see Section 7.31.D14 and 7.31.D15 in the AIA guideline [reference 120] and NFPA 99).

20. Food preparation centers shall have ventilation systems whose air supply mechanisms are interfaced appropriately with exhaust hood controls or relief vents so that exfiltration or infiltration to or from exit corridors does not compromise the exit corridor restrictions of NFPA 90A, the pressure requirements of NFPA 96, or the maximum defined in the table. The number of air changes may be reduced or varied to any extent required for odor control when the space is not in use. See Section 7.31.D1.p in the AIA guideline (reference 120).

Appendix I:

A7. Recirculating devices with HEPA filters may have potential uses in existing facilities as interim, supplemental environmental controls to meet requirements for the control of airborne infectious agents. Limitations in design must be recognized. The design of either portable or fixed systems should prevent stagnation and short circuiting of airflow. The supply and exhaust locations should direct clean air to areas where health-care workers are likely to work, across the infectious source, and then to the exhaust, so that the health-care worker is not in position between the infectious source and the exhaust location. The design of such systems should also allow for easy access for scheduled preventative maintenance and cleaning.

A11. The verification of airflow direction can include a simple visual method such as smoke trail, ball-in-tube, or flutterstrip. These devices will require a minimum differential air pressure to indicate airflow direction.

Table B.3. Pressure relationships and ventilation of certain areas of nursing facilities¹

Notes: This table is Table 8.1 in the AIA guidelines, 2001 edition. Superscripts used in this table refer to notes following the table.

Area designation	Air movement relationship to adjacent area ²	Minimum air changes of outdoor air per hour ³	Minimum total air changes per hour ⁴	All air exhausted directly to outdoors ⁵	Recirculated by means of room units ⁶	Relative humidity ⁷ (%)	Design temperature ⁸ (degrees F [C])
Resident room	–	2	2	–	–	⁹	70–75 (21–24)
Resident unit corridor	–	–	4	–	–	⁹	
Resident gathering areas	–	4	4	–	–	–	–
Toilet room	In	–	10	Yes	No	–	–
Dining rooms	–	2	4	–	–	–	75 (24)
Activity rooms, if provided	–	4	4	–	–	–	–
Physical therapy	In	2	6	–	–	–	75 (24)
Occupational therapy	In	2	6	–	–	–	75.(24)
Soiled workroom or soiled holding	In	2	10	Yes	No	–	–
Clean workroom or clean holding	Out	2	4	–	–	(Max. 70)	75 (24)
Sterilizer exhaust room	In	–	10	Yes	No	–	–
Linen and trash chute room, if provided	In	–	10	Yes	No	–	–
Laundry, general, if provided	–	2	10	Yes	No	–	–
Soiled linen sorting and storage	In	–	10	Yes	No	–	–
Clean linen storage	Out	–	2	Yes	No	–	–
Food preparation facilities ¹⁰	–	2	10	Yes	No	–	–
Dietary warewashing	In	–	10	Yes	No	–	–
Dietary storage areas	–	–	2	Yes	No	–	–
Housekeeping rooms	In	–	10	Yes	No	–	–
Bathing rooms	In	–	10	Yes	No	–	75 (24)

Notes:

1. The ventilation rates in this table cover ventilation for comfort, as well as for asepsis and odor control in areas of nursing facilities that directly affect resident care and are determined based on nursing facilities being predominantly “No Smoking” facilities. Where smoking may be allowed, ventilation rates will need adjustment. Areas where specific ventilation rates are not given in the table shall be ventilated in accordance with ASHRAE Standard 62, *Ventilation for Acceptable Indoor Air Quality*, and ASHRAE *Handbook - HVAC Applications*. OSHA standards and/or NIOSH criteria require special ventilation requirements for employee health and safety within nursing facilities.

2. Design of the ventilation system shall, insofar as possible, provide that air movement is from clean to less clean areas. However, continuous compliance may be impractical with full utilization of some forms of variable air volume and load shedding systems that may be used for energy conservation. Areas that do require positive and continuous control are noted with “Out” or “In” to indicate the required direction of air movement in relation to the space named. Rate of air movement may, of course, be varied as needed

within the limits required for positive control. Where indication of air movement direction is enclosed in parentheses, continuous directional control is required only when the specialized equipment or device is in use or where room use may otherwise compromise the intent of movement from clean to less clean. Air movement for rooms with dashes and nonpatient areas may vary as necessary to satisfy the requirements of those spaces. Additional adjustments may be needed when space is unused or unoccupied and air systems are deenergized or reduced.

3. To satisfy exhaust needs, replacement air from outside is necessary. Table B.3 does not attempt to describe specific amounts of outside air to be supplied to individual spaces except for certain areas such as those listed. Distribution of the outside air, added to the system to balance required exhaust, shall be as required by good engineering practice.
4. Number of air changes may be reduced when the room is unoccupied if provisions are made to ensure that the number of air changes indicated is reestablished any time the space is being utilized. Adjustments shall include provisions so that the direction of air movement shall remain the same when the number of air changes is reduced. Areas not indicated as having continuous directional control may have ventilation systems shut down when space is unoccupied and ventilation is not otherwise needed.
5. Air from areas with contamination and/or odor problems shall be exhausted to the outside and not recirculated to other areas. Note that individual circumstances may require special consideration for air exhaust to outside.
6. Because of cleaning difficulty and potential for buildup of contamination, recirculating room units shall not be used in areas marked "No." Isolation rooms may be ventilated by reheat induction units in which only the primary air supplied from a central system passes through the reheat unit. Gravity-type heating or cooling units such as radiators or convectors shall not be used in special care areas.
7. The ranges listed are the minimum and maximum limits where control is specifically needed. See A8.31.D in the AIA guideline (reference 120) for additional information.
8. Where temperature ranges are indicated, the systems shall be capable of maintaining the rooms at any point within the range. A single figure indicates a heating or cooling capacity of at least the indicated temperature. This is usually applicable where residents may be undressed and require a warmer environment. Nothing in these guidelines shall be construed as precluding the use of temperatures lower than those noted when the residents' comfort and medical conditions make lower temperatures desirable. Unoccupied areas such as storage rooms shall have temperatures appropriate for the function intended.
9. See A8.31.D1 in the AIA guideline (reference 120).
10. Food preparation facilities shall have ventilation systems whose air supply mechanisms are interfaced appropriately with exhaust hood controls or relief vents so that exfiltration or infiltration to or from exit corridors does not compromise the exit corridor restrictions of NFPA 90A, the pressure requirements of NFPA 96, or the maximum defined in the table. The number of air changes may be reduced or varied to any extent required for odor control when the space is not in use.

Table B.4. Filter efficiencies for central ventilation and air conditioning systems in general hospitals*

Note: This table is Table 7.3 in the AIA guidelines, 2001 edition.

Area designation	Number of filter beds	Filter bed No.1 (%)	Filter bed No. 2 (%)
All areas for inpatient care, treatment, and diagnosis, and those areas providing direct service or clean supplies, such as sterile and clean processing, etc.	2	30	90
Protective environment room	2	30	99.97
Laboratories	1	80	—
Administrative, bulk storage, soiled holding areas, food preparation areas, and laundries	1	30	—

* Additional roughing or prefilters should be considered to reduce maintenance required for filters with efficiency higher than 75 percent. The filtration efficiency ratings are based on average dust sopt efficiency per ASHRAE 52.1–1992.

Table B.5. Filter efficiencies for central ventilation and air conditioning systems in outpatient facilities*

Note: This table is Table 9.1 in the AIA guidelines, 2001 edition.

Area designation	Number of filter beds	Filter bed No. 1 (%)	Filter bed No. 2+ (%)
All areas for patient care, treatment, and/or diagnosis, and those areas providing direct service or clean supplies such as sterile and clean processing, etc.	2	30	90
Laboratories	1	80	—
Administrative, bulk storage, soiled holding areas, food preparation areas, and laundries	1	30	—

* Additional roughing or prefilters should be considered to reduce maintenance required for main filters. The filtration efficiency ratings are based on dust spot efficiency per ASHRAE 52.1–1992.

+ These requirements do not apply to small primary (e.g., neighborhood) outpatient facilities or outpatient facilities that do not perform invasive applications or procedures.

Table B.6. Filter efficiencies for central ventilation and air conditioning systems in nursing facilities

Note: This table is Table 8.2 in the AIA guidelines, 2001 edition.

Area designation	Minimum number of filter beds	Filter bed No. 1 (%)*	Filter bed No. 2 (%)*
All areas for inpatient care, treatment, and/or diagnosis, and those areas providing direct service or clean supplies	2	30	80
Administrative, bulk storage, soiled holding, laundries, and food preparation areas	1	30	–

* The filtration efficiency ratings are based on average dust spot efficiency as per ASHRAE 52.1–1992.

Table B.7. Filter efficiencies for central ventilation and air conditioning systems in psychiatric hospitals

Note: This table is Table 11.1 in the AIA guidelines, 2001 edition.

Area designation	Minimum number of filter beds	Filter bed No. 1 (%)*	Filter bed No. 2 (%)*
All areas for inpatient care, treatment, and diagnosis, and those areas providing direct services	2	30	90
Administrative, bulk storage, soiled holding, laundries, and food preparation areas	1	30	–

* The filtration efficiency ratings are based on average dust spot efficiency as per ASHRAE 52.1–1992.

Appendix C. Water

1. Biofilms

Microorganisms have a tendency to associate with and stick to surfaces. These adherent organisms can initiate and develop biofilms, which are comprised of cells embedded in a matrix of extracellularly produced polymers and associated abiotic particles.¹⁴³⁸ It is inevitable that biofilms will form in most water systems. In the health-care facility environment, biofilms may be found in the potable water supply piping, hot water tanks, air conditioning cooling towers, or in sinks, sink traps, aerators, or shower heads. Biofilms, especially in water systems, are not present as a continuous slime or film, but

are more often scanty and heterogeneous in nature.¹⁴³⁹ Biofilms may form under stagnant as well as flowing conditions, so storage tanks, in addition to water system piping, may be vulnerable to the development of biofilm, especially if water temperatures are low enough to allow the growth of thermophilic bacteria (e.g., *Legionella* spp.). Favorable conditions for biofilm formation are present if these structures and equipment are not cleaned for extended periods of time.¹⁴⁴⁰

Algae, protozoa, and fungi may be present in biofilms, but the predominant microorganisms of water system biofilms are gram-negative bacteria. Although most of these organisms will not normally pose a problem for healthy individuals, certain biofilm bacteria (e.g., *Pseudomonas aeruginosa*, *Klebsiella* spp., *Pantoea agglomerans*, and *Enterobacter cloacae*) all may be agents for opportunistic infections for immunocompromised individuals.^{1441, 1442} These biofilm organisms may easily contaminate indwelling medical devices or intravenous (IV) fluids, and they could be transferred on the hands of health-care workers.¹⁴⁴¹⁻¹⁴⁴⁴ Biofilms may potentially provide an environment for the survival of pathogenic organisms, such as *Legionella pneumophila* and *E. coli* O157:H7. Although the association of biofilms and medical devices provides a plausible explanation for a variety of health-care-associated infections, it is not clear how the presence of biofilms in the water system may influence the rates of health-care-associated waterborne infection.

Organisms within biofilms behave quite differently than their planktonic (i.e., free floating) counterparts. Research has shown that biofilm-associated organisms are more resistant to antibiotics and disinfectants than are planktonic organisms, either because the cells are protected by the polymer matrix, or because they are physiologically different.¹⁴⁴⁵⁻¹⁴⁵⁰ Nevertheless, municipal water utilities attempt to maintain a chlorine residual in the distribution system to discourage microbiological growth. Though chlorine in its various forms is a proven disinfectant, it has been shown to be less effective against biofilm bacteria.¹⁴⁴⁸ Higher levels of chlorine for longer contact times are necessary to eliminate biofilms.

Routine sampling of health-care facility water systems for biofilms is not warranted. If an epidemiologic investigation points to the water supply system as a possible source of infection, then water sampling for biofilm organisms should be considered so that prevention and control strategies can be developed. An established biofilm is difficult to remove totally in existing piping. Strategies to remediate biofilms in a water system would include flushing the system piping, hot water tank, dead legs, and those areas of the facility's water system subject to low or intermittent flow. The benefits of this treatment would include a) elimination of corrosion deposits and sludge from the bottom of hot water tanks, b) removal of biofilms from shower heads and sink aerators, and c) circulation of fresh water containing elevated chlorine residuals into the health-care facility water system.

The general strategy for evaluating water system biofilm depends on a comparison of the bacteriological quality of the incoming municipal water and that of water sampled from within facility's distribution system. Heterotrophic plate counts and coliform counts, both of which are routinely run by the municipal water utility, will at least provide an indication of the potential for biofilm formation. Heterotrophic plate count levels in potable water should be <500 CFU/mL. These levels may increase on occasion, but counts consistently >500 CFU/mL would indicate a general decrease in water quality. A direct correlation between heterotrophic plate count and biofilm levels has been demonstrated.¹⁴⁵⁰ Therefore, an increase in heterotrophic plate count would suggest a greater rate and extent of biofilm formation in a health-care facility water system. The water supplied to the facility should also contain <1 coliform bacteria/100 mL. Coliform bacteria are organisms whose presence in the distribution system could indicate fecal contamination. It has been shown that coliform bacteria can colonize biofilms within drinking water systems. Intermittant contamination of a water system with these organisms could lead to colonization of the system.

Water samples can be collected from throughout the health-care facility system, including both hot and cold water sources; samples should be cultured by standard methods.⁹⁴⁵ If heterotrophic plate counts in samples from the facility water system are higher than those from samples collected at the point of water entry to the building, it can be concluded that the facility water quality has diminished. If biofilms are detected in the facility water system and determined by an epidemiologic and environmental investigation to be a reservoir for health-care-associated pathogens, the municipal water supplier could be contacted with a request to provide higher chlorine residuals in the distribution system, or the health-care facility could consider installing a supplemental chlorination system.

Sample collection sites for biofilm in health-care facilities include a) hot water tanks; b) shower heads; and c) faucet aerators, especially in immunocompromised patient-care areas. Swabs should be placed into tubes containing phosphate buffered water, pH 7.2 or phosphate buffered saline, shipped to the laboratory under refrigeration and processed within 24 hrs. of collection. Samples are suspended by vortexing with sterile glass beads and plated onto a nonselective medium (e.g., Plate Count Agar or R2A medium) and selective media (e.g., media for *Legionella* spp. isolation) after serial dilution. If the plate counts are elevated above levels in the water (i.e. comparing the plate count per square centimeter of swabbed surface to the plate count per milliliter of water), then biofilm formation can be suspected. In the case of an outbreak, it would be advisable to isolate organisms from these plates to determine whether the suspect organisms are present in the biofilm or water samples and compare them to the organisms isolated from patient specimens.

2. Water and Dialysate Sampling Strategies in Dialysis

In order to detect the low, total viable heterotrophic plate counts outlined by the current AAMI standards for water and dialysate in dialysis settings, it is necessary to use standard quantitative culture techniques with appropriate sensitivity levels.^{792, 832, 833} The membrane filter technique is particularly suited for this application because it permits large volumes of water to be assayed.^{792, 834} Since the membrane filter technique may not be readily available in clinical laboratories, the spread plate assay can be used as an alternative.⁸³⁴ If the spread plate assay is used, however, the standard prohibits the use of a calibrated loop when applying sample to the plate.⁷⁹² The prohibition is based on the low sensitivity of the calibrated loop. A standard calibrated loop transfers 0.001 mL of sample to the culture medium, so that the minimum sensitivity of the assay is 1,000 CFU/mL. This level of sensitivity is unacceptable when the maximum allowable limit for microorganisms is 200 CFU/mL. Therefore, when the spread plate method is used, a pipette must be used to place 0.1–0.5 mL of water on the culture medium.

The current AAMI standard specifically prohibits the use of nutrient-rich media (e.g., blood agar, and chocolate agar) in dialysis water and dialysate assays because these culture media are too rich for growth of the naturally occurring organisms found in water.⁷⁹² Debate continues within AAMI, however, as to the most appropriate culture medium and incubation conditions to be used. The original clinical observations on which the microbiological requirements of this standard were based used Standard Methods Agar (SMA), a medium containing relatively few nutrients.⁶⁶⁶ The use of tryptic soy agar (TSA), a general purpose medium for isolating and cultivating microorganisms was recommended in later versions of the standard because it was thought to be more appropriate for culturing bicarbonate-containing dialysate.^{788, 789, 835} Moreover, culturing systems based on TSA are readily available from commercial sources. Several studies, however, have shown that the use of nutrient-poor media, such as R2A, results in an increased recovery of bacteria from water.^{1451, 1452} The original standard also specified incubation for 48 hours at 95°F–98.6°F (35°C–37°C) before enumeration of bacterial colonies. Extending the culturing time up to 168 hours, or 7 days and using incubation temperatures of 73.4°F–82.4°F (23°C–28°C) have also been shown to increase the recovery of bacteria.^{1451, 1452} Other

investigators, however, have not found such clear cut differences between culturing techniques.^{835, 1453} After considerable discussion, the AAMI Committee has not reached a consensus regarding changes in the assay technique, and the use of TSA or its equivalent for 48 hours at 95°F–98.6°F (35°C–37°C) remains the recommended method. It should be recognized, however, that these culturing conditions may underestimate the bacterial burden in the water and fail to identify the presence of some organisms. Specifically, the recommended method may not detect the presence of various NTM that have been associated with several outbreaks of infection in dialysis units.^{31, 32} In these instances, however, the high numbers of mycobacteria in the water were related to the total heterotrophic plate counts, each of which was significantly greater than that allowable by the AAMI standard. Additionally, the recommended method will not detect fungi and yeast, which have been shown to contaminate water used for hemodialysis applications.¹⁴⁵⁴ Biofilm on the surface of the pipes may hide viable bacterial colonies, even though no viable colonies are detected in the water using sensitive culturing techniques.¹⁴⁵⁵ Many disinfection processes remove biofilm poorly, and a rapid increase in the level of bacteria in the water following disinfection may indicate significant biofilm formation. Therefore, although the results of microbiological surveillance obtained using the test methods outlined above may be useful in guiding disinfection schedules and in demonstrating compliance with AAMI standards, they should not be taken as an indication of the absolute microbiological purity of the water.⁷⁹²

Endotoxin can be tested by one of two types of assays a) a kinetic test method [e.g., colorimetric or turbidimetric] or b) a gel-clot assay. Endotoxin units are assayed by the *Limulus* Amebocyte Lysate (LAL) method. Because endotoxins differ in their activity on a mass basis, their activity is referred to a standard *Escherichia coli* endotoxin. The current standard (EC-6) is prepared from *E. coli* O113:H10. The relationship between mass of endotoxin and its activity varies with both the lot of LAL and the lot of control standard endotoxin used. Since standards for endotoxin were harmonized in 1983 with the introduction of EC-5, the relationship between mass and activity of endotoxin has been approximately 5–10 EU/ng. Studies to harmonize standards have led to the measurement of endotoxin units (EU) where 5 EU is equivalent to 1 ng *E. coli* O55:B5 endotoxin.¹⁴⁵⁶

In summary, water used to prepare dialysate and to reprocess hemodialyzers should not contain a total microbial count >200 CFU/mL as determined by assay on TSA agar for 48 hrs. at 96.8°F (36°C), and ≤2 endotoxin units (EU) per mL. The dialysate at the end of a dialysis treatment should not contain >2,000 CFU/mL.^{31, 32, 668, 789, 792}

3. Water Sampling Strategies and Culture Techniques for Detecting Legionellae

Legionella spp. are ubiquitous and can be isolated from 20%–40% of freshwater environments, including man-made water systems.^{1457, 1458} In health-care facilities, where legionellae in potable water rarely result in disease among immunocompromised patients, courses of remedial action are unclear.

Scheduled microbiologic monitoring for legionellae remains controversial because the presence of legionellae is not necessarily evidence of a potential for causing disease.¹⁴⁵⁹ CDC recommends aggressive disinfection measures for cleaning and maintaining devices known to transmit legionellae, but does not recommend regularly scheduled microbiologic assays for the bacteria.³⁹⁶ However, scheduled monitoring of potable water within a hospital might be considered in certain settings where persons are highly susceptible to illness and mortality from *Legionella* infection (e.g., hematopoietic stem cell transplantation units and solid organ transplant units).⁹ Also, after an outbreak of

legionellosis, health officials agree monitoring is necessary to identify the source and to evaluate the efficacy of biocides or other prevention measures.

Examination of water samples is the most efficient microbiologic method for identifying sources of legionellae and is an integral part of an epidemiologic investigation into health-care-associated Legionnaires disease. Because of the diversity of plumbing and HVAC systems in health-care facilities, the number and types of sites to be tested must be determined before collection of water samples. One environmental sampling protocol that addresses sampling site selection in hospitals might serve as a prototype for sampling in other institutions.¹²⁰⁹ Any water source that might be aerosolized should be considered a potential source for transmission of legionellae. The bacteria are rarely found in municipal water supplies and tend to colonize plumbing systems and point-of-use devices. To colonize, legionellae usually require a temperature range of 77°F–108°F (25°C–42.2°C) and are most commonly located in hot water systems.¹⁴⁶⁰ Legionellae do not survive drying. Therefore, air-conditioning equipment condensate, which frequently evaporates, is not a likely source.¹⁴⁶¹

Water samples and swabs from point-of-use devices or system surfaces should be collected when sampling for legionellae (Box C.1).¹⁴³⁷ Swabs of system surfaces allow sampling of biofilms, which frequently contain legionellae. When culturing faucet aerators and shower heads, swabs of surface areas should be collected first; water samples are collected after aerators or shower heads are removed from their pipes. Collection and culture techniques are outlined (Box C.2). Swabs can be streaked directly onto buffered charcoal yeast extract agar (BCYE) plates if the plates are available at the collection site. If the swabs and water samples must be transported back to a laboratory for processing, immersing individual swabs in sample water minimizes drying during transit. Place swabs and water samples in insulated coolers to protect specimens from temperature extremes.

Box C.1. Potential sampling sites for *Legionella* spp. in health-care facilities*

-
- **Potable water systems**
incoming water main, water softener unit, holding tanks, cisterns, water heater tanks
(at the inflows and outflows)
 - **Potable water outlets, especially those in or near patient rooms**
faucets or taps, showers
 - **Cooling towers and evaporative condensers**
makeup water (e.g., added to replace water lost because of evaporation, drift, or leakage),
basin (i.e., area under the tower for collection of cooled water), sump (i.e., section of basin
from which cooled water returns to heat source), heat sources (e.g., chillers)
 - **Humidifiers (e.g., nebulizers)**
bubblers for oxygen, water used for respiratory therapy equipment
 - **Other sources**
decorative fountains, irrigation equipment, fire sprinkler system (if recently used), whirlpools,
spas
-

* Material in this box is adapted from reference 1209.

Box C.2. Procedures for collecting and processing environmental specimens for *Legionella* spp.*

1. Collect water (1-liter samples, if possible) in sterile, screw-top bottles.
2. Collect culture swabs of internal surfaces of faucets, aerators, and shower heads in a sterile, screw-top container (e.g., 50 mL plastic centrifuge tube). Submerge each swab in 5–10 mL of sample water taken from the same device from which the sample was obtained.
3. Transport samples and process in a laboratory proficient at culturing water specimens for *Legionella* spp. as soon as possible after collection.+
4. Test samples for the presence of *Legionella* spp. by using semiselective culture media using procedures specific to the cultivation and detection of *Legionella* spp.§¶

* Material in this table is compiled from references 1209, 1437, 1462–1465.

+ Samples may be transported at room temperature but must be protected from temperature extremes. Samples not processed within 24 hours of collection should be refrigerated.

§ Detection of *Legionella* spp. antigen by the direct fluorescent antibody technique is not suitable for environmental samples.

¶ Use of polymerase chain reaction for identification of *Legionella* spp. is not recommended until more data regarding the sensitivity and specificity of this procedure are available.

4. Procedure for Cleaning Cooling Towers and Related Equipment

- I. Perform these steps prior to chemical disinfection and mechanical cleaning.
 - A. Provide protective equipment to workers who perform the disinfection, to prevent their exposure to chemicals used for disinfection and aerosolized water containing *Legionella* spp. Protective equipment may include full-length protective clothing, boots, gloves, goggles, and a full- or half-face mask that combines a HEPA filter and chemical cartridges to protect against airborne chlorine levels of up to 10 mg/L.
 - B. Shut off cooling tower.
 1. Shut off the heat source, if possible.
 2. Shut off fans, if present, on the cooling tower/evaporative condenser (CT/EC).
 3. Shut off the system blowdown (i.e., purge) valve.
 4. Shut off the automated blowdown controller, if present, and set the system controller to manual.
 5. Keep make-up water valves open.
 6. Close building air-intake vents within at least 30 meters of the CT/EC until after the cleaning procedure is complete.
 7. Continue operating pumps for water circulation through the CT/EC.
- II. Perform these chemical disinfection procedures.
 - A. Add fast-release, chlorine-containing disinfectant in pellet, granular, or liquid form, and follow safety instructions on the product label. Use EPA-registered products, if available. Examples of disinfectants include sodium hypochlorite (NaOCl) or calcium hypochlorite (Ca[OCl]₂), calculated to achieve initial free residual chlorine (FRC) of 50 mg/L: either a) 3.0 lbs [1.4 kg] industrial grade NaOCl [12%–15% available Cl] per 1,000 gallons of CT/EC water; b) 10.5 lbs [4.8 kg] domestic grade NaOCl [3%–5% available Cl] per 1,000 gallons of CT/EC water; or c)

0.6 lb [0.3 kg] $\text{Ca}[\text{OCl}]_2$ per 1,000 gallons of CT/EC water. If significant biodeposits are present, additional chlorine may be required. If the volume of water in the CT/EC is unknown, it can be estimated (in gallons) by multiplying either the recirculation rate in gallons per minute by 10 or the refrigeration capacity in tons by 30. Other appropriate compounds may be suggested by a water-treatment specialist.

- B. Record the type and quality of all chemicals used for disinfection, the exact time the chemicals were added to the system, and the time and results of FRC and pH measurements.
- C. Add dispersant simultaneously with or within 15 minutes of adding disinfectant. The dispersant is best added by first dissolving it in water and adding the solution to a turbulent zone in the water system. Automatic-dishwasher compounds are examples of low- or nonfoaming, silicate-based dispersants. Dispersants are added at 10–25 lbs (4.5–11.25 kg) per 1,000 gallons of CT/EC water.
- D. After adding disinfectant and dispersant, continue circulating the water through the system. Monitor the FRC by using an FRC-measuring device with the DPD method (e.g., a swimming-pool test kit), and measure the pH with a pH meter every 15 minutes for 2 hours. Add chlorine as needed to maintain the FRC at ≥ 10 mg/L. Because the biocidal effect of chlorine is reduced at a higher pH, adjust the pH to 7.5–8.0. The pH may be lowered by using any acid (e.g., muriatic acid or sulfuric acid used for maintenance of swimming pools) that is compatible with the treatment chemicals.
- E. Two hours after adding disinfectant and dispersant or after the FRC level is stable at ≥ 10 mg/L, monitor at 2-hour intervals and maintain the FRC at ≥ 10 mg/L for 24 hours.
- F. After the FRC level has been maintained at ≥ 10 mg/L for 24 hours, drain the system. CT/EC water may be drained safely into the sanitary sewer. Municipal water and sewerage authorities should be contacted regarding local regulations. If a sanitary sewer is not available, consult local or state authorities (e.g., a department of natural resources or environmental protection) regarding disposal of water. If necessary, the drain-off may be dechlorinated by dissipation or chemical neutralization with sodium bisulfite.
- G. Refill the system with water and repeat the procedure outline in steps 2–7 in I-B above.

III. Perform mechanical cleaning.

- A. After water from the second chemical disinfection has been drained, shut down the CT/EC.
- B. Inspect all water-contact areas for sediment, sludge, and scale. Using brushes and/or a low-pressure water hose, thoroughly clean all CT/EC water-contact areas, including the basin, sump, fill, spray nozzles, and fittings. Replace components as needed.
- C. If possible, clean CT/EC water-contact areas within the chillers.

IV. Perform these procedures after mechanical cleaning.

- A. Fill the system with water and add chlorine to achieve an FRC level of 10 mg/L.
- B. Circulate the water for 1 hour, then open the blowdown valve and flush the entire system until the water is free of turbidity.
- C. Drain the system.
- D. Open any air-intake vents that were closed before cleaning.
- E. Fill the system with water. The CT/EC may be put back into service using an effective water-treatment program.

5. Maintenance Procedures Used to Decrease Survival and Multiplications of *Legionella* spp. in Potable-Water Distribution Systems

Wherever allowable by state code, provide water at $\geq 124^{\circ}\text{F}$ ($\geq 51^{\circ}\text{C}$) at all points in the heated water system, including the taps. This requires that water in calorifiers (e.g., water heaters) be maintained at $\geq 140^{\circ}\text{F}$ ($\geq 60^{\circ}\text{C}$). In the United Kingdom, where maintenance of water temperatures at $\geq 122^{\circ}\text{F}$ ($\geq 50^{\circ}\text{C}$) in hospitals has been mandated, installation of blending or mixing valves at or near taps to reduce the water temperature to $\leq 109.4^{\circ}\text{F}$ ($\leq 63^{\circ}\text{C}$) has been recommended in certain settings to reduce the risk for scald injury to patients, visitors, and health care workers.⁷²⁶ However, *Legionella* spp. can multiply even in short segments of pipe containing water at this temperature. Increasing the flow rate from the hot-water-circulation system may help lessen the likelihood of water stagnation and cooling.^{711, 1465} Insulation of plumbing to ensure delivery of cold ($< 68^{\circ}\text{F}$ [$< 20^{\circ}\text{C}$]) water to water heaters (and to cold-water outlets) may diminish the opportunity for bacterial multiplication.⁴⁵⁶ Both dead legs and capped spurs within the plumbing system provide areas of stagnation and cooling to $< 122^{\circ}\text{F}$ ($< 50^{\circ}\text{C}$) regardless of the circulating water temperature; these segments may need to be removed to prevent colonization.⁷⁰⁴ Rubber fittings within plumbing systems have been associated with persistent colonization, and replacement of these fittings may be required for *Legionella* spp. eradication.¹⁴⁶⁷

Continuous chlorination to maintain concentrations of free residual chlorine at 1–2 mg/L (1–2 ppm) at the tap is an alternative option for treatment. This requires the placement of flow-adjusted, continuous injectors of chlorine throughout the water distribution system. Adverse effects of continuous chlorination can include accelerated corrosion of plumbing (resulting in system leaks) and production of potentially carcinogenic trihalomethanes. However, when levels of free residual chlorine are below 3 mg/L (3 ppm), trihalomethane levels are kept below the maximum safety level recommended by the EPA.^{727, 1468, 1469}

Appendix D. Insects and Microorganisms

Table D.1. Microorganisms isolated from arthropods in health-care settings

Insect	Microorganism category	Microorganisms	References
Cockroaches	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Citrobacter freundii</i> ; <i>Enterobacter</i> spp., <i>E. cloacae</i> ; <i>Escherichia coli</i> ; <i>Flavobacterium</i> spp.; <i>Klebsiella</i> spp.; <i>Proteus</i> spp.; <i>Pseudomonas</i> spp., <i>P. aeruginosa</i> , <i>P. fluorescens</i> , <i>P. putida</i> ; <i>Salmonella</i> spp.; <i>Serratia</i> spp., <i>S. marcescens</i> ; <i>Shigella boydii</i>	1048, 1051, 1056, 1058, 1059, 1062
	Gram-positive bacteria	<i>Bacillus</i> spp.; <i>Enterococcus faecalis</i> ; <i>Micrococcus</i> spp.; <i>Staphylococcus aureus</i> , <i>S. epidermidis</i> ; <i>Streptococcus</i> spp., <i>S. viridans</i>	1056, 1058, 1059
	Acid-fast bacteria	<i>Mycobacterium tuberculosis</i>	1065
	Fungi	<i>Aspergillus niger</i> ; <i>Mucor</i> spp.; <i>Rhizopus</i> spp.	1052, 1059
	Parasites	<i>Endolimax nana</i> ; <i>Entamoeba coli</i>	1059
Houseflies	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Campulobacter fetus</i> subsp. <i>Jejuni</i> ; <i>Chlamydia</i> spp.; <i>Citrobacter freundii</i> ; <i>Enterobacter</i> spp.; <i>Escherichia coli</i> ; <i>Helicobacter pylori</i> ; <i>Klebsiella</i> spp.; <i>Proteus</i> spp.; <i>Pseudomonas aeruginosa</i> ; <i>Serratia marcescens</i> ; <i>Shigella</i> spp.	1047, 1048, 1050, 1053–1055, 1060
	Gram-positive bacteria	<i>Bacillus</i> spp.; <i>Enterococcus faecalis</i> ; <i>Micrococcus</i> spp.; <i>Staphylococcus</i> spp. (coagulase-negative), <i>S. aureus</i> ; <i>Streptococcus</i> spp., <i>S. viridans</i>	1048, 1060
	Fungi / yeasts	<i>Candida</i> spp.; <i>Geotrichum</i> spp.	1060
	Parasites	<i>Endolimax nana</i> ; <i>Entamoeba coli</i>	1060
	Viruses	Rotaviruses	1049
Ants	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Escherichia coli</i> ; <i>Klebsiella</i> spp.; <i>Neisseria sicca</i> ; <i>Proteus</i> spp.; <i>Providencia</i> spp.; <i>Pseudomonas aeruginosa</i> , <i>P. fluorescens</i>	1057
	Gram-positive bacteria	<i>Bacillus</i> spp., <i>B. cereus</i> , <i>B. pumilis</i> ; <i>Clostridium cochlearium</i> , <i>C. welchii</i> ; <i>Enterococcus faecalis</i> ; <i>Staphylococcus</i> spp. (coagulase-negative), <i>S. aureus</i> ; <i>Streptococcus pyrogenes</i>	1057
Spiders	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Citrobacter freundii</i> ; <i>Enterobacter aerogenes</i> ; <i>Morganella morganii</i>	1048
	Gram-positive bacteria	<i>Staphylococcus</i> spp. (coagulase-negative)	1048
Mites, midges	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Burkholderia cepacia</i> ; <i>Enterobacter agglomerans</i> , <i>E. aerogenes</i> ; <i>Hafnia alvei</i> ; <i>Pseudomonas aeruginosa</i>	1048
	Gram-positive bacteria	<i>Staphylococcus</i> spp. (coagulase-negative)	1048
Mosquitoes	Gram-negative bacteria	<i>Acinetobacter calcoaceticus</i> ; <i>Enterobacter cloacae</i>	1048
	Gram-positive bacteria	<i>Enterococcus</i> spp.; <i>Staphylococcus</i> spp. (coagulase-negative)	1048

Appendix E. Information Resources

The following sources of information may be helpful to the reader. Some of these are available at no charge, while others are available for purchase from the publisher.

Air and Water

- Jensen PA, Schafer MP. Sampling and characterization of bioaerosols. NIOSH Manual of Analytical Methods; revised 6/99. www.cdc.gov/niosh/nmam/pdfs/chapter-j.pdf
- American Institutes of Architects. *Guidelines for Design and Construction of Hospital and Health Care Facilities*. Washington DC; American Institute of Architects Press; 2001. AIA, 1735 New York Avenue, NW, Washington DC 20006. 1-800-AIA-3837 or (202) 626-7541
- ASHRAE. Standard 62, and Standard 12-2000. These documents may be purchased from: American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc. 1791 Tullie Circle, NE, Atlanta GA 30329 1-800-527-4723 or (404) 636-8400.
- University of Minnesota websites: www.dehs.umn.edu Indoor air quality site: www.dehs.umn.edu/resources.htm#indoor Water infiltration and use of the wet test (moisture) meter: www.dehs.umn.edu/remangi.html
- The CDC website for bioterrorism information contains the interim intervention plan for smallpox. The plan discusses infection control issues both for home-based care and hospital-based patient management. www.bt.cdc.gov/agent/smallpox/response-plan/index.asp

Environmental Sampling

- ISO. Sterilization of medical devices – microbiological methods, Part 1. ISO standard 11737-1. Paramus NJ; International Organization for Standardization; 1995.

Animals in Health-Care Facilities

- Service animal information with respect to the Americans with Disabilities Act. Contact the U.S. Department of Justice ADA Information Line at (800) 514-0301 (voice) or (800) 514-0383 (TDD), or visit the ADA website at: www.usdoj.gov/crt/ada/adahom1.htm

Regulated Medical Waste

- U.S. Environmental Protection Agency. This is the Internet address on their Internet web site that will link to any state for information about medical waste rules and regulations at the state level: www.epa.gov/epaoswer/other/medical/stregs.htm

General Resources

- APIC Text of Infection Control and Epidemiology. Association for Professionals in Infection Control and Epidemiology, Inc. Washington DC; 2000. (Two binder volumes, or CD-ROM)
- Abrutyn E, Goldmann DA, Scheckler WE. Saunders Infection Control Reference Service, 2nd Edition. Philadelphia PA; WB Saunders; 2000.
- ECRI publications are available on a variety of healthcare topics. Contact ECRI at (610) 825-6000. CRI, 5200 Butler Pike, Plymouth Meeting, PA 19462-1298.

Appendix F. Areas of Future Research

Air

- Standardize the methodology and interpretation of microbiologic air sampling (e.g., determine action levels or minimum infectious dose for aspergillosis, and evaluate the significance of airborne bacteria and fungi in the surgical field and the impact on postoperative SSI).
- Develop new molecular typing methods to better define the epidemiology of health-care–associated outbreaks of aspergillosis and to associate isolates recovered from both clinical and environmental sources.
- Develop new methods for the diagnosis of aspergillosis that can lead reliably to early recognition of infection.
- Assess the value of laminar flow technology for surgeries other than for joint replacement surgery.
- Determine if particulate sampling can be routinely performed in lieu of microbiologic sampling for purposes such as determining air quality of clean environments (e.g., operating rooms, HSCT units).

Water

- Evaluate new methods of water treatment, both in the facility and at the water utility (e.g., ozone, chlorine dioxide, copper/silver/monochloramine) and perform cost-benefit analyses of treatment in preventing health-care–associated legionellosis.
- Evaluate the role of biofilms in overall water quality and determine the impact of water treatments for the control of biofilm in distribution systems.
- Determine if the use of ultrapure fluids in dialysis is feasible and warranted, and determine the action level for the final bath.
- Develop quality assurance protocols and validated methods for sampling filtered rinse water used with AERs and determine acceptable microbiologic quality of AER rinse water.

Environmental Services

- Evaluate the innate resistance of microorganisms to the action of chemical germicides, and determine what, if any, linkage there may be between antibiotic resistance and resistance to disinfectants.

Laundry and Bedding

- Evaluate the microbial inactivation capabilities of new laundry detergents, bleach substitutes, other laundry additives, and new laundry technologies.

Animals in Health-Care Facilities

- Conduct surveillance to monitor incidence of infections among patients in facilities that use animal programs, and conduct investigations to determine new infection control strategies to prevent these infections.
- Evaluate the epidemiologic impact of performing procedures on animals (e.g., surgery or imaging) in human health-care facilities.

Regulated Medical Waste

- Determine the efficiency of current medical waste treatment technologies to inactivate emerging pathogens that may be present in medical waste (e.g., SARS-coV).
- Explore options to enable health-care facilities to reinstate the capacity to inactivate microbiological cultures and stocks on-site.

Index—Parts I and IV

A

AAMI standards 59, 60, 62, 222, 223
Acinetobacter spp. 11, 20, 43, 44, 99, 104
aerators 47, 48, 94, 220–222, 224, 225
aerosols... 12, 27, 41, 47, 56, 59, 67, 75, 76, 78, 80, 85, 89,
90, 98, 106, 111, 113, 114
AIA guidelines..... 17, 18, 19, 25, 37, 39, 99
AII rooms 35–37
air changes per hour (ACH)..... 6, 12, 16, 18, 31, 111, 210
air conditioners 8, 22
air conditioning systems 13, 20, 57, 59
air filtration..... 111
air intakes 31, 226
air sampling 26, 29, 89, 90, 91, 93, 210
airborne infection isolation (AII)..... 6, 19
airborne transmission..... 12
air-fluidized beds 104
alcohol-based hand rubs 53
alkaline glutaraldehyde..... 70
allergens..... 17, 80, 107
American Institute of Architects (AIA)..... 13
Americans with Disabilities Act..... 108, 110
amplified stocks and cultures..... 114, 115
Animal Assisted Activities 106, 107
Animal Assisted Therapy 106, 107
animal bites..... 107
animal handler 107
animal patient 110
Animal Welfare Act..... 112
anterooms 12, 25, 33, 36–38
ants 81
ASHRAE 13, 47, 49
aspergillosis 7, 8, 16, 19, 21, 35, 79, 80
Aspergillus fumigatus 7, 8, 29
Aspergillus spp. 5, 7, 20, 21, 28, 32, 34, 81
automated cyclers 65
automated endoscope reprocessor..... 50, 69, 70
autopsy suites/rooms..... 12, 87

B

bacterial spores 73, 84, 89
bank of filters..... 14
barrier 34
barrier precautions/protection..... 74, 109, 116
barriers..... 27, 31, 33
bassinets..... 76
biofilms..... 46, 54, 64, 71, 220–222, 224
biosafety level..... 114
bioterrorism 89, 114
bird droppings..... 9, 20, 22
birthing tanks..... 67, 69

blood.. 12, 64, 69, 75, 77–79, 86, 87, 98, 99, 102, 113, 116
bloodborne pathogens..... 73, 116
boil water advisory 51, 52

C

calibrated loop 222
carpet cleaning 79
carpet tiles..... 79
carpeting 22, 25, 52, 78, 79
cats..... 105, 106, 108, 109
chain of infection 4, 87
chemical germicides 73, 74, 77, 80, 84
chloramine/chloramine-T 68
chlorine..... 46, 50, 53, 69, 84, 221, 226
chlorine bleach..... 78, 101
chlorine residual 50, 68, 69, 94, 101, 221
cleaning .. 68, 70–72, 74, 78, 80, 83, 85, 86, 107, 109, 112,
225
cleaning cloths 75
cleaning solutions 75, 76
Clostridium difficile 5, 84
cloth chairs..... 79
cockroaches 81
coliform bacteria..... 221
colonization 42–44, 68, 70, 83, 99, 106, 227
colony counts..... 211
commissioning..... 29, 89
construction 7, 13, 14, 21, 23, 26, 27, 29, 31, 37, 76, 89
construction workers..... 24, 26, 31
contact precautions 85
contact time 74, 84, 88, 221
contaminants..... 14, 18, 19, 59, 210
contaminated fabrics..... 98, 101, 102
contingency plans 21, 50
continuous chlorination 227
cooling tower..... 41, 53, 55, 57–59, 220, 225
copper/silver ions..... 54
copper-8-quinolinolate..... 35
Creutzfeldt-Jakob disease 86, 116
Cryptosporidium parvum..... 46

D

dead legs 59, 64, 221
decorative fountains..... 47, 49, 224
demolition..... 23, 25, 26, 29
dental unit water lines..... 71
detergent/disinfectant..... 74–76
dialysate 59–62
dialysis machines 64
dialysis water 222
dialyzer 62
dialyzer membranes..... 61

dialyzer reprocessing 59
 dioctylphthalate (DOP) particle test 15
 direct contact 6, 41, 67, 85, 86, 98, 108, 111
 direct threat 109
 disinfectant fogging 75
 disinfectant residuals 96
 disinfectants 21, 76, 225
 disinfecting 71, 74, 80, 83, 85, 86, 112, 226
 disinfection 63, 64, 68, 70
 dispersant 226
 disposal (of medical waste) 113
 distribution system 94, 221
 dogs 105, 106, 108, 109
 drift eliminators 58
 drinking water 71
 droplet nuclei 6, 7, 10, 12, 89
 droplets 6, 55, 85, 86, 89
 dry cleaning 102
 drying 11
 dual-duct system 20
 duct cleaning 21
 ductwork 20, 22
 dust 8, 20, 24, 27, 30, 32, 74, 79, 93
 dust-spot test 15

E

education 24
 electrical generators 53
 emergency 53
 endotoxin 60–62, 64, 223
 engineering controls 36
 enteric viruses 85
 environmental cultures 83, 88
 Environmental Protection Agency (EPA) 21, 73–75, 77, 103, 227
 environmental sampling 88, 95
 environmental surfaces 11, 44, 71, 72, 74, 82–86, 88, 98, 107
 environmental surveillance 54, 55
 EPA registration 73, 76, 83
 EPA-registered germicides 75, 78, 85, 86
 evaporative condensers 41, 57–59
 exclusion (of a service animal) 109
 exotic animals 110

F

fan-coil units 18
 faucets 47, 54, 94, 222, 224, 225
 fecal contamination 84
 FIFRA 75, 103
 filter efficiency 27, 29
 filtration 15
 fire codes 31
 fish 105, 108
 fish tanks 108
 flies 81
 flooding 51

floors 25, 75, 82, 83
 flowers 80
 flush times 51
 flutter strips 20, 34, 36
 fomites 3, 4, 7, 85
 Food and Drug Administration (FDA) 69, 73, 103
 free residual chlorine 51, 54, 225–227
 fungal spores 8, 15, 16, 19–21, 26–28, 31, 34, 38, 79, 89, 93
 fungi 8
 furniture 52, 79, 82

G

gram-negative bacteria 11, 41, 42, 48, 50, 60, 63, 64, 221
 gram-positive bacteria 11, 84

H

hand hygiene 25, 71, 107, 109
 hand transferral 3, 44, 65, 82–84, 106, 221
 handwashing 25, 80, 84, 99, 107
 hantaviruses 12
 hematopoietic stem cell transplant 6
 hemodiafiltration 62
 hemodialysis 59, 60, 62, 64, 223
 hemodialysis patients 7
 hemofiltration 62
 HEPA filtration/filters 6, 12, 14, 15, 17, 31, 32, 36, 76
 hepatitis B virus 40, 73, 98
 heterotrophic plate counts 51, 62, 66, 95, 221–223
 high-flux membranes 61
 high-level disinfectants 73
 high-level disinfection 60, 69, 72
 high-temperature flushing 50
 high-touch surfaces 75, 83–85
 holding tank 47
 hospital disinfectant 73
 hot water system 51, 54
 hot water tanks 53, 220–222
 hot water temperature 49
 housekeeping surfaces 3, 64, 72, 74–77, 83
 HSCT patients 6, 37
 HSCT units 11, 26, 79, 80, 107
 Hubbard tanks 68
 human health-care facilities 110, 111
 human immunodeficiency virus 73
 humidifiers 17, 23, 41
 humidity 13, 14, 17, 20, 38, 90
 HVAC systems 13, 14, 16, 17, 19–21, 27, 30, 51
 hydrotherapy equipment 67–69
 hydrotherapy pools 68
 hydrotherapy tanks 67, 68, 82
 hygienically-clean laundry 98–100, 102
 hyperchlorination 50, 53, 54, 59

I, J

iatrogenic cases.....	87
ice machines and ice.....	25, 48, 65, 66
ice-storage chests.....	66
immunocompromised patients.....	6, 7, 9, 26, 29–31, 34, 42, 47, 56, 66, 80, 107, 108, 223
impaction.....	90, 211
impactors.....	28, 93
impingement.....	90, 93, 211
impingers.....	211
inactivation studies.....	87
incineration.....	113, 114
incubators (nursery).....	76
indirect transmission.....	6
indirect contact.....	41
indoor air.....	21, 24, 26, 27, 90
industrial-grade HEPA filter.....	16, 31, 38, 39
infection-control risk assessment (ICRA)....	26, 29, 31, 35, 108, 111
influenza viruses.....	6, 12, 73, 85
innate resistance.....	72, 73, 84
insects.....	67, 81
insulation material.....	20
intermediate-level disinfectants.....	73, 78, 83, 85, 86
intermediate-level disinfection.....	72
isolation/isolation areas.....	11, 36, 100
JCAHO.....	13, 14, 51, 59

L

laboratories.....	12, 13, 32, 47, 78, 79, 83, 105, 111, 112, 114, 222
laboratory confirmation.....	55
laminar airflow.....	18, 34, 38
laser plumes.....	40
laundry.....	49
laundry bags.....	100
laundry chutes.....	100
laundry cycles.....	101
laundry disinfection.....	101
laundry facility.....	99
laundry packaging.....	100, 101
laundry process.....	98, 99, 102
laundry services.....	99
laundry transport.....	100, 101
<i>Legionella pneumophila</i>	210, 221
<i>Legionella</i> spp.	41, 42, 50, 54–57, 59, 71, 222, 223, 225, 227
legionellae.....	41, 54, 211, 223
legionellosis.....	53–56, 224
Legionnaires disease.....	41, 47, 57, 58, 224
liquid chemical sterilant.....	70
low-level disinfectants.....	72, 73, 83, 86
low-level disinfection.....	60, 64

M

manufacturer's instructions.....	67, 69, 74, 86, 102, 116
material safety data sheets (MSDS).....	75, 87
mattress cover.....	77, 104
mattresses.....	77, 104
medical equipment.....	74, 83
medical equipment surfaces.....	72
medical gas piping.....	30
medical records.....	51
medical waste.....	112, 113, 117
medical waste management.....	112
membrane filtration.....	70, 95, 96, 222
methicillin-resistant <i>Staphylococcus aureus</i> (MRSA) ...	82, 83, 104, 105
microbial inactivation.....	72
microbial resistance.....	70
microbiologic air sampling.....	27
microbiologic cultures and stocks.....	112
microbiologic sampling.....	64
microbiologic sampling of laundry.....	102
microbiological wastes.....	112, 114
moisture.....	20, 24, 32, 51, 70, 96
moisture meters.....	51
molecular typing.....	28
monochloramine.....	54
mop heads.....	75
multidisciplinary team.....	23, 91
municipal water.....	47, 50, 224
municipal water systems/utilities.....	45, 221
<i>Mycobacterium tuberculosis</i>	5, 7, 10, 73, 114
myiasis.....	81

N

negative air pressure.....	6, 12, 18, 19, 21, 36, 99, 100, 104, 111
neutralizer chemicals.....	96
NIOSH.....	40
nontuberculous mycobacteria (NTM).....	5, 41, 44–46, 60, 63, 70, 71, 223

O

operating rooms.....	13, 15, 17, 34, 38, 76, 82, 87, 109, 111
opportunistic infections.....	4, 5
organic matter.....	78
OSHA.....	13, 73, 77, 79, 98, 100, 113
outdoor air.....	14, 15, 18, 25, 91
oxygen-based laundry detergents.....	101

P

particle sampling.....	27, 33, 89
performance measures.....	2
periodic culturing.....	57
peritoneal dialysis.....	64, 65

personal protective equipment 77, 98, 99, 112, 114, 225
 persons with disabilities 108, 109
 person-to-person transmission 12, 85
 pest control 82
 phenolics 76
 pillows 104
 pipes 64, 69, 221, 223, 224
 planktonic organisms 221
 plastic enclosures 31
 plastic wrapping 74
Pneumocystis carinii 9
 pneumonia 42, 55
 point-of-use fixtures 47, 51, 224
 polyvinylchloride (PVC) 46, 64
 pools 67
 positive air pressure 18, 38
 potable water 220
 potted plants 8, 80
 pressure differentials 18, 19, 25, 30, 38
 primates 105, 106, 111
 prions 86, 116
 privacy curtains 75
 product water 64, 70
 protective environment (PE) 6, 18, 19, 34, 56, 108
Pseudomonas aeruginosa 5, 11, 20, 42, 68, 70, 71, 73, 79,
 80, 96, 104, 221
 pseudo-outbreaks 44, 70
 pyrogenic reactions 60, 61

Q

quality assurance 89, 94, 95

R

R2A media 222
 rank order 27
 recirculation 16, 18
 recirculation loops 46
 recreational equipment 69
 reduced nutrient media 94
 reducing agent 94
 relative humidity 17
 renovation 13, 14, 23, 37
 repairs 31
 reprocess hemodialyzers 61, 223
 research animals 111
 reservoirs 3, 6, 41, 42, 71, 79, 83, 95, 105, 211
 resident animals 107
 respirable particles 27, 28, 90
 respirators 26, 40
 respiratory protection 36, 78
 respiratory syncytial virus (RSV) 6, 12, 85
 respiratory therapy equipment 224
 return air 14
 return temperature 54
 reverse osmosis (RO) 52, 54, 59, 60, 63
 rewiring 25
 rinse water monitoring 70

RODAC plates 102
 rodents 67
 rooftops 30

S

Sabouraud dextrose agar 96
 sample/rinse methods 97
 sanitary sewer 116, 117
 SARS 86
 SARS-CoV 86
 scalding 49, 51
 screens 82
 scrub suits 98, 99
 sealed windows 19, 26, 29, 89
 sedimentation 90, 93, 211
 select agents 114, 115
 self-closing doors 19
 semicritical device 70
 service animal 105, 108–110
 settle plates 28, 90, 93, 211
 sewage spills 51
 sharps containers 113
 shock decontamination 51
 shower heads 47, 49, 54, 220, 221, 222, 224, 225
 showers 47, 48
 skin antiseptics 73
 smallpox 36
 smallpox virus 7, 12
 smoke tubes 20, 34, 36
 sodium hydroxide 87
 sodium hypochlorite 67, 69, 73, 77, 83, 84, 87, 88, 94, 225
 solid-organ transplant program 56
 sorting (laundry) 98, 100
 Spaulding classification 71, 98
 spills 75, 77, 79
 standard precautions 100, 111
 standards 2, 14, 71, 88, 90, 112, 223
Staphylococcus aureus 10, 11, 38, 64, 73, 99, 104
 state codes/regulations 55, 69
 steam jet 101
 steam sterilization (of medical waste) 113, 114
 sterile water 55
 storage tanks 63, 64
 streptococci 10, 38
 supplemental treatment methods 53
 surgical gowns and drapes 103
 surgical site infections (SSI) 11, 38, 65
 surgical smoke 40
 surveillance 26, 51, 57, 99, 223
 swabs 224

T

tacky mats 76
 tap water 42, 44, 57, 65, 66, 70
 TB patients 38
 temperature (air) 13, 14, 17, 89
 temperature (water) 40, 45, 49, 68, 101, 221, 227

thermostatic mixing valves	49
transport and storage (of medical waste)	113
treated items/products	79, 103
tryptic soy agar	94, 96, 222
tub liners	68
tuberculocidal claim	73
tuberculosis (TB)	35

U

ultrapure dialysate	61
ultraviolet germicidal irradiation (UVGI)	14, 16, 17, 36, 38
uniforms	98, 99

V

vacuum breakers	47, 50
vacuum cleaners	76, 79
vacuuming	79
vancomycin-resistant enterococci (VRE)	3, 5, 82, 83, 105
vancomycin-resistant <i>Staphylococcus aureus</i> (VRSA)	83
variable air ventilation	20, 38
varicella-zoster virus (VZV)	5, 7, 40
vase water	81
vegetative bacteria	73
ventilation rates	18
ventilation systems	8, 9, 111
viable particles	9, 91

viral hemorrhagic fever	12
viral particles	11
viruses	11, 85
visual monitoring device	34
volumetric air samplers	29
volumetric sampling methods	28

W

wallboard	8, 22, 52
walls	25
washing machines and dryers	102
water conditioning	68
water distribution systems	64, 221, 227
water droplets	58
water pipes	46
water pressure	50
water quality	71, 94
water sampling	54, 94, 221, 224
water stagnation	227
water treatment system	63
waterborne transmission	46
weight-arrestance test	15
wet cleaning	79
whirlpool spas	59, 67, 69
whirlpools	68, 69
window chutes	33
windows	22, 59
wood	8, 9, 15, 35

The recommendations in this guideline for Ebola Virus Disease have been superseded by CDC's [Infection Prevention and Control Recommendations for Hospitalized Patients with Known or Suspected Ebola Virus Disease in U.S. Hospitals](#).

This information is in [Appendix A](#).

Click here for current information on [how Ebola virus is transmitted](#).

The recommendations in this guideline for Measles have been superseded by [CDC's Immunization of Healthcare Personnel: Recommendations of the Advisory Committee on Immunization Practices \(ACIP\)](#).

2007 Guideline for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings

**Jane D. Siegel, MD; Emily Rhinehart, RN MPH CIC; Marguerite Jackson, PhD;
Linda Chiarello, RN MS; the Healthcare Infection Control Practices Advisory
Committee**

Acknowledgement: The authors and HICPAC gratefully acknowledge Dr. Larry Strausbaugh for his many contributions and valued guidance in the preparation of this guideline.

Suggested citation: Siegel JD, Rhinehart E, Jackson M, Chiarello L, and the Healthcare Infection Control Practices Advisory Committee, 2007 Guideline for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings
<http://www.cdc.gov/ncidod/dhqp/pdf/isolation2007.pdf>

**Healthcare Infection Control Practices
Advisory Committee (HICPAC):**

Chair

Patrick J. Brennan, MD
Professor of Medicine
Division of Infectious Diseases
University of Pennsylvania Medical School

Executive Secretary

Michael Bell, MD
Division of Healthcare Quality Promotion
National Center for Infectious Diseases
Centers for Disease Control and Prevention

Members

BRINSKO, Vicki L., RN, BA Infection
Control Coordinator Vanderbilt
University Medical Center

DELLINGER, E. Patchen., MD
Professor of Surgery
University of Washington School of
Medicine

ENGEL, Jeffrey, MD
Head General Communicable Disease
Control
Branch
North Carolina State Epidemiologist

GORDON, Steven M., MD
Chairman, Department of Infections
Diseases
Hospital Epidemiologist
Cleveland Clinic Foundation
Department of Infectious Disease

HARRELL, Lizzie J., PhD, D(ABMM)
Research Professor of Molecular Genetics,
Microbiology and Pathology
Associate Director, Clinical Microbiology
Duke University Medical Center

O'BOYLE, Carol, PhD, RN
Assistant Professor, School of Nursing
University of Minnesota

PEGUES, David Alexander, MD
Division of Infectious Diseases
David Geffen School of Medicine at UCLA

PERROTTA, Dennis M. PhD., CIC
Adjunct Associate Professor of
Epidemiology
University of Texas School of Public Health
Texas A&M University School of Rural
Public
Health

PITT, Harriett M., MS, CIC, RN
Director, Epidemiology
Long Beach Memorial Medical Center

RAMSEY, Keith M., MD
Professor of Medicine
Medical Director of Infection Control
The Brody School of Medicine at East
Carolina
University

SINGH, Nalini, MD, MPH
Professor of Pediatrics
Epidemiology and International Health
The George Washington University
Children's National
Medical Center

STEVENSON, Kurt Brown, MD, MPH
Division of Infectious Diseases
Department of Internal Medicine
The Ohio State University Medical Center

SMITH, Philip W., MD
Chief, Section of Infectious Diseases
Department of Internal Medicine
University of Nebraska Medical Center

HICPAC membership (past)

Robert A. Weinstein, MD (Chair)
Cook County Hospital
Chicago, IL

Jane D. Siegel, MD (Co-Chair)
University of Texas Southwestern Medical
Center
Dallas, TX

Michele L. Pearson, MD
(Executive Secretary)
Centers for Disease Control and Prevention
Atlanta, GA

Raymond Y.W. Chinn, MD
Sharp Memorial Hospital
San Diego, CA

Alfred DeMaria, Jr, MD
Massachusetts Department of Public Health
Jamaica Plain, MA

James T. Lee, MD, PhD
University of Minnesota
Minneapolis, MN

William A. Rutala, PhD, MPH
University of North Carolina Health Care
System
Chapel Hill, NC

William E. Scheckler, MD
University of Wisconsin
Madison, WI

Beth H. Stover, RN
Kosair Children's Hospital
Louisville, KY

Marjorie A. Underwood, RN, BSN CIC
Mt. Diablo Medical Center
Concord, CA

HICPAC Liaisons

William B. Baine, MD
Liaison to Agency for Healthcare Quality
Research

Joan Blanchard, RN, MSN, CNOR
Liaison to Association of periOperative
Registered Nurses

Patrick J. Brennan, MD
Liaison to Board of Scientific Counselors

Nancy Bjerke, RN, MPH, CIC
Liaison to Association of Professionals in
Infection Prevention and Control

Jeffrey P. Engel, MD
Liaison to Advisory Committee on
Elimination of
Tuberculosis

David Henderson, MD
Liaison to National Institutes of Health

Lorine J. Jay MPH, RN, CPHQ
Liaison to Healthcare Resources Services
Administration

Stephen F. Jencks, MD, MPH
Liaison to Center for Medicare and Medicaid
Services

Sheila A. Murphey, MD
Liaison to Food and Drug Administration

Mark Russi, MD, MPH
Liaison to American College of Occupational
and
Environmental Medicine

Rachel L. Stricof, MPH
Liaison to Advisory Committee on
Elimination of
Tuberculosis

Michael L. Tapper, MD
Liaison to Society for Healthcare
Epidemiology of
America

Robert A. Wise, MD
Liaison to Joint Commission on the
Accreditation of
Healthcare Organizations

Authors' Associations

Jane D. Siegel, MD
Professor of Pediatrics
Department of Pediatrics
University of Texas Southwestern Medical
Center

Emily Rhinehart RN MPH CIC CPHQ
Vice President
AIG Consultants, Inc.

Marguerite Jackson, RN PhD CIC
Director, Administrative Unit, National
Tuberculosis
Curriculum Consortium, Department of
Medicine
University of California San Diego

Linda Chiarello, RN MS
Division of Healthcare Quality Promotion
National Center for Infectious Diseases,
CDC

TABLE OF CONTENTS

Executive Summary	7
Abbreviations.....	11
Part I: Review of the Scientific Data Regarding Transmission of Infectious Agents in Healthcare Settings	12
I.A. Evolution of the 2007 document	12
I.B. Rationale for Standard and Transmission-Based Precautions in healthcare settings.....	14
I.B.1. Source of infectious agents.....	14
I.B.2. Susceptible hosts.....	14
I.B.3. Modes of transmission	15
I.B.3.a. Contact transmission.....	15
I.B.3.a.i. Direct contact transmission	16
I.B.3.a.ii. Indirect contact transmission	16
I.B.3.b. Droplet transmission	17
I.B.3.c. Airborne transmission.....	18
I.B.3.d. Emerging issues and controversies concerning bioaerosols and airborne transmission of infectious agents.....	19
I.B.3.d.i. Transmission from patients.....	19
I.B.3.d.ii. Transmission from the environment.....	20
I.B.3.e. Other sources of infection	20
I.C. Infectious agents of special infection control interest for healthcare settings.....	20
I.C.1. Epidemiologically important organisms.....	21
I.C.1.a. <i>Clostridium difficile</i>	21
I.C.1.b. Multidrug-resistant organisms(MDROs).....	22
I.C.2. Agents of bioterrorism.....	23
I.C.3. Prions	24
I.C.4. Severe acute respiratory syndrome (SARS)	26
I.C.5. Monkeypox	28
I.C.6. Noroviruses.....	28
I.C.7. Hemorrhagic fever viruses.....	29
I.D. Transmission risks associated with specific types of healthcare settings	31
I.D.1. Hospitals.....	31
I.D.1.a. Intensive care units	31
I.D.1.b. Burn units.....	32
I.D.1.c. Pediatrics	33
I.D.2. Non-acute care settings.....	34
I.D.2.a. Long term care	34
I.D.2.b. Ambulatory care settings	35
I.D.2.c. Home care	36
I.D.2.d. Other sites of healthcare delivery	37
I.E. Transmission risks associated with special patient populations.....	38
I.E.1. Immunocompromised patients	38
I.E.2. Cystic fibrosis patients	39

I.F. New therapies with potential transmissible infectious agents.....	39
I.F.1. Gene therapy	39
I.F.2. Infections Transmitted through Blood, Organs and Tissues.....	40
I.F.3. Xenotransplantation and tissue allografts	40

Part II. Fundamental Elements to Prevent Transmission of Infectious Agents in Healthcare Settings..... 41

II.A. Healthcare system components that influence the effectiveness of precautions to prevent transmission	41
II.A.1. Administrative measures.....	41
II.A.1.a. Scope of Work and Staffing Needs for Infection Control Professionals (ICP).....	42
II.A.1.a.i. Infection Control Liaison Nurse.....	43
II.A.1.b. Bedside nurse staffing.....	43
II.A.1.c. Clinical microbiology laboratory support.....	43
II.A.2. Institutional safety culture and organizational characteristics.....	45
II.A.3. Adherence of healthcare personnel to recommended guidelines....	45
II.B. Surveillance for healthcare-associated infections (HAIs).....	46
II.C. Education of healthcare workers, patients, and families	47
II.D. Hand hygiene.....	49
II.E. Personal protective equipment for healthcare personnel	49
II.E.1. Gloves.....	50
II.E.2. Isolation gowns	51
II.E.3. Face protection: masks, goggles, face shields.....	52
II.E.3.a. Masks.....	52
II.E.3.b. Goggles, face shields.....	52
II.E.4. Respiratory protection	53
II.F. Safe work practices to prevent HCW exposure to bloodborne pathogens	55
II.F.1. Prevention of needlesticks and other sharps-related injuries	55
II.F.2. Prevention of mucous membrane contact.....	56
II.F.2.a. Precautions during aerosol-generating procedures.....	56
II.G. Patient placement.....	56
II.G.1. Hospitals and long-term care settings	56
II.G.2. Ambulatory care settings	58
II.G.3. Home care	59
II.H. Transport of patients.....	59
II.I. Environmental measures.....	60
II.J. Patient care equipment, instruments/devices	61
II.K. Textiles and laundry	61
II.L. Solid waste.....	62
II.M. Dishware and eating utensils.....	62
II.N. Adjunctive measures	63
II.N.1. Chemoprophylaxis	63
II.N.2. Immunoprophylaxis.....	63

II.N.3. Management of visitors.....	64
II.N.3.a. Visitors as sources of infection.....	64
II.N.3.b. Use of barrier precautions by visitors.....	65
Part III. HICPAC Precautions to Prevent Transmission of Infectious Agents	66
III.A. Standard Precautions	66
III.A.1. New Standard Precautions for patients.....	67
III.A.1.a. Respiratory Hygiene/Cough Etiquette.....	67
III.A.1.b. Safe Injection Practices.....	68
III.A.1.c. Infection Control Practices for Special Lumbar Puncture Procedures.....	69
III.B. Transmission-Based Precautions	69
III.B.1. Contact Precautions.....	70
III.B.2. Droplet Precautions	70
III.B.3. Airborne Infection Isolation Precautions.....	71
III.C. Syndromic or empiric application of Transmission-Based Precautions.....	71
III.D. Discontinuation of precautions.....	72
III.E. Application of Transmission-Based Precautions in ambulatory and home care settings.....	72
III.F. Protective environment (PE)	73
Part IV: Recommendations.....	74
Appendix A. Type and duration of precautions needed for selected infections and conditions	93
Tables	
Table 1. Recent history of guidelines for prevention of healthcare-associated infections.....	
Table 2. Clinical syndromes or conditions warranting additional empiric transmission-based precautions pending confirmation of diagnosis.....	
Table 3. Infection control considerations for high-priority (CDC Category A) diseases that may result from bioterrorist attacks or are considered to be bioterrorist threats ..	
Table 4. Recommendations for application of Standard Precautions for the care of all patients in all healthcare settings.....	
Table 5. Components of a Protective Environment	
Figure Sequence for donning and removing PPE	
Glossary.....	
References.....	

EXECUTIVE SUMMARY

The *Guideline for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings 2007* updates and expands the *1996 Guideline for Isolation Precautions in Hospitals*. The following developments led to revision of the 1996 guideline:

1. The transition of healthcare delivery from primarily acute care hospitals to other healthcare settings (e.g., home care, ambulatory care, free-standing specialty care sites, long-term care) created a need for recommendations that can be applied in all healthcare settings using common principles of infection control practice, yet can be modified to reflect setting-specific needs. Accordingly, the revised guideline addresses the spectrum of healthcare delivery settings. Furthermore, the term “nosocomial infections” is replaced by “healthcare-associated infections” (HAIs) to reflect the changing patterns in healthcare delivery and difficulty in determining the geographic site of exposure to an infectious agent and/or acquisition of infection.
2. The emergence of new pathogens (e.g., SARS-CoV associated with the severe acute respiratory syndrome [SARS], Avian influenza in humans), renewed concern for evolving known pathogens (e.g., *C. difficile*, noroviruses, community-associated MRSA [CA-MRSA]), development of new therapies (e.g., gene therapy), and increasing concern for the threat of bioweapons attacks, established a need to address a broader scope of issues than in previous isolation guidelines.
3. The successful experience with Standard Precautions, first recommended in the 1996 guideline, has led to a reaffirmation of this approach as the foundation for preventing transmission of infectious agents in all healthcare settings. New additions to the recommendations for Standard Precautions are Respiratory Hygiene/Cough Etiquette and safe injection practices, including the use of a mask when performing certain high-risk, prolonged procedures involving spinal canal punctures (e.g., myelography, epidural anesthesia). The need for a recommendation for Respiratory Hygiene/Cough Etiquette grew out of observations during the SARS outbreaks where failure to implement simple source control measures with patients, visitors, and healthcare personnel with respiratory symptoms may have contributed to SARS coronavirus (SARS-CoV) transmission. The recommended practices have a strong evidence base. The continued occurrence of outbreaks of hepatitis B and hepatitis C viruses in ambulatory settings indicated a need to re-iterate safe injection practice recommendations as part of Standard Precautions. The addition of a mask for certain spinal injections grew from recent evidence of an associated risk for developing meningitis caused by respiratory flora.
4. The accumulated evidence that environmental controls decrease the risk of life-threatening fungal infections in the most severely immunocompromised patients (allogeneic hematopoietic stem-cell transplant patients) led to the update on the components of the Protective Environment (PE).
5. Evidence that organizational characteristics (e.g., nurse staffing levels and composition, establishment of a safety culture) influence healthcare personnel adherence to recommended infection control practices, and therefore are important factors in preventing transmission of infectious agents, led to a new

emphasis and recommendations for administrative involvement in the development and support of infection control programs.

6. Continued increase in the incidence of HAIs caused by multidrug-resistant organisms (MDROs) in all healthcare settings and the expanded body of knowledge concerning prevention of transmission of MDROs created a need for more specific recommendations for surveillance and control of these pathogens that would be practical and effective in various types of healthcare settings.

This document is intended for use by infection control staff, healthcare epidemiologists, healthcare administrators, nurses, other healthcare providers, and persons responsible for developing, implementing, and evaluating infection control programs for healthcare settings across the continuum of care. The reader is referred to other guidelines and websites for more detailed information and for recommendations concerning specialized infection control problems.

Parts I - III: Review of the Scientific Data Regarding Transmission of Infectious Agents in Healthcare Settings

Part I reviews the relevant scientific literature that supports the recommended prevention and control practices. As with the 1996 guideline, the modes and factors that influence transmission risks are described in detail. New to the section on transmission are discussions of bioaerosols and of how droplet and airborne transmission may contribute to infection transmission. This became a concern during the SARS outbreaks of 2003, when transmission associated with aerosol-generating procedures was observed. Also new is a definition of “epidemiologically important organisms” that was developed to assist in the identification of clusters of infections that require investigation (i.e. multidrug-resistant organisms, *C. difficile*). Several other pathogens that hold special infection control interest (i.e., norovirus, SARS, Category A bioterrorist agents, prions, monkeypox, and the hemorrhagic fever viruses) also are discussed to present new information and infection control lessons learned from experience with these agents. This section of the guideline also presents information on infection risks associated with specific healthcare settings and patient populations.

Part II updates information on the basic principles of hand hygiene, barrier precautions, safe work practices and isolation practices that were included in previous guidelines. However, new to this guideline, is important information on healthcare system components that influence transmission risks, including those under the influence of healthcare administrators. An important administrative priority that is described is the need for appropriate infection control staffing to meet the ever-expanding role of infection control professionals in the modern, complex healthcare system. Evidence presented also demonstrates another administrative concern, the importance of nurse staffing levels, including numbers of appropriately trained nurses in ICUs for preventing HAIs. The role of the clinical microbiology laboratory in supporting infection control is described to emphasize the need for this service in healthcare facilities. Other factors that influence transmission risks are discussed i.e., healthcare worker adherence to recommended infection control practices, organizational safety culture or climate, education and training. Discussed for the first time in an isolation guideline is surveillance of healthcare-associated infections. The information presented will be useful to new infection control professionals as

well as persons involved in designing or responding to state programs for public reporting of HAI rates.

Part III describes each of the categories of precautions developed by the Healthcare Infection Control Practices Advisory Committee (HICPAC) and the Centers for Disease Control and Prevention (CDC) and provides guidance for their application in various healthcare settings. The categories of Transmission-Based Precautions are unchanged from those in the 1996 guideline: Contact, Droplet, and Airborne. One important change is the recommendation to don the indicated personal protective equipment (gowns, gloves, mask) *upon entry into the patient's room* for patients who are on Contact and/or Droplet Precautions since the nature of the interaction with the patient cannot be predicted with certainty and contaminated environmental surfaces are important sources for transmission of pathogens.

In addition, the Protective Environment (PE) for allogeneic hematopoietic stem cell transplant patients, described in previous guidelines, has been updated.

Tables, Appendices, and other Information

There are several tables that summarize important information: 1) a summary of the evolution of this document; 2) guidance on using empiric isolation precautions according to a clinical syndrome; 3) a summary of infection control recommendations for category A agents of bioterrorism; 4) components of Standard Precautions and recommendations for their application; 5) components of the Protective Environment; and 6) a glossary of definitions used in this guideline. New in this guideline is a figure that shows a recommended sequence for donning and removing personal protective equipment used for isolation precautions to optimize safety and prevent self-contamination during removal.

Appendix A: Type and Duration of Precautions Recommended for Selected Infections and Conditions

Appendix A consists of an updated alphabetical list of most infectious agents and clinical conditions for which isolation precautions are recommended. A preamble to the Appendix provides a rationale for recommending the use of one or more Transmission-Based Precautions, in addition to Standard Precautions, based on a review of the literature and evidence demonstrating a real or potential risk for person-to-person transmission in healthcare settings. The type and duration of recommended precautions are presented with additional comments concerning the use of adjunctive measures or other relevant considerations to prevent transmission of the specific agent. Relevant citations are included.

Pre- Publication of the Guideline on Preventing Transmission of MDROs

New to this guideline is a comprehensive review and detailed recommendations for prevention of transmission of MDROs. This portion of the guideline was published electronically in October 2006 and updated in November, 2006 (Siegel JD, Rhinehart E, Jackson M, Chiarello L and HICPAC. Management of Multidrug-Resistant Organisms in Healthcare Settings 2006 www.cdc.gov/ncidod/dhqp/pdf/ar/mdroGuideline2006.pdf), and is considered a part of the Guideline for Isolation Precautions. This section provides a detailed review of the complex topic of MDRO control in healthcare settings and is intended to provide a context for evaluation of MDRO at individual healthcare settings. A rationale and

institutional requirements for developing an effective MDRO control program are summarized. Although the focus of this guideline is on measures to prevent *transmission* of MDROs in healthcare settings, information concerning the judicious use of antimicrobial agents is presented since such practices are intricately related to the size of the reservoir of MDROs which in turn influences transmission (e.g. colonization pressure). There are two tables that summarize recommended prevention and control practices using the following seven categories of interventions to control MDROs: administrative measures, education of healthcare personnel, judicious antimicrobial use, surveillance, infection control precautions, environmental measures, and decolonization. Recommendations for each category apply to and are adapted for the various healthcare settings. With the increasing incidence and prevalence of MDROs, all healthcare facilities must prioritize effective control of MDRO transmission. Facilities should identify prevalent MDROs at the facility, implement control measures, assess the effectiveness of control programs, and demonstrate decreasing MDRO rates. A set of intensified MDRO prevention interventions is presented to be added 1) if the incidence of transmission of a target MDRO is NOT decreasing despite implementation of basic MDRO infection control measures, and 2) when the *first* case(s) of an epidemiologically important MDRO is identified within a healthcare facility.

Summary

This updated guideline responds to changes in healthcare delivery and addresses new concerns about transmission of infectious agents to patients and healthcare workers in the United States and infection control. The primary objective of the guideline is to improve the safety of the nation's healthcare delivery system by reducing the rates of HAIs.

Abbreviations Used in the Guideline

AIIR	Airborne infection isolation room
CDC	Centers for Disease Control and Prevention
CF	Cystic fibrosis
CJD	Creutzfeld-Jakob Disease
CLSI	Clinical Laboratory Standards Institute
ESBL	Extended spectrum beta-lactamases
FDA	Food and Drug Administration
HAI	Healthcare-associated infections
HBV	Hepatitis B virus
HCV	Hepatitis C virus
HEPA	High efficiency particulate air [filtration]
HICPAC	Healthcare Infection Control Practices Advisory Committee
HIV	Human immunodeficiency virus
HCW	Healthcare worker
HSCT	Hematopoietic stem-cell transplant
ICU	Intensive care unit LTCF Long-term care facility
MDRO	Multidrug-resistant organism
MDR-GNB	Multidrug-resistant gram-negative bacilli
MRSA	Methicillin-resistant <i>Staphylococcus aureus</i>
NCCLS	National Committee for Clinical Laboratory Standards
NICU	Neonatal intensive care unit
NIOSH	National Institute for Occupational Safety and Health, CDC
NNIS	National Nosocomial Infection Surveillance
NSSP	Nonsusceptible <i>Streptococcus pneumoniae</i>
OSHA	Occupational Safety and Health Administration
PICU	Pediatric intensive care unit
PPE	Personal protective equipment
RSV	Respiratory syncytial virus
SARS	Severe acquired respiratory syndrome
vCJD	variant Creutzfeld-Jakob Disease
VRE	Vancomycin-resistant enterococci
WHO	World Health Organization

Part I:

Review of Scientific Data Regarding Transmission of Infectious Agents in Healthcare Settings

I.A. Evolution of the 2007 Document

The *Guideline for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings 2007* builds upon a series of isolation and infection prevention documents promulgated since 1970. These previous documents are summarized and referenced in Table 1 and in Part I of the *1996 Guideline for Isolation Precautions in Hospitals*¹.

Objectives and methods The objectives of this guideline are to 1) provide infection control recommendations for all components of the healthcare delivery system, including hospitals, long-term care facilities, ambulatory care, home care and hospice; 2) reaffirm Standard Precautions as the foundation for preventing transmission during patient care in all healthcare settings; 3) reaffirm the importance of implementing Transmission-Based Precautions based on the clinical presentation or syndrome and likely pathogens until the infectious etiology has been determined (Table 2); and 4) provide epidemiologically sound and, whenever possible, evidence-based recommendations.

This guideline is designed for use by individuals who are charged with administering infection control programs in hospitals and other healthcare settings. The information also will be useful for other healthcare personnel, healthcare administrators, and anyone needing information about infection control measures to prevent transmission of infectious agents. Commonly used abbreviations are provided on page 12 and terms used in the guideline are defined in the Glossary (page 137).

Med-line and Pub Med were used to search for relevant studies published in English, focusing on those published since 1996. Much of the evidence cited for preventing transmission of infectious agents in healthcare settings is derived from studies that used “quasi-experimental designs”, also referred to as nonrandomized, pre- post-intervention study designs². Although these types of studies can provide valuable information regarding the effectiveness of various interventions, several factors decrease the certainty of attributing improved outcome to a specific intervention. These include: difficulties in controlling for important confounding variables; the use of multiple interventions during an outbreak; and results that are explained by the statistical principle of regression to the mean, (e.g., improvement over time without any intervention)³.

Observational studies remain relevant and have been used to evaluate infection control interventions^{4,5}. The quality of studies, consistency of results and correlation with results from randomized, controlled trials when available were considered during the literature review and assignment of evidence-based categories (See Part IV: Recommendations) to the recommendations in this guideline. Several authors have summarized properties to consider when evaluating studies for the purpose of determining if the results should change practice or in designing new studies^{2,6,7}.

Changes or clarifications in terminology This guideline contains four changes in terminology from the 1996 guideline:

- f The term *nosocomial infection* is retained to refer only to infections acquired in hospitals. The term *healthcare-associated infection (HAI)* is used to refer to infections associated with healthcare delivery in any setting (e.g., hospitals, long-term care facilities, ambulatory settings, home care). This term reflects the inability to determine with certainty where the pathogen is acquired since patients may be colonized with or exposed to potential pathogens outside of the healthcare setting, before receiving health care, or may develop infections caused by those pathogens when exposed to the conditions associated with delivery of healthcare. Additionally, patients frequently move among the various settings within a healthcare system⁸.
- f A new addition to the practice recommendations for Standard Precautions is *Respiratory Hygiene/Cough Etiquette*. While Standard Precautions generally apply to the recommended practices of healthcare personnel during patient care, Respiratory Hygiene/Cough Etiquette applies broadly to all persons who enter a healthcare setting, including healthcare personnel, patients and visitors. These recommendations evolved from observations during the SARS epidemic that failure to implement basic source control measures with patients, visitors, and healthcare personnel with signs and symptoms of respiratory tract infection may have contributed to SARS coronavirus (SARS-CoV) transmission. This concept has been incorporated into CDC planning documents for SARS and pandemic influenza^{9, 10}.
- f The term “*Airborne Precautions*” has been supplemented with the term “*Airborne Infection Isolation Room (AIIR)*” for consistency with the *Guidelines for Environmental Infection Control in Healthcare Facilities*¹¹, the *Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health-Care Settings 2005*¹² and the American Institute of Architects (AIA) guidelines for design and construction of hospitals, 2006¹³.
- f A set of prevention measures termed *Protective Environment* has been added to the precautions used to prevent HAIs. These measures, which have been defined in other guidelines, consist of engineering and design interventions that decrease the risk of exposure to environmental fungi for severely immunocompromised allogeneic hematopoietic stem cell transplant (HSCT) patients during their highest risk phase, usually the first 100 days post transplant, or longer in the presence of graft-versus-host disease^{11, 13-15}. Recommendations for a Protective Environment apply only to acute care hospitals that provide care to HSCT patients.

Scope This guideline, like its predecessors, focuses primarily on interactions between patients and healthcare providers. The Guidelines for the Prevention of MDRO Infection were published separately in November 2006, and are available online at www.cdc.gov/ncidod/dhqp/index.html. Several other HICPAC

guidelines to prevent transmission of infectious agents associated with healthcare delivery are cited; e.g., *Guideline for Hand Hygiene*, *Guideline for Environmental Infection Control*, *Guideline for Prevention of Healthcare-Associated Pneumonia*, and *Guideline for Infection Control in Healthcare Personnel*^{11, 14, 16, 17}. In combination, these provide comprehensive guidance on the primary infection control measures for ensuring a safe environment for patients and healthcare personnel.

This guideline does not discuss in detail specialized infection control issues in defined populations that are addressed elsewhere, (e.g., *Recommendations for Preventing Transmission of Infections among Chronic Hemodialysis Patients*, *Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health-Care Facilities 2005*, *Guidelines for Infection Control in Dental Health-Care Settings* and *Infection Control Recommendations for Patients with Cystic Fibrosis*^{12, 18-20}). An exception has been made by including abbreviated guidance for a Protective Environment used for allogeneic HSCT recipients because components of the Protective Environment have been more completely defined since publication of the *Guidelines for Preventing Opportunistic Infections Among HSCT Recipients in 2000* and the *Guideline for Environmental Infection Control in Healthcare Facilities*^{11, 15}.

I.B. Rationale for Standard and Transmission-Based Precautions in healthcare settings

Transmission of infectious agents within a healthcare setting requires three elements: a source (or reservoir) of infectious agents, a susceptible host with a portal of entry receptive to the agent, and a mode of transmission for the agent. This section describes the interrelationship of these elements in the epidemiology of HAIs.

I.B.1. Sources of infectious agents Infectious agents transmitted during healthcare derive primarily from human sources but inanimate environmental sources also are implicated in transmission. Human reservoirs include patients²⁰⁻²⁸, healthcare personnel^{29-35 17, 36-39}, and household members and other visitors⁴⁰⁻⁴⁵. Such source individuals may have active infections, may be in the asymptomatic and/or incubation period of an infectious disease, or may be transiently or chronically colonized with pathogenic microorganisms, particularly in the respiratory and gastrointestinal tracts. The endogenous flora of patients (e.g., bacteria residing in the respiratory or gastrointestinal tract) also are the source of HAIs⁴⁶⁻⁵⁴.

I.B.2. Susceptible hosts Infection is the result of a complex interrelationship between a potential host and an infectious agent. Most of the factors that influence infection and the occurrence and severity of disease are related to the host. However, characteristics of the host-agent interaction as it relates to

pathogenicity, virulence and antigenicity are also important, as are the infectious dose, mechanisms of disease production and route of exposure⁵⁵. There is a spectrum of possible outcomes following exposure to an infectious agent. Some persons exposed to pathogenic microorganisms never develop symptomatic disease while others become severely ill and even die. Some individuals are prone to becoming transiently or permanently colonized but remain asymptomatic. Still others progress from colonization to symptomatic disease either immediately following exposure, or after a period of asymptomatic colonization. The immune state at the time of exposure to an infectious agent, interaction between pathogens, and virulence factors intrinsic to the agent are important predictors of an individual's outcome. Host factors such as extremes of age and underlying disease (e.g. diabetes^{56,57}), human immunodeficiency virus/acquired immune deficiency syndrome [HIV/AIDS]^{58,59}, malignancy, and transplants^{18,60,61} can increase susceptibility to infection as do a variety of medications that alter the normal flora (e.g., antimicrobial agents, gastric acid suppressants, corticosteroids, antirejection drugs, antineoplastic agents, and immunosuppressive drugs). Surgical procedures and radiation therapy impair defenses of the skin and other involved organ systems. Indwelling devices such as urinary catheters, endotracheal tubes, central venous and arterial catheters^{62,64} and synthetic implants facilitate development of HAIs by allowing potential pathogens to bypass local defenses that would ordinarily impede their invasion and by providing surfaces for development of biofilms that may facilitate adherence of microorganisms and protect from antimicrobial activity⁶⁵. Some infections associated with invasive procedures result from transmission within the healthcare facility; others arise from the patient's endogenous flora⁴⁶⁻⁵⁰. High-risk patient populations with noteworthy risk factors for infection are discussed further in Sections I.D, I.E., and I.F.

I.B.3. Modes of transmission Several classes of pathogens can cause infection, including bacteria, viruses, fungi, parasites, and prions. The modes of transmission vary by type of organism and some infectious agents may be transmitted by more than one route: some are transmitted primarily by direct or indirect contact, (e.g., *Herpes simplex* virus [HSV], respiratory syncytial virus, *Staphylococcus aureus*), others by the droplet, (e.g., influenza virus, *B. pertussis*) or airborne routes (e.g., *M. tuberculosis*). Other infectious agents, such as bloodborne viruses (e.g., hepatitis B and C viruses [HBV, HCV] and HIV are transmitted rarely in healthcare settings, via percutaneous or mucous membrane exposure. Importantly, not all infectious agents are transmitted from person to person. These are distinguished in Appendix A. The three principal routes of transmission are summarized below.

I.B.3.a. Contact transmission The most common mode of transmission, contact transmission is divided into two subgroups: direct contact and indirect contact.

I.B.3.a.i. Direct contact transmission Direct transmission occurs when microorganisms are transferred from one infected person to another person without a contaminated intermediate object or person. Opportunities for direct contact transmission between patients and healthcare personnel have been summarized in the Guideline for Infection Control in Healthcare Personnel, 1998¹⁷ and include:

- blood or other blood-containing body fluids from a patient directly enters a caregiver's body through contact with a mucous membrane⁶⁶ or breaks (i.e., cuts, abrasions) in the skin⁶⁷.
- mites from a scabies-infested patient are transferred to the skin of a caregiver while he/she is having direct ungloved contact with the patient's skin^{68, 69}.
- a healthcare provider develops herpetic whitlow on a finger after contact with HSV when providing oral care to a patient without using gloves or HSV is transmitted to a patient from a herpetic whitlow on an ungloved hand of a healthcare worker (HCW)^{70, 71}.

I.B.3.a.ii. Indirect contact transmission Indirect transmission involves the transfer of an infectious agent through a contaminated intermediate object or person. In the absence of a point-source outbreak, it is difficult to determine how indirect transmission occurs. However, extensive evidence cited in the Guideline for Hand Hygiene in Health-Care Settings suggests that the contaminated hands of healthcare personnel are important contributors to indirect contact transmission¹⁶. Examples of opportunities for indirect contact transmission include:

- Hands of healthcare personnel may transmit pathogens after touching an infected or colonized body site on one patient or a contaminated inanimate object, if hand hygiene is not performed before touching another patient.^{72, 73}
- Patient-care devices (e.g., electronic thermometers, glucose monitoring devices) may transmit pathogens if devices contaminated with blood or body fluids are shared between patients without cleaning and disinfecting between patients^{74 75-77}.
- Shared toys may become a vehicle for transmitting respiratory viruses (e.g., respiratory syncytial virus^{24, 78, 79} or pathogenic bacteria (e.g., *Pseudomonas aeruginosa*⁸⁰) among pediatric patients.
- Instruments that are inadequately cleaned between patients before disinfection or sterilization (e.g., endoscopes or surgical instruments)⁸¹⁻⁸⁵ or that have manufacturing defects that interfere with the effectiveness of reprocessing^{86, 87} may transmit bacterial and viral pathogens.

Clothing, uniforms, laboratory coats, or isolation gowns used as personal protective equipment (PPE), may become contaminated with potential pathogens after care of a patient colonized or infected with an infectious agent, (e.g., MRSA⁸⁸, VRE⁸⁹, and *C. difficile*⁹⁰). Although contaminated clothing has not been

implicated directly in transmission, the potential exists for soiled garments to transfer infectious agents to successive patients.

I.B.3.b. Droplet transmission Droplet transmission is, technically, a form of contact transmission, and some infectious agents transmitted by the droplet route also may be transmitted by the direct and indirect contact routes. However, in contrast to contact transmission, respiratory droplets carrying infectious pathogens transmit infection when they travel directly from the respiratory tract of the infectious individual to susceptible mucosal surfaces of the recipient, generally over short distances, necessitating facial protection. Respiratory droplets are generated when an infected person coughs, sneezes, or talks^{91, 92} or during procedures such as suctioning, endotracheal intubation,⁹³⁻⁹⁶ cough induction by chest physiotherapy⁹⁷ and cardiopulmonary resuscitation^{98, 99}. Evidence for droplet transmission comes from epidemiological studies of disease outbreaks¹⁰⁰⁻¹⁰³, experimental studies¹⁰⁴ and from information on aerosol dynamics^{91, 105}. Studies have shown that the nasal mucosa, conjunctivae and less frequently the mouth, are susceptible portals of entry for respiratory viruses¹⁰⁶. The maximum distance for droplet transmission is currently unresolved, although pathogens transmitted by the droplet route have not been transmitted through the air over long distances, in contrast to the airborne pathogens discussed below. Historically, the area of defined risk has been a distance of ≤ 3 feet around the patient and is based on epidemiologic and simulated studies of selected infections^{103, 104}. Using this distance for donning masks has been effective in preventing transmission of infectious agents via the droplet route. However, experimental studies with smallpox^{107, 108} and investigations during the global SARS outbreaks of 2003¹⁰¹ suggest that droplets from patients with these two infections could reach persons located 6 feet or more from their source. It is likely that the distance droplets travel depends on the velocity and mechanism by which respiratory droplets are propelled from the source, the density of respiratory secretions, environmental factors such as temperature and humidity, and the ability of the pathogen to maintain infectivity over that distance¹⁰⁵. Thus, a distance of ≤ 3 feet around the patient is best viewed as an *example* of what is meant by “a short distance from a patient” and should not be used as the sole *criterion* for deciding when a mask should be donned to protect from droplet exposure. Based on these considerations, it may be prudent to don a mask when within 6 to 10 feet of the patient or upon entry into the patient’s room, especially when exposure to emerging or highly virulent pathogens is likely. More studies are needed to improve understanding of droplet transmission under various circumstances.

Droplet size is another variable under discussion. Droplets traditionally have been defined as being $>5 \mu\text{m}$ in size. Droplet nuclei, particles arising from desiccation of suspended droplets, have been associated with airborne transmission and defined as $\leq 5 \mu\text{m}$ in size¹⁰⁵, a reflection of the pathogenesis of pulmonary tuberculosis which is not generalizable to other organisms. Observations of particle dynamics have demonstrated that a range of droplet sizes, including those with diameters of $30\mu\text{m}$ or greater, can remain suspended

in the air¹⁰⁹. The behavior of droplets and droplet nuclei affect recommendations for preventing transmission. Whereas fine airborne particles containing pathogens that are able to remain infective may transmit infections over long distances, requiring AIIR to prevent its dissemination within a facility; organisms transmitted by the droplet route do not remain infective over long distances, and therefore do not require special air handling and ventilation. Examples of infectious agents that are transmitted via the droplet route include *Bordetella pertussis*¹¹⁰, influenza virus²³, adenovirus¹¹¹, rhinovirus¹⁰⁴, *Mycoplasma pneumoniae*¹¹², SARS-associated coronavirus (SARS-CoV)^{21, 96, 113}, group A streptococcus¹¹⁴, and *Neisseria meningitidis*^{95, 103, 115}. Although respiratory syncytial virus may be transmitted by the droplet route, direct contact with infected respiratory secretions is the most important determinant of transmission and consistent adherence to Standard plus Contact Precautions prevents transmission in healthcare settings^{24, 116, 117}.

Rarely, pathogens that are not transmitted routinely by the droplet route are dispersed into the air over short distances. For example, although *S. aureus* is transmitted most frequently by the contact route, viral upper respiratory tract infection has been associated with increased dispersal of *S. aureus* from the nose into the air for a distance of 4 feet under both outbreak and experimental conditions and is known as the “cloud baby” and “cloud adult” phenomenon¹¹⁸⁻¹²⁰.

I.B.3.c. Airborne transmission Airborne transmission occurs by dissemination of either airborne droplet nuclei or small particles in the respirable size range containing infectious agents that remain infective over time and distance (e.g., spores of *Aspergillus* spp, and *Mycobacterium tuberculosis*). Microorganisms carried in this manner may be dispersed over long distances by air currents and may be inhaled by susceptible individuals who have not had face-to-face contact with (or been in the same room with) the infectious individual¹²¹⁻¹²⁴. Preventing the spread of pathogens that are transmitted by the airborne route requires the use of special air handling and ventilation systems (e.g., AIIRs) to contain and then safely remove the infectious agent^{11, 12}. Infectious agents to which this applies include *Mycobacterium tuberculosis*¹²⁴⁻¹²⁷, rubeola virus (measles)¹²², and varicella-zoster virus (chickenpox)¹²³. In addition, published data suggest the possibility that variola virus (smallpox) may be transmitted over long distances through the air under unusual circumstances and AIIRs are recommended for this agent as well; however, droplet and contact routes are the more frequent routes of transmission for smallpox^{108, 128, 129}. In addition to AIIRs, respiratory protection with NIOSH certified N95 or higher level respirator is recommended for healthcare personnel entering the AIIR to prevent acquisition of airborne infectious agents such as *M. tuberculosis*¹².

For certain other respiratory infectious agents, such as influenza^{130, 131} and rhinovirus¹⁰⁴, and even some gastrointestinal viruses (e.g., norovirus¹³² and rotavirus¹³³) there is some evidence that the pathogen may be transmitted via small-particle aerosols, under natural and experimental conditions. Such transmission has occurred over distances longer than 3 feet but within a defined

airspace (e.g., patient room), suggesting that it is unlikely that these agents remain viable on air currents that travel long distances. AIIRs are not required routinely to prevent transmission of these agents. Additional issues concerning examples of small particle aerosol transmission of agents that are most frequently transmitted by the droplet route are discussed below.

I.B.3.d. Emerging issues concerning airborne transmission of infectious agents.

I.B.3.d.i. *Transmission from patients* The emergence of SARS in 2002, the importation of monkeypox into the United States in 2003, and the emergence of avian influenza present challenges to the assignment of isolation categories because of conflicting information and uncertainty about possible routes of transmission. Although SARS-CoV is transmitted primarily by contact and/or droplet routes, airborne transmission over a limited distance (e.g. within a room), has been suggested, though not proven¹³⁴⁻¹⁴¹. This is true of other infectious agents such as influenza virus¹³⁰ and noroviruses^{132, 142, 143}. Influenza viruses are transmitted primarily by close contact with respiratory droplets^{23, 102} and acquisition by healthcare personnel has been prevented by Droplet Precautions, even when positive pressure rooms were used in one center¹⁴⁴. However, inhalational transmission could not be excluded in an outbreak of influenza in the passengers and crew of a single aircraft¹³⁰. Observations of a protective effect of UV lights in preventing influenza among patients with tuberculosis during the influenza pandemic of 1957-'58 have been used to suggest airborne transmission^{145, 146}.

In contrast to the strict interpretation of an airborne route for transmission (i.e., long distances beyond the patient room environment), short distance transmission by small particle aerosols generated under specific circumstances (e.g., during endotracheal intubation) to persons in the immediate area near the patient has been demonstrated. Also, aerosolized particles <100 µm can remain suspended in air when room air current velocities exceed the terminal settling velocities of the particles¹⁰⁹. SARS-CoV transmission has been associated with endotracheal intubation, noninvasive positive pressure ventilation, and cardio•pulmonary resuscitation^{93, 94, 96, 98, 141}. Although the most frequent routes of transmission of noroviruses are contact and food and waterborne routes, several reports suggest that noroviruses may be transmitted through aerosolization of infectious particles from vomitus or fecal material^{142, 143, 147, 148}. It is hypothesized that the aerosolized particles are inhaled and subsequently swallowed.

Roy and Milton proposed a new classification for aerosol transmission when evaluating routes of SARS transmission: 1) *obligate*: under natural conditions, disease occurs following transmission of the agent only through inhalation of small particle aerosols (e.g., tuberculosis); 2) *preferential*: natural infection results from transmission through multiple routes, but small particle aerosols are the predominant route (e.g. measles, varicella); and 3) *opportunistic*: agents that naturally cause disease through other routes, but under special circumstances

may be transmitted via fine particle aerosols¹⁴⁹. This conceptual framework can explain rare occurrences of airborne transmission of agents that are transmitted most frequently by other routes (e.g., smallpox, SARS, influenza, noroviruses). Concerns about unknown or possible routes of transmission of agents associated with severe disease and no known treatment often result in more extreme prevention strategies than may be necessary; therefore, recommended precautions could change as the epidemiology of an emerging infection is defined and controversial issues are resolved.

I.B.3.d.ii. *Transmission from the environment* Some airborne infectious agents are derived from the environment and do not usually involve person-to-person transmission. For example, anthrax spores present in a finely milled powdered preparation can be aerosolized from contaminated environmental surfaces and inhaled into the respiratory tract^{150, 151}. Spores of environmental fungi (e.g., *Aspergillus spp.*) are ubiquitous in the environment and may cause disease in immunocompromised patients who inhale aerosolized (e.g., via construction dust) spores^{152, 153}. As a rule, neither of these organisms is subsequently transmitted from infected patients. However, there is one well-documented report of person-to-person transmission of *Aspergillus sp.* in the ICU setting that was most likely due to the aerosolization of spores during wound debridement¹⁵⁴. A Protective Environment refers to isolation practices designed to decrease the risk of exposure to environmental fungal agents in allogeneic HSCT patients^{11, 14, 15, 155-158}.

Environmental sources of respiratory pathogens (eg. Legionella) transmitted to humans through a common aerosol source is distinct from direct patient-to-patient transmission.

I.B.3.e. Other sources of infection Transmission of infection from sources other than infectious individuals include those associated with *common environmental sources or vehicles* (e.g. contaminated food, water, or medications (e.g. intravenous fluids). Although *Aspergillus spp.* have been recovered from hospital water systems¹⁵⁹, the role of water as a reservoir for immunosuppressed patients remains uncertain. *Vectorborne transmission* of infectious agents from mosquitoes, flies, rats, and other vermin also can occur in healthcare settings. Prevention of vector borne transmission is not addressed in this document.

I.C. Infectious agents of special infection control interest for healthcare settings

Several infectious agents with important infection control implications that either were not discussed extensively in previous isolation guidelines or have emerged recently are discussed below. These are epidemiologically important organisms (e.g., *C. difficile*), agents of bioterrorism, prions, SARS-CoV, monkeypox, noroviruses, and the hemorrhagic fever viruses. Experience with these agents has broadened the understanding of modes of transmission and effective

preventive measures. These agents are included for purposes of information and, for some (i.e., SARS-CoV, monkeypox), because of the lessons that have been learned about preparedness planning and responding effectively to new infectious agents.

I.C.1. Epidemiologically important organisms Any infectious agents transmitted in healthcare settings may, under defined conditions, become targeted for control because they are epidemiologically important. *C. difficile* is specifically discussed below because of wide recognition of its current importance in U.S. healthcare facilities. In determining what constitutes an “epidemiologically important organism”, the following characteristics apply:

- A propensity for transmission within healthcare facilities based on published reports and the occurrence of temporal or geographic clusters of > 2 patients, (e.g., *C. difficile*, norovirus, respiratory syncytial virus (RSV), influenza, rotavirus, *Enterobacter* spp; *Serratia* spp., group A streptococcus). A single case of healthcare-associated invasive disease caused by certain pathogens (e.g., group A streptococcus post-operatively¹⁶⁰, in burn units¹⁶¹, or in a LTCF¹⁶²; *Legionella* sp.^{14, 163}, *Aspergillus* sp.¹⁶⁴) is generally considered a trigger for investigation and enhanced control measures because of the risk of additional cases and severity of illness associated with these infections. Antimicrobial resistance
- Resistance to first-line therapies (e.g., MRSA, VISA, VRSA, VRE, ESBL-producing organisms).
- Common and uncommon microorganisms with unusual patterns of resistance within a facility (e.g., the first isolate of *Burkholderia cepacia* complex or *Ralstonia* spp. in non-CF patients or a quinolone-resistant strain of *Pseudomonas aeruginosa* in a facility).
- Difficult to treat because of innate or acquired resistance to multiple classes of antimicrobial agents (e.g., *Stenotrophomonas maltophilia*, *Acinetobacter* spp.).
- Association with serious clinical disease, increased morbidity and mortality (e.g., MRSA and MSSA, group A streptococcus)
- A newly discovered or reemerging pathogen

I.C.1.a. *C. difficile* *C. difficile* is a spore-forming gram positive anaerobic bacillus that was first isolated from stools of neonates in 1935¹⁶⁵ and identified as the most commonly identified causative agent of antibiotic-associated diarrhea and pseudomembranous colitis in 1977¹⁶⁶. This pathogen is a major cause of healthcare-associated diarrhea and has been responsible for many large outbreaks in healthcare settings that were extremely difficult to control. Important factors that contribute to healthcare-associated outbreaks include environmental contamination, persistence of spores for prolonged periods of time, resistance of spores to routinely used disinfectants and antiseptics, hand carriage by healthcare personnel to other patients, and exposure of patients to frequent courses of antimicrobial agents¹⁶⁷. Antimicrobials most frequently associated

with increased risk of *C. difficile* include third generation cephalosporins, clindamycin, vancomycin, and fluoroquinolones.

Since 2001, outbreaks and sporadic cases of *C. difficile* with increased morbidity and mortality have been observed in several U.S. states, Canada, England and the Netherlands¹⁶⁸⁻¹⁷². The same strain of *C. difficile* has been implicated in these outbreaks¹⁷³. This strain, toxinotype III, North American PFGE type 1, and PCR-ribotype 027 (NAP1/027). has been found to hyperproduce toxin A (16 fold increase) and toxin B (23 fold increase) compared with isolates from 12 different pulsed-field gel electrophoresis PFGE types. A recent survey of U.S. infectious disease physicians found that 40% perceived recent increases in the incidence and severity of *C. difficile* disease¹⁷⁴. Standardization of testing methodology and surveillance definitions is needed for accurate comparisons of trends in rates among hospitals¹⁷⁵. It is hypothesized that the incidence of disease and apparent heightened transmissibility of this new strain may be due, at least in part, to the greater production of toxins A and B, increasing the severity of diarrhea and resulting in more environmental contamination. Considering the greater morbidity, mortality, length of stay, and costs associated with *C. difficile* disease in both acute care and long term care facilities, control of this pathogen is now even more important than previously. Prevention of transmission focuses on syndromic application of Contact Precautions for patients with diarrhea, accurate identification of patients, environmental measures (e.g., rigorous cleaning of patient rooms) and consistent hand hygiene. Use of soap and water, rather than alcohol based handrubs, for mechanical removal of spores from hands, and a bleach-containing disinfectant (5000 ppm) for environmental disinfection, may be valuable when there is transmission in a healthcare facility. See Appendix A for specific recommendations.

I.C.1. b. Multidrug-Resistant Organisms (MDROs) In general, MDROs are defined as microorganisms – predominantly bacteria – that are resistant to one or more classes of antimicrobial agents¹⁷⁶. Although the names of certain MDROs suggest resistance to only one agent (e.g., methicillin-resistant *Staphylococcus aureus* [MRSA], vancomycin resistant enterococcus [VRE]), these pathogens are usually resistant to all but a few commercially available antimicrobial agents. This latter feature defines MDROs that are considered to be epidemiologically important and deserve special attention in healthcare facilities¹⁷⁷. Other MDROs of current concern include multidrug-resistant *Streptococcus pneumoniae* (MDRSP) which is resistant to penicillin and other broad-spectrum agents such as macrolides and fluoroquinolones, multidrug-resistant gram-negative bacilli (MDR- GNB), especially those producing extended spectrum beta-lactamases (ESBLs); and strains of *S. aureus* that are intermediate or resistant to vancomycin (i.e., VISA and VRSA)^{178-197 198}.

MDROs are transmitted by the same routes as antimicrobial susceptible infectious agents. Patient-to-patient transmission in healthcare settings, usually via hands of HCWs, has been a major factor accounting for the increase in MDRO incidence and prevalence, especially for MRSA and VRE in acute care

facilities¹⁹⁹⁻²⁰¹. Preventing the emergence and transmission of these pathogens requires a comprehensive approach that includes administrative involvement and measures (e.g., nurse staffing, communication systems, performance improvement processes to ensure adherence to recommended infection control measures), education and training of medical and other healthcare personnel, judicious antibiotic use, comprehensive surveillance for targeted MDROs, application of infection control precautions during patient care, environmental measures (e.g., cleaning and disinfection of the patient care environment and equipment, dedicated single-patient-use of non-critical equipment), and decolonization therapy when appropriate.

The prevention and control of MDROs is a national priority - one that requires that all healthcare facilities and agencies assume responsibility and participate in community-wide control programs^{176, 177}. A detailed discussion of this topic and recommendations for prevention was published in 2006 may be found at <http://www.cdc.gov/ncidod/dhqp/pdf/ar/mdroGuideline2006.pdf>

I.C.2. Agents of bioterrorism CDC has designated the agents that cause anthrax, smallpox, plague, tularemia, viral hemorrhagic fevers, and botulism as Category A (high priority) because these agents can be easily disseminated environmentally and/or transmitted from person to person; can cause high mortality and have the potential for major public health impact; might cause public panic and social disruption; and require special action for public health preparedness²⁰². General information relevant to infection control in healthcare settings for Category A agents of bioterrorism is summarized in Table 3. Consult www.bt.cdc.gov for additional, updated Category A agent information as well as information concerning Category B and C agents of bioterrorism and updates. Category B and C agents are important but are not as readily disseminated and cause less morbidity and mortality than Category A agents.

Healthcare facilities confront a different set of issues when dealing with a suspected bioterrorism event as compared with other communicable diseases. An understanding of the epidemiology, modes of transmission, and clinical course of each disease, as well as carefully drafted plans that provide an approach and relevant websites and other resources for disease-specific guidance to healthcare, administrative, and support personnel, are essential for responding to and managing a bioterrorism event. Infection control issues to be addressed include: 1) identifying persons who may be exposed or infected; 2) preventing transmission among patients, healthcare personnel, and visitors; 3) providing treatment, chemoprophylaxis or vaccine to potentially large numbers of people; 4) protecting the environment including the logistical aspects of securing sufficient numbers of AIIRs or designating areas for patient cohorts when there are an insufficient number of AIIRs available; 5) providing adequate quantities of appropriate personal protective equipment; and 6) identifying appropriate staff to care for potentially infectious patients (e.g., vaccinated healthcare personnel for

care of patients with smallpox). The response is likely to differ for exposures resulting from an intentional release compared with naturally occurring disease because of the large number of persons that can be exposed at the same time and possible differences in pathogenicity.

A variety of sources offer guidance for the management of persons exposed to the most likely agents of bioterrorism. Federal agency websites (e.g., www.usamriid.army.mil/publications/index.html, www.bt.cdc.gov) and state and county health department web sites should be consulted for the most up-to-date information. Sources of information on specific agents include: anthrax²⁰³; smallpox²⁰⁴⁻²⁰⁶; plague^{207, 208}; botulinum toxin²⁰⁹; tularemia²¹⁰; and hemorrhagic fever viruses:^{211, 212}.

I.C.2.a. Pre-event administration of smallpox (vaccinia) vaccine to healthcare personnel Vaccination of personnel in preparation for a possible smallpox exposure has important infection control implications²¹³⁻²¹⁵. These include the need for meticulous screening for vaccine contraindications in persons who are at increased risk for adverse vaccinia events; containment and monitoring of the vaccination site to prevent transmission in the healthcare setting and at home; and the management of patients with vaccinia-related adverse events^{216, 217}. The pre-event U.S. smallpox vaccination program of 2003 is an example of the effectiveness of carefully developed recommendations for both screening potential vaccinees for contraindications and vaccination site care and monitoring. Approximately 760,000 individuals were vaccinated in the Department of Defense and 40,000 in the civilian or public health populations from December 2002 to February 2005, including approximately 70,000 who worked in healthcare settings. There were no cases of eczema vaccinatum, progressive vaccinia, fetal vaccinia, or contact transfer of vaccinia in healthcare settings or in military workplaces^{218, 219}. Outside the healthcare setting, there were 53 cases of contact transfer from military vaccinees to close personal contacts (e.g., bed partners or contacts during participation in sports such as wrestling²²⁰). All contact transfers were from individuals who were not following recommendations to cover their vaccination sites. Vaccinia virus was confirmed by culture or PCR in 30 cases, and two of the confirmed cases resulted from tertiary transfer. All recipients, including one breast-fed infant, recovered without complication. Subsequent studies using viral culture and PCR techniques have confirmed the effectiveness of semipermeable dressings to contain vaccinia²²¹⁻²²⁴. This experience emphasizes the importance of ensuring that newly vaccinated healthcare personnel adhere to recommended vaccination-site care, especially if they are to care for high-risk patients. Recommendations for pre-event smallpox vaccination of healthcare personnel and vaccinia-related infection control recommendations are published in the MMWR^{216, 225} with updates posted on the CDC bioterrorism web site²⁰⁵.

I.C.3. Prions Creutzfeldt-Jakob disease (CJD) is a rapidly progressive, degenerative, neurologic disorder of humans with an incidence in the United States of approximately 1 person/million population/year^{226, 227}

(www.cdc.gov/ncidod/diseases/cjd/cjd.htm). CJD is believed to be caused by a transmissible proteinaceous infectious agent termed a prion. Infectious prions are isoforms of a host-encoded glycoprotein known as the prion protein. The incubation period (i.e., time between exposure and onset of symptoms) varies from two years to many decades. However, death typically occurs within 1 year of the onset of symptoms. Approximately 85% of CJD cases occur sporadically with no known environmental source of infection and 10% are familial. Iatrogenic transmission has occurred with most resulting from treatment with human cadaveric pituitary-derived growth hormone or gonadotropin^{228, 229}, from implantation of contaminated human dura mater grafts²³⁰ or from corneal transplants²³¹). Transmission has been linked to the use of contaminated neurosurgical instruments or stereotactic electroencephalogram electrodes^{232, 233, 234, 235}.

Prion diseases in animals include scrapie in sheep and goats, bovine spongiform encephalopathy (BSE, or “mad cow disease”) in cattle, and chronic wasting disease in deer and elk²³⁶. BSE, first recognized in the United Kingdom (UK) in 1986, was associated with a major epidemic among cattle that had consumed contaminated meat and bone meal.

The possible transmission of BSE to humans causing variant CJD (vCJD) was first described in 1996 and subsequently found to be associated with consumption of BSE-contaminated cattle products primarily in the United Kingdom. There is strong epidemiologic and laboratory evidence for a causal association between the causative agent of BSE and vCJD²³⁷. Although most cases of vCJD have been reported from the UK, a few cases also have been reported from Europe, Japan, Canada, and the United States. Most vCJD cases worldwide lived in or visited the UK during the years of a large outbreak of BSE (1980-96) and may have consumed contaminated cattle products during that time (www.cdc.gov/ncidod/diseases/cjd/cjd.htm). Although there has been no indigenously acquired vCJD in the United States, the sporadic occurrence of BSE in cattle in North America has heightened awareness of the possibility that such infections could occur and have led to increased surveillance activities. Updated information may be found on the following website: www.cdc.gov/ncidod/diseases/cjd/cjd.htm. The public health impact of prion diseases has been reviewed²³⁸.

vCJD in humans has different clinical and pathologic characteristics from sporadic or classic CJD²³⁹, including the following: 1) younger median age at death: 28 (range 16-48) vs. 68 years; 2) longer duration of illness: median 14 months vs. 4-6 months; 3) increased frequency of sensory symptoms and early psychiatric symptoms with delayed onset of frank neurologic signs; and 4) detection of prions in tonsillar and other lymphoid tissues from vCJD patients but not from sporadic CJD patients²⁴⁰. Similar to sporadic CJD, there have been no reported cases of direct human-to-human transmission of vCJD by casual or environmental contact, droplet, or airborne routes. Ongoing blood safety surveillance in the U.S. has not detected sporadic CJD transmission through

blood transfusion²⁴¹⁻²⁴³. However, bloodborne transmission of vCJD is believed to have occurred in two UK patients^{244, 245}. The following FDA websites provide information on steps that are being taken in the US to protect the blood supply from CJD and vCJD: <http://www.fda.gov/cber/gdlns/cjdvcjd.htm>;

<http://www.fda.gov/cber/gdlns/cjdvcjdq&a.htm>.

Standard Precautions are used when caring for patients with suspected or confirmed CJD or vCJD. However, special precautions are recommended for tissue handling in the histology laboratory and for conducting an autopsy, embalming, and for contact with a body that has undergone autopsy²⁴⁶. Recommendations for reprocessing surgical instruments to prevent transmission of CJD in healthcare settings have been published by the World Health Organization (WHO) and are currently under review at CDC.

Questions concerning notification of patients potentially exposed to CJD or vCJD through contaminated instruments and blood products from patients with CJD or vCJD or at risk of having vCJD may arise. The risk of transmission associated with such exposures is believed to be extremely low but may vary based on the specific circumstance. Therefore consultation on appropriate options is advised. The United Kingdom has developed several documents that clinicians and patients in the US may find useful (http://www.hpa.org.uk/infections/topics_az/cjd/information_documents.htm).

I.C.4. Severe Acute Respiratory Syndrome (SARS) SARS is a newly discovered respiratory disease that emerged in China late in 2002 and spread to several countries^{135, 140}; Mainland China, Hong Kong, Hanoi, Singapore, and Toronto were affected significantly. SARS is caused by SARS CoV, a previously unrecognized member of the coronavirus family^{247, 248}. The incubation period from exposure to the onset of symptoms is 2 to 7 days but can be as long as 10 days and uncommonly even longer²⁴⁹. The illness is initially difficult to distinguish from other common respiratory infections. Signs and symptoms usually include fever >38.0°C and chills and rigors, sometimes accompanied by headache, myalgia, and mild to severe respiratory symptoms. Radiographic finding of atypical pneumonia is an important clinical indicator of possible SARS. Compared with adults, children have been affected less frequently, have milder disease, and are less likely to transmit SARS-CoV^{135, 249-251}. The overall case fatality rate is approximately 6.0%; underlying disease and advanced age increase the risk of mortality (www.who.int/csr/sarsarchive/2003_05_07a/en/).

Outbreaks in healthcare settings, with transmission to large numbers of healthcare personnel and patients have been a striking feature of SARS; undiagnosed, infectious patients and visitors were important initiators of these outbreaks^{21, 252-254}. The relative contribution of potential modes of transmission is not precisely known. There is ample evidence for droplet and contact transmission^{96, 101, 113}; however, opportunistic airborne transmission cannot be excluded^{101, 135-139, 149, 255}. For example, exposure to aerosol-generating

procedures (e.g., endotracheal intubation, suctioning) was associated with transmission of infection to large numbers of healthcare personnel outside of the United States^{93, 94, 96, 98, 253}. Therefore, aerosolization of small infectious particles generated during these and other similar procedures could be a risk factor for transmission to others within a multi-bed room or shared airspace. A review of the infection control literature generated from the SARS outbreaks of 2003 concluded that the greatest risk of transmission is to those who have close contact, are not properly trained in use of protective infection control procedures, do not consistently use PPE; and that N95 or higher respirators may offer additional protection to those exposed to aerosol-generating procedures and high risk activities^{256, 257}. Organizational and individual factors that affected adherence to infection control practices for SARS also were identified²⁵⁷.

Control of SARS requires a coordinated, dynamic response by multiple disciplines in a healthcare setting⁹. Early detection of cases is accomplished by screening persons with symptoms of a respiratory infection for history of travel to areas experiencing community transmission or contact with SARS patients, followed by implementation of Respiratory Hygiene/Cough Etiquette (i.e., placing a mask over the patient's nose and mouth) and physical separation from other patients in common waiting areas. The precise combination of precautions to protect healthcare personnel has not been determined. At the time of this publication, CDC recommends Standard Precautions, with emphasis on the use of hand hygiene, Contact Precautions with emphasis on environmental cleaning due to the detection of SARS CoV RNA by PCR on surfaces in rooms occupied by SARS patients^{138, 254, 258}, Airborne Precautions, including use of fit-tested NIOSH-approved N95 or higher level respirators, and eye protection²⁵⁹. In Hong Kong, the use of Droplet and Contact Precautions, which included use of a mask but not a respirator, was effective in protecting healthcare personnel¹¹³. However, in Toronto, consistent use of an N95 respirator was slightly more protective than a mask⁹³. It is noteworthy that there was no transmission of SARS-CoV to public hospital workers in Vietnam despite inconsistent use of infection control measures, including use of PPE, which suggests other factors (e.g., severity of disease, frequency of high risk procedures or events, environmental features) may influence opportunities for transmission²⁶⁰.

SARS-CoV also has been transmitted in the laboratory setting through breaches in recommended laboratory practices. Research laboratories where SARS-CoV was under investigation were the source of most cases reported after the first series of outbreaks in the winter and spring of 2003^{261, 262}. Studies of the SARS outbreaks of 2003 and transmissions that occurred in the laboratory re-affirm the effectiveness of recommended infection control precautions and highlight the importance of consistent adherence to these measures.

Lessons from the SARS outbreaks are useful for planning to respond to future public health crises, such as pandemic influenza and bioterrorism events. Surveillance for cases among patients and healthcare personnel, ensuring availability of adequate supplies and staffing, and limiting access to healthcare

facilities were important factors in the response to SARS that have been summarized⁹. Guidance for infection control precautions in various settings is available at www.cdc.gov/ncidod/sars.

I.C.5. Monkeypox Monkeypox is a rare viral disease found mostly in the rain forest countries of Central and West Africa. The disease is caused by an orthopoxvirus that is similar in appearance to smallpox but causes a milder disease. The only recognized outbreak of human monkeypox in the United States was detected in June 2003 after several people became ill following contact with sick pet prairie dogs. Infection in the prairie dogs was subsequently traced to their contact with a shipment of animals from Africa, including giant Gambian rats²⁶³. This outbreak demonstrates the importance of recognition and prompt reporting of unusual disease presentations by clinicians to enable prompt identification of the etiology; and the potential of epizootic diseases to spread from animal reservoirs to humans through personal and occupational exposure²⁶⁴.

Limited data on transmission of monkeypox are available. Transmission from infected animals and humans is believed to occur primarily through direct contact with lesions and respiratory secretions; airborne transmission from animals to humans is unlikely but cannot be excluded, and may have occurred in veterinary practices (e.g., during administration of nebulized medications to ill prairie dogs²⁶⁵). Among humans, four instances of monkeypox transmission within hospitals have been reported in Africa among children, usually related to sharing the same ward or bed^{266, 267}. Additional recent literature documents transmission of Congo Basin monkeypox in a hospital compound for an extended number of generations²⁶⁸.

There has been no evidence of airborne or any other person-to-person transmission of monkeypox in the United States, and no new cases of monkeypox have been identified since the outbreak in June 2003²⁶⁹. The outbreak strain is a clade of monkeypox distinct from the Congo Basin clade and may have different epidemiologic properties (including human-to-human transmission potential) from monkeypox strains of the Congo Basin²⁷⁰; this awaits further study. Smallpox vaccine is 85% protective against Congo Basin monkeypox²⁷¹. Since there is an associated case fatality rate of $\leq 10\%$, administration of smallpox vaccine within 4 days to individuals who have had direct exposure to patients or animals with monkeypox is a reasonable consideration²⁷². For the most current information on monkeypox, see www.cdc.gov/ncidod/monkeypox/clinicians.htm.

I.C.6. Noroviruses Noroviruses, formerly referred to as Norwalk-like viruses, are members of the *Caliciviridae* family. These agents are transmitted via contaminated food or water and from person-to-person, causing explosive outbreaks of gastrointestinal disease²⁷³. Environmental contamination also has been documented as a contributing factor in ongoing transmission during outbreaks^{274, 275}. Although noroviruses cannot be propagated in cell culture,

DNA detection by molecular diagnostic techniques has facilitated a greater appreciation of their role in outbreaks of gastrointestinal disease²⁷⁶. Reported outbreaks in hospitals^{132, 142, 277}, nursing homes^{275, 278-283}, cruise ships^{284, 285}, hotels^{143, 147}, schools¹⁴⁸, and large crowded shelters established for hurricane evacuees²⁸⁶, demonstrate their highly contagious nature, the disruptive impact they have in healthcare facilities and the community, and the difficulty of controlling outbreaks in settings where people share common facilities and space. Of note, there is nearly a 5 fold increase in the risk to patients in outbreaks where a patient is the index case compared with exposure of patients during outbreaks where a staff member is the index case²⁸⁷.

The average incubation period for gastroenteritis caused by noroviruses is 12-48 hours and the clinical course lasts 12-60 hours²⁷³. Illness is characterized by acute onset of nausea, vomiting, abdominal cramps, and/or diarrhea. The disease is largely self-limited; rarely, death caused by severe dehydration can occur, particularly among the elderly with debilitating health conditions.

The epidemiology of norovirus outbreaks shows that even though primary cases may result from exposure to a fecally-contaminated food or water, secondary and tertiary cases often result from person-to-person transmission that is facilitated by contamination of fomites^{273, 288} and dissemination of infectious particles, especially during the process of vomiting^{132, 142, 143, 147, 148, 273, 279, 280}. Widespread, persistent and inapparent contamination of the environment and fomites can make outbreaks extremely difficult to control^{147, 275, 284}. These clinical observations and the detection of norovirus DNA on horizontal surfaces 5 feet above the level that might be touched normally suggest that, under certain circumstances, aerosolized particles may travel distances beyond 3 feet¹⁴⁷. It is hypothesized that infectious particles may be aerosolized from vomitus, inhaled, and swallowed. In addition, individuals who are responsible for cleaning the environment may be at increased risk of infection. Development of disease and transmission may be facilitated by the low infectious dose (i.e., <100 viral particles)²⁸⁹ and the resistance of these viruses to the usual cleaning and disinfection agents (i.e., may survive ≤ 10 ppm chlorine)²⁹⁰⁻²⁹². An alternate phenolic agent that was shown to be effective against feline calicivirus was used for environmental cleaning in one outbreak^{275, 293}. There are insufficient data to determine the efficacy of alcohol-based hand rubs against noroviruses when the hands are not visibly soiled²⁹⁴. Absence of disease in certain individuals during an outbreak may be explained by protection from infection conferred by the B histo-blood group antigen²⁹⁵. Consultation on outbreaks of gastroenteritis is available through CDC's Division of Viral and Rickettsial Diseases²⁹⁶.

I.C.7. Hemorrhagic fever viruses (HFV) The hemorrhagic fever viruses are a mixed group of viruses that cause serious disease with high fever, skin rash, bleeding diathesis, and in some cases, high mortality; the disease caused is referred to as viral hemorrhagic fever (VHF). Among the more commonly known HFVs are Ebola and Marburg viruses (Filoviridae), Lassa virus (Arenaviridae), Crimean-Congo hemorrhagic fever and Rift Valley Fever virus (Bunyaviridae),

and Dengue and Yellow fever viruses (Flaviviridae)^{212, 297}. These viruses are transmitted to humans via contact with infected animals or via arthropod vectors. While none of these viruses is endemic in the United States, outbreaks in affected countries provide potential opportunities for importation by infected humans and animals. Furthermore, there are concerns that some of these agents could be used as bioweapons²¹². Person-to-person transmission is documented for Ebola, Marburg, Lassa and Crimean-Congo hemorrhagic fever viruses. In resource-limited healthcare settings, transmission of these agents to healthcare personnel, patients and visitors has been described and in some outbreaks has accounted for a large proportion of cases²⁹⁸⁻³⁰⁰. Transmissions within households also have occurred among individuals who had direct contact with ill persons or their body fluids, but not to those who did not have such contact³⁰¹.

Evidence concerning the transmission of HFVs has been summarized^{212, 302}. Person-to-person transmission is associated primarily with direct blood and body fluid contact. Percutaneous exposure to contaminated blood carries a particularly high risk for transmission and increased mortality^{303, 304}. The finding of large numbers of Ebola viral particles in the skin and the lumina of sweat glands has raised concern that transmission could occur from direct contact with intact skin though epidemiologic evidence to support this is lacking³⁰⁵. Postmortem handling of infected bodies is an important risk for transmission^{301, 306, 307}. In rare situations, cases in which the mode of transmission was unexplained among individuals with no known direct contact, have led to speculation that airborne transmission could have occurred²⁹⁸. However, airborne transmission of naturally occurring HFVs in humans has not been seen. In one study of airplane passengers exposed to an in-flight index case of Lassa fever, there was no transmission to any passengers³⁰⁸.

In the laboratory setting, animals have been infected experimentally with Marburg or Ebola viruses via direct inoculation of the nose, mouth and/or conjunctiva^{309, 310} and by using mechanically generated virus-containing aerosols^{311, 312}. Transmission of Ebola virus among laboratory primates in an animal facility has been described³¹³. Secondarily infected animals were in individual cages and separated by approximately 3 meters. Although the possibility of airborne transmission was suggested, the authors were not able to exclude droplet or indirect contact transmission in this incidental observation.

Guidance on infection control precautions for HFVs that are transmitted person-to-person have been published by CDC^{1, 211} and by the Johns Hopkins Center for Civilian Biodefense Strategies²¹². The most recent recommendations at the time of publication of this document were posted on the CDC website on 5/19/05³¹⁴. Inconsistencies among the various recommendations have raised questions about the appropriate precautions to use in U.S. hospitals. In less developed countries, outbreaks of HFVs have been controlled with basic hygiene, barrier precautions, safe injection practices, and safe burial practices^{299, 306}. The preponderance of evidence on HFV transmission indicates that Standard, Contact and Droplet Precautions with eye protection are effective in protecting

healthcare personnel and visitors who may attend an infected patient. Single gloves are adequate for routine patient care; double-gloving is advised during invasive procedures (e.g., surgery) that pose an increased risk for blood exposure. Routine eye protection (i.e. goggles or face shield) is particularly important. Fluid-resistant gowns should be worn for all patient contact. Airborne Precautions are not required for routine patient care; however, use of AIIRs is prudent when procedures that could generate infectious aerosols are performed (e.g., endotracheal intubation, bronchoscopy, suctioning, autopsy procedures involving oscillating saws). N95 or higher level respirators may provide added protection for individuals in a room during aerosol-generating procedures (Table 3, Appendix A). When a patient with a syndrome consistent with hemorrhagic fever also has a history of travel to an endemic area, precautions are initiated upon presentation and then modified as more information is obtained (Table 2). Patients with hemorrhagic fever syndrome in the setting of a suspected bioweapon attack should be managed using Airborne Precautions, including AIIRs, since the epidemiology of a potentially weaponized hemorrhagic fever virus is unpredictable.

I.D. Transmission risks associated with specific types of healthcare settings

Numerous factors influence differences in transmission risks among the various healthcare settings. These include the population characteristics (e.g., increased susceptibility to infections, type and prevalence of indwelling devices), intensity of care, exposure to environmental sources, length of stay, and frequency of interaction between patients/residents with each other and with HCWs. These factors, as well as organizational priorities, goals, and resources, influence how different healthcare settings adapt transmission prevention guidelines to meet their specific needs^{315, 316}. Infection control management decisions are informed by data regarding institutional experience/epidemiology, trends in community and institutional HAIs, local, regional, and national epidemiology, and emerging infectious disease threats.

I.D.1. Hospitals Infection transmission risks are present in all hospital settings. However, certain hospital settings and patient populations have unique conditions that predispose patients to infection and merit special mention. These are often sentinel sites for the emergence of new transmission risks that may be unique to that setting or present opportunities for transmission to other settings in the hospital.

I.D.1.a. Intensive Care Units Intensive care units (ICUs) serve patients who are immunocompromised by disease state and/or by treatment modalities, as well as patients with major trauma, respiratory failure and other life-threatening conditions (e.g., myocardial infarction, congestive heart failure, overdoses, strokes, gastrointestinal bleeding, renal failure, hepatic failure, multi-organ system failure, and the extremes of age). Although ICUs account for a relatively

small proportion of hospitalized patients, infections acquired in these units accounted for >20% of all HAIs³¹⁷. In the National Nosocomial Infection Surveillance (NNIS) system, 26.6% of HAIs were reported from ICU and high risk nursery (NICU) patients in 2002 (NNIS, unpublished data). This patient population has increased susceptibility to colonization and infection, especially with MDROs and *Candida* sp.^{318, 319}, because of underlying diseases and conditions, the invasive medical devices and technology used in their care (e.g. central venous catheters and other intravascular devices, mechanical ventilators, extracorporeal membrane oxygenation (ECMO), hemodialysis/-filtration, pacemakers, implantable left ventricular assist devices), the frequency of contact with healthcare personnel, prolonged length of stay, and prolonged exposure to antimicrobial agents³²⁰⁻³³¹. Furthermore, adverse patient outcomes in this setting are more severe and are associated with a higher mortality³³². Outbreaks associated with a variety of bacterial, fungal and viral pathogens due to common-source and person-to-person transmissions are frequent in adult and pediatric ICUs^{31, 333-336, 337, 338}.

I.D.1.b. Burn Units Burn wounds can provide optimal conditions for colonization, infection, and transmission of pathogens; infection acquired by burn patients is a frequent cause of morbidity and mortality^{320, 339, 340}. In patients with a burn injury involving $\geq 30\%$ of the total body surface area (TBSA), the risk of invasive burn wound infection is particularly high^{341, 342}. Infections that occur in patients with burn injury involving <30% TBSA are usually associated with the use of invasive devices. Methicillin-susceptible *Staphylococcus aureus*, MRSA, enterococci, including VRE, gram-negative bacteria, and candida are prevalent pathogens in burn infections^{53, 340, 343-350} and outbreaks of these organisms have been reported³⁵¹⁻³⁵⁴. Shifts over time in the predominance of pathogens causing infections among burn patients often lead to changes in burn care practices^{343, 355-358}. Burn wound infections caused by *Aspergillus* sp. or other environmental molds may result from exposure to supplies contaminated during construction³⁵⁹ or to dust generated during construction or other environmental disruption³⁶⁰.

Hydrotherapy equipment is an important environmental reservoir of gram-negative organisms. Its use for burn care is discouraged based on demonstrated associations between use of contaminated hydrotherapy equipment and infections. Burn wound infections and colonization, as well as bloodstream infections, caused by multidrug-resistant *P. aeruginosa*³⁶¹, *A. baumannii*³⁶², and MRSA³⁵² have been associated with hydrotherapy; excision of burn wounds in operating rooms is preferred.

Advances in burn care, specifically early excision and grafting of the burn wound, use of topical antimicrobial agents, and institution of early enteral feeding, have led to decreased infectious complications. Other advances have included prophylactic antimicrobial usage, selective digestive decontamination (SDD), and use of antimicrobial-coated catheters (ACC), but few epidemiologic studies and no efficacy studies have been performed to show the relative benefit of these measures³⁵⁷.

There is no consensus on the most effective infection control practices to prevent transmission of infections to and from patients with serious burns (e.g., single-bed rooms³⁵⁸, laminar flow³⁶³ and high efficiency particulate air filtration [HEPA]³⁶⁰ or maintaining burn patients in a separate unit without exposure to patients or equipment from other units³⁶⁴). There also is controversy regarding the need for and type of barrier precautions for routine care of burn patients. One retrospective study demonstrated efficacy and cost effectiveness of a simplified barrier isolation protocol for wound colonization, emphasizing handwashing and use of gloves, caps, masks and plastic impermeable aprons (rather than isolation gowns) for direct patient contact³⁶⁵. However, there have been no studies that define the most effective combination of infection control precautions for use in burn settings. Prospective studies in this area are needed.

I.D.1.c. Pediatrics Studies of the epidemiology of HAIs in children have identified unique infection control issues in this population^{63, 64, 366-370}. Pediatric intensive care unit (PICU) patients and the lowest birthweight babies in the high-risk nursery (HRN) monitored in the NNIS system have had high rates of central venous catheter-associated bloodstream infections^{64, 320, 369-372}. Additionally, there is a high prevalence of community-acquired infections among hospitalized infants and young children who have not yet become immune either by vaccination or by natural infection. The result is more patients and their sibling visitors with transmissible infections present in pediatric healthcare settings, especially during seasonal epidemics (e.g., pertussis^{36, 40, 41}, respiratory viral infections including those caused by RSV²⁴, influenza viruses³⁷³, parainfluenza virus³⁷⁴, human metapneumovirus³⁷⁵, and adenoviruses³⁷⁶, rubeola [measles]³⁴, varicella [chickenpox]³⁷⁷, and rotavirus^{38, 378}).

Close physical contact between healthcare personnel and infants and young children (eg. cuddling, feeding, playing, changing soiled diapers, and cleaning copious uncontrolled respiratory secretions) provides abundant opportunities for transmission of infectious material. Practices and behaviors such as congregation of children in play areas where toys and bodily secretions are easily shared and family members rooming-in with pediatric patients can further increase the risk of transmission. Pathogenic bacteria have been recovered from toys used by hospitalized patients³⁷⁹; contaminated bath toys were implicated in an outbreak of multidrug-resistant *P. aeruginosa* on a pediatric oncology unit⁸⁰. In addition, several patient factors increase the likelihood that infection will result from exposure to pathogens in healthcare settings (e.g., immaturity of the neonatal immune system, lack of previous natural infection and resulting immunity, prevalence of patients with congenital or acquired immune deficiencies, congenital anatomic anomalies, and use of life-saving invasive devices in neonatal and pediatric intensive care units)⁶³. There are theoretical concerns that infection risk will increase in association with innovative practices used in the NICU for the purpose of improving developmental outcomes. Such factors include co-bedding³⁸⁰ and kangaroo care³⁸¹ that may increase opportunity for skin-to-skin exposure of multiple gestation infants to each other and to their mothers, respectively; although infection risk may actually be

reduced among infants receiving kangaroo care³⁸². Children who attend child care centers^{383, 384} and pediatric rehabilitation units³⁸⁵ may increase the overall burden of antimicrobial resistance (eg. by contributing to the reservoir of community-associated MRSA [CA-MRSA])³⁸⁶⁻³⁹¹. Patients in chronic care facilities may have increased rates of colonization with resistant GNBs and may be sources of introduction of resistant organisms to acute care settings⁵⁰.

I.D.2. Nonacute healthcare settings Healthcare is provided in various settings outside of hospitals including facilities, such as long-term care facilities (LTCF) (e.g. nursing homes), homes for the developmentally disabled, settings where behavioral health services are provided, rehabilitation centers and hospices³⁹². In addition, healthcare may be provided in nonhealthcare settings such as workplaces with occupational health clinics, adult day care centers, assisted living facilities, homeless shelters, jails and prisons, school clinics and infirmaries. Each of these settings has unique circumstances and population risks to consider when designing and implementing an infection control program. Several of the most common settings and their particular challenges are discussed below. While this Guideline does not address each setting, the principles and strategies provided may be adapted and applied as appropriate.

I.D.2.a. Long-term care The designation LTCF applies to a diverse group of residential settings, ranging from institutions for the developmentally disabled to nursing homes for the elderly and pediatric chronic-care facilities³⁹³⁻³⁹⁵. Nursing homes for the elderly predominate numerically and frequently represent long-term care as a group of facilities. Approximately 1.8 million Americans reside in the nation's 16,500 nursing homes³⁹⁶. Estimates of HAI rates of 1.8 to 13.5 per 1000 resident-care days have been reported with a range of 3 to 7 per 1000 resident-care days in the more rigorous studies³⁹⁷⁻⁴⁰¹. The infrastructure described in the Department of Veterans Affairs nursing home care units is a promising example for the development of a nationwide HAI surveillance system for LTCFs⁴⁰².

LTCFs are different from other healthcare settings in that elderly patients at increased risk for infection are brought together in one setting and remain in the facility for extended periods of time; for most residents, it is their home. An atmosphere of community is fostered and residents share common eating and living areas, and participate in various facility-sponsored activities^{403, 404}. Since able residents interact freely with each other, controlling transmission of infection in this setting is challenging⁴⁰⁵. Residents who are colonized or infected with certain microorganisms are, in some cases, restricted to their room. However, because of the psychosocial risks associated with such restriction, it has been recommended that psychosocial needs be balanced with infection control needs in the LTCF setting⁴⁰⁶⁻⁴⁰⁹. Documented LTCF outbreaks have been caused by various viruses (e.g., influenza virus^{35, 410-412}, rhinovirus⁴¹³, adenovirus (conjunctivitis)⁴¹⁴, norovirus^{278, 279 275, 281}) and bacteria, including group A streptococcus¹⁶², *B. pertussis*⁴¹⁵, non-susceptible *S. pneumoniae*^{197, 198}, other MDROs, and *Clostridium difficile*⁴¹⁶) These pathogens can lead to substantial

morbidity and mortality, and increased medical costs; prompt detection and implementation of effective control measures are required.

Risk factors for infection are prevalent among LTCF residents^{395, 417, 418}. Age-related declines in immunity may affect responses to immunizations for influenza and other infectious agents, and increase susceptibility to tuberculosis. Immobility, incontinence, dysphagia, underlying chronic diseases, poor functional status, and age-related skin changes increase susceptibility to urinary, respiratory and cutaneous and soft tissue infections, while malnutrition can impair wound healing⁴¹⁹⁻⁴²³. Medications (e.g., drugs that affect level of consciousness, immune function, gastric acid secretions, and normal flora, including antimicrobial therapy) and invasive devices (e.g., urinary catheters and feeding tubes) heighten susceptibility to infection and colonization in LTCF residents⁴²⁴⁻⁴²⁶. Finally, limited functional status and total dependence on healthcare personnel for activities of daily living have been identified as independent risk factors for infection^{401, 417, 427} and for colonization with MRSA^{428, 429} and ESBL-producing *K. pneumoniae*⁴³⁰. Several position papers and review articles have been published that provide guidance on various aspects of infection control and antimicrobial resistance in LTCFs^{406-408, 431-436}. The Centers for Medicare and Medicaid Services (CMS) have established regulations for the prevention of infection in LTCFs⁴³⁷.

Because residents of LTCFs are hospitalized frequently, they can transfer pathogens between LTCFs and healthcare facilities in which they receive care^{8, 438-441}. This is also true for pediatric long-term care populations. Pediatric chronic care facilities have been associated with importing extended-spectrum cephalosporin-resistant, gram-negative bacilli into one PICU⁵⁰. Children from pediatric rehabilitation units may contribute to the reservoir of community-associated MRSA^{385, 389-391}.

I.D.2.b. Ambulatory Care In the past decade, healthcare delivery in the United States has shifted from the acute, inpatient hospital to a variety of ambulatory and community-based settings, including the home. Ambulatory care is provided in hospital-based outpatient clinics, nonhospital-based clinics and physician offices, public health clinics, free-standing dialysis centers, ambulatory surgical centers, urgent care centers, and many others. In 2000, there were 83 million visits to hospital outpatient clinics and more than 823 million visits to physician offices⁴⁴²; ambulatory care now accounts for most patient encounters with the health care system⁴⁴³. In these settings, adapting transmission prevention guidelines is challenging because patients remain in common areas for prolonged periods waiting to be seen by a healthcare provider or awaiting admission to the hospital, examination or treatment rooms are turned around quickly with limited cleaning, and infectious patients may not be recognized immediately. Furthermore, immunocompromised patients often receive chemotherapy in infusion rooms where they stay for extended periods of time along with other types of patients.

There are few data on the risk of HAIs in ambulatory care settings, with the exception of hemodialysis centers^{18, 444, 445}. Transmission of infections in outpatient settings has been reviewed in three publications⁴⁴⁶⁻⁴⁴⁸. Goodman and Solomon summarized 53 clusters of infections associated with the outpatient setting from 1961-1990⁴⁴⁶. Overall, 29 clusters were associated with common source transmission from contaminated solutions or equipment, 14 with person-to-person transmission from or involving healthcare personnel and ten associated with airborne or droplet transmission among patients and healthcare workers. Transmission of bloodborne pathogens (i.e., hepatitis B and C viruses and, rarely, HIV) in outbreaks, sometimes involving hundreds of patients, continues to occur in ambulatory settings. These outbreaks often are related to common source exposures, usually a contaminated medical device, multi-dose vial, or intravenous solution^{82, 449-453}. In all cases, transmission has been attributed to failure to adhere to fundamental infection control principles, including safe injection practices and aseptic technique. This subject has been reviewed and recommended infection control and safe injection practices summarized⁴⁵⁴.

Airborne transmission of *M. tuberculosis* and measles in ambulatory settings, most frequently emergency departments, has been reported^{34, 127, 446, 448, 455-457}. Measles virus was transmitted in physician offices and other outpatient settings during an era when immunization rates were low and measles outbreaks in the community were occurring regularly^{34, 122, 458}. Rubella has been transmitted in the outpatient obstetric setting³³; there are no published reports of varicella transmission in the outpatient setting. In the ophthalmology setting, adenovirus type 8 epidemic keratoconjunctivitis has been transmitted via incompletely disinfected ophthalmology equipment and/or from healthcare workers to patients, presumably by contaminated hands^{17, 446, 448, 459-462}.

If transmission in outpatient settings is to be prevented, screening for potentially infectious symptomatic and asymptomatic individuals, especially those who may be at risk for transmitting airborne infectious agents (e.g., *M. tuberculosis*, varicella-zoster virus, rubeola [measles]), is necessary at the start of the initial patient encounter. Upon identification of a potentially infectious patient, implementation of prevention measures, including prompt separation of potentially infectious patients and implementation of appropriate control measures (e.g., Respiratory Hygiene/Cough Etiquette and Transmission-Based Precautions) can decrease transmission risks^{9, 12}. Transmission of MRSA and VRE in outpatient settings has not been reported, but the association of CA-MRSA in healthcare personnel working in an outpatient HIV clinic with environmental CA-MRSA contamination in that clinic, suggests the possibility of transmission in that setting⁴⁶³. Patient-to-patient transmission of *Burkholderia species* and *Pseudomonas aeruginosa* in outpatient clinics for adults and children with cystic fibrosis has been confirmed^{464, 465}.

I.D.2.c. Home Care Home care in the United States is delivered by over 20,000 provider agencies that include home health agencies, hospices, durable medical equipment providers, home infusion therapy services, and personal care and

support services providers. Home care is provided to patients of all ages with both acute and chronic conditions. The scope of services ranges from assistance with activities of daily living and physical and occupational therapy to the care of wounds, infusion therapy, and chronic ambulatory peritoneal dialysis (CAPD).

The incidence of infection in home care patients, other than those associated with infusion therapy is not well studied⁴⁶⁶⁻⁴⁷¹. However, data collection and calculation of infection rates have been accomplished for central venous catheter-associated bloodstream infections in patients receiving home infusion therapy⁴⁷⁰⁻⁴⁷⁴ and for the risk of blood contact through percutaneous or mucosal exposures, demonstrating that surveillance can be performed in this setting⁴⁷⁵. Draft definitions for home care associated infections have been developed⁴⁷⁶.

Transmission risks during home care are presumed to be minimal. The main transmission risks to home care patients are from an infectious healthcare provider or contaminated equipment; providers also can be exposed to an infectious patient during home visits. Since home care involves patient care by a limited number of personnel in settings without multiple patients or shared equipment, the potential reservoir of pathogens is reduced. Infections of home care providers, that could pose a risk to home care patients include infections transmitted by the airborne or droplet routes (e.g., chickenpox, tuberculosis, influenza), and skin infestations (e.g., scabies⁶⁹ and lice) and infections (e.g., impetigo) transmitted by direct or indirect contact. There are no published data on indirect transmission of MDROs from one home care patient to another, although this is theoretically possible if contaminated equipment is transported from an infected or colonized patient and used on another patient. Of note, investigation of the first case of VISA in homecare¹⁸⁶ and the first 2 reported cases of VRSA^{178, 180, 181, 183} found no evidence of transmission of VISA or VRSA to other home care recipients. Home health care also may contribute to antimicrobial resistance; a review of outpatient vancomycin use found 39% of recipients did not receive the antibiotic according to recommended guidelines⁴⁷⁷.

Although most home care agencies implement policies and procedures to prevent transmission of organisms, the current approach is based on the adaptation of the *1996 Guideline for Isolation Precautions in Hospitals*¹ as well as other professional guidance^{478, 479}. This issue has been very challenging in the home care industry and practice has been inconsistent and frequently not evidence-based. For example, many home health agencies continue to observe “nursing bag technique,” a practice that prescribes the use of barriers between the nursing bag and environmental surfaces in the home⁴⁸⁰. While the home environment may not always appear clean, the use of barriers between two non-critical surfaces has been questioned^{481, 482}. Opportunities exist to conduct research in home care related to infection transmission risks⁴⁸³.

I.D.2.d. Other sites of healthcare delivery Facilities that are not primarily healthcare settings but in which healthcare is delivered include clinics in correctional facilities and shelters. Both settings can have suboptimal features,

such as crowded conditions and poor ventilation. Economically disadvantaged individuals who may have chronic illnesses and healthcare problems related to alcoholism, injection drug use, poor nutrition, and/or inadequate shelter often receive their primary healthcare at sites such as these⁴⁸⁴. Infectious diseases of special concern for transmission include tuberculosis, scabies, respiratory infections (e.g., *N. meningitides*, *S. pneumoniae*), sexually transmitted and bloodborne diseases (e.g., HIV, HBV, HCV, syphilis, gonorrhea), hepatitis A virus (HAV), diarrheal agents such as norovirus, and foodborne diseases^{286, 485-488}. A high index of suspicion for tuberculosis and CA-MRSA in these populations is needed as outbreaks in these settings or among the populations they serve have been reported⁴⁸⁹⁻⁴⁹⁷.

Patient encounters in these types of facilities provide an opportunity to deliver recommended immunizations and screen for *M. tuberculosis* infection in addition to diagnosing and treating acute illnesses⁴⁹⁸. Recommended infection control measures in these non-traditional areas designated for healthcare delivery are the same as for other ambulatory care settings. Therefore, these settings must be equipped to observe Standard Precautions and, when indicated, Transmission-based Precautions.

I.E. Transmission risks associated with special patient populations

As new treatments emerge for complex diseases, unique infection control challenges associated with special patient populations need to be addressed.

I.E.1. Immunocompromised patients Patients who have congenital primary immune deficiencies or acquired disease (eg. treatment-induced immune deficiencies) are at increased risk for numerous types of infections while receiving healthcare and may be located throughout the healthcare facility. The specific defects of the immune system determine the types of infections that are most likely to be acquired (e.g., viral infections are associated with T-cell defects and fungal and bacterial infections occur in patients who are neutropenic). As a general group, immunocompromised patients can be cared for in the same environment as other patients; however, it is always advisable to minimize exposure to other patients with transmissible infections such as influenza and other respiratory viruses^{499, 500}. The use of more intense chemotherapy regimens for treatment of childhood leukemia may be associated with prolonged periods of neutropenia and suppression of other components of the immune system, extending the period of infection risk and raising the concern that additional precautions may be indicated for select groups^{501, 502}. With the application of newer and more intense immunosuppressive therapies for a variety of medical conditions (e.g., rheumatologic disease^{503, 504}, inflammatory bowel disease⁵⁰⁵), immunosuppressed patients are likely to be more widely distributed throughout a healthcare facility rather than localized to single patient units (e.g.

hematology-oncology). Guidelines for preventing infections in certain groups of immunocompromised patients have been published ^{15, 506, 507}.

Published data provide evidence to support placing allogeneic HSCT patients in a Protective Environment ^{15, 157, 158}. Also, three guidelines have been developed that address the special requirements of these immunocompromised patients, including use of antimicrobial prophylaxis and engineering controls to create a Protective Environment for the prevention of infections caused by *Aspergillus* spp. and other environmental fungi ^{11, 14, 15}. As more intense chemotherapy regimens associated with prolonged periods of neutropenia or graft-versus-host disease are implemented, the period of risk and duration of environmental protection may need to be prolonged beyond the traditional 100 days ⁵⁰⁸.

I.E.2. Cystic fibrosis patients Patients with cystic fibrosis (CF) require special consideration when developing infection control guidelines. Compared to other patients, CF patients require additional protection to prevent transmission from contaminated respiratory therapy equipment ⁵⁰⁹⁻⁵¹³. Infectious agents such as *Burkholderia cepacia* complex and *P. aeruginosa* ^{464, 465, 514, 515} have unique clinical and prognostic significance. In CF patients, *B. cepacia* infection has been associated with increased morbidity and mortality ⁵¹⁶⁻⁵¹⁸, while delayed acquisition of chronic *P. aeruginosa* infection may be associated with an improved long-term clinical outcome ^{519, 520}.

Person-to-person transmission of *B. cepacia* complex has been demonstrated among children ⁵¹⁷ and adults ⁵²¹ with CF in healthcare settings ^{464, 522}, during various social contacts ⁵²³, most notably attendance at camps for patients with CF ⁵²⁴, and among siblings with CF ⁵²⁵. Successful infection control measures used to prevent transmission of respiratory secretions include segregation of CF patients from each other in ambulatory and hospital settings (including use of private rooms with separate showers), environmental decontamination of surfaces and equipment contaminated with respiratory secretions, elimination of group chest physiotherapy sessions, and disbanding of CF camps ^{97, 526}. The Cystic Fibrosis Foundation published a consensus document with evidence-based recommendations for infection control practices for CF patients ²⁰.

I.F. New therapies associated with potentially transmissible infectious agents

I.F.1. Gene therapy Gene therapy has been attempted using a number of different viral vectors, including nonreplicating retroviruses, adenoviruses, adeno-associated viruses, and replication-competent strains of poxviruses. Unexpected adverse events have restricted the prevalence of gene therapy protocols.

The infectious hazards of gene therapy are theoretical at this time, but require meticulous surveillance due to the possible occurrence of in vivo recombination

and the subsequent emergence of a transmissible genetically altered pathogen. Greatest concern attends the use of replication-competent viruses, especially vaccinia. As of the time of publication, no reports have described transmission of a vector virus from a gene therapy recipient to another individual, but surveillance is ongoing. Recommendations for monitoring infection control issues throughout the course of gene therapy trials have been published⁵²⁷⁻⁵²⁹.

I.F.2. Infections transmitted through blood, organs and other tissues The potential hazard of transmitting infectious pathogens through biologic products is a small but ever present risk, despite donor screening. Reported infections transmitted by transfusion or transplantation include West Nile Virus infection⁵³⁰, cytomegalovirus infection⁵³¹, Creutzfeldt-Jacob disease²³⁰, hepatitis C⁵³², infections with *Clostridium* spp.⁵³³ and group A streptococcus⁵³⁴, malaria⁵³⁵, babesiosis⁵³⁶, Chagas disease⁵³⁷, lymphocytic choriomeningitis⁵³⁸, and rabies^{539, 540}. Therefore, it is important to consider receipt of biologic products when evaluating patients for potential sources of infection.

I.F.3. Xenotransplantation The transplantation of nonhuman cells, tissues, and organs into humans potentially exposes patients to zoonotic pathogens. Transmission of known zoonotic infections (e.g., trichinosis from porcine tissue), constitutes one concern, but also of concern is the possibility that transplantation of nonhuman cells, tissues, or organs may transmit previously unknown zoonotic infections (xenozoonoses) to immunosuppressed human recipients. Potential infections that might accompany transplantation of porcine organs have been described⁵⁴¹. Guidelines from the U.S. Public Health Service address many infectious diseases and infection control issues that surround the developing field of xenotransplantation⁵⁴²); work in this area is ongoing.

Part II:

Fundamental elements needed to prevent transmission of infectious agents in healthcare settings

II.A. Healthcare system components that influence the effectiveness of precautions to prevent transmission

II.A.1. Administrative measures Healthcare organizations can demonstrate a commitment to preventing transmission of infectious agents by incorporating infection control into the objectives of the organization's patient and occupational safety programs⁵⁴³⁻⁵⁴⁷. An infrastructure to guide, support, and monitor adherence to Standard and Transmission-Based Precautions^{434, 548, 549} will facilitate fulfillment of the organization's mission and achievement of the Joint Commission on Accreditation of Healthcare Organization's patient safety goal to decrease HAIs⁵⁵⁰. Policies and procedures that explain how Standard and Transmission-Based Precautions are applied, including systems used to identify and communicate information about patients with potentially transmissible infectious agents, are essential to ensure the success of these measures and may vary according to the characteristics of the organization.

A key administrative measure is provision of fiscal and human resources for maintaining infection control and occupational health programs that are responsive to emerging needs. Specific components include bedside nurse⁵⁵¹ and infection prevention and control professional (ICP) staffing levels⁵⁵², inclusion of ICPs in facility construction and design decisions¹¹, clinical microbiology laboratory support^{553, 554}, adequate supplies and equipment including facility ventilation systems¹¹, adherence monitoring⁵⁵⁵, assessment and correction of system failures that contribute to transmission^{556, 557}, and provision of feedback to healthcare personnel and senior administrators^{434, 548, 549, 558}. The positive influence of institutional leadership has been demonstrated repeatedly in studies of HCW adherence to recommended hand hygiene practices^{176, 177, 434, 548, 549, 559-564}. Healthcare administrator involvement in infection control processes can improve administrators' awareness of the rationale and resource requirements for following recommended infection control practices.

Several administrative factors may affect the transmission of infectious agents in healthcare settings: institutional culture, individual worker behavior, and the work environment. Each of these areas is suitable for performance improvement monitoring and incorporation into the organization's patient safety goals^{543, 544, 546, 565}.

II.A.1.a.Scope of work and staffing needs for infection control professionals

The effectiveness of infection surveillance and control programs in preventing nosocomial infections in United States hospitals was assessed by the CDC through the Study on the Efficacy of Nosocomial Infection Control (SENIC Project) conducted 1970-76⁵⁶⁶. In a representative sample of US general hospitals, those with a trained infection control physician or microbiologist involved in an infection control program, and at least one infection control nurse per 250 beds, were associated with a 32% lower rate of four infections studied (CVC-associated bloodstream infections, ventilator-associated pneumonias, catheter-related urinary tract infections, and surgical site infections).

Since that landmark study was published, responsibilities of ICPs have expanded commensurate with the growing complexity of the healthcare system, the patient populations served, and the increasing numbers of medical procedures and devices used in all types of healthcare settings. The scope of work of ICPs was first assessed in 1982⁵⁶⁷⁻⁵⁶⁹ by the Certification Board of Infection Control (CBIC), and has been re-assessed every five years since that time^{558, 570-572}. The findings of these task analyses have been used to develop and update the Infection Control Certification Examination, offered for the first time in 1983. With each survey, it is apparent that the role of the ICP is growing in complexity and scope, beyond traditional infection control activities in acute care hospitals. Activities currently assigned to ICPs in response to emerging challenges include: 1) surveillance and infection prevention at facilities other than acute care hospitals e.g., ambulatory clinics, day surgery centers, long term care facilities, rehabilitation centers, home care; 2) oversight of employee health services related to infection prevention, e.g. assessment of risk and administration of recommended treatment following exposure to infectious agents, tuberculosis screening, influenza vaccination, respiratory protection fit testing, and administration of other vaccines as indicated, such as smallpox vaccine in 2003; 3) preparedness planning for annual influenza outbreaks, pandemic influenza, SARS, bioweapons attacks; 4) adherence monitoring for selected infection control practices; 5) oversight of risk assessment and implementation of prevention measures associated with construction and renovation; 6) prevention of transmission of MDROs; 7) evaluation of new medical products that could be associated with increased infection risk. e.g., intravenous infusion materials; 9) communication with the public, facility staff, and state and local health departments concerning infection control-related issues; and 10) participation in local and multi-center research projects^{434, 549, 552, 558, 573, 574}.

None of the CBIC job analyses addressed specific staffing requirements for the identified tasks, although the surveys did include information about hours worked; the 2001 survey included the number of ICPs assigned to the responding facilities⁵⁵⁸. There is agreement in the literature that 1 ICP per 250 acute care beds is no longer adequate to meet current infection control needs; a Delphi project that assessed staffing needs of infection control programs in the 21st century concluded that a ratio of 0.8 to 1.0 ICP per 100 occupied acute care beds is an appropriate level of staffing⁵⁵². A survey of participants in the National

Nosocomial Infections Surveillance (NNIS) system found the average daily census per ICP was 115³¹⁶. Results of other studies have been similar: 3 per 500 beds for large acute care hospitals, 1 per 150-250 beds in long term care facilities, and 1.56 per 250 in small rural hospitals^{573, 575}. The foregoing demonstrates that infection control staffing can no longer be based on patient census alone, but rather must be determined by the scope of the program, characteristics of the patient population, complexity of the healthcare system, tools available to assist personnel to perform essential tasks (e.g., electronic tracking and laboratory support for surveillance), and unique or urgent needs of the institution and community⁵⁵². Furthermore, appropriate training is required to optimize the quality of work performed^{558, 572, 576}.

II.A.1.a.i. Infection Control Nurse Liaison Designating a bedside nurse on a patient care unit as an infection control liaison or “link nurse” is reported to be an effective adjunct to enhance infection control at the unit level⁵⁷⁷⁻⁵⁸². Such individuals receive training in basic infection control and have frequent communication with the ICPs, but maintain their primary role as bedside caregiver on their units. The infection control nurse liaison increases the awareness of infection control at the unit level. He or she is especially effective in implementation of new policies or control interventions because of the rapport with individuals on the unit, an understanding of unit-specific challenges, and ability to promote strategies that are most likely to be successful in that unit. This position is an adjunct to, not a replacement for, fully trained ICPs. Furthermore, the infection control liaison nurses should not be counted when considering ICP staffing.

II.A.1.b. Bedside nurse staffing There is increasing evidence that the level of bedside nurse-staffing influences the quality of patient care^{583, 584}. If there are adequate nursing staff, it is more likely that infection control practices, including hand hygiene and Standard and Transmission-Based Precautions, will be given appropriate attention and applied correctly and consistently⁵⁵². A national multicenter study reported strong and consistent inverse relationships between nurse staffing and five adverse outcomes in medical patients, two of which were HAIs: urinary tract infections and pneumonia⁵⁸³. The association of nursing staff shortages with increased rates of HAIs has been demonstrated in several outbreaks in hospitals and long term care settings, and with increased transmission of hepatitis C virus in dialysis units^{22, 418, 551, 585-597}. In most cases, when staffing improved as part of a comprehensive control intervention, the outbreak ended or the HAI rate declined. In two studies^{590, 596}, the composition of the nursing staff (“pool” or “float” vs. regular staff nurses) influenced the rate of primary bloodstream infections, with an increased infection rate occurring when the proportion of regular nurses decreased and pool nurses increased.

II.A.1.c. Clinical microbiology laboratory support The critical role of the clinical microbiology laboratory in infection control and healthcare epidemiology is described well^{553, 554, 598-600} and is supported by the Infectious Disease Society

of America policy statement on consolidation of clinical microbiology laboratories published in 2001⁵⁵³. The clinical microbiology laboratory contributes to preventing transmission of infectious diseases in healthcare settings by promptly detecting and reporting epidemiologically important organisms, identifying emerging patterns of antimicrobial resistance, and assisting in assessment of the effectiveness of recommended precautions to limit transmission during outbreaks⁵⁹⁸. Outbreaks of infections may be recognized first by laboratorians¹⁶². Healthcare organizations need to ensure the availability of the recommended scope and quality of laboratory services, a sufficient number of appropriately trained laboratory staff members, and systems to promptly communicate epidemiologically important results to those who will take action (e.g., providers of clinical care, infection control staff, healthcare epidemiologists, and infectious disease consultants)⁶⁰¹. As concerns about emerging pathogens and bioterrorism grow, the role of the clinical microbiology laboratory takes on even greater importance. For healthcare organizations that outsource microbiology laboratory services (e.g., ambulatory care, home care, LTCFs, smaller acute care hospitals), it is important to specify by contract the types of services (e.g., periodic institution-specific aggregate susceptibility reports) required to support infection control.

Several key functions of the clinical microbiology laboratory are relevant to this guideline:

- Antimicrobial susceptibility by testing and interpretation in accordance with current guidelines developed by the National Committee for Clinical Laboratory Standards (NCCLS), known as the Clinical and Laboratory Standards Institute (CLSI) since 2005⁶⁰², for the detection of emerging resistance patterns^{603, 604}, and for the preparation, analysis, and distribution of periodic cumulative antimicrobial susceptibility summary reports⁶⁰⁵⁻⁶⁰⁷. While not required, clinical laboratories ideally should have access to rapid genotypic identification of bacteria and their antibiotic resistance genes⁶⁰⁸.
- Performance of surveillance cultures when appropriate (including retention of isolates for analysis) to assess patterns of infection transmission and effectiveness of infection control interventions at the facility or organization. Microbiologists assist in decisions concerning the indications for initiating and discontinuing active surveillance programs and optimize the use of laboratory resources.
- Molecular typing, on-site or outsourced, in order to investigate and control healthcare-associated outbreaks⁶⁰⁹.
- Application of rapid diagnostic tests to support clinical decisions involving patient treatment, room selection, and implementation of control measures including barrier precautions and use of vaccine or chemoprophylaxis agents (e.g., influenza⁶¹⁰⁻⁶¹², B. pertussis⁶¹³, RSV^{614, 615}, and enteroviruses⁶¹⁶). The microbiologist provides guidance to limit rapid testing to clinical situations in which rapid results influence patient

- management decisions, as well as providing oversight of point-of-care testing performed by non-laboratory healthcare workers ⁶¹⁷.
- Detection and rapid reporting of epidemiologically important organisms, including those that are reportable to public health agencies.
 - Implementation of a quality control program that ensures testing services are appropriate for the population served, and stringently evaluated for sensitivity, specificity, applicability, and feasibility.
 - Participation in a multidisciplinary team to develop and maintain an effective institutional program for the judicious use of antimicrobial agents ^{618, 619}.

II.A.2. Institutional safety culture and organizational characteristics Safety culture (or safety climate) refers to a work environment where a shared commitment to safety on the part of management and the workforce is understood and followed ^{557, 620, 621}. The authors of the Institute of Medicine Report, *To Err is Human* ⁵⁴³, acknowledge that causes of medical error are multifaceted but emphasize repeatedly the pivotal role of system failures and the benefits of a safety culture. A safety culture is created through 1) the actions management takes to improve patient and worker safety; 2) worker participation in safety planning; 3) the availability of appropriate protective equipment; 4) influence of group norms regarding acceptable safety practices; and 5) the organization's socialization process for new personnel. Safety and patient outcomes can be enhanced by improving or creating organizational characteristics within patient care units as demonstrated by studies of surgical ICUs ^{622, 623}. Each of these factors has a direct bearing on adherence to transmission prevention recommendations ²⁵⁷. Measurement of an institutional culture of safety is useful for designing improvements in healthcare ^{624, 625}. Several hospital-based studies have linked measures of safety culture with both employee adherence to safe practices and reduced exposures to blood and body fluids ⁶²⁶⁻⁶³². One study of hand hygiene practices concluded that improved adherence requires integration of infection control into the organization's safety culture ⁵⁶¹. Several hospitals that are part of the Veterans Administration Healthcare System have taken specific steps toward improving the safety culture, including error reporting mechanisms, performing root cause analysis on problems identified, providing safety incentives, and employee education. ⁶³³⁻⁶³⁵.

II.A.3. Adherence of healthcare personnel to recommended guidelines Adherence to recommended infection control practices decreases transmission of infectious agents in healthcare settings ^{116, 562, 636-640}. However, several observational studies have shown limited adherence to recommended practices by healthcare personnel ^{559, 640-657}. Observed adherence to universal precautions ranged from 43% to 89% ^{641, 642, 649, 651, 652}. However, the degree of adherence depended frequently on the practice that was assessed and, for glove use, the circumstance in which they were used. Appropriate glove use has ranged from a low of 15% ⁶⁴⁵ to a high of 82% ⁶⁵⁰. However, 92% and 98% adherence with glove use have been reported during arterial blood gas collection and

resuscitation, respectively, procedures where there may be considerable blood contact^{643, 656}. Differences in observed adherence have been reported among occupational groups in the same healthcare facility⁶⁴¹ and between experienced and nonexperienced professionals⁶⁴⁵. In surveys of healthcare personnel, self-reported adherence was generally higher than that reported in observational studies. Furthermore, where an observational component was included with a self-reported survey, self-perceived adherence was often greater than observed adherence⁶⁵⁷. Among nurses and physicians, increasing years of experience is a negative predictor of adherence^{645, 651}. Education to improve adherence is the primary intervention that has been studied. While positive changes in knowledge and attitude have been demonstrated,^{640, 658} there often has been limited or no accompanying change in behavior^{642, 644}. Self-reported adherence is higher in groups that have received an educational intervention^{630, 659}. Educational interventions that incorporated videotaping and performance feedback were successful in improving adherence during the period of study; the long-term effect of these interventions is not known⁶⁵⁴. The use of videotape also served to identify system problems (e.g., communication and access to personal protective equipment) that otherwise may not have been recognized.

Use of engineering controls and facility design concepts for improving adherence is gaining interest. While introduction of automated sinks had a negative impact on consistent adherence to hand washing⁶⁶⁰, use of electronic monitoring and voice prompts to remind healthcare workers to perform hand hygiene, and improving accessibility to hand hygiene products, increased adherence and contributed to a decrease in HAIs in one study⁶⁶¹. More information is needed regarding how technology might improve adherence.

Improving adherence to infection control practices requires a multifaceted approach that incorporates continuous assessment of both the individual and the work environment^{559, 561}. Using several behavioral theories, Kretzer and Larson concluded that a single intervention (e.g., a handwashing campaign or putting up new posters about transmission precautions) would likely be ineffective in improving healthcare personnel adherence⁶⁶². Improvement requires that the organizational leadership make prevention an institutional priority and integrate infection control practices into the organization's safety culture⁵⁶¹. A recent review of the literature concluded that variations in organizational factors (e.g., safety climate, policies and procedures, education and training) and individual factors (e.g., knowledge, perceptions of risk, past experience) were determinants of adherence to infection control guidelines for protection against SARS and other respiratory pathogens²⁵⁷.

II.B. Surveillance for healthcare-associated infections (HAIs)

Surveillance is an essential tool for case-finding of single patients or clusters of patients who are infected or colonized with epidemiologically important organisms (e.g., susceptible bacteria such as *S. aureus*, *S. pyogenes* [Group A streptococcus] or *Enterobacter-Klebsiella* spp; MRSA, VRE, and other MDROs; *C. difficile*; RSV; influenza virus) for which transmission-based precautions may

be required. Surveillance is defined as the ongoing, systematic collection, analysis, interpretation, and dissemination of data regarding a health-related event for use in public health action to reduce morbidity and mortality and to improve health⁶⁶³. The work of Ignaz Semmelweis that described the role of person-to-person transmission in puerperal sepsis is the earliest example of the use of surveillance data to reduce transmission of infectious agents⁶⁶⁴. Surveillance of both process measures and the infection rates to which they are linked are important for evaluating the effectiveness of infection prevention efforts and identifying indications for change^{555, 665-668}.

The Study on the Efficacy of Nosocomial Infection Control (SENIC) found that different combinations of infection control practices resulted in reduced rates of nosocomial surgical site infections, pneumonia, urinary tract infections, and bacteremia in acute care hospitals⁵⁶⁶; however, surveillance was the only component essential for reducing all four types of HAIs. Although a similar study has not been conducted in other healthcare settings, a role for surveillance and the need for novel strategies have been described in LTCFs^{398, 434, 669, 670} and in home care⁴⁷⁰⁻⁴⁷³. The essential elements of a surveillance system are: 1) standardized definitions; 2) identification of patient populations at risk for infection; 3) statistical analysis (e.g. risk-adjustment, calculation of rates using appropriate denominators, trend analysis using methods such as statistical process control charts); and 4) feedback of results to the primary caregivers⁶⁷¹⁻⁶⁷⁶. Data gathered through surveillance of high-risk populations, device use, procedures, and/or facility locations (e.g., ICUs) are useful for detecting transmission trends⁶⁷¹⁻⁶⁷³. Identification of clusters of infections should be followed by a systematic epidemiologic investigation to determine commonalities in persons, places, and time; and guide implementation of interventions and evaluation of the effectiveness of those interventions.

Targeted surveillance based on the highest risk areas or patients has been preferred over facility-wide surveillance for the most effective use of resources^{673, 676}. However, surveillance for certain epidemiologically important organisms may need to be facility-wide. Surveillance methods will continue to evolve as healthcare delivery systems change^{392, 677} and user-friendly electronic tools become more widely available for electronic tracking and trend analysis^{674, 678, 679}. Individuals with experience in healthcare epidemiology and infection control should be involved in selecting software packages for data aggregation and analysis to assure that the need for efficient and accurate HAI surveillance will be met. Effective surveillance is increasingly important as legislation requiring public reporting of HAI rates is passed and states work to develop effective systems to support such legislation⁶⁸⁰.

II.C. Education of HCWs, patients, and families

Education and training of healthcare personnel are a prerequisite for ensuring that policies and procedures for Standard and Transmission-Based Precautions are understood and practiced. Understanding the scientific rationale for the

precautions will allow HCWs to apply procedures correctly, as well as safely modify precautions based on changing requirements, resources, or healthcare settings^{14, 655, 681-688}. In one study, the likelihood of HCWs developing SARS was strongly associated with less than 2 hours of infection control training and lack of understanding of infection control procedures⁶⁸⁹. Education about the important role of vaccines (e.g., influenza, measles, varicella, pertussis, pneumococcal) in protecting healthcare personnel, their patients, and family members can help improve vaccination rates⁶⁹⁰⁻⁶⁹³.

Education on the principles and practices for preventing transmission of infectious agents should begin during training in the health professions and be provided to anyone who has an opportunity for contact with patients or medical equipment (e.g., nursing and medical staff; therapists and technicians, including respiratory, physical, occupational, radiology, and cardiology personnel; phlebotomists; housekeeping and maintenance staff; and students). In healthcare facilities, education and training on Standard and Transmission-Based Precautions are typically provided at the time of orientation and should be repeated as necessary to maintain competency; updated education and training are necessary when policies and procedures are revised or when there is a special circumstance, such as an outbreak that requires modification of current practice or adoption of new recommendations. Education and training materials and methods appropriate to the HCW's level of responsibility, individual learning habits, and language needs, can improve the learning experience^{658, 694-702}.

Education programs for healthcare personnel have been associated with sustained improvement in adherence to best practices and a related decrease in device-associated HAIs in teaching and non-teaching settings^{639, 703} and in medical and surgical ICUs {Coopersmith, 2002 #2149; Babcock, 2004 #2126; Berenholtz, 2004 #2289; www.ihl.org/IHI/Programs/Campaign, #2563}. Several studies have shown that, in addition to targeted education to improve specific practices, periodic assessment and feedback of the HCWs knowledge, and adherence to recommended practices are necessary to achieve the desired changes and to identify continuing education needs^{562, 704-708}. Effectiveness of this approach for isolation practices has been demonstrated for control of RSV^{116, 684}.

Patients, family members, and visitors can be partners in preventing transmission of infections in healthcare settings^{9, 42, 709-711}. Information about Standard Precautions, especially hand hygiene, Respiratory Hygiene/Cough Etiquette, vaccination (especially against influenza) and other routine infection prevention strategies may be incorporated into patient information materials that are provided upon admission to the healthcare facility. Additional information about Transmission-Based Precautions is best provided at the time they are initiated. Fact sheets, pamphlets, and other printed material may include information on the rationale for the additional precautions, risks to household members, room assignment for Transmission-Based Precautions purposes, explanation about the use of personal protective equipment by HCWs, and directions for use of

such equipment by family members and visitors. Such information may be particularly helpful in the home environment where household members often have primary responsibility for adherence to recommended infection control practices. Healthcare personnel must be available and prepared to explain this material and answer questions as needed.

II.D. Hand hygiene

Hand hygiene has been cited frequently as the single most important practice to reduce the transmission of infectious agents in healthcare settings^{559, 712, 713} and is an essential element of Standard Precautions. The term “hand hygiene” includes both handwashing with either plain or antiseptic-containing soap and water, and use of alcohol-based products (gels, rinses, foams) that do not require the use of water. In the absence of visible soiling of hands, approved alcohol-based products for hand disinfection are preferred over antimicrobial or plain soap and water because of their superior microbiocidal activity, reduced drying of the skin, and convenience⁵⁵⁹. Improved hand hygiene practices have been associated with a sustained decrease in the incidence of MRSA and VRE infections primarily in the ICU^{561, 562, 714-717}. The scientific rationale, indications, methods, and products for hand hygiene are summarized in other publications^{559, 717}.

The effectiveness of hand hygiene can be reduced by the type and length of fingernails^{559, 718, 719}. Individuals wearing artificial nails have been shown to harbor more pathogenic organisms, especially gram negative bacilli and yeasts, on the nails and in the subungual area than those with native nails^{720, 721}. In 2002, CDC/HICPAC recommended (Category IA) that artificial fingernails and extenders not be worn by healthcare personnel who have contact with high-risk patients (e.g., those in ICUs, ORs) due to the association with outbreaks of gram-negative bacillus and candidal infections as confirmed by molecular typing of isolates^{30, 31, 559, 722-725}. The need to restrict the wearing of artificial fingernails by all healthcare personnel who provide direct patient care or by healthcare personnel who have contact with other high risk groups (e.g., oncology, cystic fibrosis patients), has not been studied, but has been recommended by some experts²⁰. At this time such decisions are at the discretion of an individual facility’s infection control program. There is less evidence that jewelry affects the quality of hand hygiene. Although hand contamination with potential pathogens is increased with ring-wearing^{559, 726}, no studies have related this practice to HCW-to-patient transmission of pathogens.

II.E. Personal protective equipment (PPE) for healthcare personnel

PPE refers to a variety of barriers and respirators used alone or in combination to protect mucous membranes, airways, skin, and clothing from contact with infectious agents. The selection of PPE is based on the nature of the patient

interaction and/or the likely mode(s) of transmission. Guidance on the use of PPE is discussed in Part III. A suggested procedure for donning and removing PPE that will prevent skin or clothing contamination is presented in the Figure. Designated containers for used disposable or reusable PPE should be placed in a location that is convenient to the site of removal to facilitate disposal and containment of contaminated materials. Hand hygiene is always the final step after removing and disposing of PPE. The following sections highlight the primary uses and methods for selecting this equipment.

II.E.1. Gloves Gloves are used to prevent contamination of healthcare personnel hands when 1) anticipating direct contact with blood or body fluids, mucous membranes, nonintact skin and other potentially infectious material; 2) having direct contact with patients who are colonized or infected with pathogens transmitted by the contact route e.g., VRE, MRSA, RSV^{559, 727, 728}; or 3) handling or touching visibly or potentially contaminated patient care equipment and environmental surfaces^{72, 73, 559}. Gloves can protect both patients and healthcare personnel from exposure to infectious material that may be carried on hands⁷³. The extent to which gloves will protect healthcare personnel from transmission of bloodborne pathogens (e.g., HIV, HBV, HCV) following a needlestick or other puncture that penetrates the glove barrier has not been determined. Although gloves may reduce the volume of blood on the external surface of a sharp by 46-86%⁷²⁹, the residual blood in the lumen of a hollowbore needle would not be affected; therefore, the effect on transmission risk is unknown. Gloves manufactured for healthcare purposes are subject to FDA evaluation and clearance⁷³⁰. Nonsterile disposable medical gloves made of a variety of materials (e.g., latex, vinyl, nitrile) are available for routine patient care⁷³¹. The selection of glove type for non-surgical use is based on a number of factors, including the task that is to be performed, anticipated contact with chemicals and chemotherapeutic agents, latex sensitivity, sizing, and facility policies for creating a latex-free environment^{17, 732-734}. For contact with blood and body fluids during non-surgical patient care, a single pair of gloves generally provides adequate barrier protection⁷³⁴. However, there is considerable variability among gloves; both the quality of the manufacturing process and type of material influence their barrier effectiveness⁷³⁵. While there is little difference in the barrier properties of unused intact gloves⁷³⁶, studies have shown repeatedly that vinyl gloves have higher failure rates than latex or nitrile gloves when tested under simulated and actual clinical conditions^{731, 735-738}. For this reason either latex or nitrile gloves are preferable for clinical procedures that require manual dexterity and/or will involve more than brief patient contact. It may be necessary to stock gloves in several sizes. Heavier, reusable utility gloves are indicated for non-patient care activities, such as handling or cleaning contaminated equipment or surfaces^{11, 14, 739}.

During patient care, transmission of infectious organisms can be reduced by adhering to the principles of working from “clean” to “dirty”, and confining or limiting contamination to surfaces that are directly needed for patient care. It may be necessary to change gloves during the care of a single patient to prevent

cross-contamination of body sites^{559, 740}. It also may be necessary to change gloves if the patient interaction also involves touching portable computer keyboards or other mobile equipment that is transported from room to room. Discarding gloves between patients is necessary to prevent transmission of infectious material. Gloves must not be washed for subsequent reuse because microorganisms cannot be removed reliably from glove surfaces and continued glove integrity cannot be ensured. Furthermore, glove reuse has been associated with transmission of MRSA and gram-negative bacilli⁷⁴¹⁻⁷⁴³.

When gloves are worn in combination with other PPE, they are put on last. Gloves that fit snugly around the wrist are preferred for use with an isolation gown because they will cover the gown cuff and provide a more reliable continuous barrier for the arms, wrists, and hands. Gloves that are removed properly will prevent hand contamination (Figure). Hand hygiene following glove removal further ensures that the hands will not carry potentially infectious material that might have penetrated through unrecognized tears or that could contaminate the hands during glove removal^{559, 728, 741}.

II.E.2. Isolation gowns Isolation gowns are used as specified by Standard and Transmission-Based Precautions, to protect the HCW's arms and exposed body areas and prevent contamination of clothing with blood, body fluids, and other potentially infectious material^{24, 88, 262, 744-746}. The need for and type of isolation gown selected is based on the nature of the patient interaction, including the anticipated degree of contact with infectious material and potential for blood and body fluid penetration of the barrier. The wearing of isolation gowns and other protective apparel is mandated by the OSHA Bloodborne Pathogens Standard⁷³⁹. Clinical and laboratory coats or jackets worn over personal clothing for comfort and/or purposes of identity are not considered PPE.

When applying Standard Precautions, an isolation gown is worn only if contact with blood or body fluid is anticipated. However, when Contact Precautions are used (i.e., to prevent transmission of an infectious agent that is not interrupted by Standard Precautions alone and that is associated with environmental contamination), donning of both gown and gloves upon room entry is indicated to address unintentional contact with contaminated environmental surfaces^{54, 72, 73, 88}. The routine donning of isolation gowns upon entry into an intensive care unit or other high-risk area does not prevent or influence potential colonization or infection of patients in those areas^{365, 747-750}.

Isolation gowns are always worn in combination with gloves, and with other PPE when indicated. Gowns are usually the first piece of PPE to be donned. Full coverage of the arms and body front, from neck to the mid-thigh or below will ensure that clothing and exposed upper body areas are protected. Several gown sizes should be available in a healthcare facility to ensure appropriate coverage for staff members. Isolation gowns should be removed before leaving the patient care area to prevent possible contamination of the environment outside the patient's room. Isolation gowns should be removed in a manner that prevents contamination of clothing or skin (Figure). The outer, "contaminated", side of the

gown is turned inward and rolled into a bundle, and then discarded into a designated container for waste or linen to contain contamination.

II.E.3. Face protection: masks, goggles, face shields

II.E.3.a. Masks Masks are used for three primary purposes in healthcare settings: 1) placed on healthcare personnel to protect them from contact with infectious material from patients e.g., respiratory secretions and sprays of blood or body fluids, consistent with Standard Precautions and Droplet Precautions; 2) placed on healthcare personnel when engaged in procedures requiring sterile technique to protect patients from exposure to infectious agents carried in a healthcare worker's mouth or nose, and 3) placed on coughing patients to limit potential dissemination of infectious respiratory secretions from the patient to others (i.e., Respiratory Hygiene/Cough Etiquette). Masks may be used in combination with goggles to protect the mouth, nose and eyes, or a face shield may be used instead of a mask and goggles, to provide more complete protection for the face, as discussed below. **Masks should not be confused with particulate respirators that are used to prevent inhalation of small particles that may contain infectious agents transmitted via the airborne route as described below.**

The mucous membranes of the mouth, nose, and eyes are susceptible portals of entry for infectious agents, as can be other skin surfaces if skin integrity is compromised (e.g., by acne, dermatitis)^{66, 751-754}. Therefore, use of PPE to protect these body sites is an important component of Standard Precautions. The protective effect of masks for exposed healthcare personnel has been demonstrated^{93, 113, 755, 756}. Procedures that generate splashes or sprays of blood, body fluids, secretions, or excretions (e.g., endotracheal suctioning, bronchoscopy, invasive vascular procedures) require either a face shield (disposable or reusable) or mask and goggles^{93-95, 96, 113, 115, 262, 739, 757}. The wearing of masks, eye protection, and face shields in specified circumstances when blood or body fluid exposures are likely to occur is mandated by the OSHA Bloodborne Pathogens Standard⁷³⁹. Appropriate PPE should be selected based on the anticipated level of exposure.

Two mask types are available for use in healthcare settings: surgical masks that are cleared by the FDA and required to have fluid-resistant properties, and procedure or isolation masks^{758 #2688}. No studies have been published that compare mask types to determine whether one mask type provides better protection than another. Since procedure/isolation masks are not regulated by the FDA, there may be more variability in quality and performance than with surgical masks. Masks come in various shapes (e.g., molded and non-molded), sizes, filtration efficiency, and method of attachment (e.g., ties, elastic, ear loops). Healthcare facilities may find that different types of masks are needed to meet individual healthcare personnel needs.

II.E.3.b. Goggles, face shields Guidance on eye protection for infection control has been published⁷⁵⁹. The eye protection chosen for specific work situations (e.g., goggles or face shield) depends upon the circumstances of exposure, other

PPE used, and personal vision needs. Personal eyeglasses and contact lenses are NOT considered adequate eye protection (www.cdc.gov/niosh/topics/eye/eye-infectious.html). NIOSH states that, eye protection must be comfortable, allow for sufficient peripheral vision, and must be adjustable to ensure a secure fit. It may be necessary to provide several different types, styles, and sizes of protective equipment. Indirectly-vented goggles with a manufacturer's anti-fog coating may provide the most reliable practical eye protection from splashes, sprays, and respiratory droplets from multiple angles. Newer styles of goggles may provide better indirect airflow properties to reduce fogging, as well as better peripheral vision and more size options for fitting goggles to different workers. Many styles of goggles fit adequately over prescription glasses with minimal gaps. While effective as eye protection, goggles do not provide splash or spray protection to other parts of the face.

The role of goggles, in addition to a mask, in preventing exposure to infectious agents transmitted via respiratory droplets has been studied only for RSV. Reports published in the mid-1980s demonstrated that eye protection reduced occupational transmission of RSV^{760, 761}. Whether this was due to preventing hand-eye contact or respiratory droplet-eye contact has not been determined. However, subsequent studies demonstrated that RSV transmission is effectively prevented by adherence to Standard plus Contact Precautions and that for this virus routine use of goggles is not necessary^{24, 116, 117, 684, 762}. It is important to remind healthcare personnel that even if Droplet Precautions are not recommended for a specific respiratory tract pathogen, protection for the eyes, nose and mouth by using a mask and goggles, or face shield alone, is necessary when it is likely that there will be a splash or spray of any respiratory secretions or other body fluids as defined in Standard Precautions

Disposable or non-disposable face shields may be used as an alternative to goggles⁷⁵⁹. As compared with goggles, a face shield can provide protection to other facial areas in addition to the eyes. Face shields extending from chin to crown provide better face and eye protection from splashes and sprays; face shields that wrap around the sides may reduce splashes around the edge of the shield.

Removal of a face shield, goggles and mask can be performed safely after gloves have been removed, and hand hygiene performed. The ties, ear pieces and/or headband used to secure the equipment to the head are considered "clean" and therefore safe to touch with bare hands. The front of a mask, goggles and face shield are considered contaminated (Figure).

II.E.4. Respiratory protection The subject of respiratory protection as it applies to preventing transmission of airborne infectious agents, including the need for and frequency of fit-testing is under scientific review and was the subject of a CDC workshop in 2004⁷⁶³. Respiratory protection currently requires the use of a respirator with N95 or higher filtration to prevent inhalation of infectious particles. Information about respirators and respiratory protection programs is summarized

in the *Guideline for Preventing Transmission of Mycobacterium tuberculosis in Health-care Settings, 2005* (CDC.MMWR 2005; 54: RR-17¹²).

Respiratory protection is broadly regulated by OSHA under the general industry standard for respiratory protection (29CFR1910.134)⁷⁶⁴ which requires that U.S. employers in all employment settings implement a program to protect employees from inhalation of toxic materials. OSHA program components include medical clearance to wear a respirator; provision and use of appropriate respirators, including fit-tested NIOSH-certified N95 and higher particulate filtering respirators; education on respirator use and periodic re-evaluation of the respiratory protection program. When selecting particulate respirators, models with inherently good fit characteristics (i.e., those expected to provide protection factors of 10 or more to 95% of wearers) are preferred and could theoretically relieve the need for fit testing^{765, 766}. Issues pertaining to respiratory protection remain the subject of ongoing debate. Information on various types of respirators may be found at www.cdc.gov/niosh/npptl/respirators/respsars.html and in published studies^{765, 767, 768}. A user-seal check (formerly called a “fit check”) should be performed by the wearer of a respirator each time a respirator is donned to minimize air leakage around the facepiece⁷⁶⁹. The optimal frequency of fit-testing has not been determined; re-testing may be indicated if there is a change in facial features of the wearer, onset of a medical condition that would affect respiratory function in the wearer, or a change in the model or size of the initially assigned respirator¹².

Respiratory protection was first recommended for protection of preventing U.S. healthcare personnel from exposure to *M. tuberculosis* in 1989. That recommendation has been maintained in two successive revisions of the Guidelines for Prevention of Transmission of Tuberculosis in Hospitals and other Healthcare Settings^{12, 126}. The incremental benefit from respirator use, in addition to administrative and engineering controls (i.e., AIIRs, early recognition of patients likely to have tuberculosis and prompt placement in an AIIR, and maintenance of a patient with suspected tuberculosis in an AIIR until no longer infectious), for preventing transmission of airborne infectious agents (e.g., *M. tuberculosis*) is undetermined. Although some studies have demonstrated effective prevention of *M. tuberculosis* transmission in hospitals where surgical masks, instead of respirators, were used in conjunction with other administrative and engineering controls^{637, 770, 771}, CDC currently recommends N95 or higher level respirators for personnel exposed to patients with suspected or confirmed tuberculosis. Currently this is also true for other diseases that could be transmitted through the airborne route, including SARS²⁶² and smallpox^{108, 129, 772}, until inhalational transmission is better defined or healthcare-specific protective equipment more suitable for preventing infection are developed. Respirators are also currently recommended to be worn during the performance of aerosol-generating procedures (e.g., intubation, bronchoscopy, suctioning) on patients with SARS Co-V infection, avian influenza and pandemic influenza (See Appendix A).

Although Airborne Precautions are recommended for preventing airborne transmission of measles and varicella-zoster viruses, there are no data upon

which to base a recommendation for respiratory protection to protect susceptible personnel against these two infections; transmission of varicella-zoster virus has been prevented among pediatric patients using negative pressure isolation alone⁷⁷³. Whether respiratory protection (i.e., wearing a particulate respirator) would enhance protection from these viruses has not been studied. Since the majority of healthcare personnel have natural or acquired immunity to these viruses, only immune personnel generally care for patients with these infections⁷⁷⁴⁻⁷⁷⁷. Although there is no evidence to suggest that masks are not adequate to protect healthcare personnel in these settings, for purposes of consistency and simplicity, or because of difficulties in ascertaining immunity, some facilities may require the use of respirators for entry into all AIRs, regardless of the specific infectious agent.

Procedures for safe removal of respirators are provided (Figure). In some healthcare settings, particulate respirators used to provide care for patients *with M. tuberculosis* are reused by the same HCW. This is an acceptable practice providing the respirator is not damaged or soiled, the fit is not compromised by change in shape, and the respirator has not been contaminated with blood or body fluids. There are no data on which to base a recommendation for the length of time a respirator may be reused.

II.F. Safe work practices to prevent HCW exposure to bloodborne pathogens

II.F.1. Prevention of needlesticks and other sharps-related injuries Injuries due to needles and other sharps have been associated with transmission of HBV, HCV and HIV to healthcare personnel^{778, 779}. The prevention of sharps injuries has always been an essential element of Universal and now Standard Precautions^{1, 780}. These include measures to handle needles and other sharp devices in a manner that will prevent injury to the user and to others who may encounter the device during or after a procedure. These measures apply to routine patient care and do not address the prevention of sharps injuries and other blood exposures during surgical and other invasive procedures that are addressed elsewhere⁷⁸¹⁻⁷⁸⁵.

Since 1991, when OSHA first issued its Bloodborne Pathogens Standard to protect healthcare personnel from blood exposure, the focus of regulatory and legislative activity has been on implementing a hierarchy of control measures. This has included focusing attention on removing sharps hazards through the development and use of engineering controls. The federal Needlestick Safety and Prevention Act signed into law in November, 2000 authorized OSHA's revision of its Bloodborne Pathogens Standard to more explicitly require the use of safety-engineered sharp devices⁷⁸⁶. CDC has provided guidance on sharps injury prevention^{787, 788}, including for the design, implementation and evaluation of a comprehensive sharps injury prevention program⁷⁸⁹.

II.F.2. Prevention of mucous membrane contact Exposure of mucous membranes of the eyes, nose and mouth to blood and body fluids has been associated with the transmission of bloodborne viruses and other infectious agents to healthcare personnel^{66, 752, 754, 779}. The prevention of mucous membrane exposures has always been an element of Universal and now Standard Precautions for routine patient care^{1, 753} and is subject to OSHA bloodborne pathogen regulations. Safe work practices, in addition to wearing PPE, are used to protect mucous membranes and non-intact skin from contact with potentially infectious material. These include keeping gloved and ungloved hands that are contaminated from touching the mouth, nose, eyes, or face; and positioning patients to direct sprays and splatter away from the face of the caregiver. Careful placement of PPE before patient contact will help avoid the need to make PPE adjustments and possible face or mucous membrane contamination during use.

In areas where the need for resuscitation is unpredictable, mouthpieces, pocket resuscitation masks with one-way valves, and other ventilation devices provide an alternative to mouth-to-mouth resuscitation, preventing exposure of the caregiver's nose and mouth to oral and respiratory fluids during the procedure.

II.F.2.a. Precautions during aerosol-generating procedures The performance of procedures that can generate small particle aerosols (aerosol-generating procedures), such as bronchoscopy, endotracheal intubation, and open suctioning of the respiratory tract, have been associated with transmission of infectious agents to healthcare personnel, including *M. tuberculosis*⁷⁹⁰, SARS-CoV^{93, 94, 98} and *N. meningitidis*⁹⁵. Protection of the eyes, nose and mouth, in addition to gown and gloves, is recommended during performance of these procedures in accordance with Standard Precautions. Use of a particulate respirator is recommended during aerosol-generating procedures when the aerosol is likely to contain *M. tuberculosis*, SARS-CoV, or avian or pandemic influenza viruses.

II.G. Patient placement

II.G.1. Hospitals and long-term care settings Options for patient placement include single patient rooms, two patient rooms, and multi-bed wards. Of these, single patient rooms are preferred when there is a concern about transmission of an infectious agent. Although some studies have failed to demonstrate the efficacy of single patient rooms to prevent HAIs⁷⁹¹, other published studies, including one commissioned by the American Institute of Architects and the Facility Guidelines Institute, have documented a beneficial relationship between private rooms and reduction in infectious and noninfectious adverse patient outcomes^{792, 793}. The AIA notes that private rooms are the trend in hospital planning and design. However, most hospitals and long-term care facilities have multi-bed rooms and must consider many competing priorities when determining the appropriate room placement for patients (e.g., reason for admission; patient characteristics, such as age, gender, mental status; staffing needs; family

requests; psychosocial factors; reimbursement concerns). In the absence of obvious infectious diseases that require specified airborne infection isolation rooms (e.g., tuberculosis, SARS, chickenpox), the risk of transmission of infectious agents is not always considered when making placement decisions. When there are only a limited number of single-patient rooms, it is prudent to prioritize them for those patients who have conditions that facilitate transmission of infectious material to other patients (e.g., draining wounds, stool incontinence, uncontained secretions) and for those who are at increased risk of acquisition and adverse outcomes resulting from HAI (e.g., immunosuppression, open wounds, indwelling catheters, anticipated prolonged length of stay, total dependence on HCWs for activities of daily living)^{15, 24, 43, 430, 794, 795}. Single-patient rooms are always indicated for patients placed on Airborne Precautions and in a Protective Environment and are preferred for patients who require Contact or Droplet Precautions^{23, 24, 410, 435, 796, 797}. During a suspected or proven outbreak caused by a pathogen whose reservoir is the gastrointestinal tract, use of single patient rooms with private bathrooms limits opportunities for transmission, especially when the colonized or infected patient has poor personal hygiene habits, fecal incontinence, or cannot be expected to assist in maintaining procedures that prevent transmission of microorganisms (e.g., infants, children, and patients with altered mental status or developmental delay). In the absence of continued transmission, it is not necessary to provide a private bathroom for patients colonized or infected with enteric pathogens as long as personal hygiene practices and Standard Precautions, especially hand hygiene and appropriate environmental cleaning, are maintained. Assignment of a dedicated commode to a patient, and cleaning and disinfecting fixtures and equipment that may have fecal contamination (e.g., bathrooms, commodes⁷⁹⁸, scales used for weighing diapers) and the adjacent surfaces with appropriate agents may be especially important when a single-patient room can not be used since environmental contamination with intestinal tract pathogens is likely from both continent and incontinent patients^{54, 799}. Results of several studies to determine the benefit of a single-patient room to prevent transmission of *Clostridium difficile* are inconclusive^{167, 800-802}. Some studies have shown that being in the same room with a colonized or infected patient is not necessarily a risk factor for transmission^{791, 803-805}. However, for children, the risk of healthcare-associated diarrhea is increased with the increased number of patients per room⁸⁰⁶. Thus, patient factors are important determinants of infection transmission risks, and the need for a single-patient room and/or private bathroom for any patient is best determined on a case-by-case basis.

Cohorting is the practice of grouping together patients who are colonized or infected with the same organism to confine their care to one area and prevent contact with other patients. Cohorts are created based on clinical diagnosis, microbiologic confirmation when available, epidemiology, and mode of transmission of the infectious agent. It is generally preferred not to place severely immunosuppressed patients in rooms with other patients. Cohorting has been used extensively for managing outbreaks of MDROs including MRSA^{22, 807}, VRE^{638, 808, 809}, MDR-ESBLs⁸¹⁰, *Pseudomonas aeruginosa*²⁹; methicillin-susceptible

*Staphylococcus aureus*⁸¹¹; RSV^{812, 813}; adenovirus keratoconjunctivitis⁸¹⁴; rotavirus⁸¹⁵; and SARS⁸¹⁶. Modeling studies provide additional support for cohorting patients to control outbreaks Talon⁸¹⁷⁻⁸¹⁹. However, cohorting often is implemented only after routine infection control measures have failed to control an outbreak.

Assigning or cohorting healthcare personnel to care only for patients infected or colonized with a single target pathogen limits further transmission of the target pathogen to uninfected patients^{740, 819} but is difficult to achieve in the face of current staffing shortages in hospitals⁵⁸³ and residential healthcare sites⁸²⁰⁻⁸²². However, when continued transmission is occurring after implementing routine infection control measures and creating patient cohorts, cohorting of healthcare personnel may be beneficial.

During the seasons when RSV, human metapneumovirus⁸²³, parainfluenza, influenza, other respiratory viruses⁸²⁴, and rotavirus are circulating in the community, cohorting based on the presenting clinical syndrome is often a priority in facilities that care for infants and young children⁸²⁵. For example, during the respiratory virus season, infants may be cohorted based solely on the clinical diagnosis of bronchiolitis due to the logistical difficulties and costs associated with requiring microbiologic confirmation prior to room placement, and the predominance of RSV during most of the season. However, when available, single patient rooms are always preferred since a common clinical presentation (e.g., bronchiolitis), can be caused by more than one infectious agent^{823, 824, 826}. Furthermore, the inability of infants and children to contain body fluids, and the close physical contact that occurs during their care, increases infection transmission risks for patients and personnel in this setting^{24, 795}.

II.G.2. Ambulatory settings Patients actively infected with or incubating transmissible infectious diseases are seen frequently in ambulatory settings (e.g., outpatient clinics, physicians' offices, emergency departments) and potentially expose healthcare personnel and other patients, family members and visitors^{21, 34, 127, 135, 142, 827}. In response to the global outbreak of SARS in 2003 and in preparation for pandemic influenza, healthcare providers working in outpatient settings are urged to implement source containment measures (e.g., asking coughing patients to wear a surgical mask or cover their coughs with tissues) to prevent transmission of respiratory infections, beginning at the point of initial patient encounter^{9, 262, 828} as described below in section III.A.1.a. Signs can be posted at the entrance to facilities or at the reception or registration desk requesting that the patient or individuals accompanying the patient promptly inform the receptionist if there are symptoms of a respiratory infection (e.g., cough, flu-like illness, increased production of respiratory secretions). The presence of diarrhea, skin rash, or known or suspected exposure to a transmissible disease (e.g., measles, pertussis, chickenpox, tuberculosis) also could be added. Placement of potentially infectious patients without delay in an examination room limits the number of exposed individuals, e.g., in the common waiting area.

In waiting areas, maintaining a distance between symptomatic and non-symptomatic patients (e.g., >3 feet), in addition to source control measures, may limit exposures. However, infections transmitted via the airborne route (e.g., *M. tuberculosis*, measles, chickenpox) require additional precautions^{12, 125, 829}. Patients suspected of having such an infection can wear a surgical mask for source containment, if tolerated, and should be placed in an examination room, preferably an AIIR, as soon as possible. If this is not possible, having the patient wear a mask and segregate him/herself from other patients in the waiting area will reduce opportunities to expose others. Since the person(s) accompanying the patient also may be infectious, application of the same infection control precautions may need to be extended to these persons if they are symptomatic^{21, 252, 830}. For example, family members accompanying children admitted with suspected *M. tuberculosis* have been found to have unsuspected pulmonary tuberculosis with cavitory lesions, even when asymptomatic^{42, 831}. Patients with underlying conditions that increase their susceptibility to infection (e.g., those who are immunocompromised^{43, 44} or have cystic fibrosis²⁰) require special efforts to protect them from exposures to infected patients in common waiting areas. By informing the receptionist of their infection risk upon arrival, appropriate steps may be taken to further protect them from infection. In some cystic fibrosis clinics, in order to avoid exposure to other patients who could be colonized with *B. cepacia*, patients have been given beepers upon registration so that they may leave the area and receive notification to return when an examination room becomes available⁸³².

II.G.3. Home care In home care, the patient placement concerns focus on protecting others in the home from exposure to an infectious household member. For individuals who are especially vulnerable to adverse outcomes associated with certain infections, it may be beneficial to either remove them from the home or segregate them within the home. Persons who are not part of the household may need to be prohibited from visiting during the period of infectivity. For example, if a patient with pulmonary tuberculosis is contagious and being cared for at home, very young children (<4 years of age)⁸³³ and immunocompromised persons who have not yet been infected should be removed or excluded from the household. During the SARS outbreak of 2003, segregation of infected persons during the communicable phase of the illness was beneficial in preventing household transmission^{249, 834}.

II.H. Transport of patients

Several principles are used to guide transport of patients requiring Transmission-Based Precautions. In the inpatient and residential settings these include 1) limiting transport of such patients to essential purposes, such as diagnostic and therapeutic procedures that cannot be performed in the patient's room; 2) when transport is necessary, using appropriate barriers on the patient (e.g., mask, gown, wrapping in sheets or use of impervious dressings to cover the affected area(s) when infectious skin lesions or drainage are present, consistent with the route and risk of transmission; 3) notifying healthcare personnel in the receiving

area of the impending arrival of the patient and of the precautions necessary to prevent transmission; and 4) for patients being transported outside the facility, informing the receiving facility and the medi-van or emergency vehicle personnel in advance about the type of Transmission-Based Precautions being used. For tuberculosis, additional precautions may be needed in a small shared air space such as in an ambulance ¹².

II.I. Environmental measures

Cleaning and disinfecting non-critical surfaces in patient-care areas are part of Standard Precautions. In general, these procedures do not need to be changed for patients on Transmission-Based Precautions. The cleaning and disinfection of all patient-care areas is important for frequently touched surfaces, especially those closest to the patient, that are most likely to be contaminated (e.g., bedrails, bedside tables, commodes, doorknobs, sinks, surfaces and equipment in close proximity to the patient) ^{11, 72, 73, 835}. The frequency or intensity of cleaning may need to change based on the patient's level of hygiene and the degree of environmental contamination and for certain for infectious agents whose reservoir is the intestinal tract ⁵⁴. This may be especially true in LTCFs and pediatric facilities where patients with stool and urine incontinence are encountered more frequently. Also, increased frequency of cleaning may be needed in a Protective Environment to minimize dust accumulation ¹¹. Special recommendations for cleaning and disinfecting environmental surfaces in dialysis centers have been published ¹⁸. In all healthcare settings, administrative, staffing and scheduling activities should prioritize the proper cleaning and disinfection of surfaces that could be implicated in transmission. During a suspected or proven outbreak where an environmental reservoir is suspected, routine cleaning procedures should be reviewed, and the need for additional trained cleaning staff should be assessed. Adherence should be monitored and reinforced to promote consistent and correct cleaning is performed.

EPA-registered disinfectants or detergents/disinfectants that best meet the overall needs of the healthcare facility for routine cleaning and disinfection should be selected ^{11, 836}. In general, use of the existing facility detergent/disinfectant according to the manufacturer's recommendations for amount, dilution, and contact time is sufficient to remove pathogens from surfaces of rooms where colonized or infected individuals were housed. This includes those pathogens that are resistant to multiple classes of antimicrobial agents (e.g., *C. difficile*, VRE, MRSA, MDR-GNB ^{11, 24, 88, 435, 746, 796, 837}). Most often, environmental reservoirs of pathogens during outbreaks are related to a failure to follow recommended procedures for cleaning and disinfection rather than the specific cleaning and disinfectant agents used ⁸³⁸⁻⁸⁴¹.

Certain pathogens (e.g., rotavirus, noroviruses, *C. difficile*) may be resistant to some routinely used hospital disinfectants ^{275, 292, 842-847}. The role of specific disinfectants in limiting transmission of rotavirus has been demonstrated experimentally ⁸⁴². Also, since *C. difficile* may display increased levels of spore production when exposed to non-chlorine-based cleaning agents, and the spores are more resistant than vegetative cells to commonly used surface disinfectants,

some investigators have recommended the use of a 1:10 dilution of 5.25% sodium hypochlorite (household bleach) and water for routine environmental disinfection of rooms of patients with *C. difficile* when there is continued transmission^{844, 848}. In one study, the use of a hypochlorite solution was associated with a decrease in rates of *C. difficile* infections⁸⁴⁷. The need to change disinfectants based on the presence of these organisms can be determined in consultation with the infection control committee^{11, 847, 848}. Detailed recommendations for disinfection and sterilization of surfaces and medical equipment that have been in contact with prion-containing tissue or high risk body fluids, and for cleaning of blood and body substance spills, are available in the Guidelines for Environmental Infection Control in Health-Care Facilities¹¹ and in the Guideline for Disinfection and Sterilization⁸⁴⁸.

II.J. Patient care equipment and instruments/devices

Medical equipment and instruments/devices must be cleaned and maintained according to the manufacturers' instructions to prevent patient-to-patient transmission of infectious agents^{86, 87, 325, 849}. Cleaning to remove organic material must always precede high level disinfection and sterilization of critical and semi-critical instruments and devices because residual proteinaceous material reduces the effectiveness of the disinfection and sterilization processes^{836, 848}. Noncritical equipment, such as commodes, intravenous pumps, and ventilators, must be thoroughly cleaned and disinfected before use on another patient. All such equipment and devices should be handled in a manner that will prevent HCW and environmental contact with potentially infectious material. It is important to include computers and personal digital assistants (PDAs) used in patient care in policies for cleaning and disinfection of non-critical items. The literature on contamination of computers with pathogens has been summarized⁸⁵⁰ and two reports have linked computer contamination to colonization and infections in patients^{851, 852}. Although keyboard covers and washable keyboards that can be easily disinfected are in use, the infection control benefit of those items and optimal management have not been determined.

In all healthcare settings, providing patients who are on Transmission-Based Precautions with dedicated noncritical medical equipment (e.g., stethoscope, blood pressure cuff, electronic thermometer) has been beneficial for preventing transmission^{74, 89, 740, 853, 854}. When this is not possible, disinfection after use is recommended. Consult other guidelines for detailed guidance in developing specific protocols for cleaning and reprocessing medical equipment and patient care items in both routine and special circumstances^{11, 14, 18, 20, 740, 836, 848}.

In home care, it is preferable to remove visible blood or body fluids from durable medical equipment before it leaves the home. Equipment can be cleaned on-site using a detergent/disinfectant and, when possible, should be placed in a single plastic bag for transport to the reprocessing location^{20, 739}.

II.K. Textiles and laundry

Soiled textiles, including bedding, towels, and patient or resident clothing may be contaminated with pathogenic microorganisms. However, the risk of disease

transmission is negligible if they are handled, transported, and laundered in a safe manner ^{11, 855, 856}. Key principles for handling soiled laundry are 1) not shaking the items or handling them in any way that may aerosolize infectious agents; 2) avoiding contact of one's body and personal clothing with the soiled items being handled; and 3) containing soiled items in a laundry bag or designated bin. When laundry chutes are used, they must be maintained to minimize dispersion of aerosols from contaminated items ¹¹. The methods for handling, transporting, and laundering soiled textiles are determined by organizational policy and any applicable regulations ⁷³⁹; guidance is provided in the Guidelines for Environmental Infection Control ¹¹. Rather than rigid rules and regulations, hygienic and common sense storage and processing of clean textiles is recommended ^{11, 857}. When laundering occurs outside of a healthcare facility, the clean items must be packaged or completely covered and placed in an enclosed space during transport to prevent contamination with outside air or construction dust that could contain infectious fungal spores that are a risk for immunocompromised patients ¹¹.

Institutions are required to launder garments used as personal protective equipment and uniforms visibly soiled with blood or infective material ⁷³⁹. There are few data to determine the safety of home laundering of HCW uniforms, but no increase in infection rates was observed in the one published study ⁸⁵⁸ and no pathogens were recovered from home- or hospital-laundered scrubs in another study ⁸⁵⁹. In the home, textiles and laundry from patients with potentially transmissible infectious pathogens do not require special handling or separate laundering, and may be washed with warm water and detergent ^{11, 858, 859}.

II.L. Solid waste

The management of solid waste emanating from the healthcare environment is subject to federal and state regulations for medical and non-medical waste ^{860, 861}. No additional precautions are needed for non-medical solid waste that is being removed from rooms of patients on Transmission-Based Precautions. Solid waste may be contained in a single bag (as compared to using two bags) of sufficient strength. ⁸⁶²

II.M. Dishware and eating utensils

The combination of hot water and detergents used in dishwashers is sufficient to decontaminate dishware and eating utensils. Therefore, no special precautions are needed for dishware (e.g., dishes, glasses, cups) or eating utensils; reusable dishware and utensils may be used for patients requiring Transmission-Based Precautions. In the home and other communal settings, eating utensils and drinking vessels that are being used should not be shared, consistent with principles of good personal hygiene and for the purpose of preventing transmission of respiratory viruses, *Herpes simplex* virus, and infectious agents that infect the gastrointestinal tract and are transmitted by the fecal/oral route (e.g., hepatitis A virus, noroviruses). If adequate resources for cleaning utensils and dishes are not available, disposable products may be used.

II.N. Adjunctive measures

Important adjunctive measures that are not considered primary components of programs to prevent transmission of infectious agents, but improve the effectiveness of such programs, include 1) antimicrobial management programs; 2) postexposure chemoprophylaxis with antiviral or antibacterial agents; 3) vaccines used both for pre and postexposure prevention; and 4) screening and restricting visitors with signs of transmissible infections. Detailed discussion of judicious use of antimicrobial agents is beyond the scope of this document; however the topic is addressed in the MDRO section (Management of Multidrug-Resistant Organisms in Healthcare Settings 2006.

www.cdc.gov/ncidod/dhqp/pdf/ar/mdroGuideline2006.pdf).

II.N.1. Chemoprophylaxis Antimicrobial agents and topical antiseptics may be used to prevent infection and potential outbreaks of selected agents. Infections for which postexposure chemoprophylaxis is recommended under defined conditions include *B. pertussis*^{17, 863}, *N. meningitidis*⁸⁶⁴, *B. anthracis* after environmental exposure to aerosolizable material⁸⁶⁵, influenza virus⁶¹¹, HIV⁸⁶⁶, and group A streptococcus¹⁶⁰. Orally administered antimicrobials may also be used under defined circumstances for MRSA decolonization of patients or healthcare personnel⁸⁶⁷.

Another form of chemoprophylaxis is the use of topical antiseptic agents. For example, triple dye is used routinely on the umbilical cords of term newborns to reduce the risk of colonization, skin infections, and omphalitis caused by *S. aureus*, including MRSA, and group A streptococcus^{868, 869}. Extension of the use of triple dye to low birth weight infants in the NICU was one component of a program that controlled one longstanding MRSA outbreak²². Topical antiseptics are also used for decolonization of healthcare personnel or selected patients colonized with MRSA, using mupirocin as discussed in the MDRO guideline⁸⁷⁰
^{867, 871-873}.

II.N.2. Immunoprophylaxis Certain immunizations recommended for susceptible healthcare personnel have decreased the risk of infection and the potential for transmission in healthcare facilities^{17, 874}. The OSHA mandate that requires employers to offer hepatitis B vaccination to HCWs played a substantial role in the sharp decline in incidence of occupational HBV infection^{778, 875}. The use of varicella vaccine in healthcare personnel has decreased the need to place susceptible HCWs on administrative leave following exposure to patients with varicella⁷⁷⁵. Also, reports of healthcare-associated transmission of rubella in obstetrical clinics^{33, 876} and measles in acute care settings³⁴ demonstrate the importance of immunization of susceptible healthcare personnel against childhood diseases. Many states have requirements for HCW vaccination for measles and rubella in the absence of evidence of immunity. Annual influenza vaccine campaigns targeted to patients and healthcare personnel in LTCFs and acute-care settings have been instrumental in preventing or limiting institutional

outbreaks and increasing attention is being directed toward improving influenza vaccination rates in healthcare personnel^{35, 611, 690, 877, 878, 879}.

Transmission of *B. pertussis* in healthcare facilities has been associated with large and costly outbreaks that include both healthcare personnel and patients^{17, 36, 41, 100, 683, 827, 880, 881}.

HCWs who have close contact with infants with pertussis are at particularly high risk because of waning immunity and, until 2005, the absence of a vaccine that could be used in adults. However, two acellular pertussis vaccines were licensed in the United States in 2005, one for use in individuals aged 11-18 and one for use in ages 10-64 years⁸⁸². Provisional ACIP recommendations at the time of publication of this document include adolescents and adults, especially those with contact with infants < 12 months of age and healthcare personnel with direct patient contact^{883 884}.

Immunization of children and adults will help prevent the introduction of vaccine-preventable diseases into healthcare settings. The recommended immunization schedule for children is published annually in the January issues of the *Morbidity Mortality Weekly Report* with interim updates as needed^{885, 886}. An adult immunization schedule also is available for healthy adults and those with special immunization needs due to high risk medical conditions⁸⁸⁷.

Some vaccines are also used for postexposure prophylaxis of susceptible individuals, including varicella⁸⁸⁸, influenza⁶¹¹, hepatitis B⁷⁷⁸, and smallpox²²⁵ vaccines^{17, 874}. In the future, administration of a newly developed *S. aureus* conjugate vaccine (still under investigation) to selected patients may provide a novel method of preventing healthcare-associated *S. aureus*, including MRSA, infections in high-risk groups (e.g., hemodialysis patients and candidates for selected surgical procedures)^{889, 890}.

Immune globulin preparations also are used for postexposure prophylaxis of certain infectious agents under specified circumstances (e.g., varicella-zoster virus [VZIG], hepatitis B virus [HBIG], rabies [RIG], measles and hepatitis A virus [IG]^{17, 833, 874}). The RSV monoclonal antibody preparation, Palivizumab, may have contributed to controlling a nosocomial outbreak of RSV in one NICU, but there is insufficient evidence to support a routine recommendation for its use in this setting⁸⁹¹.

II.N. 3. Management of visitors

II.N.3.a. Visitors as sources of infection Visitors have been identified as the source of several types of HAIs (e.g., pertussis^{40, 41}, *M. tuberculosis*^{42, 892}, influenza, and other respiratory viruses^{24, 43, 44, 373} and SARS^{21, 252-254}). However, effective methods for visitor screening in healthcare settings have not been studied. Visitor screening is especially important during community outbreaks of infectious diseases and for high risk patient units. Sibling visits are often encouraged in birthing centers, post partum rooms and in pediatric inpatient units, ICUs, and in residential settings for children; in hospital settings, a child visitor should visit only his or her own sibling. Screening of visiting siblings and other children before they are allowed into clinical areas is necessary to prevent the introduction of childhood illnesses and common respiratory infections.

Screening may be passive through the use of signs to alert family members and visitors with signs and symptoms of communicable diseases not to enter clinical areas. More active screening may include the completion of a screening tool or questionnaire which elicits information related to recent exposures or current symptoms. That information is reviewed by the facility staff and the visitor is either permitted to visit or is excluded⁸³³.

Family and household members visiting pediatric patients with pertussis and tuberculosis may need to be screened for a history of exposure as well as signs and symptoms of current infection. Potentially infectious visitors are excluded until they receive appropriate medical screening, diagnosis, or treatment. If exclusion is not considered to be in the best interest of the patient or family (i.e., primary family members of critically or terminally ill patients), then the symptomatic visitor must wear a mask while in the healthcare facility and remain in the patient's room, avoiding exposure to others, especially in public waiting areas and the cafeteria.

Visitor screening is used consistently on HSCT units^{15, 43}. However, considering the experience during the 2003 SARS outbreaks and the potential for pandemic influenza, developing effective visitor screening systems will be beneficial⁹.

Education concerning Respiratory Hygiene/Cough Etiquette is a useful adjunct to visitor screening.

II.N.3.b. Use of barrier precautions by visitors The use of gowns, gloves, or masks by visitors in healthcare settings has not been addressed specifically in the scientific literature. Some studies included the use of gowns and gloves by visitors in the control of MDRO's, but did not perform a separate analysis to determine whether their use by visitors had a measurable impact⁸⁹³⁻⁸⁹⁵. Family members or visitors who are providing care or having very close patient contact (e.g., feeding, holding) may have contact with other patients and could contribute to transmission if barrier precautions are not used correctly. Specific recommendations may vary by facility or by unit and should be determined by the level of interaction.

Part III:

Precautions to Prevent Transmission of Infectious Agents There are two tiers of HICPAC/CDC precautions to prevent transmission of infectious agents, Standard Precautions and Transmission-Based Precautions. Standard Precautions are intended to be applied to the care of all patients in all healthcare settings, regardless of the suspected or confirmed presence of an infectious agent. **Implementation of *Standard Precautions* constitutes the primary strategy for the prevention of healthcare-associated transmission of infectious agents among patients and healthcare personnel.**

Transmission-Based Precautions are for patients who are known or suspected to be infected or colonized with infectious agents, including certain epidemiologically important pathogens, which require additional control measures to effectively prevent transmission. Since the infecting agent often is not known at the time of admission to a healthcare facility, Transmission-Based Precautions are used empirically, according to the clinical syndrome and the likely etiologic agents at the time, and then modified when the pathogen is identified or a transmissible infectious etiology is ruled out. Examples of this syndromic approach are presented in Table 2. The HICPAC/CDC Guidelines also include recommendations for creating a Protective Environment for allogeneic HSCT patients.

The specific elements of Standard and Transmission-Based Precautions are discussed in Part II of this guideline. In Part III, the circumstances in which Standard Precautions, Transmission-Based Precautions, and a Protective Environment are applied are discussed. See Tables 4 and 5 for summaries of the key elements of these sets of precautions

III.A. Standard Precautions Standard Precautions combine the major features of Universal Precautions (UP)^{780, 896} and Body Substance Isolation (BSI)⁶⁴⁰ and are based on the principle that all blood, body fluids, secretions, excretions except sweat, nonintact skin, and mucous membranes may contain transmissible infectious agents. Standard Precautions include a group of infection prevention practices that apply to all patients, regardless of suspected or confirmed infection status, in any setting in which healthcare is delivered (Table 4). These include: hand hygiene; use of gloves, gown, mask, eye protection, or face shield, depending on the anticipated exposure; and safe injection practices. Also, equipment or items in the patient environment likely to have been contaminated with infectious body fluids must be handled in a manner to prevent transmission of infectious agents (e.g. wear gloves for direct contact, contain heavily soiled equipment, properly clean and disinfect or sterilize reusable equipment before use on another patient).

The application of Standard Precautions during patient care is determined by the nature of the HCW-patient interaction and the extent of anticipated blood, body fluid, or pathogen exposure. For some interactions (e.g., performing venipuncture), only gloves may be needed; during other interactions (e.g., intubation), use of gloves, gown, and face shield or mask and goggles is necessary. Education and training on the principles and rationale for

recommended practices are critical elements of Standard Precautions because they facilitate appropriate decision-making and promote adherence when HCWs are faced with new circumstances^{655, 681-686}. An example of the importance of the use of Standard Precautions is intubation, especially under emergency circumstances when infectious agents may not be suspected, but later are identified (e.g., SARS-CoV, *N. meningitidis*). The application of Standard Precautions is described below and summarized in Table 4. Guidance on donning and removing gloves, gowns and other PPE is presented in the Figure. Standard Precautions are also intended to protect patients by ensuring that healthcare personnel do not carry infectious agents to patients on their hands or via equipment used during patient care.

III.A.1. New Elements of Standard Precautions Infection control problems that are identified in the course of outbreak investigations often indicate the need for new recommendations or reinforcement of existing infection control recommendations to protect patients. Because such recommendations are considered a standard of care and may not be included in other guidelines, they are added here to Standard Precautions. Three such areas of practice that have been added are: Respiratory Hygiene/Cough Etiquette, safe injection practices, and use of masks for insertion of catheters or injection of material into spinal or epidural spaces via lumbar puncture procedures (e.g., myelogram, spinal or epidural anesthesia). While most elements of Standard Precautions evolved from Universal Precautions that were developed for protection of healthcare personnel, these new elements of Standard Precautions focus on protection of patients.

III.A.1.a. Respiratory Hygiene/Cough Etiquette The transmission of SARS-CoV in emergency departments by patients and their family members during the widespread SARS outbreaks in 2003 highlighted the need for vigilance and prompt implementation of infection control measures at the first point of encounter within a healthcare setting (e.g., reception and triage areas in emergency departments, outpatient clinics, and physician offices)^{21, 254, 897}. The strategy proposed has been termed Respiratory Hygiene/Cough Etiquette^{9, 828} and is intended to be incorporated into infection control practices as a new component of Standard Precautions. The strategy is targeted at patients and accompanying family members and friends with undiagnosed transmissible respiratory infections, and applies to any person with signs of illness including cough, congestion, rhinorrhea, or increased production of respiratory secretions when entering a healthcare facility^{40, 41, 43}. The term *cough etiquette* is derived from recommended source control measures for *M. tuberculosis*^{12, 126}. The elements of Respiratory Hygiene/Cough Etiquette include 1) education of healthcare facility staff, patients, and visitors; 2) posted signs, in language(s) appropriate to the population served, with instructions to patients and accompanying family members or friends; 3) source control measures (e.g., covering the mouth/nose with a tissue when coughing and prompt disposal of used tissues, using surgical masks on the coughing person when tolerated and

appropriate); 4) hand hygiene after contact with respiratory secretions; and 5) spatial separation, ideally >3 feet, of persons with respiratory infections in common waiting areas when possible. Covering sneezes and coughs and placing masks on coughing patients are proven means of source containment that prevent infected persons from dispersing respiratory secretions into the air^{107, 145, 898, 899}. Masking may be difficult in some settings, (e.g., pediatrics, in which case, the emphasis by necessity may be on cough etiquette⁹⁰⁰. Physical proximity of <3 feet has been associated with an increased risk for transmission of infections via the droplet route (e.g., *N. meningitidis*¹⁰³ and group A streptococcus¹¹⁴ and therefore supports the practice of distancing infected persons from others who are not infected. The effectiveness of good hygiene practices, especially hand hygiene, in preventing transmission of viruses and reducing the incidence of respiratory infections both within and outside⁹⁰¹⁻⁹⁰³ healthcare settings is summarized in several reviews^{559, 717, 904}.

These measures should be effective in decreasing the risk of transmission of pathogens contained in large respiratory droplets (e.g., influenza virus²³, adenovirus¹¹¹, *B. pertussis*⁸²⁷ and *Mycoplasma pneumoniae*¹¹². Although fever will be present in many respiratory infections, patients with pertussis and mild upper respiratory tract infections are often afebrile. Therefore, the absence of fever does not always exclude a respiratory infection. Patients who have asthma, allergic rhinitis, or chronic obstructive lung disease also may be coughing and sneezing. While these patients often are not infectious, cough etiquette measures are prudent.

Healthcare personnel are advised to observe Droplet Precautions (i.e., wear a mask) and hand hygiene when examining and caring for patients with signs and symptoms of a respiratory infection. Healthcare personnel who have a respiratory infection are advised to avoid direct patient contact, especially with high risk patients. If this is not possible, then a mask should be worn while providing patient care.

III.A.1.b. Safe Injection Practices The investigation of four large outbreaks of HBV and HCV among patients in ambulatory care facilities in the United States identified a need to define and reinforce safe injection practices⁴⁵³. The four outbreaks occurred in a private medical practice, a pain clinic, an endoscopy clinic, and a hematology/oncology clinic. The primary breaches in infection control practice that contributed to these outbreaks were 1) reinsertion of used needles into a multiple-dose vial or solution container (e.g., saline bag) and 2) use of a single needle/syringe to administer intravenous medication to multiple patients. In one of these outbreaks, preparation of medications in the same workspace where used needle/syringes were dismantled also may have been a contributing factor. These and other outbreaks of viral hepatitis could have been prevented by adherence to basic principles of aseptic technique for the preparation and administration of parenteral medications^{453, 454}. These include the use of a sterile, single-use, disposable needle and syringe for each injection given and prevention of contamination of injection equipment and medication.

Whenever possible, use of single-dose vials is preferred over multiple-dose vials, especially when medications will be administered to multiple patients. Outbreaks related to unsafe injection practices indicate that some healthcare personnel are unaware of, do not understand, or do not adhere to basic principles of infection control and aseptic technique. A survey of US healthcare workers who provide medication through injection found that 1% to 3% reused the same needle and/or syringe on multiple patients⁹⁰⁵. Among the deficiencies identified in recent outbreaks were a lack of oversight of personnel and failure to follow-up on reported breaches in infection control practices in ambulatory settings. Therefore, to ensure that all healthcare workers understand and adhere to recommended practices, principles of infection control and aseptic technique need to be reinforced in training programs and incorporated into institutional policies that are monitored for adherence⁴⁵⁴.

III.A.1.c. Infection Control Practices for Special Lumbar Puncture

Procedures In 2004, CDC investigated eight cases of post-myelography meningitis that either were reported to CDC or identified through a survey of the Emerging Infections Network of the Infectious Disease Society of America. Blood and/or cerebrospinal fluid of all eight cases yielded streptococcal species consistent with oropharyngeal flora and there were changes in the CSF indices and clinical status indicative of bacterial meningitis. Equipment and products used during these procedures (e.g., contrast media) were excluded as probable sources of contamination. Procedural details available for seven cases determined that antiseptic skin preparations and sterile gloves had been used. However, none of the clinicians wore a face mask, giving rise to the speculation that droplet transmission of oropharyngeal flora was the most likely explanation for these infections. Bacterial meningitis following myelogram and other spinal procedures (e.g., lumbar puncture, spinal and epidural anesthesia, intrathecal chemotherapy) has been reported previously⁹⁰⁶⁻⁹¹⁵. As a result, the question of whether face masks should be worn to prevent droplet spread of oral flora during spinal procedures (e.g., myelogram, lumbar puncture, spinal anesthesia) has been debated^{916,917}. Face masks are effective in limiting the dispersal of oropharyngeal droplets⁹¹⁸ and are recommended for the placement of central venous catheters⁹¹⁹. In October 2005, the Healthcare Infection Control Practices Advisory Committee (HICPAC) reviewed the evidence and concluded that there is sufficient experience to warrant the additional protection of a face mask for the individual placing a catheter or injecting material into the spinal or epidural space.

III.B. Transmission-Based Precautions There are three categories of Transmission-Based Precautions: Contact Precautions, Droplet Precautions, and Airborne Precautions. Transmission-Based Precautions are used when the route(s) of transmission is (are) not completely interrupted using Standard Precautions alone. For some diseases that have multiple routes of transmission (e.g., SARS), more than one Transmission-Based Precautions category may be used. When used either singly or in combination, they are always used in

addition to Standard Precautions. See Appendix A for recommended precautions for specific infections. When Transmission-Based Precautions are indicated, efforts must be made to counteract possible adverse effects on patients (i.e., anxiety, depression and other mood disturbances⁹²⁰⁻⁹²², perceptions of stigma⁹²³, reduced contact with clinical staff⁹²⁴⁻⁹²⁶, and increases in preventable adverse events⁵⁶⁵ in order to improve acceptance by the patients and adherence by HCWs.

III.B.1. Contact Precautions Contact Precautions are intended to prevent transmission of infectious agents, including epidemiologically important microorganisms, which are spread by direct or indirect contact with the patient or the patient's environment as described in I.B.3.a. The specific agents and circumstance for which Contact Precautions are indicated are found in Appendix A. The application of Contact Precautions for patients infected or colonized with MDROs is described in the 2006 HICPAC/CDC MDRO guideline⁹²⁷. Contact Precautions also apply where the presence of excessive wound drainage, fecal incontinence, or other discharges from the body suggest an increased potential for extensive environmental contamination and risk of transmission. A single-patient room is preferred for patients who require Contact Precautions. When a single-patient room is not available, consultation with infection control personnel is recommended to assess the various risks associated with other patient placement options (e.g., cohorting, keeping the patient with an existing roommate). In multi-patient rooms, ≥ 3 feet spatial separation between beds is advised to reduce the opportunities for inadvertent sharing of items between the infected/colonized patient and other patients. Healthcare personnel caring for patients on Contact Precautions wear a gown and gloves for all interactions that may involve contact with the patient or potentially contaminated areas in the patient's environment. Donning PPE upon room entry and discarding before exiting the patient room is done to contain pathogens, especially those that have been implicated in transmission through environmental contamination (e.g., VRE, *C. difficile*, noroviruses and other intestinal tract pathogens; RSV)^{54, 72, 73, 78, 274, 275, 740}.

III.B.2. Droplet Precautions Droplet Precautions are intended to prevent transmission of pathogens spread through close respiratory or mucous membrane contact with respiratory secretions as described in I.B.3.b. Because these pathogens do not remain infectious over long distances in a healthcare facility, special air handling and ventilation are not required to prevent droplet transmission. Infectious agents for which Droplet Precautions are indicated are found in Appendix A and include *B. pertussis*, influenza virus, adenovirus, rhinovirus, *N. meningitides*, and group A streptococcus (for the first 24 hours of antimicrobial therapy). A single patient room is preferred for patients who require Droplet Precautions. When a single-patient room is not available, consultation with infection control personnel is recommended to assess the various risks associated with other patient placement options (e.g., cohorting, keeping the patient with an existing roommate). Spatial separation of ≥ 3 feet and drawing

the curtain between patient beds is especially important for patients in multi-bed rooms with infections transmitted by the droplet route. Healthcare personnel wear a mask (a respirator is not necessary) for close contact with infectious patient; the mask is generally donned upon room entry. Patients on Droplet Precautions who must be transported outside of the room should wear a mask if tolerated and follow Respiratory Hygiene/Cough Etiquette.

III.B.3. Airborne Precautions Airborne Precautions prevent transmission of infectious agents that remain infectious over long distances when suspended in the air (e.g., rubeola virus [measles], varicella virus [chickenpox], *M. tuberculosis*, and possibly SARS-CoV) as described in I.B.3.c and Appendix A. The preferred placement for patients who require Airborne Precautions is in an airborne infection isolation room (AIIR). An AIIR is a single-patient room that is equipped with special air handling and ventilation capacity that meet the American Institute of Architects/Facility Guidelines Institute (AIA/FGI) standards for AIIRs (i.e., monitored negative pressure relative to the surrounding area, 12 air exchanges per hour for new construction and renovation and 6 air exchanges per hour for existing facilities, air exhausted directly to the outside or recirculated through HEPA filtration before return)^{12, 13}. Some states require the availability of such rooms in hospitals, emergency departments, and nursing homes that care for patients with *M. tuberculosis*. A respiratory protection program that includes education about use of respirators, fit-testing, and user seal checks is required in any facility with AIIRs. In settings where Airborne Precautions cannot be implemented due to limited engineering resources (e.g., physician offices), masking the patient, placing the patient in a private room (e.g., office examination room) with the door closed, and providing N95 or higher level respirators or masks if respirators are not available for healthcare personnel will reduce the likelihood of airborne transmission until the patient is either transferred to a facility with an AIIR or returned to the home environment, as deemed medically appropriate. Healthcare personnel caring for patients on Airborne Precautions wear a mask or respirator, depending on the disease-specific recommendations (Respiratory Protection II.E.4, Table 2, and Appendix A), that is donned prior to room entry. Whenever possible, non-immune HCWs should not care for patients with vaccine-preventable airborne diseases (e.g., measles, chickenpox, and smallpox).

III.C. Syndromic and empiric applications of Transmission-Based Precautions Diagnosis of many infections requires laboratory confirmation. Since laboratory tests, especially those that depend on culture techniques, often require two or more days for completion, Transmission-Based Precautions must be implemented while test results are pending based on the clinical presentation and likely pathogens. Use of appropriate Transmission-Based Precautions at the time a patient develops symptoms or signs of transmissible infection, or arrives at a healthcare facility for care, reduces transmission opportunities. While it is not possible to identify prospectively all patients needing Transmission-Based Precautions, certain clinical syndromes and conditions carry a sufficiently high

risk to warrant their use empirically while confirmatory tests are pending (Table 2). Infection control professionals are encouraged to modify or adapt this table according to local conditions.

III.D. Discontinuation of Transmission-Based Precautions Transmission-Based Precautions remain in effect for limited periods of time (i.e., while the risk for transmission of the infectious agent persists or for the duration of the illness (Appendix A). For most infectious diseases, this duration reflects known patterns of persistence and shedding of infectious agents associated with the natural history of the infectious process and its treatment. For some diseases (e.g., pharyngeal or cutaneous diphtheria, RSV), Transmission-Based Precautions remain in effect until culture or antigen-detection test results document eradication of the pathogen and, for RSV, symptomatic disease is resolved. For other diseases, (e.g., *M. tuberculosis*) state laws and regulations, and healthcare facility policies, may dictate the duration of precautions¹²). In immunocompromised patients, viral shedding can persist for prolonged periods of time (many weeks to months) and transmission to others may occur during that time; therefore, the duration of contact and/or droplet precautions may be prolonged for many weeks^{500, 928-933}.

The duration of Contact Precautions for patients who are colonized or infected with MDROs remains undefined. MRSA is the only MDRO for which effective decolonization regimens are available⁸⁶⁷. However, carriers of MRSA who have negative nasal cultures after a course of systemic or topical therapy may resume shedding MRSA in the weeks that follow therapy^{934, 935}. Although early guidelines for VRE suggested discontinuation of Contact Precautions after three stool cultures obtained at weekly intervals proved negative⁷⁴⁰, subsequent experiences have indicated that such screening may fail to detect colonization that can persist for >1 year^{27, 936-938}. Likewise, available data indicate that colonization with VRE, MRSA⁹³⁹, and possibly MDR-GNB, can persist for many months, especially in the presence of severe underlying disease, invasive devices, and recurrent courses of antimicrobial agents.

It may be prudent to assume that MDRO carriers are colonized permanently and manage them accordingly. Alternatively, an interval free of hospitalizations, antimicrobial therapy, and invasive devices (e.g., 6 or 12 months) before reculturing patients to document clearance of carriage may be used. Determination of the best strategy awaits the results of additional studies. See the 2006 HICPAC/CDC MDRO guideline⁹²⁷ for discussion of possible criteria to discontinue Contact Precautions for patients colonized or infected with MDROs.

III.E. Application of Transmission-Based Precautions in ambulatory and home care settings Although Transmission-Based Precautions generally apply in all healthcare settings, exceptions exist. For example, in home care, AIIRs are not available. Furthermore, family members already exposed to diseases such as varicella and tuberculosis would not use masks or respiratory protection, but visiting HCWs would need to use such protection. Similarly, management of patients colonized or infected with MDROs may necessitate

Contact Precautions in acute care hospitals and in some LTCFs when there is continued transmission, but the risk of transmission in ambulatory care and home care, has not been defined. Consistent use of Standard Precautions may suffice in these settings, but more information is needed.

III.F. Protective Environment A Protective Environment is designed for allogeneic HSCT patients to minimize fungal spore counts in the air and reduce the risk of invasive environmental fungal infections (see Table 5 for specifications)^{11, 13-15}. The need for such controls has been demonstrated in studies of aspergillus outbreaks associated with construction^{11, 14, 15, 157, 158}. As defined by the American Institute of Architecture¹³ and presented in detail in the Guideline for Environmental Infection Control 2003^{11, 861}, air quality for HSCT patients is improved through a combination of environmental controls that include 1) HEPA filtration of incoming air; 2) directed room air flow; 3) positive room air pressure relative to the corridor; 4) well-sealed rooms (including sealed walls, floors, ceilings, windows, electrical outlets) to prevent flow of air from the outside; 5) ventilation to provide ≥ 12 air changes per hour; 6) strategies to minimize dust (e.g., scrubbable surfaces rather than upholstery⁹⁴⁰ and carpet⁹⁴¹, and routinely cleaning crevices and sprinkler heads); and 7) prohibiting dried and fresh flowers and potted plants in the rooms of HSCT patients. The latter is based on molecular typing studies that have found indistinguishable strains of *Aspergillus terreus* in patients with hematologic malignancies and in potted plants in the vicinity of the patients⁹⁴²⁻⁹⁴⁴. The desired quality of air may be achieved without incurring the inconvenience or expense of laminar airflow^{15, 157}. To prevent inhalation of fungal spores during periods when construction, renovation, or other dust-generating activities that may be ongoing in and around the health-care facility, it has been advised that severely immunocompromised patients wear a high-efficiency respiratory-protection device (e.g., an N95 respirator) when they leave the Protective Environment^{11, 14, 945}). The use of masks or respirators by HSCT patients when they are outside of the Protective Environment for prevention of environmental fungal infections in the absence of construction has not been evaluated. A Protective Environment does not include the use of barrier precautions beyond those indicated for Standard and Transmission-Based Precautions. No published reports support the benefit of placing solid organ transplants or other immunocompromised patients in a Protective Environment.

Part IV:

Recommendations

These recommendations are designed to prevent transmission of infectious agents among patients and healthcare personnel in all settings where healthcare is delivered. As in other CDC/HICPAC guidelines, each recommendation is categorized on the basis of existing scientific data, theoretical rationale, applicability, and when possible, economic impact. The CDC/HICPAC system for categorizing recommendations is as follows:

Category IA Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiologic studies.

Category IB Strongly recommended for implementation and supported by some experimental, clinical, or epidemiologic studies and a strong theoretical rationale.

Category IC Required for implementation, as mandated by federal and/or state regulation or standard.

Category II Suggested for implementation and supported by suggestive clinical or epidemiologic studies or a theoretical rationale.

No recommendation; unresolved issue. Practices for which insufficient evidence or no consensus regarding efficacy exists.

I. Administrative Responsibilities

Healthcare organization administrators should ensure the implementation of recommendations in this section.

- I.A. Incorporate preventing transmission of infectious agents into the objectives of the organization's patient and occupational safety programs^{543-546, 561, 620, 626, 946}. *Category IB/IC*
- I.B. Make preventing transmission of infectious agents a priority for the healthcare organization. Provide administrative support, including fiscal and human resources for maintaining infection control programs^{434, 548, 549, 559, 561, 566, 662 552, 562-564, 946}. *Category IB/IC*
 - I.B.1. Assure that individuals with training in infection control are employed by or are available by contract to all healthcare facilities so that the infection control program is managed by one or more qualified individuals^{552, 566 316, 575, 947 573, 576, 946}. *Category IB/IC*
 - I.B.1.a. Determine the specific infection control full-time equivalents (FTEs) according to the scope of the infection control program, the complexity of the healthcare facility or system, the characteristics of the patient population, the unique or urgent needs of the facility and community, and proposed staffing levels based on survey results and recommendations from professional organizations^{434, 549 552, 566 316, 569, 573, 575 948 949}. *Category IB*
 - I.B.2. Include prevention of healthcare-associated infections (HAI) as one determinant of bedside nurse staffing levels and composition,

- especially in high-risk units^{585-589 590 592 593 551, 594, 595 418, 596, 597 583}.
Category IB
- I.B.3. Delegate authority to infection control personnel or their designees (e.g., patient care unit charge nurses) for making infection control decisions concerning patient placement and assignment of Transmission-Based Precautions^{549 434, 857, 946}. *Category IC*
- I.B.4. Involve infection control personnel in decisions on facility construction and design, determination of AIIR and Protective Environment capacity needs and environmental assessments^{11, 13, 950 951 12}. *Category IB/IC*
- I.B.4.a. Provide ventilation systems required for a sufficient number of AIIRs (as determined by a risk assessment) and Protective Environments in healthcare facilities that provide care to patients for whom such rooms are indicated, according to published recommendations^{11-13, 15}. *Category IB/IC*
- I.B.5. Involve infection control personnel in the selection and post-implementation evaluation of medical equipment and supplies and changes in practice that could affect the risk of HAI^{952, 953}. *Category IC*
- I.B.6. Ensure availability of human and fiscal resources to provide clinical microbiology laboratory support, including a sufficient number of medical technologists trained in microbiology, appropriate to the healthcare setting, for monitoring transmission of microorganisms, planning and conducting epidemiologic investigations, and detecting emerging pathogens. Identify resources for performing surveillance cultures, rapid diagnostic testing for viral and other selected pathogens, preparation of antimicrobial susceptibility summary reports, trend analysis, and molecular typing of clustered isolates (performed either on-site or in a reference laboratory) and use these resources according to facility-specific epidemiologic needs, in consultation with clinical microbiologists^{553, 609, 610, 612, 617, 954 614 603, 615, 616 605 599 554 598, 606, 607}. *Category IB*
- I.B.7. Provide human and fiscal resources to meet occupational health needs related to infection control (e.g., healthcare personnel immunization, post-exposure evaluation and care, evaluation and management of healthcare personnel with communicable infections^{739 12 17, 879-881, 955 134 690}). *Category IB/IC*
- I.B.8. In all areas where healthcare is delivered, provide supplies and equipment necessary for the consistent observance of Standard Precautions, including hand hygiene products and personal protective equipment (e.g., gloves, gowns, face and eye protection)^{739 559 946}. *Category IB/IC*
- I.B.9. Develop and implement policies and procedures to ensure that reusable patient care equipment is cleaned and reprocessed appropriately before use on another patient^{11, 956 957, 958 959 836 87 11, 960 961}. *Category IA/IC*

- I.C. Develop and implement processes to ensure oversight of infection control activities appropriate to the healthcare setting and assign responsibility for oversight of infection control activities to an individual or group within the healthcare organization that is knowledgeable about infection control ^{434, 549, 566}. *Category II*
- I.D. Develop and implement systems for early detection and management (e.g., use of appropriate infection control measures, including isolation precautions, PPE) of potentially infectious persons at initial points of patient encounter in outpatient settings (e.g., triage areas, emergency departments, outpatient clinics, physician offices) and at the time of admission to hospitals and long-term care facilities (LTCF) ^{9, 122, 134, 253, 827}. *Category IB*
- I.E. Develop and implement policies and procedures to limit patient visitation by persons with signs or symptoms of a communicable infection. Screen visitors to high-risk patient care areas (e.g., oncology units, hematopoietic stem cell transplant [HSCT] units, intensive care units, other severely immunocompromised patients) for possible infection ^{43 24, 41, 962, 963}. *Category IB*
- I.F. Identify performance indicators of the effectiveness of organization-specific measures to prevent transmission of infectious agents (Standard and Transmission-Based Precautions), establish processes to monitor adherence to those performance measures and provide feedback to staff members ^{704 739 705 708 666, 964 667 668 555}. *Category IB*

II. Education and Training

- II.A. Provide job- or task-specific education and training on preventing transmission of infectious agents associated with healthcare during orientation to the healthcare facility; update information periodically during ongoing education programs. Target all healthcare personnel for education and training, including but not limited to medical, nursing, clinical technicians, laboratory staff; property service (housekeeping), laundry, maintenance and dietary workers; students, contract staff and volunteers. Document competency initially and repeatedly, as appropriate, for the specific staff positions. Develop a system to ensure that healthcare personnel employed by outside agencies meet these education and training requirements through programs offered by the agencies or by participation in the healthcare facility's program designed for full-time personnel ^{126, 559, 561, 562, 655, 681-684, 686, 688, 689, 702, 893, 919, 965}. *Category IB*
 - II.A.1. Include in education and training programs, information concerning use of vaccines as an adjunctive infection control measure ^{17, 611, 690, 874}. *Category IB*
 - II.A.2. Enhance education and training by applying principles of adult learning, using reading level and language appropriate material for the target audience, and using online educational tools available to the institution ^{658, 694, 695, 697, 698, 700, 966}. *Category IB*

- II.B. Provide instructional materials for patients and visitors on recommended hand hygiene and Respiratory Hygiene/Cough Etiquette practices and the application of Transmission-Based Precautions^{9, 709, 710, 963}. *Category II*

III. Surveillance

- III.A. Monitor the incidence of epidemiologically-important organisms and targeted HAIs that have substantial impact on outcome and for which effective preventive interventions are available; use information collected through surveillance of high-risk populations, procedures, devices and highly transmissible infectious agents to detect transmission of infectious agents in the healthcare facility^{566, 671, 672, 675, 687, 919, 967, 968 673 969 970}.
Category IA
- III.B. Apply the following epidemiologic principles of infection surveillance^{671, 967 673 969 663 664}. *Category IB*
- y Use standardized definitions of infection
 - y Use laboratory-based data (when available)
 - y Collect epidemiologically-important variables (e.g., patient locations and/or clinical service in hospitals and other large multi-unit facilities, population-specific risk factors [e.g., low birth-weight neonates], underlying conditions that predispose to serious adverse outcomes)
 - y Analyze data to identify trends that may indicated increased rates of transmission
 - y Feedback information on trends in the incidence and prevalence of HAIs, probable risk factors, and prevention strategies and their impact to the appropriate healthcare providers, organization administrators, and as required by local and state health authorities
- III.C. Develop and implement strategies to reduce risks for transmission and evaluate effectiveness^{566, 673, 684, 970 963 971}. *Category IB*
- III.D. When transmission of epidemiologically-important organisms continues despite implementation and documented adherence to infection prevention and control strategies, obtain consultation from persons knowledgeable in infection control and healthcare epidemiology to review the situation and recommend additional measures for control^{566 247 687}.
Category IB
- III.E. Review periodically information on community or regional trends in the incidence and prevalence of epidemiologically-important organisms (e.g., influenza, RSV, pertussis, invasive group A streptococcal disease, MRSA, VRE) (including in other healthcare facilities) that may impact transmission of organisms within the facility^{398, 687, 972, 973 974}. *Category II*

IV. Standard Precautions

Assume that every person is potentially infected or colonized with an organism that could be transmitted in the healthcare setting and apply the following infection control practices during the delivery of health care.

- IV.A. Hand Hygiene

- IV.A.1. During the delivery of healthcare, avoid unnecessary touching of surfaces in close proximity to the patient to prevent both contamination of clean hands from environmental surfaces and transmission of pathogens from contaminated hands to surfaces^{72, 73, 739, 800, 975}(CDC, 2001 #970). *Category IB/IC*
- IV.A.2. When hands are visibly dirty, contaminated with proteinaceous material, or visibly soiled with blood or body fluids, wash hands with either a nonantimicrobial soap and water or an antimicrobial soap and water⁵⁵⁹. *Category IA*
- IV.A.3. If hands are not visibly soiled, or after removing visible material with nonantimicrobial soap and water, decontaminate hands in the clinical situations described in IV.A.2.a-f. The preferred method of hand decontamination is with an alcohol-based hand rub^{562, 978}. Alternatively, hands may be washed with an antimicrobial soap and water. Frequent use of alcohol-based hand rub immediately following handwashing with nonantimicrobial soap may increase the frequency of dermatitis⁵⁵⁹. *Category IB*
Perform hand hygiene:
 - IV.A.3.a. Before having direct contact with patients^{664, 979}. *Category IB*
 - IV.A.3.b. After contact with blood, body fluids or excretions, mucous membranes, nonintact skin, or wound dressings⁶⁶⁴. *Category IA*
 - IV.A.3.c. After contact with a patient's intact skin (e.g., when taking a pulse or blood pressure or lifting a patient)^{167, 976, 979, 980}. *Category IB*
 - IV.A.3.d. If hands will be moving from a contaminated-body site to a clean-body site during patient care. *Category II*
 - IV.A.3.e. After contact with inanimate objects (including medical equipment) in the immediate vicinity of the patient^{72, 73, 88, 800, 981, 982}. *Category II*
 - IV.A.3.f. After removing gloves^{728, 741, 742}. *Category IB*
- IV.A.4. Wash hands with non-antimicrobial soap and water or with antimicrobial soap and water if contact with spores (e.g., *C. difficile* or *Bacillus anthracis*) is likely to have occurred. The physical action of washing and rinsing hands under such circumstances is recommended because alcohols, chlorhexidine, iodophors, and other antiseptic agents have poor activity against spores^{559, 956, 983}. *Category II*
- IV.A.5. Do not wear artificial fingernails or extenders if duties include direct contact with patients at high risk for infection and associated adverse outcomes (e.g., those in ICUs or operating rooms)^{30, 31, 559, 722-724}. *Category IA*
 - IV.A.5.a. Develop an organizational policy on the wearing of non-natural nails by healthcare personnel who have direct contact with patients outside of the groups specified above⁹⁸⁴. *Category II*
- IV.B. Personal protective equipment (PPE) (see Figure)
 - IV.B.1. Observe the following principles of use:

- IV.B.1.a. Wear PPE, as described in IV.B.2-4, when the nature of the anticipated patient interaction indicates that contact with blood or body fluids may occur^{739, 780, 896}. *Category IB/IC*
- IV.B.1.b. Prevent contamination of clothing and skin during the process of removing PPE (see Figure). *Category II*
- IV.B.1.c. Before leaving the patient's room or cubicle, remove and discard PPE^{18, 739}. *Category IB/IC*
- IV.B.2. Gloves
 - IV.B.2.a. Wear gloves when it can be reasonably anticipated that contact with blood or other potentially infectious materials, mucous membranes, nonintact skin, or potentially contaminated intact skin (e.g., of a patient incontinent of stool or urine) could occur^{18, 728, 739, 741, 780, 985}. *Category IB/IC*
 - IV.B.2.b. Wear gloves with fit and durability appropriate to the task^{559, 731, 732, 739, 986, 987}. *Category IB*
 - IV.B.2.b.i. Wear disposable medical examination gloves for providing direct patient care.
 - IV.B.2.b.ii. Wear disposable medical examination gloves or reusable utility gloves for cleaning the environment or medical equipment.
 - IV.B.2.c. Remove gloves after contact with a patient and/or the surrounding environment (including medical equipment) using proper technique to prevent hand contamination (see Figure). Do not wear the same pair of gloves for the care of more than one patient. Do not wash gloves for the purpose of reuse since this practice has been associated with transmission of pathogens^{559, 728, 741-743, 988}. *Category IB*
 - IV.B.2.d. Change gloves during patient care if the hands will move from a contaminated body-site (e.g., perineal area) to a clean body-site (e.g., face). *Category II*
- IV.B.3. Gowns
 - IV.B.3.a. Wear a gown, that is appropriate to the task, to protect skin and prevent soiling or contamination of clothing during procedures and patient-care activities when contact with blood, body fluids, secretions, or excretions is anticipated^{739, 780, 896}. *Category IB/IC*
 - IV.B.3.a.i. Wear a gown for direct patient contact if the patient has uncontained secretions or excretions^{24, 88, 89, 739, 744}. *Category IB/IC*
 - IV.B.3.a.ii. Remove gown and perform hand hygiene before leaving the patient's environment^{24, 88, 89, 739, 744}. *Category IB/IC*
 - IV.B.3.b. Do not reuse gowns, even for repeated contacts with the same patient. *Category II*
 - IV.B.3.c. Routine donning of gowns upon entrance into a high risk unit (e.g., ICU, NICU, HSCT unit) is not indicated^{365, 747-750}. *Category IB*
- IV.B.4. Mouth, nose, eye protection

- IV.B.4.a. Use PPE to protect the mucous membranes of the eyes, nose and mouth during procedures and patient-care activities that are likely to generate splashes or sprays of blood, body fluids, secretions and excretions. Select masks, goggles, face shields, and combinations of each according to the need anticipated by the task performed^{113, 739, 780, 896}. *Category IB/IC*
- IV.B.5. During aerosol-generating procedures (e.g., bronchoscopy, suctioning of the respiratory tract [if not using in-line suction catheters], endotracheal intubation) in patients who are not suspected of being infected with an agent for which respiratory protection is otherwise recommended (e.g., *M. tuberculosis*, SARS or hemorrhagic fever viruses), wear one of the following: a face shield that fully covers the front and sides of the face, a mask with attached shield, or a mask and goggles (in addition to gloves and gown)^{95, 96, 113, 126 93 94, 134}. *Category IB*
- IV.C. Respiratory Hygiene/Cough Etiquette
 - IV.C.1. Educate healthcare personnel on the importance of source control measures to contain respiratory secretions to prevent droplet and fomite transmission of respiratory pathogens, especially during seasonal outbreaks of viral respiratory tract infections (e.g., influenza, RSV, adenovirus, parainfluenza virus) in communities^{14, 24, 684 10, 262}. *Category IB*
 - IV.C.2. Implement the following measures to contain respiratory secretions in patients and accompanying individuals who have signs and symptoms of a respiratory infection, beginning at the point of initial encounter in a healthcare setting (e.g., triage, reception and waiting areas in emergency departments, outpatient clinics and physician offices)^{20, 24, 145, 902, 989}.
 - IV.C.2.a. Post signs at entrances and in strategic places (e.g., elevators, cafeterias) within ambulatory and inpatient settings with instructions to patients and other persons with symptoms of a respiratory infection to cover their mouths/noses when coughing or sneezing, use and dispose of tissues, and perform hand hygiene after hands have been in contact with respiratory secretions. *Category II*
 - IV.C.2.b. Provide tissues and no-touch receptacles (e.g., foot-pedal-operated lid or open, plastic-lined waste basket) for disposal of tissues²⁰. *Category II*
 - IV.C.2.c. Provide resources and instructions for performing hand hygiene in or near waiting areas in ambulatory and inpatient settings; provide conveniently-located dispensers of alcohol-based hand rubs and, where sinks are available, supplies for handwashing^{559, 903}. *Category IB*
 - IV.C.2.d. During periods of increased prevalence of respiratory infections in the community (e.g., as indicated by increased school absenteeism, increased number of patients seeking care for a

respiratory infection), offer masks to coughing patients and other symptomatic persons (e.g., persons who accompany ill patients) upon entry into the facility or medical office^{126, 899, 898} and encourage them to maintain special separation, ideally a distance of at least 3 feet, from others in common waiting areas^{23, 103, 111, 114, 20, 134}. *Category IB*

IV.C.2.d.i. Some facilities may find it logistically easier to institute this recommendation year-round as a standard of practice. *Category II*

IV.D. Patient placement

IV.D.1. Include the potential for transmission of infectious agents in patient-placement decisions. Place patients who pose a risk for transmission to others (e.g., uncontained secretions, excretions or wound drainage; infants with suspected viral respiratory or gastrointestinal infections) in a single-patient room when available^{24, 430, 435, 796, 797, 806, 990, 410, 793}. *Category IB*

IV.D.2. Determine patient placement based on the following principles:

- y Route(s) of transmission of the known or suspected infectious agent
- y Risk factors for transmission in the infected patient
- y Risk factors for adverse outcomes resulting from an HAI in other patients in the area or room being considered for patient-placement
- y Availability of single-patient rooms
- y Patient options for room-sharing (e.g., cohorting patients with the same infection) *Category II*

IV.E. Patient-care equipment and instruments/devices⁹⁵⁶

IV.E.1. Establish policies and procedures for containing, transporting, and handling patient-care equipment and instruments/devices that may be contaminated with blood or body fluids^{18, 739, 975}. *Category IB/IC*

IV.E.2. Remove organic material from critical and semi-critical instrument/devices, using recommended cleaning agents before high level disinfection and sterilization to enable effective disinfection and sterilization processes^{836, 991, 992}. *Category IA*

IV.E.3. Wear PPE (e.g., gloves, gown), according to the level of anticipated contamination, when handling patient-care equipment and instruments/devices that is visibly soiled or may have been in contact with blood or body fluids^{18, 739, 975}. *Category IB/IC*

IV.F. Care of the environment¹¹

IV.F.1. Establish policies and procedures for routine and targeted cleaning of environmental surfaces as indicated by the level of patient contact and degree of soiling¹¹. *Category II*

IV.F.2. Clean and disinfect surfaces that are likely to be contaminated with pathogens, including those that are in close proximity to the patient (e.g., bed rails, over bed tables) and frequently-touched surfaces in the patient care environment (e.g., door knobs, surfaces in and

- surrounding toilets in patients' rooms) on a more frequent schedule compared to that for other surfaces (e.g., horizontal surfaces in waiting rooms) ^{11 73, 740, 746, 993, 994 72, 800, 835 995}. *Category IB*
- IV.F.3. Use EPA-registered disinfectants that have microbiocidal (i.e., killing) activity against the pathogens most likely to contaminate the patient-care environment. Use in accordance with manufacturer's instructions ^{842-844, 956, 996}. *Category IB/IC*
- IV.F.3.a. Review the efficacy of in-use disinfectants when evidence of continuing transmission of an infectious agent (e.g., rotavirus, *C. difficile*, norovirus) may indicate resistance to the in-use product and change to a more effective disinfectant as indicated ^{275, 842, 847}. *Category II*
- IV.F.4. In facilities that provide health care to pediatric patients or have waiting areas with child play toys (e.g., obstetric/gynecology offices and clinics), establish policies and procedures for cleaning and disinfecting toys at regular intervals ^{379 80}. *Category IB*
- Use the following principles in developing this policy and procedures: *Category II*
 - y Select play toys that can be easily cleaned and disinfected
 - y Do not permit use of stuffed furry toys if they will be shared
 - y Clean and disinfect large stationary toys (e.g., climbing equipment) at least weekly and whenever visibly soiled
 - y If toys are likely to be mouthed, rinse with water after disinfection; alternatively wash in a dishwasher
 - y When a toy requires cleaning and disinfection, do so immediately or store in a designated labeled container separate from toys that are clean and ready for use
- IV.F.5. Include multi-use electronic equipment in policies and procedures for preventing contamination and for cleaning and disinfection, especially those items that are used by patients, those used during delivery of patient care, and mobile devices that are moved in and out of patient rooms frequently (e.g., daily) ^{850 851, 852, 997}. *Category IB*
- IV.F.5.a. No recommendation for use of removable protective covers or washable keyboards. *Unresolved issue*
- IV.G. Textiles and laundry
- IV.G.1. Handle used textiles and fabrics with minimum agitation to avoid contamination of air, surfaces and persons ^{739, 998, 999}. *Category IB/IC*
- IV.G.2. If laundry chutes are used, ensure that they are properly designed, maintained, and used in a manner to minimize dispersion of aerosols from contaminated laundry ^{11, 13, 1000, 1001}. *Category IB/IC*
- IV.H. Safe injection practices
- The following recommendations apply to the use of needles, cannulas that replace needles, and, where applicable intravenous delivery systems ⁴⁵⁴

- IV.H.1. Use aseptic technique to avoid contamination of sterile injection equipment ^{1002, 1003}. *Category IA*
- IV.H.2. Do not administer medications from a syringe to multiple patients, even if the needle or cannula on the syringe is changed. Needles, cannulae and syringes are sterile, single-use items; they should not be reused for another patient nor to access a medication or solution that might be used for a subsequent patient ^{453, 919, 1004, 1005}.
Category IA
- IV.H.3. Use fluid infusion and administration sets (i.e., intravenous bags, tubing and connectors) for one patient only and dispose appropriately after use. Consider a syringe or needle/cannula contaminated once it has been used to enter or connect to a patient's intravenous infusion bag or administration set ⁴⁵³.
Category IB
- IV.H.4. Use single-dose vials for parenteral medications whenever possible ⁴⁵³. *Category IA*
- IV.H.5. Do not administer medications from single-dose vials or ampules to multiple patients or combine leftover contents for later use ^{369 453, 1005}. *Category IA*
- IV.H.6. If multidose vials must be used, both the needle or cannula and syringe used to access the multidose vial must be sterile ^{453, 1002}.
Category IA
- IV.H.7. Do not keep multidose vials in the immediate patient treatment area and store in accordance with the manufacturer's recommendations; discard if sterility is compromised or questionable ^{453, 1003}. *Category IA*
- IV.H.8. Do not use bags or bottles of intravenous solution as a common source of supply for multiple patients ^{453, 1006}. *Category IB*
- IV.I. Infection control practices for special lumbar puncture procedures
Wear a surgical mask when placing a catheter or injecting material into the spinal canal or subdural space (i.e., during myelograms, lumbar puncture and spinal or epidural anesthesia ^{906 907-909 910, 911 912-914, 918 1007}). *Category IB*
- IV.J. Worker safety
Adhere to federal and state requirements for protection of healthcare personnel from exposure to bloodborne pathogens ⁷³⁹. *Category IC*

V. Transmission-Based Precautions

V.A. General principles

- V.A.1. In addition to Standard Precautions, use Transmission-Based Precautions for patients with documented or suspected infection or colonization with highly transmissible or epidemiologically-important pathogens for which additional precautions are needed to prevent transmission (see Appendix A) ^{24, 93, 126, 141, 306, 806, 1008}. *Category IA*
- V.A.2. Extend duration of Transmission-Based Precautions, (e.g., Droplet, Contact) for immunosuppressed patients with viral infections due to

prolonged shedding of viral agents that may be transmitted to others^{928, 931-933, 1009-1011}.

Category IA

V.B. Contact Precautions

V.B.1. Use Contact Precautions as recommended in Appendix A for patients with known or suspected infections or evidence of syndromes that represent an increased risk for contact transmission. For specific recommendations for use of Contact Precautions for colonization or infection with MDROs, go to the MDRO guideline:

www.cdc.gov/ncidod/dhqp/pdf/ar/mdroGuideline2006.pdf⁸⁷⁰.

V.B.2. Patient placement

V.B.2.a. In *acute care hospitals*, place patients who require Contact Precautions in a single-patient room when available^{24, 687, 793, 796, 797, 806, 837, 893, 1012, 1013} *Category IB*

When single-patient rooms are in short supply, apply the following principles for making decisions on patient placement:

- y Prioritize patients with conditions that may facilitate transmission (e.g., uncontained drainage, stool incontinence) for single-patient room placement. *Category II*
- y Place together in the same room (cohort) patients who are infected or colonized with the same pathogen and are suitable roommates^{29, 638, 808, 811-813, 815, 818, 819} *Category IB*
- y If it becomes necessary to place a patient who requires Contact Precautions in a room with a patient who is not infected or colonized with the same infectious agent:
 - o Avoid placing patients on Contact Precautions in the same room with patients who have conditions that may increase the risk of adverse outcome from infection or that may facilitate transmission (e.g., those who are immunocompromised, have open wounds, or have anticipated prolonged lengths of stay). *Category II*
 - o Ensure that patients are physically separated (i.e., >3 feet apart) from each other. Draw the privacy curtain between beds to minimize opportunities for direct contact.) *Category II*
 - o Change protective attire and perform hand hygiene between contact with patients in the same room, regardless of whether one or both patients are on Contact Precautions^{728, 741, 742, 988, 1014, 1015}. *Category IB*

V.B.2.b. In *long-term care and other residential settings*, make decisions regarding patient placement on a case-by-case basis, balancing infection risks to other patients in the room, the presence of risk factors that increase the likelihood of transmission, and the

- potential adverse psychological impact on the infected or colonized patient^{920, 921}. *Category II*
- V.B.2.c. In *ambulatory settings*, place patients who require Contact Precautions in an examination room or cubicle as soon as possible²⁰. *Category II*
- V.B.3. Use of personal protective equipment
- V.B.3.a. Gloves
Wear gloves whenever touching the patient's intact skin^{24, 89, 134, 559, 746, 837} or surfaces and articles in close proximity to the patient (e.g., medical equipment, bed rails)^{72, 73, 88, 837}. Don gloves upon entry into the room or cubicle. *Category IB*
- V.B.3.b. Gowns
- V.B.3.b.i. Wear a gown whenever anticipating that clothing will have direct contact with the patient or potentially contaminated environmental surfaces or equipment in close proximity to the patient. Don gown upon entry into the room or cubicle. Remove gown and observe hand hygiene before leaving the patient-care environment^{24, 88, 134, 745, 837}. *Category IB*
- V.B.3.b.ii. After gown removal, ensure that clothing and skin do not contact potentially contaminated environmental surfaces that could result in possible transfer of microorganism to other patients or environmental surfaces^{72, 73}. *Category II*
- V.B.4. Patient transport
- V.B.4.a. In *acute care hospitals and long-term care and other residential settings*, limit transport and movement of patients outside of the room to medically-necessary purposes. *Category II*
- V.B.4.b. When transport or movement in any healthcare setting is necessary, ensure that infected or colonized areas of the patient's body are contained and covered. *Category II*
- V.B.4.c. Remove and dispose of contaminated PPE and perform hand hygiene prior to transporting patients on Contact Precautions. *Category II*
- V.B.4.d. Don clean PPE to handle the patient at the transport destination. *Category II*
- V.B.5. Patient-care equipment and instruments/devices
- V.B.5.a. Handle patient-care equipment and instruments/devices according to Standard Precautions^{739, 836}. *Category IB/IC*
- V.B.5.b. In *acute care hospitals and long-term care and other residential settings*, use disposable noncritical patient-care equipment (e.g., blood pressure cuffs) or implement patient-dedicated use of such equipment. If common use of equipment for multiple patients is unavoidable, clean and disinfect such equipment before use on another patient^{24, 88, 796, 836, 837, 854, 1016}. *Category IB*
- V.B.5.c. In *home care settings*

- V.B.5.c.i. Limit the amount of non-disposable patient-care equipment brought into the home of patients on Contact Precautions. Whenever possible, leave patient-care equipment in the home until discharge from home care services. *Category II*
- V.B.5.c.ii. If noncritical patient-care equipment (e.g., stethoscope) cannot remain in the home, clean and disinfect items before taking them from the home using a low- to intermediate-level disinfectant. Alternatively, place contaminated reusable items in a plastic bag for transport and subsequent cleaning and disinfection. *Category II*
- V.B.5.d. In *ambulatory settings*, place contaminated reusable noncritical patient-care equipment in a plastic bag for transport to a soiled utility area for reprocessing. *Category II*
- V.B.6. Environmental measures
 - Ensure that rooms of patients on Contact Precautions are prioritized for frequent cleaning and disinfection (e.g., at least daily) with a focus on frequently-touched surfaces (e.g., bed rails, overbed table, bedside commode, lavatory surfaces in patient bathrooms, doorknobs) and equipment in the immediate vicinity of the patient^{11, 24, 88, 746, 837}. *Category IB*
- V.B.7. Discontinue Contact Precautions after signs and symptoms of the infection have resolved or according to pathogen-specific recommendations in Appendix A. *Category IB*
- V.C. Droplet Precautions
 - V.C.1. Use Droplet Precautions as recommended in Appendix A for patients known or suspected to be infected with pathogens transmitted by respiratory droplets (i.e., large-particle droplets >5 μ in size) that are generated by a patient who is coughing, sneezing or talking^{14, 23, Steinberg, 1969 #1708, 41, 95, 103, 111, 112, 755, 756, 989, 1017}. *Category IB*
 - V.C.2. Patient placement
 - V.C.2.a. In acute care hospitals, place patients who require Droplet Precautions in a single-patient room when available *Category II*
When single-patient rooms are in short supply, apply the following principles for making decisions on patient placement:
 - y Prioritize patients who have excessive cough and sputum production for single-patient room placement *Category II*
 - y Place together in the same room (cohort) patients who are infected the same pathogen and are suitable roommates⁸¹⁴
⁸¹⁶. *Category IB*
 - y If it becomes necessary to place patients who require Droplet Precautions in a room with a patient who does not have the same infection:
 - y Avoid placing patients on Droplet Precautions in the same room with patients who have conditions that may increase

- the risk of adverse outcome from infection or that may facilitate transmission (e.g., those who are immunocompromised, have or have anticipated prolonged lengths of stay). *Category II*
- y Ensure that patients are physically separated (i.e., >3 feet apart) from each other. Draw the privacy curtain between beds to minimize opportunities for close contact ^{103, 104, 410}. *Category IB*
 - y Change protective attire and perform hand hygiene between contact with patients in the same room, regardless of whether one patient or both patients are on Droplet Precautions ^{741-743, 988, 1014, 1015}. *Category IB*
- V.C.2.b. In *long-term care and other residential settings*, make decisions regarding patient placement on a case-by-case basis after considering infection risks to other patients in the room and available alternatives ⁴¹⁰. *Category II*
- V.C.2.c. In *ambulatory settings*, place patients who require Droplet Precautions in an examination room or cubicle as soon as possible. Instruct patients to follow recommendations for Respiratory Hygiene/Cough Etiquette ^{447, 448, 9, 828}. *Category II*
- V.C.3. Use of personal protective equipment
- V.C.3.a. Don a mask upon entry into the patient room or cubicle ^{14, 23, 41, 103, 111, 113, 115, 827}. *Category IB*
 - V.C.3.b. No recommendation for routinely wearing eye protection (e.g., goggle or face shield), in addition to a mask, for close contact with patients who require Droplet Precautions. *Unresolved issue*
 - V.C.3.c. For patients with suspected or proven SARS, avian influenza or pandemic influenza, refer to the following websites for the most current recommendations (www.cdc.gov/ncidod/sars/ ; www.cdc.gov/flu/avian/ ; www.pandemicflu.gov/) ^{134, 1018, 1019}
- V.C.4. Patient transport
- V.C.4.a. In *acute care hospitals and long-term care and other residential settings*, limit transport and movement of patients outside of the room to medically-necessary purposes. *Category II*
 - V.C.4.b. If transport or movement in any healthcare setting is necessary, instruct patient to wear a mask and follow Respiratory Hygiene/Cough Etiquette (www.cdc.gov/flu/professionals/infectioncontrol/resphygiene.htm) . *Category IB*
 - V.C.4.c. No mask is required for persons transporting patients on Droplet Precautions. *Category II*
 - V.C.4.d. Discontinue Droplet Precautions after signs and symptoms have resolved or according to pathogen-specific recommendations in Appendix A. *Category IB*
- V.D. Airborne Precautions

- V.D.1. Use Airborne Precautions as recommended in Appendix A for patients known or suspected to be infected with infectious agents transmitted person-to-person by the airborne route (e.g., *M tuberculosis*¹², measles^{34, 122, 1020}, chickenpox^{123, 773, 1021}, disseminated herpes zoster¹⁰²²). *Category IA/IC*
- V.D.2. Patient placement
- V.D.2.a. In *acute care hospitals and long-term care settings*, place patients who require Airborne Precautions in an AIIR that has been constructed in accordance with current guidelines¹¹⁻¹³. *Category IA/IC*
- V.D.2.a.i. Provide at least six (existing facility) or 12 (new construction/renovation) air changes per hour.
- V.D.2.a.ii. Direct exhaust of air to the outside. If it is not possible to exhaust air from an AIIR directly to the outside, the air may be returned to the air-handling system or adjacent spaces if all air is directed through HEPA filters.
- V.D.2.a.iii. Whenever an AIIR is in use for a patient on Airborne Precautions, monitor air pressure daily with visual indicators (e.g., smoke tubes, flutter strips), regardless of the presence of differential pressure sensing devices (e.g., manometers)^{11, 12, 1023, 1024}.
- V.D.2.a.iv. Keep the AIIR door closed when not required for entry and exit.
- V.D.2.b. When an AIIR is not available, transfer the patient to a facility that has an available AIIR¹². *Category II*
- V.D.2.c. In the event of an outbreak or exposure involving large numbers of patients who require Airborne Precautions:
- y Consult infection control professionals before patient placement to determine the safety of alternative room that do not meet engineering requirements for an AIIR.
 - y Place together (cohort) patients who are presumed to have the same infection(based on clinical presentation and diagnosis when known) in areas of the facility that are away from other patients, especially patients who are at increased risk for infection (e.g., immunocompromised patients).
 - y Use temporary portable solutions (e.g., exhaust fan) to create a negative pressure environment in the converted area of the facility. Discharge air directly to the outside, away from people and air intakes, or direct all the air through HEPA filters before it is introduced to other air spaces¹². *Category II*
- V.D.2.d. In *ambulatory settings*:
- V.D.2.d.i. Develop systems (e.g., triage, signage) to identify patients with known or suspected infections that require Airborne Precautions upon entry into ambulatory settings^{9, 12, 34, 127, 134}. *Category IA*

- V.D.2.d.ii. Place the patient in an AIIR as soon as possible. If an AIIR is not available, place a surgical mask on the patient and place him/her in an examination room. Once the patient leaves, the room should remain vacant for the appropriate time, generally one hour, to allow for a full exchange of air ^{11, 12, 122}. *Category IB/IC*
- V.D.2.d.iii. Instruct patients with a known or suspected airborne infection to wear a surgical mask and observe Respiratory Hygiene/Cough Etiquette. Once in an AIIR, the mask may be removed; the mask should remain on if the patient is not in an AIIR ^{12, 107, 145, 899}. *Category IB/IC*
- V.D.3. Personnel restrictions
Restrict susceptible healthcare personnel from entering the rooms of patients known or suspected to have measles (rubeola), varicella (chickenpox), disseminated zoster, or smallpox if other immune healthcare personnel are available ^{17, 775}. *Category IB*
- V.D.4. Use of PPE
 - V.D.4.a. Wear a fit-tested NIOSH-approved N95 or higher level respirator for respiratory protection when entering the room or home of a patient when the following diseases are suspected or confirmed:
 - y Infectious pulmonary or laryngeal tuberculosis or when infectious tuberculosis skin lesions are present and procedures that would aerosolize viable organisms (e.g., irrigation, incision and drainage, whirlpool treatments) are performed ^{12, 1025, 1026}. *Category IB*
 - y Smallpox (vaccinated and unvaccinated). Respiratory protection is recommended for all healthcare personnel, including those with a documented “take” after smallpox vaccination due to the risk of a genetically engineered virus against which the vaccine may not provide protection, or of exposure to a very large viral load (e.g., from high-risk aerosol-generating procedures, immunocompromised patients, hemorrhagic or flat smallpox ^{108, 129}. *Category II*
 - V.D.4.b. No recommendation is made regarding the use of PPE by healthcare personnel who are presumed to be immune to measles (rubeola) or varicella-zoster based on history of disease, vaccine, or serologic testing when caring for an individual with known or suspected measles, chickenpox or disseminated zoster, due to difficulties in establishing definite immunity ^{1027, 1028}. *Unresolved issue*
 - V.D.4.c. No recommendation is made regarding the type of personal protective equipment (i.e., surgical mask or respiratory protection with a N95 or higher respirator) to be worn by susceptible healthcare personnel who must have contact with patients with known or suspected measles, chickenpox or disseminated herpes zoster. *Unresolved issue*

- V.D.5. Patient transport
- V.D.5.a. In *acute care hospitals and long-term care and other residential settings*, limit transport and movement of patients outside of the room to medically-necessary purposes. *Category II*
 - V.D.5.b. If transport or movement outside an AIIR is necessary, instruct patients to wear a surgical mask, if possible, and observe Respiratory Hygiene/Cough Etiquette ¹². *Category II*
 - V.D.5.c. For patients with skin lesions associated with varicella or smallpox or draining skin lesions caused by *M. tuberculosis*, cover the affected areas to prevent aerosolization or contact with the infectious agent in skin lesions ^{108, 1025, 1026, 1029-1031}. *Category IB*
 - V.D.5.d. Healthcare personnel transporting patients who are on Airborne Precautions do not need to wear a mask or respirator during transport if the patient is wearing a mask and infectious skin lesions are covered. *Category II*
- V.D.6. Exposure management
 Immunize or provide the appropriate immune globulin to susceptible persons as soon as possible following unprotected contact (i.e., exposed) to a patient with measles, varicella or smallpox: *Category IA*
- y Administer measles vaccine to exposed susceptible persons within 72 hours after the exposure or administer immune globulin within six days of the exposure event for high-risk persons in whom vaccine is contraindicated ^{17, 1032-1035}.
 - y Administer varicella vaccine to exposed susceptible persons within 120 hours after the exposure or administer varicella immune globulin (VZIG or alternative product), when available, within 96 hours for high-risk persons in whom vaccine is contraindicated (e.g., immunocompromised patients, pregnant women, newborns whose mother's varicella onset was <5 days before or within 48 hours after delivery ^{888, 1035-1037}).
 - y Administer smallpox vaccine to exposed susceptible persons within 4 days after exposure ^{108, 1038-1040}.
- V.D.7. Discontinue Airborne Precautions according to pathogen-specific recommendations in Appendix A. *Category IB*
- V.D.8. Consult CDC's "Guidelines for Preventing the Transmission of *Mycobacterium tuberculosis* in Health-Care Settings, 2005" ¹² and the "Guideline for Environmental Infection Control in Health-Care Facilities" ¹¹ for additional guidance on environment strategies for preventing transmission of tuberculosis in healthcare settings. The environmental recommendations in these guidelines may be applied to patients with other infections that require Airborne Precautions.

- VI. Protective Environment (Table 4)**
- VI.A. Place allogeneic hematopoietic stem cell transplant (HSCT) patients in a Protective Environment as described in the “Guideline to Prevent Opportunistic Infections in HSCT Patients”¹⁵, the “Guideline for Environmental Infection Control in Health-Care Facilities”¹¹, and the “Guidelines for Preventing Health-Care-Associated Pneumonia, 2003”¹⁴ to reduce exposure to environmental fungi (e.g., *Aspergillus* sp)^{157, 158}.
Category IB
- VI.B. No recommendation for placing patients with other medical conditions that are associated with increased risk for environmental fungal infections (e.g., aspergillosis) in a Protective Environment¹¹. *Unresolved issue*
- VI.C. For patients who require a Protective Environment, implement the following (see Table 5)^{11, 15}
- VI.C.1. Environmental controls
- VI.C.1.a. Filtered incoming air using central or point-of-use high efficiency particulate (HEPA) filters capable of removing 99.97% of particles $\geq 0.3 \mu\text{m}$ in diameter¹³. *Category IB*
- VI.C.1.b. Directed room airflow with the air supply on one side of the room that moves air across the patient bed and out through an exhaust on the opposite side of the room¹³. *Category IB*
- VI.C.1.c. Positive air pressure in room relative to the corridor (pressure differential of ≥ 12.5 Pa [0.01-in water gauge])¹³. *Category IB*
- VI.C.1.c.i. Monitor air pressure daily with visual indicators (e.g., smoke tubes, flutter strips)^{11, 1024}. *Category IA*
- VI.C.1.d. Well-sealed rooms that prevent infiltration of outside air¹³.
Category IB
- VI.C.1.e. At least 12 air changes per hour¹³. *Category IB*
- VI.C.2. Lower dust levels by using smooth, nonporous surfaces and finishes that can be scrubbed, rather than textured material (e.g., upholstery). Wet dust horizontal surfaces whenever dust detected and routinely clean crevices and sprinkler heads where dust may accumulate^{940, 941}. *Category II*
- VI.C.3. Avoid carpeting in hallways and patient rooms in areas⁹⁴¹.
Category IB
- VI.C.4. Prohibit dried and fresh flowers and potted plants⁹⁴²⁻⁹⁴⁴. *Category II*
- VI.D. Minimize the length of time that patients who require a Protective Environment are outside their rooms for diagnostic procedures and other activities^{11, 158, 945}. *Category IB*
- VI.E. During periods of construction, to prevent inhalation of respirable particles that could contain infectious spores, provide respiratory protection (e.g., N95 respirator) to patients who are medically fit to tolerate a respirator when they are required to leave the Protective Environment^{945 158}.
Category II
- VI.E.1.a. No recommendation for fit-testing of patients who are using respirators. *Unresolved issue*

- VI.E.1.b. No recommendation for use of particulate respirators when leaving the Protective Environment in the absence of construction. *Unresolved issue*
- VI.F. Use of Standard and Transmission-Based Precautions in a Protective Environment.
 - VI.F.1. Use Standard Precautions as recommended for all patient interactions. *Category IA*
 - VI.F.2. Implement Droplet and Contact Precautions as recommended for diseases listed in Appendix A. Transmission-Based precautions for viral infections may need to be prolonged because of the patient's immunocompromised state and prolonged shedding of viruses^{930 1010 928, 932 1011}. *Category IB*
 - VI.F.3. Barrier precautions, (e.g., masks, gowns, gloves) are not required for healthcare personnel in the absence of suspected or confirmed infection in the patient or if they are not indicated according to Standard Precautions¹⁵. *Category II*
 - VI.F.4. Implement Airborne Precautions for patients who require a Protective Environment room and who also have an airborne infectious disease (e.g., pulmonary or laryngeal tuberculosis, acute varicella-zoster). *Category IA*
 - VI.F.4.a. Ensure that the Protective Environment is designed to maintain positive pressure¹³. *Category IB*
 - VI.F.4.b. Use an anteroom to further support the appropriate air-balance relative to the corridor and the Protective Environment; provide independent exhaust of contaminated air to the outside or place a HEPA filter in the exhaust duct if the return air must be recirculated^{13, 1041}. *Category IB*
 - VI.F.4.c. If an anteroom is not available, place the patient in an AIIR and use portable, industrial-grade HEPA filters in the room to enhance filtration of spores¹⁰⁴². *Category II*

Note: The recommendations in this guideline for Ebola Virus Disease has been superseded by CDC's Infection Prevention and Control Recommendations for Hospitalized Patients with Known or Suspected Ebola Virus Disease in U.S. Hospitals.

This information is in Appendix A.

[Click here for current information on how Ebola virus is transmitted.](#)

Appendix A:

Preamble The mode(s) and risk of transmission for each specific disease agent included in Appendix A were reviewed. Principle sources consulted for the development of disease-specific recommendations for Appendix A included infectious disease manuals and textbooks^{833, 1043, 1044}. The published literature was searched for evidence of person-to-person transmission in healthcare and non-healthcare settings with a focus on reported outbreaks that would assist in developing recommendations for all settings where healthcare is delivered. Criteria used to assign Transmission-Based Precautions categories follow:

- A Transmission-Based Precautions category was assigned if there was strong evidence for person-to-person transmission via droplet, contact, or airborne routes in healthcare or non-healthcare settings and/or if patient factors (e.g., diapered infants, diarrhea, draining wounds) increased the risk of transmission
- Transmission-Based Precautions category assignments reflect the predominant mode(s) of transmission
- If there was no evidence for person-to-person transmission by droplet, contact or airborne routes, Standard Precautions were assigned
- If there was a low risk for person-to-person transmission and no evidence of healthcare-associated transmission, Standard Precautions were assigned
- Standard Precautions were assigned for bloodborne pathogens (e.g., hepatitis B and C viruses, human immunodeficiency virus) as per CDC recommendations for Universal Precautions issued in 1988⁷⁸⁰. Subsequent experience has confirmed the efficacy of Standard Precautions to prevent exposure to infected blood and body fluid^{778, 779, 866}.

Additional information relevant to use of precautions was added in the comments column to assist the caregiver in decision-making. Citations were added as needed to support a change in or provide additional evidence for recommendations for a specific disease and for new infectious agents (e.g., SARS-CoV, avian influenza) that have been added to Appendix A. The reader may refer to more detailed discussion concerning modes of transmission and emerging pathogens in the background text and for MDRO control in Appendix B.

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
Abscess			
Draining, major	C	DI	No dressing or containment of drainage; until drainage stops or can be contained by dressing
Draining, minor or limited	S		Dressing covers and contains drainage
Acquired human immunodeficiency syndrome (HIV)	S		Post-exposure chemoprophylaxis for some blood exposures ⁸⁶⁶ .
Actinomycosis	S		Not transmitted from person to person
Adenovirus infection (see agent-specific guidance under gastroenteritis, conjunctivitis, pneumonia)			
Amebiasis	S		Person to person transmission is rare. Transmission in settings for the mentally challenged and in a family group has been reported ¹⁰⁴⁵ . Use care when handling diapered infants and mentally challenged persons ¹⁰⁴⁶ .
Anthrax	S		Infected patients do not generally pose a transmission risk.
Cutaneous	S		Transmission through non-intact skin contact with draining lesions possible, therefore use Contact Precautions if large amount of uncontained drainage. Handwashing with soap and water preferable to use of waterless alcohol based antiseptics since alcohol does not

¹ Type of Precautions: A, Airborne Precautions; C, Contact; D, Droplet; S, Standard; when A, C, and D are specified, also use S.

[†] Duration of precautions: CN, until off antimicrobial treatment and culture-negative; DI, duration of illness (with wound lesions, DI means until wounds stop draining); DE, until environment completely decontaminated; U, until time specified in hours (hrs) after initiation of effective therapy; Unknown: criteria for establishing eradication of pathogen has not been determined

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
			have sporocidal activity ⁹⁸³ .
Pulmonary	S		Not transmitted from person to person
Environmental: aerosolizable spore-containing powder or other substance		DE	Until decontamination of environment complete ²⁰³ . Wear respirator (N95 mask or PAPRs), protective clothing; decontaminate persons with powder on them (http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5135a3.htm) Hand hygiene: Handwashing for 30-60 seconds with soap and water or 2% chlorhexidine gluconate after spore contact (alcohol handrubs inactive against spores ⁹⁸³ . Post-exposure prophylaxis following environmental exposure: 60 days of antimicrobials (either doxycycline, ciprofloxacin, or levofloxacin) and post-exposure vaccine under IND
Antibiotic-associated colitis (see <i>Clostridium difficile</i>)			
Arthropod-borne viral encephalitides (eastern, western, Venezuelan equine encephalomyelitis; St Louis, California encephalitis; West Nile Virus) and viral fevers (dengue, yellow fever, Colorado tick fever)	S		Not transmitted from person to person except rarely by transfusion, and for West Nile virus by organ transplant, breastmilk or transplacentally ^{530, 1047} . Install screens in windows and doors in endemic areas Use DEET-containing mosquito repellants and clothing to cover extremities
Ascariasis	S		Not transmitted from person to person
Aspergillosis	S		Contact Precautions and Airborne Precautions if massive soft tissue infection with copious drainage and repeated irrigations required ¹⁵⁴ .
Avian influenza (see influenza, avian below)			
Babesiosis	S		Not transmitted from person to person except rarely by transfusion,
Blastomycosis, North American, cutaneous or pulmonary	S		Not transmitted from person to person
Botulism	S		Not transmitted from person to person
Bronchiolitis (see respiratory infections in infants and young children)	C	DI	Use mask according to Standard Precautions.

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
Brucellosis (undulant, Malta, Mediterranean fever)	S		Not transmitted from person to person except rarely via banked spermatozoa and sexual contact ^{1048, 1049} . Provide antimicrobial prophylaxis following laboratory exposure ¹⁰⁵⁰ .
<i>Campylobacter</i> gastroenteritis (see gastroenteritis)			
Candidiasis, all forms including mucocutaneous	S		
Cat-scratch fever (benign inoculation lymphoreticulosis)	S		Not transmitted from person to person
Cellulitis	S		
Chancroid (soft chancre) (<i>H. ducreyi</i>)	S		Transmitted sexually from person to person
Chickenpox (see varicella)			
<i>Chlamydia trachomatis</i>			
Conjunctivitis	S		
Genital (lymphogranuloma venereum)	S		
Pneumonia (infants \leq 3 mos. of age))	S		
<i>Chlamydia pneumoniae</i>	S		Outbreaks in institutionalized populations reported, rarely ^{1051, 1052}
Cholera (see gastroenteritis)			
Closed-cavity infection			
Open drain in place; limited or minor drainage	S		Contact Precautions if there is copious uncontained drainage
No drain or closed drainage system in place	S		
<i>Clostridium</i>			
<i>C. botulinum</i>	S		Not transmitted from person to person
<i>C. difficile</i> (see Gastroenteritis, <i>C. difficile</i>)	C	DI	
<i>C. perfringens</i>			
Food poisoning	S		Not transmitted from person to person
Gas gangrene	S		Transmission from person to person rare; one outbreak in a surgical setting reported ¹⁰⁵³ . Use Contact Precautions if wound drainage is

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
			extensive.
Coccidioidomycosis (valley fever)			
Draining lesions	S		Not transmitted from person to person except under extraordinary circumstances because the infectious arthroconidial form of <i>Coccidioides immitis</i> is not produced in humans ¹⁰⁵⁴ .
Pneumonia	S		Not transmitted from person to person except under extraordinary circumstances, (e.g., inhalation of aerosolized tissue phase endospores during necropsy, transplantation of infected lung) because the infectious arthroconidial form of <i>Coccidioides immitis</i> is not produced in humans ^{1054, 1055} .
Colorado tick fever	S		Not transmitted from person to person
Congenital rubella	C	Until 1 yr of age	Standard Precautions if nasopharyngeal and urine cultures repeatedly neg. after 3 mos. of age
Conjunctivitis			
Acute bacterial	S		
<i>Chlamydia</i>	S		
Gonococcal	S		
Acute viral (acute hemorrhagic)	C	DI	Adenovirus most common; enterovirus 70 ¹⁰⁵⁶ , Coxsackie virus A24 ¹⁰⁵⁷) also associated with community outbreaks. Highly contagious; outbreaks in eye clinics, pediatric and neonatal settings, institutional settings reported. Eye clinics should follow Standard Precautions when handling patients with conjunctivitis. Routine use of infection control measures in the handling of instruments and equipment will prevent the occurrence of outbreaks in this and other settings. ^{460, 814, 1058, 1059 461, 1060} .
Corona virus associated with SARS (SARS-CoV) (see severe acute respiratory syndrome)			

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
Coxsackie virus disease (see enteroviral infection)			
Creutzfeldt-Jakob disease CJD, vCJD	S		Use disposable instruments or special sterilization/disinfection for surfaces, objects contaminated with neural tissue if CJD or vCJD suspected and has not been R/O; No special burial procedures ¹⁰⁶¹
Croup (see respiratory infections in infants and young children)			
Crimean-Congo Fever (see Viral Hemorrhagic Fever)	S		
Cryptococcosis	S		Not transmitted from person to person, except rarely via tissue and corneal transplant ^{1062, 1063}
Cryptosporidiosis (see gastroenteritis)			
Cysticercosis	S		Not transmitted from person to person
Cytomegalovirus infection, including in neonates and immunosuppressed patients	S		No additional precautions for pregnant HCWs
Decubitus ulcer (see Pressure ulcer)			
Dengue fever	S		Not transmitted from person to person
Diarrhea, acute-infective etiology suspected (see gastroenteritis)			
Diphtheria			
Cutaneous	C	CN	Until 2 cultures taken 24 hrs. apart negative
Pharyngeal	D	CN	Until 2 cultures taken 24 hrs. apart negative
Ebola virus (see viral hemorrhagic fevers)			
Echinococcosis (hydatidosis)	S		Not transmitted from person to person
Echovirus (see enteroviral infection)			
Encephalitis or encephalomyelitis (see specific etiologic agents)			
Endometritis (endomyometritis)	S		
Enterobiasis (pinworm disease, oxyuriasis)	S		
<i>Enterococcus</i> species (see multidrug-resistant organisms if			

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
epidemiologically significant or vancomycin resistant)			
Enterocolitis, <i>C. difficile</i> (see <i>C. difficile</i> , gastroenteritis)			
Enteroviral infections (i.e., Group A and B Coxsackie viruses and Echo viruses) (excludes polio virus)	S		Use Contact Precautions for diapered or incontinent children for duration of illness and to control institutional outbreaks
Epiglottitis, due to <i>Haemophilus influenzae</i> type b	D	U 24 hrs	See specific disease agents for epiglottitis due to other etiologies)
Epstein-Barr virus infection, including infectious mononucleosis	S		
Erythema infectiosum (also see Parvovirus B19)			
<i>Escherichia coli</i> gastroenteritis (see gastroenteritis)			
Food poisoning			
Botulism	S		Not transmitted from person to person
<i>C. perfringens</i> or <i>welchii</i>	S		Not transmitted from person to person
Staphylococcal	S		Not transmitted from person to person
Furunculosis, staphylococcal	S		Contact if drainage not controlled. Follow institutional policies if MRSA
Infants and young children	C	DI	
Gangrene (gas gangrene)	S		Not transmitted from person to person
Gastroenteritis	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks for gastroenteritis caused by all of the agents below
Adenovirus	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
<i>Campylobacter</i> species	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
Cholera (<i>Vibrio cholerae</i>)	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
<i>C. difficile</i>	C	DI	Discontinue antibiotics if appropriate. Do not share electronic thermometers ^{853, 854} ; ensure consistent environmental cleaning and

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
			disinfection. Hypochlorite solutions may be required for cleaning if transmission continues ⁸⁴⁷ . Handwashing with soap and water preferred because of the absence of sporicidal activity of alcohol in waterless antiseptic handrubs ⁹⁸³ .
<i>Cryptosporidium species</i>	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
<i>E. coli</i>			
Enteropathogenic O157:H7 and other shiga toxin-producing Strains	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
Other species	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
<i>Giardia lamblia</i>	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
Noroviruses	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks. Persons who clean areas heavily contaminated with feces or vomitus may benefit from wearing masks since virus can be aerosolized from these body substances ^{142, 147 148} ; ensure consistent environmental cleaning and disinfection with focus on restrooms even when apparently unsoiled ^{273, 1064}). Hypochlorite solutions may be required when there is continued transmission ²⁹⁰⁻²⁹² . Alcohol is less active, but there is no evidence that alcohol antiseptic handrubs are not effective for hand decontamination ²⁹⁴ . Cohorting of affected patients to separate airspaces and toilet facilities may help interrupt transmission during outbreaks.
Rotavirus	C	DI	Ensure consistent environmental cleaning and disinfection and frequent removal of soiled diapers. Prolonged shedding may occur in

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
			both immunocompetent and immunocompromised children and the elderly ^{932, 933}
<i>Salmonella</i> species (including <i>S. typhi</i>)	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
<i>Shigella</i> species (Bacillary dysentery)	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
<i>Vibrio parahaemolyticus</i>	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
Viral (if not covered elsewhere)	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
<i>Yersinia enterocolitica</i>	S		Use Contact Precautions for diapered or incontinent persons for the duration of illness or to control institutional outbreaks
German measles (see rubella; see congenital rubella)			
Giardiasis (see gastroenteritis)			
Gonococcal ophthalmia neonatorum (gonorrheal ophthalmia, acute conjunctivitis of newborn)	S		
Gonorrhea	S		
Granuloma inguinale (Donovanosis, granuloma venereum)	S		
Guillain-Barré' syndrome	S		Not an infectious condition
<i>Haemophilus influenzae</i> (see disease-specific recommendations)			
Hand, foot, and mouth disease (see enteroviral infection)			
Hansen's Disease (see Leprosy)			
Hantavirus pulmonary syndrome	S		Not transmitted from person to person
<i>Helicobacter pylori</i>	S		
Hepatitis, viral			
Type A	S		Provide hepatitis A vaccine post-exposure as recommended ¹⁰⁶⁵
Diapered or incontinent patients	C		Maintain Contact Precautions in infants and children <3 years of age

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
			for duration of hospitalization; for children 3-14 yrs. of age for 2 weeks after onset of symptoms; >14 yrs. of age for 1 week after onset of symptoms ^{833, 1066, 1067} .
Type B-HBsAg positive; acute or chronic	S		See specific recommendations for care of patients in hemodialysis centers ⁷⁷⁸
Type C and other unspecified non-A, non-B	S		See specific recommendations for care of patients in hemodialysis centers ⁷⁷⁸
Type D (seen only with hepatitis B)	S		
Type E	S		Use Contact Precautions for diapered or incontinent individuals for the duration of illness ¹⁰⁶⁸
Type G	S		
Herpangina (see enteroviral infection)			
Hookworm	S		
Herpes simplex (<i>Herpesvirus hominis</i>)			
Encephalitis	S		
Mucocutaneous, disseminated or primary, severe	C	Until lesions dry and crusted	
Mucocutaneous, recurrent (skin, oral, genital)	S		
Neonatal	C	Until lesions dry and crusted	Also, for asymptomatic, exposed infants delivered vaginally or by C-section and if mother has active infection and membranes have been ruptured for more than 4 to 6 hrs until infant surface cultures obtained at 24-36 hrs. of age negative after 48 hrs incubation ^{1069, 1070}
Herpes zoster (varicella-zoster) (shingles)			
Disseminated disease in any patient Localized disease in immunocompromised patient until disseminated infection ruled out	A,C	DI	Susceptible HCWs should not enter room if immune caregivers are available; no recommendation for protection of immune HCWs; no recommendation for type of protection, i.e. surgical mask or respirator; for susceptible HCWs.

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
Localized in patient with intact immune system with lesions that can be contained/covered	S	DI	Susceptible HCWs should not provide direct patient care when other immune caregivers are available.
Histoplasmosis	S		Not transmitted from person to person
Human immunodeficiency virus (HIV)	S		Post-exposure chemoprophylaxis for some blood exposures ⁸⁶⁶ .
Human metapneumovirus	C	DI	HAI reported ¹⁰⁷¹ , but route of transmission not established ⁸²³ . Assumed to be Contact transmission as for RSV since the viruses are closely related and have similar clinical manifestations and epidemiology. Wear masks according to Standard Precautions..
Impetigo	C	U 24 hrs	
Infectious mononucleosis	S		
Influenza			
Human (seasonal influenza)			See www.cdc.gov/flu/professionals/infectioncontrol/healthcaresettings.htm for current seasonal influenza guidance.
Avian (e.g., H5N1, H7, H9 strains))			See www.cdc.gov/flu/avian/professional/infect-control.htm for current avian influenza guidance.
Pandemic influenza (also a human influenza virus)	D	5 days from onset of symptoms	See http://www.pandemicflu.gov for current pandemic influenza guidance.
Kawasaki syndrome	S		Not an infectious condition
Lassa fever (see viral hemorrhagic fevers)			

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
Legionnaires' disease	S		Not transmitted from person to person
Leprosy	S		
Leptospirosis	S		Not transmitted from person to person
Lice			http://www.cdc.gov/ncidod/dpd/parasites/lice/default.htm
Head (pediculosis)	C	U 24 hrs	
Body	S		Transmitted person to person through infested clothing. Wear gown and gloves when removing clothing; bag and wash clothes according to CDC guidance above
Pubic	S		Transmitted person to person through sexual contact
Listeriosis (<i>listeria monocytogenes</i>)	S		Person-to-person transmission rare; cross-transmission in neonatal settings reported ^{1072, 1073 1074, 1075}
Lyme disease	S		Not transmitted from person to person
Lymphocytic choriomeningitis	S		Not transmitted from person to person
Lymphogranuloma venereum	S		
Malaria	S		Not transmitted from person to person except through transfusion rarely and through a failure to follow Standard Precautions during patient care ¹⁰⁷⁶⁻¹⁰⁷⁹ . Install screens in windows and doors in endemic areas. Use DEET-containing mosquito repellants and clothing to cover extremities
Marburg virus disease (see viral hemorrhagic fevers)			
Measles (rubeola)	A	4 days after onset of rash; DI in immune compromised	Susceptible HCWs should not enter room if immune care providers are available; no recommendation for face protection for immune HCW; no recommendation for type of face protection for susceptible HCWs, i.e., mask or respirator ^{1027, 1028} . For exposed susceptibles, post-exposure vaccine within 72 hrs. or immune globulin within 6 days when available ^{17, 1032, 1034} . Place exposed susceptible patients on Airborne Precautions and exclude susceptible healthcare personnel

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
			from duty from day 5 after first exposure to day 21 after last exposure, regardless of post-exposure vaccine ¹⁷ .
Melioidosis, all forms	S		Not transmitted from person to person
Meningitis			
Aseptic (nonbacterial or viral; also see enteroviral infections)	S		Contact for infants and young children
Bacterial, gram-negative enteric, in neonates	S		
Fungal	S		
<i>Haemophilus influenzae</i> , type b known or suspected	D	U 24 hrs	
<i>Listeria monocytogenes</i> (See Listeriosis)	S		
<i>Neisseria meningitidis</i> (meningococcal) known or suspected	D	U 24 hrs	See meningococcal disease below
<i>Streptococcus pneumoniae</i>	S		
<i>M. tuberculosis</i>	S		Concurrent, active pulmonary disease or draining cutaneous lesions may necessitate addition of Contact and/or Airborne Precautions; For children, airborne precautions until active tuberculosis ruled out in visiting family members (see tuberculosis below) ⁴²
Other diagnosed bacterial	S		
Meningococcal disease: sepsis, pneumonia, meningitis	D	U 24 hrs	Postexposure chemoprophylaxis for household contacts, HCWs exposed to respiratory secretions; postexposure vaccine only to control outbreaks ^{15, 17} .
<i>Molluscum contagiosum</i>	S		
Monkeypox	A,C	A-Until monkeypox confirmed and smallpox excluded C-Until lesions crusted	Use See www.cdc.gov/ncidod/monkeypox for most current recommendations. Transmission in hospital settings unlikely ²⁶⁹ . Pre- and post-exposure smallpox vaccine recommended for exposed HCWs

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
Mucormycosis	S		
Multidrug-resistant organisms (MDROs), infection or colonization (e.g., MRSA, VRE, VISA/VRSA, ESBLs, resistant <i>S. pneumoniae</i>)	S/C		MDROs judged by the infection control program, based on local, state, regional, or national recommendations, to be of clinical and epidemiologic significance. Contact Precautions recommended in settings with evidence of ongoing transmission, acute care settings with increased risk for transmission or wounds that cannot be contained by dressings. See recommendations for management options in Management of Multidrug-Resistant Organisms In Healthcare Settings, 2006 ⁸⁷⁰ . Contact state health department for guidance regarding new or emerging MDRO.
Mumps (infectious parotitis)	D	U 9 days	After onset of swelling; susceptible HCWs should not provide care if immune caregivers are available. Note: (Recent assessment of outbreaks in healthy 18-24 year olds has indicated that salivary viral shedding occurred early in the course of illness and that 5 days of isolation after onset of parotitis may be appropriate in community settings; however the implications for healthcare personnel and high-risk patient populations remain to be clarified.)
Mycobacteria, nontuberculosis (atypical)			Not transmitted person-to-person
Pulmonary	S		
Wound	S		
<i>Mycoplasma pneumoniae</i>	D	DI	
Necrotizing enterocolitis	S		Contact Precautions when cases clustered temporally ¹⁰⁸⁰⁻¹⁰⁸³ .
Nocardiosis, draining lesions, or other presentations	S		Not transmitted person-to-person
Norovirus (see gastroenteritis)			
Norwalk agent gastroenteritis (see gastroenteritis)			
Orf	S		

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
Parainfluenza virus infection, respiratory in infants and young children	C	DI	Viral shedding may be prolonged in immunosuppressed patients ^{1009, 1010} . Reliability of antigen testing to determine when to remove patients with prolonged hospitalizations from Contact Precautions uncertain.
Parvovirus B19 (Erythema infectiosum)	D		Maintain precautions for duration of hospitalization when chronic disease occurs in an immunocompromised patient. For patients with transient aplastic crisis or red-cell crisis, maintain precautions for 7 days. Duration of precautions for immunosuppressed patients with persistently positive PCR not defined, but transmission has occurred ⁹²⁹ .
Pediculosis (lice)	C	U 24 hrs after treatment	
Pertussis (whooping cough)	D	U 5 days	Single patient room preferred. Cohorting an option. Post-exposure chemoprophylaxis for household contacts and HCWs with prolonged exposure to respiratory secretions ⁸⁶³ . Recommendations for Tdap vaccine in adults under development.
Pinworm infection (Enterobiasis)	S		
Plague (<i>Yersinia pestis</i>)			
Bubonic	S		
Pneumonic	D	U 48 hrs	Antimicrobial prophylaxis for exposed HCW ²⁰⁷ .
Pneumonia			
Adenovirus	D, C	DI	Outbreaks in pediatric and institutional settings reported ^{376, 1084-1086} . In immunocompromised hosts, extend duration of Droplet and Contact Precautions due to prolonged shedding of virus ⁹³¹
Bacterial not listed elsewhere (including gram-negative bacterial)	S		
<i>B. cepacia</i> in patients with CF, including respiratory tract colonization	C	Unknown	Avoid exposure to other persons with CF; private room preferred. Criteria for D/C precautions not established. See CF Foundation guideline ²⁰

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
<i>B. cepacia</i> in patients without CF(see Multidrug-resistant organisms)			
<i>Chlamydia</i>	S		
Fungal	S		
<i>Haemophilus influenzae</i> , type b			
Adults	S		
Infants and children	D	U 24 hrs	
<i>Legionella spp.</i>	S		
Meningococcal	D	U 24 hrs	See meningococcal disease above
Multidrug-resistant bacterial (see multidrug-resistant organisms)			
<i>Mycoplasma</i> (primary atypical pneumonia)	D	DI	
Pneumococcal pneumonia	S		Use Droplet Precautions if evidence of transmission within a patient care unit or facility ^{196-198, 1087}
<i>Pneumocystis jiroveci</i> (<i>Pneumocystis carinii</i>)	S		Avoid placement in the same room with an immunocompromised patient.
<i>Staphylococcus aureus</i>	S		For MRSA, see MDROs
<i>Streptococcus</i> , group A			
Adults	D	U 24 hrs	See streptococcal disease (group A streptococcus) below
Infants and young children	D	U 24 hrs	Contact precautions if skin lesions present
Varicella-zoster (See Varicella-Zoster)			Contact Precautions if skin lesions present
Viral			
Adults	S		
Infants and young children (see respiratory infectious disease, acute, or specific viral agent)			
Poliomyelitis	C	DI	
Pressure ulcer (decubitus ulcer, pressure sore) infected			

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
Major	C	DI	If no dressing or containment of drainage; until drainage stops or can be contained by dressing
Minor or limited	S		If dressing covers and contains drainage
Prion disease (See Creutzfeld-Jacob Disease)			
Psittacosis (ornithosis) (<i>Chlamydia psittaci</i>)	S		Not transmitted from person to person
Q fever	S		
Rabies	S		Person to person transmission rare; transmission via corneal, tissue and organ transplants has been reported ^{539, 1088} . If patient has bitten another individual or saliva has contaminated an open wound or mucous membrane, wash exposed area thoroughly and administer postexposure prophylaxis. ¹⁰⁸⁹
Rat-bite fever (<i>Streptobacillus moniliformis</i> disease, <i>Spirillum minus</i> disease)	S		Not transmitted from person to person
Relapsing fever	S		Not transmitted from person to person
Resistant bacterial infection or colonization (see multidrug-resistant organisms)			
Respiratory infectious disease, acute (if not covered elsewhere)			
Adults	S		
Infants and young children	C	DI	Also see syndromes or conditions listed in Table 2
Respiratory syncytial virus infection, in infants, young children and immunocompromised adults	C	DI	Wear mask according to Standard Precautions ²⁴ CB ^{116, 117} . In immunocompromised patients, extend the duration of Contact Precautions due to prolonged shedding ⁹²⁸). Reliability of antigen testing to determine when to remove patients with prolonged hospitalizations from Contact Precautions uncertain.
Reye's syndrome	S		Not an infectious condition
Rheumatic fever	S		Not an infectious condition
Rhinovirus	D	DI	Droplet most important route of transmission ^{104 1090} . Outbreaks have

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
			occurred in NICUs and LTCFs ^{413, 1091, 1092} . Add Contact Precautions if copious moist secretions and close contact likely to occur (e.g., young infants) ^{111, 833} .
Rickettsial fevers, tickborne (Rocky Mountain spotted fever, tickborne typhus fever)	S		Not transmitted from person to person except through transfusion, rarely
Rickettsialpox (vesicular rickettsiosis)	S		Not transmitted from person to person
Ringworm (dermatophytosis, dermatomycosis, tinea)	S		Rarely, outbreaks have occurred in healthcare settings, (e.g., NICU ¹⁰⁹³ , rehabilitation hospital ¹⁰⁹⁴). Use Contact Precautions for outbreak.
Ritter's disease (staphylococcal scalded skin syndrome)	C	DI	See staphylococcal disease, scalded skin syndrome below
Rocky Mountain spotted fever	S		Not transmitted from person to person except through transfusion, rarely
Roseola infantum (exanthem subitum; caused by HHV-6)	S		
Rotavirus infection (see gastroenteritis)			
Rubella (German measles) (also see congenital rubella)	D	U 7 days after onset of rash	Susceptible HCWs should not enter room if immune caregivers are available. No recommendation for wearing face protection (e.g., a surgical mask) if immune. Pregnant women who are not immune should not care for these patients ^{17, 33} . Administer vaccine within three days of exposure to non-pregnant susceptible individuals. Place exposed susceptible patients on Droplet Precautions; exclude susceptible healthcare personnel from duty from day 5 after first exposure to day 21 after last exposure, regardless of post-exposure vaccine.
Rubeola (see measles)			
Salmonellosis (see gastroenteritis)			
Scabies	C	U 24	
Scalded skin syndrome, staphylococcal	C	DI	See staphylococcal disease, scalded skin syndrome below)
Schistosomiasis (bilharziasis)	S		

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
Severe acute respiratory syndrome (SARS)	A, D,C	DI plus 10 days after resolution of fever, provided respiratory symptoms are absent or improving	Airborne Precautions preferred; D if AIIR unavailable. N95 or higher respiratory protection; surgical mask if N95 unavailable; eye protection (goggles, face shield); aerosol-generating procedures and “supershedders” highest risk for transmission via small droplet nuclei and large droplets ^{93, 94, 96} . Vigilant environmental disinfection (see www.cdc.gov/ncidod/sars)
Shigellosis (see gastroenteritis)			
Smallpox (variola; see vaccinia for management of vaccinated persons)	A,C	DI	Until all scabs have crusted and separated (3-4 weeks). Non-vaccinated HCWs should not provide care when immune HCWs are available; N95 or higher respiratory protection for susceptible and successfully vaccinated individuals; postexposure vaccine within 4 days of exposure protective ^{108, 129, 1038-1040} .
Sporotrichosis	S		
<i>Spirillum minor</i> disease (rat-bite fever)	S		Not transmitted from person to person
Staphylococcal disease (<i>S aureus</i>)			
Skin, wound, or burn			
Major	C	DI	No dressing or dressing does not contain drainage adequately
Minor or limited	S		Dressing covers and contains drainage adequately
Enterocolitis	S		Use Contact Precautions for diapered or incontinent children for duration of illness
Multidrug-resistant (see multidrug-resistant organisms)			
Pneumonia	S		
Scalded skin syndrome	C	DI	Consider healthcare personnel as potential source of nursery, NICU outbreak ¹⁰⁹⁵ .
Toxic shock syndrome	S		
<i>Streptobacillus moniliformis</i> disease (rat-bite fever)	S		Not transmitted from person to person

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
Streptococcal disease (group A streptococcus)			
Skin, wound, or burn			
Major	C,D	U 24 hrs	No dressing or dressing does not contain drainage adequately
Minor or limited	S		Dressing covers and contains drainage adequately
Endometritis (puerperal sepsis)	S		
Pharyngitis in infants and young children	D	U 24 hrs	
Pneumonia	D	U 24 hrs	
Scarlet fever in infants and young children	D	U 24 hrs	
Serious invasive disease	D	U24 hrs	Outbreaks of serious invasive disease have occurred secondary to transmission among patients and healthcare personnel ^{162, 972, 1096-1098} Contact Precautions for draining wound as above; follow rec. for antimicrobial prophylaxis in selected conditions ¹⁶⁰ .
Streptococcal disease (group B streptococcus), neonatal	S		
Streptococcal disease (not group A or B) unless covered elsewhere	S		
Multidrug-resistant (see multidrug-resistant organisms)			
Strongyloidiasis	S		
Syphilis			
Latent (tertiary) and seropositivity without lesions	S		
Skin and mucous membrane, including congenital, primary, Secondary	S		
Tapeworm disease			
<i>Hymenolepis nana</i>	S		Not transmitted from person to person
<i>Taenia solium</i> (pork)	S		
Other	S		
Tetanus	S		Not transmitted from person to person
Tinea (e.g., dermatophytosis, dermatomycosis, ringworm)	S		Rare episodes of person-to-person transmission

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
Toxoplasmosis	S		Transmission from person to person is rare; vertical transmission from mother to child, transmission through organs and blood transfusion rare
Toxic shock syndrome (staphylococcal disease, streptococcal disease)	S		Droplet Precautions for the first 24 hours after implementation of antibiotic therapy if Group A streptococcus is a likely etiology
Trachoma, acute	S		
Transmissible spongiform encephalopathy (see Creutzfeld-Jacob disease, CJD, vCJD)			
Trench mouth (Vincent's angina)	S		
Trichinosis	S		
Trichomoniasis	S		
Trichuriasis (whipworm disease)	S		
Tuberculosis (<i>M. tuberculosis</i>)			
Extrapulmonary, draining lesion)	A,C		Discontinue precautions only when patient is improving clinically, and drainage has ceased or there are three consecutive negative cultures of continued drainage ^{1025, 1026} . Examine for evidence of active pulmonary tuberculosis.
Extrapulmonary, no draining lesion, meningitis	S		Examine for evidence of pulmonary tuberculosis. For infants and children, use Airborne Precautions until active pulmonary tuberculosis in visiting family members ruled out ⁴²
Pulmonary or laryngeal disease, confirmed	A		Discontinue precautions only when patient on effective therapy is improving clinically and has three consecutive sputum smears negative for acid-fast bacilli collected on separate days (MMWR 2005; 54: RR-17 http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5417a1.htm?s_cid=rr5417a1_e) ¹² .
Pulmonary or laryngeal disease, suspected	A		Discontinue precautions only when the likelihood of infectious TB

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
			disease is deemed negligible, and either 1) there is another diagnosis that explains the clinical syndrome or 2) the results of three sputum smears for AFB are negative. Each of the three sputum specimens should be collected 8-24 hours apart, and at least one should be an early morning specimen
Skin-test positive with no evidence of current active disease	S		
Tularemia			
Draining lesion	S		Not transmitted from person to person
Pulmonary	S		Not transmitted from person to person
Typhoid (<i>Salmonella typhi</i>) fever (see gastroenteritis)			
Typhus			
<i>Rickettsia prowazekii</i> (Epidemic or Louse-borne typhus)	S		Transmitted from person to person through close personal or clothing contact
<i>Rickettsia typhi</i>	S		Not transmitted from person to person
Urinary tract infection (including pyelonephritis), with or without urinary catheter	S		
Vaccinia (vaccination site, adverse events following vaccination) *			Only vaccinated HCWs have contact with active vaccination sites and care for persons with adverse vaccinia events; if unvaccinated, only HCWs without contraindications to vaccine may provide care.
Vaccination site care (including autoinoculated areas)	S		Vaccination recommended for vaccinators; for newly vaccinated HCWs: semi-permeable dressing over gauze until scab separates, with dressing change as fluid accumulates, ~3-5 days; gloves, hand hygiene for dressing change; vaccinated HCW or HCW without contraindication to vaccine for dressing changes ^{205, 221, 225} .
Eczema vaccinatum	C	Until lesions dry and crusted, scabs separated	For contact with virus-containing lesions and exudative material
Fetal vaccinia	C		
Generalized vaccinia	C		

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type *	Duration †	Comments
Progressive vaccinia	C		
Postvaccinia encephalitis	S		
Blepharitis or conjunctivitis	S/C		Use Contact Precautions if there is copious drainage
Iritis or keratitis	S		
Vaccinia-associated erythema multiforme (Stevens Johnson Syndrome)	S		Not an infectious condition
Secondary bacterial infection (e.g., <i>S. aureus</i> , group A beta hemolytic streptococcus)	S/C		Follow organism-specific (strep, staph most frequent) recommendations and consider magnitude of drainage
Varicella Zoster	A,C	Until lesions dry and crusted	Susceptible HCWs should not enter room if immune caregivers are available; no recommendation for face protection of immune HCWs; no recommendation for type of protection, i.e. surgical mask or respirator for susceptible HCWs. In immunocompromised host with varicella pneumonia, prolong duration of precautions for duration of illness. Post-exposure prophylaxis: provide post-exposure vaccine ASAP but within 120 hours; for susceptible exposed persons for whom vaccine is contraindicated (immunocompromised persons, pregnant women, newborns whose mother's varicella onset is ≤5days before delivery or within 48 hrs after delivery) provide VZIG, when available, within 96 hours; if unavailable, use IVIG, Use Airborne Precautions for exposed susceptible persons and exclude exposed susceptible healthcare workers beginning 8 days after first exposure until 21 days after last exposure or 28 if received VZIG, regardless of postexposure vaccination. ¹⁰³⁶
Variola (see smallpox)			
<i>Vibrio parahaemolyticus</i> (see gastroenteritis)			
Vincent's angina (trench mouth)	S		
Viral hemorrhagic fevers	S, D, C	DI	Single-patient room preferred. Emphasize: 1) use of sharps safety

APPENDIX A¹

TYPE AND DURATION OF PRECAUTIONS RECOMMENDED FOR SELECTED INFECTIONS AND CONDITIONS

Infection/Condition	Precautions		
	Type [*]	Duration [†]	Comments
due to Lassa, Ebola, Marburg, Crimean-Congo fever viruses			devices and safe work practices, 2) hand hygiene; 3) barrier protection against blood and body fluids upon entry into room (single gloves and fluid-resistant or impermeable gown, face/eye protection with masks, goggles or face shields); and 4) appropriate waste handling. Use N95 or higher respirators when performing aerosol-generating procedures. Largest viral load in final stages of illness when hemorrhage may occur; additional PPE, including double gloves, leg and shoe coverings may be used, especially in resource-limited settings where options for cleaning and laundry are limited. Notify public health officials immediately if Ebola is suspected ^{212, 314, 740, 772} Also see Table 3 for Ebola as a bioterrorism agent
Viral respiratory diseases (not covered elsewhere)			
Adults	S		
Infants and young children (see respiratory infectious disease, acute)			
Whooping cough (see pertussis)			
Wound infections			
Major	C	DI	No dressing or dressing does not contain drainage adequately
Minor or limited	S		Dressing covers and contains drainage adequately
<i>Yersinia enterocolitica</i> gastroenteritis (see gastroenteritis)			
Zoster (varicella-zoster) (see herpes zoster)			
Zygomycosis (phycomycosis, mucormycosis)	S		Not transmitted person-to-person

TABLE 1. HISTORY OF GUIDELINES FOR ISOLATION PRECAUTIONS IN HOSPITALS*

YEAR (Ref)	DOCUMENT ISSUED	COMMENT
1970 1099	Isolation Techniques for Use in Hospitals, 1 st ed.	<ul style="list-style-type: none"> - Introduced seven isolation precaution categories with color-coded cards: Strict, Respiratory, Protective, Enteric, Wound and Skin, Discharge, and Blood - No user decision-making required - Simplicity a strength; over isolation prescribed for some infections
1975 1100	Isolation Techniques for Use in Hospitals, 2 nd ed.	<ul style="list-style-type: none"> - Same conceptual framework as 1st edition
1983 1101	CDC Guideline for Isolation Precautions in Hospitals	<ul style="list-style-type: none"> - Provided two systems for isolation: category-specific and disease-specific - Protective Isolation eliminated; Blood Precautions expanded to include Body Fluids - Categories included Strict, Contact, Respiratory, AFB, Enteric, Drainage/Secretion, Blood and Body Fluids - Emphasized decision-making by users
1985-88 780, 896	Universal Precautions	<ul style="list-style-type: none"> - Developed in response to HIV/AIDS epidemic - Dictated application of Blood and Body Fluid precautions to all patients, regardless of infection status - Did not apply to feces, nasal secretions, sputum, sweat, tears, urine, or vomitus unless contaminated by visible blood - Added personal protective equipment to protect HCWs from mucous membrane exposures - Handwashing recommended immediately after glove removal - Added specific recommendations for handling needles and other sharp devices; concept became integral to OSHA's 1991 rule on occupational exposure to blood-borne pathogens in healthcare settings

<p>1987 1102</p>	<p>Body Substance Isolation</p>	<ul style="list-style-type: none"> - Emphasized avoiding contact with all moist and potentially infectious body substances except sweat even if blood not present - Shared some features with Universal Precautions - Weak on infections transmitted by large droplets or by contact with dry surfaces - Did not emphasize need for special ventilation to contain airborne infections - Handwashing after glove removal not specified in the absence of visible soiling
<p>1996 1</p>	<p>Guideline for Isolation Precautions in Hospitals</p>	<ul style="list-style-type: none"> - Prepared by the Healthcare Infection Control Practices Advisory Committee (HICPAC) - Melded major features of Universal Precautions and Body Substance Isolation into Standard Precautions to be used with all patients at all times - Included three transmission-based precaution categories: airborne, droplet, and contact - Listed clinical syndromes that should dictate use of empiric isolation until an etiological diagnosis is established

* Derived from Garner ICHE 1996

TABLE 2. CLINICAL SYNDROMES OR CONDITIONS WARRANTING EMPIRIC TRANSMISSION-BASED PRECAUTIONS IN ADDITION TO STANDARD PRECAUTIONS PENDING CONFIRMATION OF DIAGNOSIS*

Clinical Syndrome or Condition†	Potential Pathogens‡	Empiric Precautions (Always includes Standard Precautions)
DIARRHEA		
Acute diarrhea with a likely infectious cause in an incontinent or diapered patient	Enteric pathogens§	Contact Precautions (pediatrics and adult)
MENINGITIS		
	<i>Neisseria meningitidis</i>	Droplet Precautions for first 24 hrs of antimicrobial therapy; mask and face protection for intubation
	Enteroviruses	Contact Precautions for infants and children
	<i>M. tuberculosis</i>	Airborne Precautions if pulmonary infiltrate Airborne Precautions plus Contact Precautions if potentially infectious draining body fluid present
RASH OR EXANTHEMS, GENERALIZED, ETIOLOGY UNKNOWN		
Petechial/ecchymotic with fever (general) - If positive history of travel to an area with an ongoing outbreak of VHF in the 10 days before onset of fever	<i>Neisseria meningitides</i> Ebola, Lassa, Marburg viruses	Droplet Precautions for first 24 hrs of antimicrobial therapy Droplet Precautions plus Contact Precautions, with face/eye protection, emphasizing safety sharps and barrier precautions when blood exposure likely. Use N95 or higher respiratory protection when aerosol-generating procedure performed

Vesicular	Varicella-zoster, <i>herpes simplex</i> , variola (smallpox), vaccinia viruses Vaccinia virus	Airborne plus Contact Precautions; Contact Precautions only if <i>herpes simplex</i> , localized zoster in an immunocompetent host or vaccinia viruses most likely
Maculopapular with cough, coryza and fever	Rubeola (measles) virus	Airborne Precautions

Clinical Syndrome or Condition†	Potential Pathogens‡	Empiric Precautions (Always includes Standard Precautions)
RESPIRATORY INFECTIONS		
Cough/fever/upper lobe pulmonary infiltrate in an HIV-negative patient or a patient at low risk for human immunodeficiency virus (HIV) infection	<i>M. tuberculosis</i> , Respiratory viruses, <i>S. pneumoniae</i> , <i>S. aureus</i> (MSSA or MRSA)	Airborne Precautions plus Contact precautions
Cough/fever/pulmonary infiltrate in any lung location in an HIV-infected patient or a patient at high risk for HIV infection	<i>M. tuberculosis</i> , Respiratory viruses, <i>S. pneumoniae</i> , <i>S. aureus</i> (MSSA or MRSA)	Airborne Precautions plus Contact Precautions Use eye/face protection if aerosol-generating procedure performed or contact with respiratory secretions anticipated. If tuberculosis is unlikely and there are no AIIRs and/or respirators available, use Droplet Precautions instead of Airborne Precautions Tuberculosis more likely in HIV-infected individual than in HIV negative individual
Cough/fever/pulmonary infiltrate in any lung location in a patient with a history of recent travel (10-21 days) to countries with active outbreaks of SARS, avian influenza	<i>M. tuberculosis</i> , severe acute respiratory syndrome virus (SARS-CoV), avian influenza	Airborne plus Contact Precautions plus eye protection. If SARS and tuberculosis unlikely, use Droplet Precautions instead of Airborne Precautions.
Respiratory infections, particularly bronchiolitis and pneumonia, in infants and young children	Respiratory syncytial virus, parainfluenza virus, adenovirus, influenza virus, Human metapneumovirus	Contact plus Droplet Precautions; Droplet Precautions may be discontinued when adenovirus and influenza have been ruled out

Skin or Wound Infection

Abscess or draining wound that cannot be covered	<i>Staphylococcus aureus</i> (MSSA or MRSA), group A streptococcus	Contact Precautions Add Droplet Precautions for the first 24 hours of appropriate antimicrobial therapy if invasive Group A streptococcal disease is suspected
--------------------------------------------------	--------------------------------------------------------------------	-------------------------------------------------------------------------------------------------------------------------------------------------------------------

- * Infection control professionals should modify or adapt this table according to local conditions. To ensure that appropriate empiric precautions are implemented always, hospitals must have systems in place to evaluate patients routinely according to these criteria as part of their preadmission and admission care.
- † Patients with the syndromes or conditions listed below may present with atypical signs or symptoms (e.g. neonates and adults with pertussis may not have paroxysmal or severe cough). The clinician's index of suspicion should be guided by the prevalence of specific conditions in the community, as well as clinical judgment.
- ‡ The organisms listed under the column "Potential Pathogens" are not intended to represent the complete, or even most likely, diagnoses, but rather possible etiologic agents that require additional precautions beyond Standard Precautions until they can be ruled out.
- § These pathogens include enterohemorrhagic *Escherichia coli* O157:H7, *Shigella spp*, hepatitis A virus, noroviruses, rotavirus, *C. difficile*.

TABLE 3.
INFECTION CONTROL CONSIDERATIONS FOR HIGH-PRIORITY (CDC CATEGORY A) DISEASES THAT MAY RESULT FROM BIOTERRORIST ATTACKS OR ARE CONSIDERED TO BE BIOTERRORIST THREATS

(www.bt.cdc.gov)^a

^a Abbreviations used in this table: RT = respiratory tract; GIT = gastrointestinal tract; CXR = chest x-ray; CT = computerized axial tomography; CSF = cerebrospinal fluid; and LD₅₀ – lethal dose for 50% of experimental animals; HCWs = healthcare worker; BSL = biosafety level; PAPR = powered air purifying respirator; PCR = polymerase chain reaction; IHC = immunohistochemistry

Disease	Anthrax
Site(s) of Infection; Transmission Mode Cutaneous and inhalation disease have occurred in past bioterrorist incidents	Cutaneous (contact with spores); RT (inhalation of spores); GIT (ingestion of spores - rare) Comment: Spores can be inhaled into the lower respiratory tract. The infectious dose of <i>B. anthracis</i> in humans by any route is not precisely known. In primates, the LD ₅₀ (i.e., the dose required to kill 50% of animals) for an aerosol challenge with <i>B. anthracis</i> is estimated to be 8,000–50,000 spores; the infectious dose may be as low as 1-3 spores
Incubation Period	Cutaneous: 1 to 12 days; RT: Usually 1 to 7 days but up to 43 days reported; GIT: 15-72 hours
Clinical Features	Cutaneous: Painless, reddish papule, which develops a central vesicle or bulla in 1-2 days; over next 3-7 days lesion becomes pustular, and then necrotic, with black eschar; extensive surrounding edema. RT: initial flu-like illness for 1-3 days with headache, fever, malaise, cough; by day 4 severe dyspnea and shock, and is usually fatal (85%-90% if untreated; meningitis in 50% of RT cases). GIT: ; if intestinal form, necrotic, ulcerated edematous lesions develop in intestines with fever, nausea and vomiting, progression to hematemesis and bloody diarrhea; 25-60% fatal
Diagnosis	Cutaneous: Swabs of lesion (under eschar) for IHC, PCR and culture; punch biopsy for IHC, PCR and culture; vesicular fluid aspirate for Gram stain and culture; blood culture if systemic symptoms; acute and

	<p>convalescent sera for ELISA serology</p> <p>RT: CXR or CT demonstrating wide mediastinal widening and/or pleural effusion, hilar abnormalities; blood for culture and PCR; pleural effusion for culture, PCR and IHC; CSF if meningeal signs present for IHC, PCR and culture; acute and convalescent sera for ELISA serology; pleural and/or bronchial biopsies IHC.</p> <p>GIT: blood and ascites fluid, stool samples, rectal swabs, and swabs of oropharyngeal lesions if present for culture, PCR and IHC</p>
Infectivity	<p>Cutaneous: Person-to-person transmission from contact with lesion of untreated patient possible, but extremely rare.</p> <p>RT and GIT: Person-to-person transmission does not occur.</p> <p>Aerosolized powder, environmental exposures: Highly infectious if aerosolized</p>
Recommended Precautions	<p>Cutaneous: Standard Precautions; Contact Precautions if uncontained copious drainage.</p> <p>RT and GIT: Standard Precautions.</p> <p>Aerosolized powder, environmental exposures: Respirator (N95 mask or PAPRs), protective clothing; decontamination of persons with powder on them (http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5135a3.htm)</p> <p>Hand hygiene: Handwashing for 30-60 seconds with soap and water or 2% chlorhexidine gluconate after spore contact (alcohol handrubs inactive against spores [Weber DJ JAMA 2003; 289:1274]).</p> <p>Post-exposure prophylaxis following environmental exposure: 60 days of antimicrobials (either doxycycline, ciprofloxacin, or levofloxacin) and post-exposure vaccine under IND</p>

Disease	Botulism
Site(s) of Infection; Transmission Mode	<p>GIT: Ingestion of toxin-containing food, RT: Inhalation of toxin containing aerosol cause disease.</p> <p>Comment: Toxin ingested or potentially delivered by aerosol in bioterrorist incidents. LD₅₀ for type A is 0.001 µg/ml/kg.</p>
Incubation Period	1-5 days.
Clinical Features	Ptosis, generalized weakness, dizziness, dry mouth and throat, blurred vision, diplopia, dysarthria, dysphonia, and dysphagia followed by symmetrical descending paralysis and respiratory failure.

Diagnosis	Clinical diagnosis; identification of toxin in stool, serology unless toxin-containing material available for toxin neutralization bioassays.
Infectivity	Not transmitted from person to person. Exposure to toxin necessary for disease.
Recommended Precautions	Standard Precautions.
Disease	Ebola Hemorrhagic Fever
Site(s) of Infection; Transmission Mode	As a rule infection develops after exposure of mucous membranes or RT, or through broken skin or percutaneous injury.
Incubation Period	2-19 days, usually 5-10 days
Clinical Features	Febrile illnesses with malaise, myalgias, headache, vomiting and diarrhea that are rapidly complicated by hypotension, shock, and hemorrhagic features. Massive hemorrhage in < 50% pts.
Diagnosis	Etiologic diagnosis can be made using RT-PCR, serologic detection of antibody and antigen, pathologic assessment with immunohistochemistry and viral culture with EM confirmation of morphology,
Infectivity	Person-to-person transmission primarily occurs through unprotected contact with blood and body fluids; percutaneous injuries (e.g., needlestick) associated with a high rate of transmission; transmission in healthcare settings has been reported but is prevented by use of barrier precautions.
Recommended Precautions	Hemorrhagic fever specific barrier precautions: If disease is believed to be related to intentional release of a bioweapon, epidemiology of transmission is unpredictable pending observation of disease transmission. Until the nature of the pathogen is understood and its transmission pattern confirmed, Standard, Contact and Airborne Precautions should be used. Once the pathogen is characterized, if the epidemiology of transmission is consistent with natural disease, Droplet Precautions can be substituted for Airborne Precautions. Emphasize: 1) use of sharps safety devices and safe work practices, 2) hand hygiene; 3) barrier protection against blood and body fluids upon entry into room (single gloves and fluid-resistant or impermeable gown, face/eye protection with masks, goggles or face shields); and 4) appropriate waste handling. Use N95 or higher respirators when performing aerosol-generating procedures. In settings where AIIRs are unavailable or the large numbers of patients cannot be accommodated by existing AIIRs, observe Droplet Precautions (plus Standard Precautions and Contact Precautions) and segregate patients from those not suspected of VHF infection. Limit blood draws to those essential to care. See text for discussion and Appendix A for recommendations for naturally

	occurring VHF.s.
--	------------------

Disease	Plague ²
Site(s) of Infection; Transmission Mode	RT: Inhalation of respiratory droplets. Comment: Pneumonic plague most likely to occur if used as a biological weapon, but some cases of bubonic and primary septicemia may also occur. Infective dose 100 to 500 bacteria
Incubation Period	1 to 6, usually 2 to 3 days.
Clinical Features	Pneumonic: fever, chills, headache, cough, dyspnea, rapid progression of weakness, and in a later stage hemoptysis, circulatory collapse, and bleeding diathesis
Diagnosis	Presumptive diagnosis from Gram stain or Wayson stain of sputum, blood, or lymph node aspirate; definitive diagnosis from cultures of same material, or paired acute/convalescent serology.
Infectivity	Person-to-person transmission occurs via respiratory droplets risk of transmission is low during first 20-24 hours of illness and requires close contact. Respiratory secretions probably are not infectious within a few hours after initiation of appropriate therapy.
Recommended Precautions	Standard Precautions, Droplet Precautions until patients have received 48 hours of appropriate therapy. Chemoprophylaxis: Consider antibiotic prophylaxis for HCWs with close contact exposure.

² Pneumonic plague is not as contagious as is often thought. Historical accounts and contemporary evidence indicate that persons with plague usually only transmit the infection when the disease is in the end stage. These persons cough copious amounts of bloody sputum that contains many plague bacteria. Patients in the early stage of primary pneumonic plague (approximately the first 20–24 h) apparently pose little risk [1, 2]. Antibiotic medication rapidly clears the sputum of plague bacilli, so that a patient generally is not infective within hours after initiation of effective antibiotic treatment [3]. This means that in modern times many patients will never reach a stage where they pose a significant risk to others. Even in the end stage of disease, transmission only occurs after close contact. Simple protective measures, such as wearing masks, good hygiene, and avoiding close contact, have been effective to interrupt transmission during many pneumonic plague outbreaks [2]. In the United States, the last known cases of person to person transmission of pneumonic plague occurred in 1925 [2].

1. Wu L-T. A treatise on pneumonic plague. Geneva: League of Nations, 1926. III. Health.
2. Kool JL. Risk of person to person transmission of pneumonic plague. *Clinical Infectious Diseases*, 2005; 40 (8): 1166-1172
3. Butler TC. Plague and other Yersinia infections. In: Greenough WB, ed. *Current topics in infectious disease*. New York: Plenum Medical Book Company, 1983.

Disease	Smallpox
Site(s) of Infection; Transmission Mode	RT Inhalation of droplet or, rarely, aerosols; and skin lesions (contact with virus). Comment: If used as a biological weapon, natural disease, which has not occurred since 1977, will likely result.
Incubation Period	7 to 19 days (mean 12 days)
Clinical Features	Fever, malaise, backache, headache, and often vomiting for 2-3 days; then generalized papular or maculopapular rash (more on face and extremities), which becomes vesicular (on day 4 or 5) and then pustular; lesions all in same stage.
Diagnosis	Electron microscopy of vesicular fluid or culture of vesicular fluid by WHO approved laboratory (CDC); detection by PCR available only in select LRN labs, CDC and USAMRID
Infectivity	Secondary attack rates up to 50% in unvaccinated persons; infected persons may transmit disease from time rash appears until all lesions have crusted over (about 3 weeks); greatest infectivity during first 10 days of rash.
Recommended Precautions	Combined use of Standard, Contact, and Airborne Precautions ^b until all scabs have separated (3-4 weeks). Only immune HCWs to care for pts; post-exposure vaccine within 4 days. Vaccinia: HCWs cover vaccination site with gauze and semi-permeable dressing until scab separates (≥ 21 days). Observe hand hygiene. Adverse events with virus-containing lesions: Standard plus Contact Precautions until all lesions crusted

^b Transmission by the airborne route is a rare event; Airborne Precautions is recommended when possible, but in the event of mass exposures, barrier precautions and containment within a designated area are most important^{204, 212}.

^c Vaccinia adverse events with lesions containing infectious virus include inadvertent autoinoculation, ocular lesions (blepharitis, conjunctivitis), generalized vaccinia, progressive vaccinia, eczema vaccinatum; bacterial superinfection also requires addition of contact precautions if exudates cannot be contained^{216, 217}.

Disease	Tularemia
Site(s) of Infection; Transmission Mode	RT: Inhalation of aerosolized bacteria. GIT: Ingestion of food or drink contaminated with aerosolized bacteria. Comment: Pneumonic or typhoidal disease likely to occur after bioterrorist event using aerosol delivery. Infective dose 10-50 bacteria
Incubation Period	2 to 10 days, usually 3 to 5 days
Clinical Features	Pneumonic: malaise, cough, sputum production, dyspnea; Typhoidal: fever, prostration, weight loss and frequently an associated pneumonia.
Diagnosis	Diagnosis usually made with serology on acute and convalescent serum specimens; bacterium can be detected by PCR (LRN) or isolated from blood and other body fluids on cysteine-enriched media or mouse inoculation.
Infectivity	Person-to-person spread is rare. Laboratory workers who encounter/handle cultures of this organism are at high risk for disease if exposed.
Recommended Precautions	Standard Precautions

TABLE 4.
RECOMMENDATIONS FOR APPLICATION OF STANDARD PRECAUTIONS FOR THE CARE OF ALL PATIENTS IN ALL HEALTHCARE SETTINGS
(See Sections II.D.-II.J. and III.A.1)

COMPONENT	RECOMMENDATIONS
Hand hygiene	After touching blood, body fluids, secretions, excretions, contaminated items; immediately after removing gloves; between patient contacts.
Personal protective equipment (PPE)	
Gloves	For touching blood, body fluids, secretions, excretions, contaminated items; for touching mucous membranes and nonintact skin
Gown	During procedures and patient-care activities when contact of clothing/exposed skin with blood/body fluids, secretions, and excretions is anticipated..
Mask, eye protection (goggles), face shield*	During procedures and patient-care activities likely to generate splashes or sprays of blood, body fluids, secretions, especially suctioning, endotracheal intubation
Soiled patient-care equipment	Handle in a manner that prevents transfer of microorganisms to others and to the environment; wear gloves if visibly contaminated; perform hand hygiene.
Environmental control	Develop procedures for routine care, cleaning, and disinfection of environmental surfaces, especially frequently touched surfaces in patient-care areas.
Textiles and laundry	Handle in a manner that prevents transfer of microorganisms to others and to the environment
Needles and other sharps	Do not recap, bend, break, or hand-manipulate used needles; if recapping is required, use a one-handed scoop technique only; use safety features when available; place used sharps in puncture-resistant container
Patient resuscitation	Use mouthpiece, resuscitation bag, other ventilation devices to prevent contact with mouth and oral secretions

Patient placement	Prioritize for single-patient room if patient is at increased risk of transmission, is likely to contaminate the environment, does not maintain appropriate hygiene, or is at increased risk of acquiring infection or developing adverse outcome following infection.
Respiratory hygiene/cough etiquette (source containment of infectious respiratory secretions in symptomatic patients, beginning at initial point of encounter e.g., triage and reception areas in emergency departments and physician offices)	Instruct symptomatic persons to cover mouth/nose when sneezing/coughing; use tissues and dispose in no-touch receptacle; observe hand hygiene after soiling of hands with respiratory secretions; wear surgical mask if tolerated or maintain spatial separation, >3 feet if possible.

* * During aerosol-generating procedures on patients with suspected or proven infections transmitted by respiratory aerosols (e.g., SARS), wear a fit-tested N95 or higher respirator in addition to gloves, gown, and face/eye protection.

TABLE 5. COMPONENTS OF A PROTECTIVE ENVIRONMENT

(Adapted from MMWR 2003; 52 [RR-10])

I. Patients: allogeneic hematopoietic stem cell transplant (HSCT) only

- Maintain in PE room except for required diagnostic or therapeutic procedures that cannot be performed in the room, e.g. radiology, operating room
- Respiratory protection e.g., N95 respirator, for the patient when leaving PE during periods of construction

II. Standard and Expanded Precautions

- Hand hygiene observed before and after patient contact
- Gown, gloves, mask NOT required for HCWs or visitors for routine entry into the room
- Use of gown, gloves, mask by HCWs and visitors according to Standard Precautions and as indicated for suspected or proven infections for which Transmission-Based Precautions are recommended

III. Engineering

- Central or point-of-use HEPA (99.97% efficiency) filters capable of removing particles 0.3 μm in diameter for supply (incoming) air
- Well-sealed rooms
 - Proper construction of windows, doors, and intake and exhaust ports
 - Ceilings: smooth, free of fissures, open joints, crevices
 - Walls sealed above and below the ceiling
 - If leakage detected, locate source and make necessary repairs
- Ventilation to maintain ≥ 12 ACH
- Directed air flow: air supply and exhaust grills located so that clean, filtered air enters from one side of the room, flows across the patient's bed, exits on opposite side of the room
- Positive room air pressure in relation to the corridor
 - Pressure differential of >2.5 Pa [0.01" water gauge]
- Monitor and document results of air flow patterns daily using visual methods (e.g., flutter strips, smoke tubes) or a hand held pressure gauge
- Self-closing door on all room exits
- Maintain back-up ventilation equipment (e.g., portable units for fans or filters) for emergency provision of ventilation requirements for PE areas and take immediate steps to restore the fixed ventilation system
- For patients who require both a PE and Airborne Infection Isolation, use an anteroom to ensure proper air balance relationships and provide independent exhaust of contaminated air to the outside or place a HEPA filter in the exhaust duct. If an anteroom is not available, place patient in an AIIR and use portable ventilation units, industrial-grade HEPA filters to enhance filtration of spores.

IV. Surfaces

- Daily wet-dusting of horizontal surfaces using cloths moistened with EPA-registered hospital disinfectant/detergent
- Avoid dusting methods that disperse dust
- No carpeting in patient rooms or hallways
- No upholstered furniture and furnishings

V. Other

- No flowers (fresh or dried) or potted plants in PE rooms or areas
- Use vacuum cleaner equipped with HEPA filters when vacuum cleaning is necessary

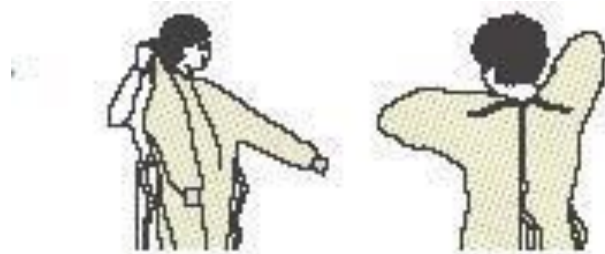
Figure.

Example of Safe Donning and Removal of Personal Protective Equipment (PPE)

DONNING PPE

GOWN

- Fully cover torso from neck to knees, arms to end of wrist, and wrap around the back
- Fasten in back at neck and waist



MASK OR RESPIRATOR

- Secure ties or elastic band at middle of head and neck
- Fit flexible band to nose bridge
- Fit snug to face and below chin
- Fit-check respirator



GOGGLES/FACE SHIELD

- Put on face and adjust to fit



GLOVES

- Use non-sterile for isolation
- Select according to hand size
- Extend to cover wrist of isolation gown



SAFE WORK PRACTICES

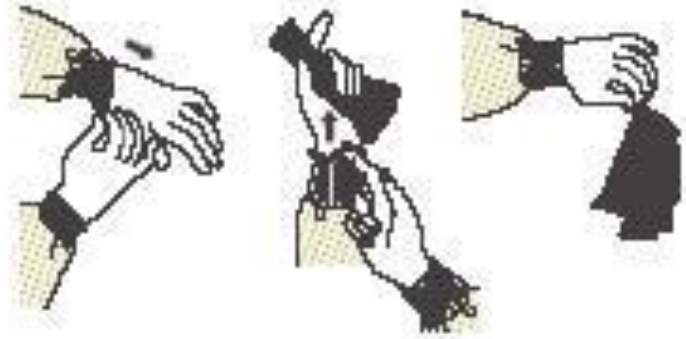
- Keep hands away from face
- Work from clean to dirty
- Limit surfaces touched
- Change when torn or heavily contaminated
- Perform hand hygiene

REMOVING PPE

Remove PPE at doorway before leaving patient room or in anteroom

GLOVES

- Outside of gloves are contaminated!
- Grasp outside of glove with opposite gloved hand; peel off
- Hold removed glove in gloved hand
- Slide fingers of ungloved hand under remaining glove at wrist



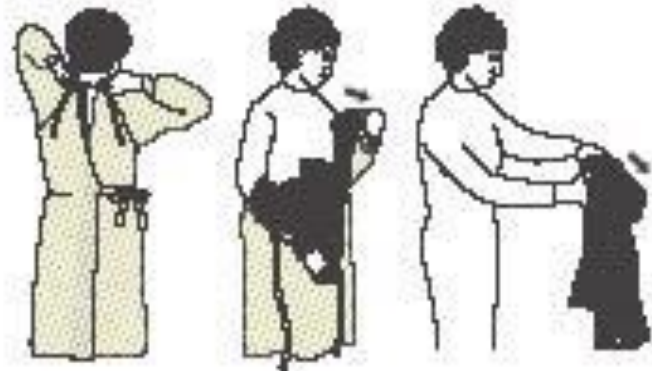
GOGGLES/FACE SHIELD

- Outside of goggles or face shield are contaminated!
- To remove, handle by “clean” head band or ear pieces
- Place in designated receptacle for reprocessing or in waste container



GOWN

- Gown front and sleeves are contaminated!
- Unfasten neck, then waist ties
- Remove gown using a peeling motion; pull gown from each shoulder toward the same hand
- Gown will turn inside out
- Hold removed gown away from body, roll into a bundle and discard into waste or linen receptacle



MASK OR RESPIRATOR

- Front of mask/respirator is contaminated – DO NOT TOUCH!
- Grasp ONLY bottom then top ties/elastics and remove
- Discard in waste container



HAND HYGIENE

Perform hand hygiene immediately after removing all PPE!

GLOSSARY

Airborne infection isolation room (AIIR). Formerly, negative pressure isolation room, an AIIR is a single-occupancy patient-care room used to isolate persons with a suspected or confirmed airborne infectious disease. Environmental factors are controlled in AIIRs to minimize the transmission of infectious agents that are usually transmitted from person to person by droplet nuclei associated with coughing or aerosolization of contaminated fluids. AIIRs should provide negative pressure in the room (so that air flows under the door gap into the room); **and** an air flow rate of 6-12 ACH (6 ACH for existing structures, 12 ACH for new construction or renovation); **and** direct exhaust of air from the room to the outside of the building or recirculation of air through a HEPA filter before retruning to circulation (MMWR 2005; 54 [RR-17]).

American Institute of Architects (AIA). A professional organization that develops standards for building ventilation, The “2001 Guidelines for Design and Construction of Hospital and Health Care Facilities”, the development of which was supported by the AIA, Academy of Architecture for Health, Facilities Guideline Institute, with assistance from the U.S. Department of Health and Human Services and the National Institutes of Health, is the primary source of guidance for creating airborne infection isolation rooms (AIIRs) and protective environments (www.aia.org/aah).

Ambulatory care settings. Facilities that provide health care to patients who do not remain overnight (e.g., hospital-based outpatient clinics, nonhospital-based clinics and physician offices, urgent care centers, surgicenters, free-standing dialysis centers, public health clinics, imaging centers, ambulatory behavioral health and substance abuse clinics, physical therapy and rehabilitation centers, and dental practices).

Bioaerosols. An airborne dispersion of particles containing whole or parts of biological entities, such as bacteria, viruses, dust mites, fungal hyphae, or fungal spores. Such aerosols usually consist of a mixture of mono-dispersed and aggregate cells, spores or viruses, carried by other materials, such as respiratory secretions and/or inert particles. Infectious bioaerosols (i.e., those that contain biological agents capable of causing an infectious disease) can be generated from human sources (e.g., expulsion from the respiratory tract during coughing, sneezing, talking or singing; during suctioning or wound irrigation), wet environmental sources (e.g. HVAC and cooling tower water with Legionella) or dry sources (e.g., construction dust with spores produced by *Aspergillus* spp.). Bioaerosols include large respiratory droplets and small droplet nuclei (Cole EC. AJIC 1998;26: 453-64).

Caregivers. All persons who are not employees of an organization, are not paid, and provide or assist in providing healthcare to a patient (e.g., family member, friend) and acquire technical training as needed based on the tasks that must be performed.

Cohorting. In the context of this guideline, this term applies to the practice of grouping patients infected or colonized with the same infectious agent together to confine their care to one area and prevent contact with susceptible patients (cohorting patients). During outbreaks, healthcare personnel may be assigned to a cohort of patients to further limit opportunities for transmission (cohorting staff).

Colonization. Proliferation of microorganisms on or within body sites without detectable host immune response, cellular damage, or clinical expression. The presence of a microorganism within a host may occur with varying duration, but may become a source of potential transmission. In many instances, colonization and carriage are synonymous.

Droplet nuclei. Microscopic particles < 5 µm in size that are the residue of evaporated droplets and are produced when a person coughs, sneezes, shouts, or sings. These particles can remain suspended in the air for prolonged periods of time and can be carried on normal air currents in a room or beyond, to adjacent spaces or areas receiving exhaust air.

Engineering controls. Removal or isolation of a workplace hazard through technology. AllRs, a Protective Environment, engineered sharps injury prevention devices and sharps containers are examples of engineering controls.

Epidemiologically important pathogens . Infectious agents that have one or more of the following characteristics: 1) are readily transmissible; 2) have a proclivity toward causing outbreaks; 3) may be associated with a severe outcome; or 4) are difficult to treat. Examples include *Acinetobacter sp.*, *Aspergillus sp.*, *Burkholderia cepacia*, *Clostridium difficile*, *Klebsiella* or *Enterobacter sp.*, extended-spectrum-beta-lactamase producing gram negative bacilli [ESBLs], methicillin-resistant *Staphylococcus aureus* [MRSA], *Pseudomonas aeruginosa*, vancomycin-resistant enterococci [VRE], methicillin resistant *Staphylococcus aureus* [MRSA], vancomycin resistant *Staphylococcus aureus* [VRSA] influenza virus, respiratory syncytial virus [RSV], rotavirus, SARS-CoV, noroviruses and the hemorrhagic fever viruses).

Hand hygiene. A general term that applies to any one of the following: 1) handwashing with plain (nonantimicrobial) soap and water); 2) antiseptic handwash (soap containing antiseptic agents and water); 3) antiseptic handrub (waterless antiseptic product, most often alcohol-based, rubbed on all surfaces of hands); or 4) surgical hand antisepsis (antiseptic handwash or antiseptic handrub performed preoperatively by surgical personnel to eliminate transient hand flora and reduce resident hand flora)⁵⁵⁹.

Healthcare-associated infection (HAI). An infection that develops in a patient who is cared for in any setting where healthcare is delivered (e.g., acute care hospital, chronic care facility, ambulatory clinic, dialysis center, surgicenter, home) and is related to receiving health care (i.e., was not incubating or present at the time healthcare was provided). In ambulatory and home settings, HAI would apply to any infection that is associated with a medical or surgical intervention. Since the geographic location of infection acquisition is often uncertain, the preferred term is considered to be *healthcare-associated* rather than *healthcare-acquired*.

Healthcare epidemiologist. A person whose primary training is medical (M.D., D.O.) and/or masters or doctorate-level epidemiology who has received advanced training in healthcare epidemiology. Typically these professionals direct or provide consultation to an infection control program in a hospital, long term care facility (LTCF), or healthcare delivery system (also see infection control professional).

Healthcare personnel, healthcare worker (HCW). All paid and unpaid persons who work in a healthcare setting (e.g. any person who has professional or technical training in a healthcare-related field and provides patient care in a healthcare setting or any person who provides services that support the delivery of healthcare such as dietary, housekeeping, engineering, maintenance personnel).

Hematopoietic stem cell transplantation (HSCT). Any transplantation of blood- or bone marrow-derived hematopoietic stem cells, regardless of donor type (e.g., allogeneic or autologous) or cell source (e.g., bone marrow, peripheral blood, or placental/umbilical cord blood); associated with periods of severe immunosuppression that vary with the source of the cells, the intensity of chemotherapy required, and the presence of graft versus host disease (MMWR 2000; 49: RR-10).

High-efficiency particulate air (HEPA) filter. An air filter that removes >99.97% of particles $\geq 0.3\mu\text{m}$ (the most penetrating particle size) at a specified flow rate of air. HEPA filters may be integrated into the central air handling systems, installed at the point of use above the ceiling of a room, or used as portable units (MMWR 2003; 52: RR-10).

Home care. A wide-range of medical, nursing, rehabilitation, hospice and social services delivered to patients in their place of residence (e.g., private residence, senior living center, assisted living facility). Home health-care services include care provided by home health aides and skilled nurses, respiratory therapists, dietitians, physicians, chaplains, and volunteers; provision of durable medical equipment; home infusion therapy; and physical, speech, and occupational therapy.

Immunocompromised patients. Those patients whose immune mechanisms are deficient because of congenital or acquired immunologic disorders (e.g., human immunodeficiency virus [HIV] infection, congenital immune deficiency syndromes), chronic diseases such as diabetes mellitus, cancer, emphysema, or cardiac failure, ICU care, malnutrition, and immunosuppressive therapy of another disease process [e.g., radiation, cytotoxic chemotherapy, anti-graft• rejection medication, corticosteroids, monoclonal antibodies directed against a specific component of the immune system]). The type of infections for which an immunocompromised patient has increased susceptibility is determined by the severity of immunosuppression and the specific component(s) of the immune system that is affected. Patients undergoing allogeneic HSCT and those with chronic graft versus host disease are considered the most vulnerable to HAIs. Immunocompromised states also make it more difficult to diagnose certain infections (e.g., tuberculosis) and are associated with more severe clinical disease states than persons with the same infection and a normal immune system.

Infection. The transmission of microorganisms into a host after evading or overcoming defense mechanisms, resulting in the organism's proliferation and invasion within host tissue(s). Host responses to infection may include clinical symptoms or may be subclinical, with manifestations of disease mediated by direct organisms pathogenesis and/or a function of cell-mediated or antibody responses that result in the destruction of host tissues.

Infection control and prevention professional (ICP). A person whose primary training is in either nursing, medical technology, microbiology, or epidemiology and who has acquired special training in infection control. Responsibilities may include collection, analysis, and feedback of infection data and trends to healthcare providers; consultation on infection risk assessment, prevention and control strategies; performance of education and training activities; implementation of evidence-based infection control practices or those mandated by regulatory and licensing agencies; application of epidemiologic principles to improve patient outcomes; participation in planning renovation and construction projects (e.g., to ensure appropriate containment of construction dust); evaluation of new products or procedures on patient outcomes; oversight of employee health services related to infection prevention; implementation of preparedness plans; communication within the healthcare setting, with local and state health departments, and with the community at large concerning infection control issues; and participation in research. Certification in infection control (CIC) is available through the Certification Board of Infection Control and Epidemiology.

Infection control and prevention program. A multidisciplinary program that includes a group of activities to ensure that recommended practices for the prevention of healthcare-associated infections are implemented and followed by HCWs, making the healthcare setting safe from infection for patients and

healthcare personnel. The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) requires the following five components of an infection control program for accreditation: 1) *surveillance*: monitoring patients and healthcare personnel for acquisition of infection and/or colonization; 2) *investigation*: identification and analysis of infection problems or undesirable trends; 3) *prevention*: implementation of measures to prevent transmission of infectious agents and to reduce risks for device- and procedure-related infections; 4) *control*: evaluation and management of outbreaks; and 5) *reporting*: provision of information to external agencies as required by state and federal law and regulation (www.jcaho.org). The infection control program staff has the ultimate authority to determine infection control policies for a healthcare organization with the approval of the organization's governing body.

Long-term care facilities (LTCFs). An array of residential and outpatient facilities designed to meet the bio-psychosocial needs of persons with sustained self-care deficits. These include skilled nursing facilities, chronic disease hospitals, nursing homes, foster and group homes, institutions for the developmentally disabled, residential care facilities, assisted living facilities, retirement homes, adult day health care facilities, rehabilitation centers, and long-term psychiatric hospitals.

Mask. A term that applies collectively to items used to cover the nose and mouth and includes both procedure masks and surgical masks (www.fda.gov/cdrh/ode/guidance/094.html#4).

Multidrug-resistant organisms (MDROs). In general, bacteria that are resistant to one or more classes of antimicrobial agents and usually are resistant to all but one or two commercially available antimicrobial agents (e.g., MRSA, VRE, extended spectrum beta-lactamase [ESBL]-producing or intrinsically resistant gram-negative bacilli) ¹⁷⁶.

Nosocomial infection. A term that is derived from two Greek words "nosos" (disease) and "komeion" (to take care of) and refers to any infection that develops during or as a result of an admission to an acute care facility (hospital) and was not incubating at the time of admission.

Personal protective equipment (PPE). A variety of barriers used alone or in combination to protect mucous membranes, skin, and clothing from contact with infectious agents. PPE includes gloves, masks, respirators, goggles, face shields, and gowns.

Procedure Mask. A covering for the nose and mouth that is intended for use in general patient care situations. These masks generally attach to the face with ear loops rather than ties or elastic. Unlike surgical masks, procedure masks are not regulated by the Food and Drug Administration.

Protective Environment. A specialized patient-care area, usually in a hospital, that has a positive air flow relative to the corridor (i.e., air flows from the room to the outside adjacent space). The combination of high-efficiency particulate air (HEPA) filtration, high numbers (≥ 12) of air changes per hour (ACH), and minimal leakage of air into the room creates an environment that can safely accommodate patients with a severely compromised immune system (e.g., those who have received allogeneic hemopoietic stem-cell transplant [HSCT]) and decrease the risk of exposure to spores produced by environmental fungi. Other components include use of scrubbable surfaces instead of materials such as upholstery or carpeting, cleaning to prevent dust accumulation, and prohibition of fresh flowers or potted plants.

Quasi-experimental studies. Studies to evaluate interventions but do not use randomization as part of the study design. These studies are also referred to as nonrandomized, pre-post-intervention study designs. These studies aim to demonstrate causality between an intervention and an outcome but cannot achieve the level of confidence concerning attributable benefit obtained through a randomized, controlled trial. In hospitals and public health settings, randomized control trials often cannot be implemented due to ethical, practical and urgency reasons; therefore, quasi-experimental design studies are used commonly. However, even if an intervention appears to be effective statistically, the question can be raised as to the possibility of alternative explanations for the result.. Such study design is used when it is not logistically feasible or ethically possible to conduct a randomized, controlled trial, (e.g., during outbreaks). Within the classification of quasi-experimental study designs, there is a hierarchy of design features that may contribute to validity of results (Harris et al. CID 2004:38: 1586).

Residential care setting. A facility in which people live, minimal medical care is delivered, and the psychosocial needs of the residents are provided for.

Respirator. A personal protective device worn by healthcare personnel to protect them from inhalation exposure to airborne infectious agents that are $< 5 \mu\text{m}$ in size. These include infectious droplet nuclei from patients with *M. tuberculosis*, variola virus [smallpox], SARS-CoV), and dust particles that contain infectious particles, such as spores of environmental fungi (e.g., *Aspergillus* sp.). The CDC's National Institute for Occupational Safety and Health (NIOSH) certifies respirators used in healthcare settings (www.cdc.gov/niosh/topics/respirators/). The N95 disposable particulate, air purifying, respirator is the type used most commonly by healthcare personnel. Other respirators used include N-99 and N-100 particulate respirators, powered air-purifying respirators (PAPRS) with high efficiency filters; and non-powered full-facepiece elastomeric negative pressure respirators. A listing of NIOSH-approved respirators can be found at www.cdc.gov/niosh/npptl/respirators/disp_part/particlist.html. Respirators must be used in conjunction with a complete Respiratory Protection Program, as

required by the Occupational Safety and Health Administration (OSHA), that includes fit testing, training, proper selection of respirators, medical clearance and respirator maintenance.

Respiratory Hygiene/ Cough Etiquette. A combination of measures designed to minimize the transmission of respiratory pathogens via droplet or airborne routes in healthcare settings. The components of Respiratory Hygiene/Cough Etiquette are 1) covering the mouth and nose during coughing and sneezing, 2) using tissues to contain respiratory secretions with prompt disposal into a no-touch receptacle, 3) offering a surgical mask to persons who are coughing to decrease contamination of the surrounding environment, and 4) turning the head away from others and maintaining spatial separation, ideally >3 feet, when coughing. These measures are targeted to all patients with symptoms of respiratory infection and their accompanying family members or friends beginning at the point of initial encounter with a healthcare setting (e.g., reception/triage in emergency departments, ambulatory clinics, healthcare provider offices) ¹²⁶ (Srinivasin A ICHE 2004; 25: 1020; www.cdc.gov/flu/professionals/infectioncontrol/resphygiene.htm).

Safety culture/climate. The shared perceptions of workers and management regarding the expectations of safety in the work environment. A hospital safety climate includes the following six organizational components: 1) senior management support for safety programs; 2) absence of workplace barriers to safe work practices; 3) cleanliness and orderliness of the worksite; 4) minimal conflict and good communication among staff members; 5) frequent safety-related feedback/training by supervisors; and 6) availability of PPE and engineering controls ⁶²⁰.

Source Control. The process of containing an infectious agent either at the portal of exit from the body or within a confined space. The term is applied most frequently to containment of infectious agents transmitted by the respiratory route but could apply to other routes of transmission, (e.g., a draining wound, vesicular or bullous skin lesions). Respiratory Hygiene/Cough Etiquette that encourages individuals to “cover your cough” and/or wear a mask is a source control measure. The use of enclosing devices for local exhaust ventilation (e.g., booths for sputum induction or administration of aerosolized medication) is another example of source control.

Standard Precautions. A group of infection prevention practices that apply to all patients, regardless of suspected or confirmed diagnosis or presumed infection status. Standard Precautions is a combination and expansion of Universal Precautions ⁷⁸⁰ and Body Substance Isolation ¹¹⁰². Standard Precautions is based on the principle that all blood, body fluids, secretions, excretions except sweat, nonintact skin, and mucous membranes may contain transmissible infectious agents. Standard Precautions includes hand hygiene, and depending on the anticipated exposure, use of gloves, gown, mask, eye protection, or face shield. Also, equipment or items in the patient environment

likely to have been contaminated with infectious fluids must be handled in a manner to prevent transmission of infectious agents, (e.g. wear gloves for handling, contain heavily soiled equipment, properly clean and disinfect or sterilize reusable equipment before use on another patient).

Surgical mask. A device worn over the mouth and nose by operating room personnel during surgical procedures to protect both surgical patients and operating room personnel from transfer of microorganisms and body fluids. Surgical masks also are used to protect healthcare personnel from contact with large infectious droplets (>5 μm in size). According to draft guidance issued by the Food and Drug Administration on May 15, 2003, surgical masks are evaluated using standardized testing procedures for fluid resistance, bacterial filtration efficiency, differential pressure (air exchange), and flammability in order to mitigate the risks to health associated with the use of surgical masks. These specifications apply to any masks that are labeled surgical, laser, isolation, or dental or medical procedure_ (www.fda.gov/cdrh/ode/guidance/094.html#4). Surgical masks do not protect against inhalation of small particles or droplet nuclei and should not be confused with particulate respirators that are recommended for protection against selected airborne infectious agents, (e.g., *Mycobacterium tuberculosis*).

References:

1. Garner JS. Guideline for isolation precautions in hospitals. The Hospital Infection Control Practices Advisory Committee. *Infect Control Hosp Epidemiol* 1996;17(1):53-80.(s).
2. Harris AD, Bradham DD, Baumgarten M, Zuckerman IH, Fink JC, Perencevich EN. The use and interpretation of quasi-experimental studies in infectious diseases. *Clin Infect Dis* 2004;38(11):1586-91.
3. Morton V, Torgerson DJ. Effect of regression to the mean on decision making in health care. *Bmj* 2003;326(7398):1083-4.
4. Pocock SJ, Elbourne DR. Randomized trials or observational tribulations? *N Engl J Med* 2000;342(25):1907-9.
5. Ioannidis JP, Haidich AB, Pappa M, et al. Comparison of evidence of treatment effects in randomized and nonrandomized studies. *Jama* 2001;286(7):821-30.
6. Bent S, Shojania KG, Saint S. The use of systematic reviews and meta-analyses in infection control and hospital epidemiology. *Am J Infect Control* 2004;32(4):246-54.
7. Harris AD, Lautenbach E, Perencevich E. A systematic review of quasi-experimental study designs in the fields of infection control and antibiotic resistance. *Clin Infect Dis* 2005;41(1):77-82.
8. Evans R, Lloyd JF, Abouzelof RH, Taylor CW, Anderson VR, Samore MH. System-wide Surveillance for Clinical Encounters by Patients Previously Identified with MRSA and VRE. *Medinfo* 2004;2004:212-6.
9. Srinivasan A, McDonald LC, Jernigan D, et al. Foundations of the severe acute respiratory syndrome preparedness and response plan for healthcare facilities. *Infect Control Hosp Epidemiol* 2004;25(12):1020-5.
10. www.cdc.gov/flu/avian/professional/infect-control.htm
11. CDC. Guidelines for Environmental Infection Control in Health-Care Facilities. Recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). *MMWR* 2003;52(RR10);1-42.
12. CDC. Guidelines for preventing the transmission of Mycobacterium tuberculosis in health-care settings, 2005. *MMWR Recomm Rep* 2005;54(17):1-141.
13. AIA. Guidelines for Design and Construction of Hospital and Health Care Facilities. In: American Institute of Architects. Washington, DC: American Institute of Architects Press; 2006.
14. CDC. Guidelines for Preventing Health-Care-Associated Pneumonia, 2003. Recommendations of CDC and the Healthcare Infection Control

- Practices Advisory Committee. MMWR Recomm Rep 2004;53(RR-3):1-40.
15. CDC. Guidelines for preventing opportunistic infections among hematopoietic stem cell transplant recipients. Recommendations of CDC, the Infectious Disease Society of America, and the American Society of Blood and Marrow Transplantation. MMWR - Morbidity & Mortality Weekly Report 2000;49(RR-10):1-125.
 16. Boyce JM, Pittet D. Guideline for Hand Hygiene in Health-Care Settings. Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force. Society for Healthcare Epidemiology of America/Association for Professionals in Infection Control/Infectious Diseases Society of America. MMWR Recomm Rep 2002;51(RR-16):1-45, quiz CE1-4.
 17. Bolyard EA, Tablan OC, Williams WW, Pearson ML, Shapiro CN, Deitchmann SD. Guideline for infection control in healthcare personnel, 1998. Hospital Infection Control Practices Advisory Committee. Infect Control Hosp Epidemiol 1998;19(6):407-63.
 18. CDC. Recommendations for Preventing Transmission of Infections Among Chronic Hemodialysis Patients. MMWR 2001;50(RR05):1-43.
 19. Kohn WG, Collins AS, Cleveland JL, Harte JA, Eklund KJ, Malvitz DM. Guidelines for infection control in dental health-care settings--2003. MMWR Recomm Rep 2003;52(RR-17):1-61.
 20. Saiman L, Siegel J. Infection control recommendations for patients with cystic fibrosis: microbiology, important pathogens, and infection control practices to prevent patient-to-patient transmission. Infect Control Hosp Epidemiol 2003;24(5 Suppl):S6-52.
 21. Varia M, Wilson S, Sarwal S, et al. Investigation of a nosocomial outbreak of severe acute respiratory syndrome (SARS) in Toronto, Canada. Cmaj 2003;169(4):285-92.
 22. Haley RW, Cushion NB, Tenover FC, et al. Eradication of endemic methicillin-resistant Staphylococcus aureus infections from a neonatal intensive care unit. J Infect Dis 1995;171(3):614-24.
 23. Bridges CB, Kuehnert MJ, Hall CB. Transmission of influenza: implications for control in health care settings. Clin Infect Dis 2003;37(8):1094-101.
 24. Hall CB. Nosocomial respiratory syncytial virus infections: the "Cold War" has not ended. Clin Infect Dis 2000;31(2):590-6.
 25. Campbell JR, Zaccaria E, Mason EO, Jr., Baker CJ. Epidemiological analysis defining concurrent outbreaks of Serratia marcescens and methicillin-resistant Staphylococcus aureus in a neonatal intensive-care unit. Infect Control Hosp Epidemiol 1998;19(12):924-8.

26. Pena C, Pujol M, Ardanuy C, et al. Epidemiology and successful control of a large outbreak due to *Klebsiella pneumoniae* producing extended-spectrum beta-lactamases. *Antimicrob Agents Chemother* 1998;42(1):53-8.
27. Bonten MJ, Slaughter S, Ambergen AW, et al. The role of "colonization pressure" in the spread of vancomycin-resistant enterococci: an important infection control variable. *Arch Intern Med* 1998;158(10):1127-32.
28. Jensenius M, Ringertz SH, Berild D, Bell H, Espinoza R, Grinde B. Prolonged nosocomial outbreak of hepatitis A arising from an alcoholic with pneumonia. *Scand J Infect Dis* 1998;30(2):119-23.
29. Zawacki A, O'Rourke E, Potter-Bynoe G, Maccone A, Harbarth S, Goldmann D. An outbreak of *Pseudomonas aeruginosa* pneumonia and bloodstream infection associated with intermittent otitis externa in a healthcare worker. *Infect Control Hosp Epidemiol* 2004;25(12):1083-9.
30. Foca M, Jakob K, Whittier S, et al. Endemic *Pseudomonas aeruginosa* infection in a neonatal intensive care unit. *N Engl J Med* 2000;343(10):695-700.
31. Gupta A, Della-Latta P, Todd B, et al. Outbreak of extended-spectrum beta-lactamase-producing *Klebsiella pneumoniae* in a neonatal intensive care unit linked to artificial nails. *Infect Control Hosp Epidemiol* 2004;25(3):210-5.
32. Boyce JM, Opal SM, Potter-Bynoe G, Medeiros AA. Spread of methicillin-resistant *Staphylococcus aureus* in a hospital after exposure to a health care worker with chronic sinusitis. *Clin Infect Dis* 1993;17(3):496-504.
33. Fliegel PE, Weinstein WM. Rubella outbreak in a prenatal clinic: management and prevention. *Am J Infect Control* 1982;10(1):29-33.
34. Atkinson WL, Markowitz LE, Adams NC, Seastrom GR. Transmission of measles in medical settings--United States, 1985-1989. *Am J Med* 1991;91(3B):320S-4S.
35. Carman WF, Elder AG, Wallace LA, et al. Effects of influenza vaccination of health-care workers on mortality of elderly people in long-term care: a randomised controlled trial. *Lancet* 2000;355(9198):93-7.
36. CDC. Outbreaks of pertussis associated with hospitals--Kentucky, Pennsylvania, and Oregon, 2003. *MMWR Morb Mortal Wkly Rep* 2005;54(3):67-71.
37. Mermel LA, McKay M, Dempsey J, Parenteau S. *Pseudomonas* surgical-site infections linked to a healthcare worker with onychomycosis. *Infect Control Hosp Epidemiol* 2003;24(10):749-52.
38. Barnes GL, Callaghan SL, Kirkwood CD, Bogdanovic-Sakran N, Johnston LJ, Bishop RF. Excretion of serotype G1 rotavirus strains by

- asymptomatic staff: a possible source of nosocomial infection. *J Pediatr* 2003;142(6):722-5.
39. Wang JT, Chang SC, Ko WJ, et al. A hospital-acquired outbreak of methicillin-resistant *Staphylococcus aureus* infection initiated by a surgeon carrier. *J Hosp Infect* 2001;47(2):104-9.
 40. Valenti WM, Pincus PH, Messner MK. Nosocomial pertussis: possible spread by a hospital visitor. *Am J Dis Child* 1980;134(5):520-1.
 41. Christie CD, Glover AM, Willke MJ, Marx ML, Reising SF, Hutchinson NM. Containment of pertussis in the regional pediatric hospital during the Greater Cincinnati epidemic of 1993. *Infect Control Hosp Epidemiol* 1995;16(10):556-63.
 42. Munoz FM, Ong LT, Seavy D, Medina D, Correa A, Starke JR. Tuberculosis among adult visitors of children with suspected tuberculosis and employees at a children's hospital. *Infect Control Hosp Epidemiol* 2002;23(10):568-72.
 43. Garcia R, Raad I, Abi-Said D, et al. Nosocomial respiratory syncytial virus infections: prevention and control in bone marrow transplant patients. *Infect Control Hosp Epidemiol* 1997;18(6):412-6.
 44. Whimbey E, Champlin RE, Couch RB, et al. Community respiratory virus infections among hospitalized adult bone marrow transplant recipients. *Clin Infect Dis* 1996;22(5):778-82.
 45. Saiman L, O'keefe M, Graham PL, et al. Hospital transmission of community-acquired methicillin-resistant *Staphylococcus aureus* among postpartum women. *Clin Infect Dis* 2003;37(10):1313-9.
 46. Bonten MJ, Slaughter S, Hayden MK, Nathan C, van Voorhis J, Weinstein RA. External sources of vancomycin-resistant enterococci for intensive care units. *Crit Care Med* 1998;26(12):2001-4.
 47. Flynn DM, Weinstein RA, Nathan C, Gaston MA, Kabins SA. Patients' endogenous flora as the source of "nosocomial" *Enterobacter* in cardiac surgery. *J Infect Dis* 1987;156(2):363-8.
 48. Olson B, Weinstein RA, Nathan C, Chamberlin W, Kabins SA. Epidemiology of endemic *Pseudomonas aeruginosa*: why infection control efforts have failed. *J Infect Dis* 1984;150(6):808-16.
 49. Perl TM, Cullen JJ, Wenzel RP, et al. Intranasal mupirocin to prevent postoperative *Staphylococcus aureus* infections. *N Engl J Med* 2002;346(24):1871-7.
 50. Toltzis P, Hoyen C, et al. Factors that predict preexisting colonization with antibiotic-resistant gram-negative bacilli in patients admitted to a pediatric intensive care unit. *Pediatrics* 1999;103 (4 Pt1):719-23.

51. Sarginson RE, Taylor N, Reilly N, Baines PB, Van Saene HK. Infection in prolonged pediatric critical illness: A prospective four-year study based on knowledge of the carrier state. *Crit Care Med* 2004;32(3):839-47.
52. Silvestri L, Monti Bragadin C, Milanese M, et al. Are most ICU infections really nosocomial? A prospective observational cohort study in mechanically ventilated patients. *J Hosp Infect* 1999;42(2):125-33.
53. Heggens JP, Phillips LG, Boertman JA, et al. The epidemiology of methicillin-resistant *Staphylococcus aureus* in a burn center. *J Burn Care Rehabil* 1988;9(6):610-2.
54. Donskey CJ. The role of the intestinal tract as a reservoir and source for transmission of nosocomial pathogens. *Clin Infect Dis* 2004;39(2):219-26.
55. Osterholm MT, Hedberg CW, Moore KA. The epidemiology of infectious diseases. . In: G.L. M, Jr DRG, J.E. B, eds. *Principles and Practice of Infectious Diseases*. 5th ed ed. Philadelphia: Churchill Livingstone; 2000:161-3.
56. Thomsen RW, Hundborg HH, Lervang HH, Johnsen SP, Schonheyder HC, Sorensen HT. Risk of community-acquired pneumococcal bacteremia in patients with diabetes: a population-based case-control study. *Diabetes Care* 2004;27(5):1143-7.
57. Carton JA, Maradona JA, Nuno FJ, Fernandez-Alvarez R, Perez-Gonzalez F, Asensi V. Diabetes mellitus and bacteraemia: a comparative study between diabetic and non-diabetic patients. *Eur J Med* 1992;1(5):281-7.
58. Hirschtick RE, Glassroth J, Jordan MC, et al. Bacterial pneumonia in persons infected with the human immunodeficiency virus. Pulmonary Complications of HIV Infection Study Group. *N Engl J Med* 1995;333(13):845-51.
59. Rosenberg AL, Seneff MG, Atiyeh L, Wagner R, Bojanowski L, Zimmerman JE. The importance of bacterial sepsis in intensive care unit patients with acquired immunodeficiency syndrome: implications for future care in the age of increasing antiretroviral resistance. *Crit Care Med* 2001;29(3):548-56.
60. Malone JL, Ijaz K, Lambert L, et al. Investigation of healthcare-associated transmission of *Mycobacterium tuberculosis* among patients with malignancies at three hospitals and at a residential facility. *Cancer* 2004;101(12):2713-21.
61. Fishman JA, Rubin RH. Infection in organ-transplant recipients. *N Engl J Med* 1998;338(24):1741-51.
62. Safdar N, Kluger DM, Maki DG. A review of risk factors for catheter-related bloodstream infection caused by percutaneously inserted, noncuffed central venous catheters: implications for preventive strategies. *Medicine (Baltimore)* 2002;81(6):466-79.

63. Jarvis WR, Robles B. Nosocomial infections in pediatric patients. *Adv Pediatr Infect Dis* 1996;12:243-959(js).
64. Yogaraj JS, Elward AM, Fraser VJ. Rate, risk factors, and outcomes of nosocomial primary bloodstream infection in pediatric intensive care unit patients. *Pediatrics* 2002;110(3):481-5.
65. Donlan RM. Biofilms: microbial life on surfaces. *Emerg Infect Dis* 2002;8(9):881-90.
66. Rosen HR. Acquisition of hepatitis C by a conjunctival splash. *Am J Infect Control* 1997;25(3):242-7.
67. Beltrami EM, Kozak A, Williams IT, et al. Transmission of HIV and hepatitis C virus from a nursing home patient to a health care worker. *Am J Infect Control* 2003;31(3):168-75.
68. Obasanjo OO, Wu P, Conlon M, et al. An outbreak of scabies in a teaching hospital: lessons learned. *Infect Control Hosp Epidemiol* 2001;22(1):13-8.
69. Andersen BM, Haugen H, Rasch M, Heldal Haugen A, Tageson A. Outbreak of scabies in Norwegian nursing homes and home care patients: control and prevention. *J Hosp Infect* 2000;45(2):160-4.
70. Avitzur Y, Amir J. Herpetic whitlow infection in a general pediatrician--an occupational hazard. *Infection* 2002;30(4):234-6.
71. Adams G, Stover BH, Keenlyside RA, et al. Nosocomial herpetic infections in a pediatric intensive care unit. *Am J Epidemiol* 1981;113(2):126-32.
72. Bhalla A, Pultz NJ, Gries DM, et al. Acquisition of nosocomial pathogens on hands after contact with environmental surfaces near hospitalized patients. *Infect Control Hosp Epidemiol* 2004;25(2):164-7.
73. Duckro AN, Blom DW, Lyle EA, Weinstein RA, Hayden MK. Transfer of vancomycin-resistant enterococci via health care worker hands. *Arch Intern Med* 2005;165(3):302-7.
74. Brooks SE, Veal RO, Kramer M, Dore L, Schupf N, Adachi M. Reduction in the incidence of *Clostridium difficile*-associated diarrhea in an acute care hospital and a skilled nursing facility following replacement of electronic thermometers with single-use disposables. *Infect Control Hosp Epidemiol* 1992;13(2):98-103.
75. CDC. Nosocomial hepatitis B virus infection associated with reusable fingerstick blood sampling devices--Ohio and New York City, 1996. *MMWR Morb Mortal Wkly Rep* 1997;46 (10):217-21.
76. Desenclos JC, Bourdiol-Razes M, Rolin B, et al. Hepatitis C in a ward for cystic fibrosis and diabetic patients: possible transmission by spring-loaded finger-stick devices for self-monitoring of capillary blood glucose. *Infect Control Hosp Epidemiol* 2001;22(11):701-7.

77. CDC. Transmission of hepatitis B virus among persons undergoing blood glucose monitoring in long-term-care facilities--Mississippi, North Carolina, and Los Angeles County, California, 2003-2004. *MMWR Morb Mortal Wkly Rep* 2005;54(9):220-3.
78. Hall CB, Douglas RG, Jr. Modes of transmission of respiratory syncytial virus. *J Pediatr* 1981;99(1):100-3.
79. Hall CB, Douglas RG, Jr., Geiman JM. Possible transmission by fomites of respiratory syncytial virus. *J Infect Dis* 1980;141(1):98-102.
80. Buttery JP, Alabaster SJ, Heine RG, et al. Multiresistant *Pseudomonas aeruginosa* outbreak in a pediatric oncology ward related to bath toys. *Pediatr Infect Dis J* 1998;17(6):509-13.
81. Agerton T, Valway S, Gore B, et al. Transmission of a highly drug-resistant strain (strain W1) of *Mycobacterium tuberculosis*. Community outbreak and nosocomial transmission via a contaminated bronchoscope. *JAMA* 1997;278(13):1073-7.
82. Bronowicki JP, Venard V, Botte C, et al. Patient-to-patient transmission of hepatitis C virus during colonoscopy. *N Engl J Med* 1997;337(4):237-40.
83. Michele TM, Cronin WA, Graham NM, et al. Transmission of *Mycobacterium tuberculosis* by a fiberoptic bronchoscope. Identification by DNA fingerprinting. *JAMA* 1997;278(13):1093-5.
84. Schelenz S, French G. An outbreak of multidrug-resistant *Pseudomonas aeruginosa* infection associated with contamination of bronchoscopes and an endoscope washer-disinfector. *J Hosp Infect* 2000;46(1):23-30.
85. Weber DJ, Rutala WA. Lessons from outbreaks associated with bronchoscopy. *Infect Control Hosp Epidemiol* 2001;22(7):403-8.
86. Kirschke DL, Jones TF, Craig AS, et al. *Pseudomonas aeruginosa* and *Serratia marcescens* contamination associated with a manufacturing defect in bronchoscopes. *N Engl J Med* 2003;348(3):214-20.
87. Srinivasan A, Wolfenden LL, Song X, et al. An outbreak of *Pseudomonas aeruginosa* infections associated with flexible bronchoscopes. *N Engl J Med* 2003;348(3):221-7.
88. Boyce JM, Potter-Bynoe G, Chenevert C, King T. Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. *Infect Control Hosp Epidemiol* 1997;18(9):622-7.(mj).
89. Zachary KC, Bayne PS, Morrison VJ, Ford DS, Silver LC, Hooper DC. Contamination of gowns, gloves, and stethoscopes with vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 2001;22(9):560-4.
90. Perry C, Marshall R, Jones E. Bacterial contamination of uniforms. *J Hosp Infect* 2001;48(3):238-41.

91. Papineni RS, Rosenthal FS. The size distribution of droplets in the exhaled breath of healthy human subjects. *J Aerosol Med* 1997;10(2):105-16.
92. Wells WF. On airborne infection: Study II. Droplets and droplet nuclei. *Am J Hygiene* 1934;20:611-18.
93. Loeb M, McGeer A, Henry B, et al. SARS among critical care nurses, Toronto. *Emerg Infect Dis* 2004;10(2):251-5.
94. Fowler RA, Guest CB, Lapinsky SE, et al. Transmission of severe acute respiratory syndrome during intubation and mechanical ventilation. *Am J Respir Crit Care Med* 2004;169(11):1198-202.
95. Gehanno JF, Kohen-Couderc L, Lemeland JF, Leroy J. Nosocomial meningococemia in a physician. *Infect Control Hosp Epidemiol* 1999;20(8):564-5.
96. Scales D, et al. Illness in intensive-care staff after brief exposure to severe acute respiratory syndrome. *Emerg Infect Dis* 2003;9(10):1205-10.
97. Ensor E, Humphreys H, Peckham D, Webster C, Knox AJ. Is *Burkholderia* (*Pseudomonas*) *cepacia* disseminated from cystic fibrosis patients during physiotherapy? *J Hosp Infect* 1996;32(1):9-15.
98. Christian MD, Loutfy M, McDonald LC, et al. Possible SARS coronavirus transmission during cardiopulmonary resuscitation. *Emerg Infect Dis* 2004;10(2):287-93.
99. Valenzuela TD, Hooton TM, Kaplan EL, Schlievert P. Transmission of 'toxic strep' syndrome from an infected child to a firefighter during CPR. *Ann Emerg Med* 1991;20(1):90-2.
100. Bassinet L, Matrat M, Njamkepo E, Aberrane S, Housset B, Guiso N. Nosocomial pertussis outbreak among adult patients and healthcare workers. *Infect Control Hosp Epidemiol* 2004;25(11):995-7.
101. Wong TW, Lee CK, Tam W, et al. Cluster of SARS among medical students exposed to single patient, Hong Kong. *Emerg Infect Dis* 2004;10(2):269-76.
102. Pachucki CT, Pappas SA, Fuller GF, Krause SL, Lentino JR, Schaaff DM. Influenza A among hospital personnel and patients. Implications for recognition, prevention, and control. *Arch Intern Med* 1989;149(1):77-80.
103. Feigin RD, Baker CJ, Herwaldt LA, Lampe RM, Mason EO, Whitney SE. Epidemic meningococcal disease in an elementary-school classroom. *N Engl J Med* 1982;307(20):1255-7.
104. Dick EC, Jennings LC, Mink KA, Wartgow CD, Inhorn SL. Aerosol transmission of rhinovirus colds. *J Infect Dis* 1987;156(3):442-8.
105. Duguid JP. The size and duration of air-carriage of respiratory droplets and droplet nuclei. *J Hyg (Lond)* 1946;44:471-9.

106. Hall CB, Douglas RG, Jr., Schnabel KC, Geiman JM. Infectivity of respiratory syncytial virus by various routes of inoculation. *Infect Immun* 1981;33(3):779-83.
107. Downie AW, Meiklejohn M, St Vincent L, Rao AR, Sundara Babu BV, Kempe CH. The recovery of smallpox virus from patients and their environment in a smallpox hospital. *Bull World Health Organ* 1965;33(5):615-22.
108. Fenner F, Henderson DA, Arita I, Jezek Z, Ladnyi ID. The epidemiology of smallpox. In: *Smallpox and its eradication*. Switzerland: World Health Organization; 1988.
109. Cole EC, Cook CE. Characterization of infectious aerosols in health care facilities: an aid to effective engineering controls and preventive strategies. *Am J Infect Control* 1998;26(4):453-64.
110. Christie C, Mazon D, Hierholzer W, Jr., Patterson JE. Molecular heterogeneity of *Acinetobacter baumannii* isolates during seasonal increase in prevalence. *Infect Control Hosp Epidemiol* 1995;16(10):590-4.
111. Musher DM. How contagious are common respiratory tract infections? *N Engl J Med* 2003;348(13):1256-66.
112. Steinberg P, White RJ, Fuld SL, Gutekunst RR, Chanock RM, Senterfit LB. Ecology of *Mycoplasma pneumoniae* infections in marine recruits at Parris Island, South Carolina. *Am J Epidemiol* 1969;89(1):62-73.
113. Seto WH, Tsang D, Yung RW, et al. Effectiveness of precautions against droplets and contact in prevention of nosocomial transmission of severe acute respiratory syndrome (SARS). *Lancet* 2003;361(9368):1519-20.
114. Hamburger M, Robertson OH. Expulsion of group A haemolytic streptococci in droplets and droplet nuclei by sneezing, coughing and talking. *Am J Med* 1948;4:690.
115. CDC. Nosocomial meningococemia. *MMWR Morb Mortal Wkly Rep* 1978;27:358.
116. LeClair JM, Freeman J, Sullivan BF, Crowley CM, Goldmann DA. Prevention of nosocomial respiratory syncytial virus infections through compliance with glove and gown isolation precautions. *N Engl J Med* 1987;317(6):329-34.
117. Madge P, Paton JY, McColl JH, Mackie PL. Prospective controlled study of four infection-control procedures to prevent nosocomial infection with respiratory syncytial virus. *Lancet* 1992;340(8827):1079-83.
118. Bassetti S, Bischoff WE, Walter M, et al. Dispersal of *Staphylococcus aureus* Into the Air Associated With a Rhinovirus Infection. *Infect Control Hosp Epidemiol* 2005;26(2):196-203.
119. Eichenwald HF, Kotsevalov O, Fasso LA. The "cloud baby": an example of bacterial-viral interaction. *Am J Dis Child* 1960;100:161-73.

120. Sheretz RJ, Reagan DR, Hampton KD, et al. A cloud adult: the Staphylococcus aureus-virus interaction revisited. *Ann Intern Med* 1996;124(6):539-47.
121. Coronado VG, Beck-Sague CM, Hutton MD, et al. Transmission of multidrug-resistant Mycobacterium tuberculosis among persons with human immunodeficiency virus infection in an urban hospital: epidemiologic and restriction fragment length polymorphism analysis. *J Infect Dis* 1993;168(4):1052-5.
122. Bloch AB, Orenstein WA, Ewing WM, et al. Measles outbreak in a pediatric practice: airborne transmission in an office setting. *Pediatrics* 1985;75(4):676-83.
123. LeClair JM, Zaia JA, Levin MJ, Congdon RG, Goldmann DA. Airborne transmission of chickenpox in a hospital. *N Engl J Med* 1980;302(8):450-3.
124. Riley RL, Mills CC, Nyka W, et al. Aerial dissemination of pulmonary tuberculosis. A two-year study of contagion in a tuberculosis ward. 1959. *Am J Hyg* 1959;70:185-96.
125. Beck-Sague C, Dooley SW, Hutton MD, et al. Hospital outbreak of multidrug-resistant Mycobacterium tuberculosis infections. Factors in transmission to staff and HIV-infected patients. *JAMA* 1992;268(10):1280-6.
126. CDC. Guidelines for preventing the transmission of Mycobacterium tuberculosis in health-care facilities, 1994. Centers for Disease Control and Prevention. *MMWR Recomm Rep* 1994;43(RR-13):1-132.
127. Haley CE, McDonald RC, Rossi L, Jones WD, Jr., Haley RW, Luby JP. Tuberculosis epidemic among hospital personnel. *Infect Control Hosp Epidemiol* 1989;10(5):204-10.
128. Wehrle PF, Posch J, Richter KH, Henderson DA. An airborne outbreak of smallpox in a German hospital and its significance with respect to other recent outbreaks in Europe. *Bull World Health Organ* 1970;43(5):669-79.
129. Gelfand HM, Posch J. The recent outbreak of smallpox in Meschede, West Germany. *Am J Epidemiol* 1971;93(4):234-7.
130. Moser MR, Bender TR, Margolis HS, Noble GR, Kendal AP, Ritter DG. An outbreak of influenza aboard a commercial airliner. *Am J Epidemiol* 1979;110(1):1-6.
131. Alford RH, Kasel JA, Gerone PJ, Knight V. Human influenza resulting from aerosol inhalation. *Proc Soc Exp Biol Med* 1966;122(3):800-4.
132. Chadwick PR, McCann R. Transmission of a small round structured virus by vomiting during a hospital outbreak of gastroenteritis. *J Hosp Infect* 1994;26(4):251-9.

133. Prince DS, Astry C, Vonderfecht S, Jakab G, Shen FM, Yolken RH. Aerosol transmission of experimental rotavirus infection. *Pediatr Infect Dis* 1986;5(2):218-22.
134. www.cdc.gov/ncidod/sars.
135. Peiris JS, Yuen KY, Osterhaus AD, Stohr K. The severe acute respiratory syndrome. *N Engl J Med* 2003;349(25):2431-41.
136. Olsen SJ, Chang HL, Cheung TY, et al. Transmission of the severe acute respiratory syndrome on aircraft. *N Engl J Med* 2003;349(25):2416-22.
137. Wilder-Smith A, Leong HN, Villacian JS. In-flight transmission of Severe Acute Respiratory Syndrome (SARS): A Case Report. *J Travel Med* 2003;10(5):299-300.
138. Booth TF, Kournikakis B, Bastien N, et al. Detection of airborne severe acute respiratory syndrome (SARS) coronavirus and environmental contamination in SARS outbreak units. *J Infect Dis* 2005;191(9):1472-7.
139. Yu IT, Li Y, Wong TW, et al. Evidence of airborne transmission of the severe acute respiratory syndrome virus. *N Engl J Med* 2004;350(17):1731-9.
140. CDC. Update: Outbreak of severe acute respiratory syndrome--worldwide. *MMWR Morb Mortal Wkly Rep* 2003;52 (12):241-6, 8.
141. CDC. Cluster of severe acute respiratory syndrome cases among protected health-care workers--Toronto, Canada, April 2003. *MMWR Morb Mortal Wkly Rep* 2003;52(19):433-6.
142. Sawyer LA, Murphy JJ, Kaplan JE, et al. 25- to 30-nm virus particle associated with a hospital outbreak of acute gastroenteritis with evidence for airborne transmission. *Am J Epidemiol* 1988;127(6):1261-71.
143. Marks PJ, Vipond IB, Carlisle D, Deakin D, Fey RE, Caul EO. Evidence for airborne transmission of Norwalk-like virus (NLV) in a hotel restaurant. *Epidemiol Infect* 2000;124(3):481-7.
144. Salgado CD, Farr BM, Hall KK, Hayden FG. Influenza in the acute hospital setting. *Lancet Infect Dis* 2002;2(3):145-55.
145. Riley RL. Airborne infection. *Am J Med* 1974;57(3):466-75.
146. McLean R. General discussion. . *Am Rev Respir Dis* 1961;83 36-8.
147. Cheesbrough JS, Green J, Gallimore CI, Wright PA, Brown DW. Widespread environmental contamination with Norwalk-like viruses (NLV) detected in a prolonged hotel outbreak of gastroenteritis. *Epidemiol Infect* 2000;125(1):93-8.
148. Marks PJ, Vipond IB, Regan FM, Wedgwood K, Fey RE, Caul EO. A school outbreak of Norwalk-like virus: evidence for airborne transmission. *Epidemiol Infect* 2003;131(1):727-36.

149. Roy CJ, Milton DK. Airborne transmission of communicable infection--the elusive pathway. *N Engl J Med* 2004;350(17):1710-2.
150. Dull PM, Wilson KE, Kournikakis B, et al. Bacillus anthracis aerosolization associated with a contaminated mail sorting machine. *Emerg Infect Dis* 2002;8(10):1044-7.
151. Weis CP, Intrepido AJ, Miller AK, et al. Secondary aerosolization of viable Bacillus anthracis spores in a contaminated US Senate Office. *JAMA* 2002;288(22):2853-8.
152. Patterson JE, Zidouh A, Minitier P, Andriole VT, Patterson TF. Hospital epidemiologic surveillance for invasive aspergillosis: patient demographics and the utility of antigen detection. *Infect Control Hosp Epidemiol* 1997;18(2):104-8.
153. Arnow PM, Andersen RL, Mainous PD, Smith EJ. Pulmonary aspergillosis during hospital renovation. *Am Rev Respir Dis* 1978;118(1):49-53.
154. Pegues DA, Lasker BA, McNeil MM, Hamm PM, Lundal JL, Kubak BM. Cluster of cases of invasive aspergillosis in a transplant intensive care unit: evidence of person-to-person airborne transmission. *Clin Infect Dis* 2002;34(3):412-6.
155. Buffington J, Reporter R, Lasker BA, et al. Investigation of an epidemic of invasive aspergillosis: utility of molecular typing with the use of random amplified polymorphic DNA probes. *Pediatr Infect Dis J* 1994;13(5):386-93.
156. Krasinski K, Holzman RS, Hanna B, Greco MA, Graff M, Bhogal M. Nosocomial fungal infection during hospital renovation. *Infect Control* 1985;6(7):278-82.
157. Humphreys H. Positive-pressure isolation and the prevention of invasive aspergillosis. What is the evidence? *J Hosp Infect* 2004;56(2):93-100.
158. Thio CL, Smith D, Merz WG, et al. Refinements of environmental assessment during an outbreak investigation of invasive aspergillosis in a leukemia and bone marrow transplant unit. *Infect Control Hosp Epidemiol* 2000;21(1):18-23.
159. Anaissie EJ, Stratton SL, Dignani MC, et al. Pathogenic Aspergillus species recovered from a hospital water system: a 3-year prospective study. *Clin Infect Dis* 2002;34(6):780-9.
160. CDC. Prevention of invasive group A streptococcal disease among household contacts of case patients and among postpartum and postsurgical patients: recommendations from the Centers for Disease Control and Prevention. *Clin Infect Dis* 2002;35 (8):950-9.
161. Gruteke P, van Belkum A, Schouls LM, et al. Outbreak of group A streptococci in a burn center: use of pheno- and genotypic procedures for strain tracking. *J Clin Microbiol* 1996;34(1):114-8.

162. Greene CM, Van Beneden CA, Javadi M, et al. Cluster of deaths from group A streptococcus in a long-term care facility--Georgia, 2001. *Am J Infect Control* 2005;33(2):108-13.
163. Sabria M, Campins M. Legionnaires' disease: update on epidemiology and management options. *Am J Respir Med* 2003;2(3):235-43.
164. Bille J, Marchetti O, Calandra T. Changing face of health-care associated fungal infections. *Curr Opin Infect Dis* 2005;18(4):314-9.
165. Hall IC, O'Toole E. Intestinal flora in newborn infants with a description of a new pathogenic anaerobe, *Bacillus difficilis*. *Am J Dis Child* 1935;49:390-402.
166. George WL, Sutter VL, Finegold SM. Antimicrobial agent-induced diarrhea--a bacterial disease. *J Infect Dis* 1977;136(6):822-8.
167. McFarland LV, Mulligan ME, Kwok RY, Stamm WE. Nosocomial acquisition of *Clostridium difficile* infection. *N Engl J Med* 1989;320(4):204-10.
168. Pepin J, Valiquette L, Alary ME, et al. *Clostridium difficile*-associated diarrhea in a region of Quebec from 1991 to 2003: a changing pattern of disease severity. *Cmaj* 2004;171(5):466-72.
169. 169. Agency HP. Outbreak of *Clostridium difficile* infection in a hospital in south east England. *Communicable Disease Report Weekly* 2005;31(24).
170. Koopmans M, Wilbrink B, Conyn M, et al. Transmission of H7N7 avian influenza A virus to human beings during a large outbreak in commercial poultry farms in the Netherlands. *Lancet* 2004;363(9409):587-93.
171. McDonald LC, Killgore GE, Thompson A, et al. An epidemic, toxin gene-variant strain of *Clostridium difficile*. *N Engl J Med* 2005;353(23):2433-41.
172. Loo VG, Poirier L, Miller MA, et al. A predominantly clonal multi-institutional outbreak of *Clostridium difficile*-associated diarrhea with high morbidity and mortality. *N Engl J Med* 2005;353(23):2442-9.
173. Warny M, Pepin J, Fang A, et al. Toxin production by an emerging strain of *Clostridium difficile* associated with outbreaks of severe disease in North America and Europe. *Lancet* 2005;366(9491):1079-84.
174. Layton BA ML, Gerding DN, Liedtke LA, Strausbaugh LJ. Perceived increases in the incidence and severity of *Clostridium difficile* disease: an emerging threat that continues to unfold. In: 15th Annual Scientific Meeting of the Society for Healthcare Epidemiology of America;. Los Angeles, CA.; 2005.
175. Sohn S, Climo M, Diekema D, et al. Varying rates of *Clostridium difficile*-associated diarrhea at prevention epicenter hospitals. *Infect Control Hosp Epidemiol* 2005;26(8):676-9.

176. IOM. Antimicrobial Resistance: Issues and Options. Workshop report. In: Harrison PF, Lederberg J, eds. Washington, DC: National Academy Press; 1998:8-74.
177. Shlaes DM, Gerding DN, John JF, Jr., et al. Society for Healthcare Epidemiology of America and Infectious Diseases Society of America Joint Committee on the Prevention of Antimicrobial Resistance: guidelines for the prevention of antimicrobial resistance in hospitals. *Infect Control Hosp Epidemiol* 1997;18(4):275-91.
178. Whitener CJ, Park SY, Browne FA, et al. Vancomycin-resistant *Staphylococcus aureus* in the absence of vancomycin exposure. *Clin Infect Dis* 2004;38(8):1049-55.
179. CDC. *Staphylococcus aureus* with reduced susceptibility to vancomycin-- United States. *MMWR Morb Mortal Wkly Rep* 1997;46 (33):765-6.
180. CDC. *Staphylococcus aureus* resistant to vancomycin--United States, 2002. *MMWR Morb Mortal Wkly Rep* 2002;51 (26):565-7.
181. CDC. Public Health Dispatch: Vancomycin-Resistant *Staphylococcus aureus* --- Pennsylvania, 2002. *MMWR - Morbidity & Mortality Weekly Report* 2002;51(40):902.
182. CDC. Vancomycin-resistant *Staphylococcus aureus*--New York, 2004. *MMWR Morb Mortal Wkly Rep* 2004;53(15):322-3.
183. Chang S, Sievert DM, Hageman JC, et al. Infection with vancomycin-resistant *Staphylococcus aureus* containing the vanA resistance gene. *N Engl J Med* 2003;348(14):1342-7.
184. Fridkin SK, Hageman J, McDougal LK, et al. Epidemiological and microbiological characterization of infections caused by *Staphylococcus aureus* with reduced susceptibility to vancomycin, United States, 1997-2001. *Clin Infect Dis* 2003;36(4):429-39.
185. Gold HS, Moellering RC, Jr. Antimicrobial-drug resistance. *N Engl J Med* 1996;335(19):1445-53.
186. Hageman JC, Pegues DA, Jepson C, et al. Vancomycin-intermediate *Staphylococcus aureus* in a home health-care patient. *Emerg Infect Dis* 2001;7(6):1023-5.
187. Harwell JI, Brown RB. The drug-resistant pneumococcus: clinical relevance, therapy, and prevention. *Chest* 2000;117(2):530-41.
188. Jones RN. Resistance patterns among nosocomial pathogens: trends over the past few years. *Chest* 2001;119(2 Suppl):397S-404S.
189. Murray BE. Vancomycin-resistant enterococcal infections. *N Engl J Med* 2000;342(10):710-21.
190. Neuhauser MM, Weinstein RA, Rydman R, Danziger LH, Karam G, Quinn JP. Antibiotic resistance among gram-negative bacilli in US

- intensive care units: implications for fluoroquinolone use. *JAMA* 2003;289(7):885-8.
191. Pitout JD, Sanders CC, Sanders WE, Jr. Antimicrobial resistance with focus on beta-lactam resistance in gram- negative bacilli. *Am J Med* 1997;103(1):51-9.
 192. Rotun SS, McMath V, Schoonmaker DJ, et al. Staphylococcus aureus with reduced susceptibility to vancomycin isolated from a patient with fatal bacteremia. *Emerg Infect Dis* 1999;5(1):147-9.
 193. Smith TL, Pearson ML, Wilcox KR, et al. Emergence of vancomycin resistance in Staphylococcus aureus. Glycopeptide-Intermediate Staphylococcus aureus Working Group. *N Engl J Med* 1999;340(7):493-501.
 194. Srinivasan A, Dick JD, Perl TM. Vancomycin resistance in staphylococci. *Clin Microbiol Rev* 2002;15(3):430-8.
 195. Hyle EP, Lipworth AD, Zaoutis TE, et al. Risk Factors for Increasing Multidrug Resistance among Extended-Spectrum β -lactamase-Lactamase-Producing *Escheria coli* and *Klebsiella* species *Clin Infect Dis* 2005;40(9):1317-24.
 196. Gleich S, Morad Y, Echague R, et al. Streptococcus pneumoniae serotype 4 outbreak in a home for the aged: report and review of recent outbreaks. *Infect Control Hosp Epidemiol* 2000;21:711.
 197. Fry AM, Udeagu CC, Soriano-Gabarro M, et al. Persistence of fluoroquinolone-resistant, multidrug-resistant Streptococcus pneumoniae in a long-term-care facility: efforts to reduce intrafacility transmission. *Infect Control Hosp Epidemiol* 2005;26(3):239-47.
 198. Carter RJ, Sorenson G, Heffernan R, et al. Failure to control an outbreak of multidrug-resistant Streptococcus pneumoniae in a long-term-care facility: emergence and ongoing transmission of a fluoroquinolone-resistant strain. *Infect Control Hosp Epidemiol* 2005;26(3):248-55.
 199. Blok HE, Troelstra A, Kamp-Hopmans TE, et al. Role of healthcare workers in outbreaks of methicillin-resistant Staphylococcus aureus: a 10- year evaluation from a Dutch university hospital. *Infect Control Hosp Epidemiol* 2003;24(9):679-85.
 200. Muto CA, Jernigan JA, Ostrowsky BE, et al. SHEA guideline for preventing nosocomial transmission of multidrug-resistant strains of Staphylococcus aureus and enterococcus. *Infect Control Hosp Epidemiol* 2003;24(5):362-86.
 201. Tammelin A, Klotz F, Hambraeus A, Stahle E, Ransjo U. Nasal and hand carriage of Staphylococcus aureus in staff at a Department for Thoracic and Cardiovascular Surgery: endogenous or exogenous source? *Infect Control Hosp Epidemiol* 2003;24(9):686-9.

202. CDC. Biological and chemical terrorism: strategic plan for preparedness and response. Recommendations of the CDC Strategic Planning Workgroup. MMWR Recomm Rep 2000;49(RR-4):1-14.
203. Inglesby TV, O'Toole T, Henderson DA, et al. Anthrax as a biological weapon, 2002: updated recommendations for management. JAMA 2002;287(17):2236-52.
204. Henderson DA, Inglesby TV, Bartlett JG, et al. Smallpox as a biological weapon: medical and public health management. Working Group on Civilian Biodefense. JAMA 1999;281(22):2127-37.
205. www.bt.cdc.gov/agent/smallpox/
206. www.who.int/csr/disease/smallpox/en/
207. Kool JL. Risk of person-to-person transmission of pneumonic plague. Clin Infect Dis 2005;40(8):1166-72.
208. Inglesby TV, Dennis DT, Henderson DA, et al. Plague as a biological weapon: medical and public health management. Working Group on Civilian Biodefense. JAMA 2000;283(17):2281-90.
209. Arnon SS, Schechter R, Inglesby TV, et al. Botulinum toxin as a biological weapon: medical and public health management. JAMA 2001;285(8):1059-70.
210. Dennis DT, Inglesby TV, Henderson DA, et al. Tularemia as a biological weapon: medical and public health management. JAMA 2001;285(21):2763-73.
211. CDC. Notice to Readers Update: Management of Patients with Suspected Viral Hemorrhagic Fever -- United States. MMWR Recomm Rep 1995;44(25):475-9.
212. Borio L, Inglesby T, Peters CJ, et al. Hemorrhagic fever viruses as biological weapons: medical and public health management. JAMA 2002;287(18):2391-405.
213. Neff JM, Lane JM, Fulginiti VA, Henderson DA. Contact vaccinia--transmission of vaccinia from smallpox vaccination. JAMA 2002;288(15):1901-5.
214. Sepkowitz KA. How contagious is vaccinia? N Engl J Med 2003;348(5):439-46.
215. Lane JM, Fulginiti VA. Transmission of vaccinia virus and rationale for measures for prevention. Clin Infect Dis 2003;37(2):281-4.
216. CDC. Smallpox Vaccination and Adverse Reactions: Guidance for Clinicians. MMWR - Morbidity & Mortality Weekly Report 2003;52(RR04):1-28.

217. Fulginiti VA, Papier A, Lane JM, Neff JM, Henderson DA. Smallpox vaccination: a review, part II. Adverse events. *Clin Infect Dis* 2003;37(2):251-71.
218. www.smallpox.mil./event/SPSafetySum.asp
219. CDC. Update: adverse events following civilian smallpox vaccination--United States, 2003. *MMWR Morb Mortal Wkly Rep* 2003;52(18):419-20.
220. CDC. Secondary and tertiary transfer of vaccinia virus among U.U. military personnel--United States and worldwide, 2002-2004. *MMWR Morb Mortal Wkly Rep* 2004;53(5):103-5.
221. Talbot TR, Ziel E, Doersam JK, LaFleur B, Tollefson S, Edwards KM. Risk of vaccinia transfer to the hands of vaccinated persons after smallpox immunization. *Clin Infect Dis* 2004;38(4):536-41.
222. Hepburn MJ, Dooley DP, Murray CK, et al. Frequency of vaccinia virus isolation on semipermeable versus nonocclusive dressings covering smallpox vaccination sites in hospital personnel. *Am J Infect Control* 2004;32(3):126-30.
223. Waibel KH, Ager EP, Topolski RL, Walsh DS. Randomized trial comparing vaccinia on the external surfaces of 3 conventional bandages applied to smallpox vaccination sites in primary vaccinees. *Clin Infect Dis* 2004;39(7):1004-7.
224. Tenorio AR, Peoples M, Patri M, et al. Quantitative Vaccinia Cultures and Evolution of Vaccinia-Specific CD8+ Cytotoxic T-lymphocytes (CTL) Responses in Revaccinees. Abstract #823, 41st Annual Meeting IDSA, October 2003, San Diego 2003.
225. Wharton M, Strikas RA, Harpaz R, et al. Recommendations for using smallpox vaccine in a pre-event vaccination program. Supplemental recommendations of the Advisory Committee on Immunization Practices (ACIP) and the Healthcare Infection Control Practices Advisory Committee (HICPAC). *MMWR Recomm Rep* 2003;52(RR-7):1-16.
226. CDC. Surveillance for Creutzfeldt-Jakob Disease -- United States. *MMWR* 1996;45(31):665-8.
227. Johnson RT, Gibbs CJ, Jr. Creutzfeldt-Jakob disease and related transmissible spongiform encephalopathies. *N Engl J Med* 1998;339(27):1994-2004.
228. Brown P, Gajdusek DC, Gibbs CJ, Jr., Asher DM. Potential epidemic of Creutzfeldt-Jakob disease from human growth hormone therapy. *N Engl J Med* 1985;313(12):728-31.
229. Frasier SD, Foley TP, Jr. Clinical review 58: Creutzfeldt-Jakob disease in recipients of pituitary hormones. *J Clin Endocrinol Metab* 1994;78(6):1277-9.

230. CDC. Update: Creutzfeldt-Jakob disease associated with cadaveric dura mater grafts--Japan, 1979-2003. *MMWR Morb Mortal Wkly Rep* 2003;52(48):1179-81.
231. Lang CJ, Heckmann JG, Neundorfer B. Creutzfeldt-Jakob disease via dural and corneal transplants. *J Neurol Sci* 1998;160(2):128-39.
232. el Hachimi KH, Chaunu MP, Cervenakova L, Brown P, Foncin JF. Putative neurosurgical transmission of Creutzfeldt-Jakob disease with analysis of donor and recipient: agent strains. *C R Acad Sci III* 1997;320(4):319-28.
233. Will RG, Matthews WB. Evidence for case-to-case transmission of Creutzfeldt-Jakob disease. *J Neurol Neurosurg Psychiatry* 1982;45(3):235-8.
234. Bernoulli C, Siegfried J, Baumgartner G, et al. Danger of accidental person-to-person transmission of Creutzfeldt-Jakob disease by surgery. *Lancet* 1977;1(8009):478-9.
235. Rutala WA, Weber DJ. Creutzfeldt-Jakob disease: recommendations for disinfection and sterilization. *Clin Infect Dis* 2001;32(9):1348-56.
236. Belay ED, Maddox RA, Williams ES, Miller MW, Gambetti P, Schonberger LB. Chronic wasting disease and potential transmission to humans. *Emerg Infect Dis* 2004;10(6):977-84.
237. Collinge J, Sidle KC, Meads J, Ironside J, Hill AF. Molecular analysis of prion strain variation and the aetiology of 'new variant' CJD. *Nature* 1996;383(6602):685-90.
238. Belay ED, Schonberger LB. The public health impact of prion diseases. *Annu Rev Public Health* 2005;26:191-212.
239. Belay ED, Schonberger LB. Variant Creutzfeldt-Jakob disease and bovine spongiform encephalopathy. *Clin Lab Med* 2002;22(4):849-62, v-vi.
240. Hill AF, Butterworth RJ, Joiner S, et al. Investigation of variant Creutzfeldt-Jakob disease and other human prion diseases with tonsil biopsy samples. *Lancet* 1999;353(9148):183-9.
241. Evatt B. Creutzfeldt-Jakob disease and haemophilia: assessment of risk. *Haemophilia* 2000;6 Suppl 1:94-9.
242. Chamberland ME. Emerging infectious agents: do they pose a risk to the safety of transfused blood and blood products? *Clin Infect Dis* 2002;34(6):797-805.
243. www.fda.gov/cber/gdlns/cjdvcjd.htm
244. Llewelyn CA, Hewitt PE, Knight RS, et al. Possible transmission of variant Creutzfeldt-Jakob disease by blood transfusion. *Lancet* 2004;363(9407):417-21.

245. Peden AH, Head MW, Ritchie DL, Bell JE, Ironside JW. Preclinical vCJD after blood transfusion in a PRNP codon 129 heterozygous patient. *Lancet* 2004;364(9433):527-9.
246. Brown P. Guidelines for high risk autopsy cases: special precautions for Creutzfeldt-Jakob Disease. In: Hutchins G, ed. *Autopsy Performance and Reporting*, Northfield, Ill.: College of American Pathologists 1990:68-74.
247. Drosten C, Gunther S, Preiser W, et al. Identification of a novel coronavirus in patients with severe acute respiratory syndrome. *N Engl J Med* 2003;348(20):1967-76.
248. Ksiazek TG, Erdman D, Goldsmith CS, et al. A novel coronavirus associated with severe acute respiratory syndrome. *N Engl J Med* 2003;348(20):1953-66.
249. Chan WM, Kwan YW, Wan HS, Leung CW, Chiu MC. Epidemiologic linkage and public health implication of a cluster of severe acute respiratory syndrome in an extended family. *Pediatr Infect Dis J* 2004;23(12):1156-9.
250. Leung CW, Kwan YW, Ko PW, et al. Severe acute respiratory syndrome among children. *Pediatrics* 2004;113(6):e535-43.
251. Bitnun A, Allen U, Heurter H, et al. Children hospitalized with severe acute respiratory syndrome-related illness in Toronto. *Pediatrics* 2003;112(4):e261.
252. Chow KY, Lee CE, Ling ML, Heng DM, Yap SG. Outbreak of severe acute respiratory syndrome in a tertiary hospital in Singapore, linked to an index patient with atypical presentation: epidemiological study. *Bmj* 2004;328(7433):195.
253. Shen Z, Ning F, Zhou W, et al. Superspreading SARS events, Beijing, 2003. *Emerg Infect Dis* 2004;10(2):256-60.
254. Chen Y-C, Huang L-M, Chan C-C, et al. SARS in Hospital Emergency Room. *Emerg Infect Dis* 2004;10:782-8.
255. Chung Y-C, Huang L-M, Chan C-C, et al. SARS in Hospital Emergency Room. *Emerg Infect Dis* 2004;10:782-8.
256. Gamage B, Moore D, Copes R, Yassi A, Bryce E. Protecting health care workers from SARS and other respiratory pathogens: a review of the infection control literature. *Am J Infect Control* 2005;33(2):114-21.
257. Moore D, Gamage B, Bryce E, Copes R, Yassi A. Protecting health care workers from SARS and other respiratory pathogens: organizational and individual factors that affect adherence to infection control guidelines. *Am J Infect Control* 2005;33(2):88-96.
258. Dowell SF, Simmerman JM, Erdman DD, et al. Severe acute respiratory syndrome coronavirus on hospital surfaces. *Clin Infect Dis* 2004;39(5):652-7.

259. Public Health Guidance for Community-Level Preparedness and Response to Severe Acute Respiratory Syndrome (SARS). 2004. (Accessed at <http://www.cdc.gov/ncidod/sars/guidance/I/occupational.htm>.)
260. Le DH, Bloom SA, Nguyen QH, et al. Lack of SARS transmission among public hospital workers, Vietnam. *Emerg Infect Dis* 2004;10(2):265-8.
261. Lim PL, Kurup A, Gopalakrishna G, et al. Laboratory-acquired severe acute respiratory syndrome. *N Engl J Med* 2004;350(17):1740-5.
262. CDC. www.cdc.gov/ncidod/sars. 2003.
263. Reed KD, Melski JW, Graham MB, et al. The detection of monkeypox in humans in the Western Hemisphere. *N Engl J Med* 2004;350(4):342-50.
264. Anderson MG, Frenkel LD, Homann S, Guffey J. A case of severe monkeypox virus disease in an American child: emerging infections and changing professional values. *Pediatr Infect Dis J* 2003;22:1093-6.
265. Jezek Z, Fenner F. Human monkey pox. In: Melnick JL ed. *Monographs in virology*. Vol. 17. Basel, Switzerland: S Karger AG. 1988:81-102.
266. Marennikova SS, Jezek Z, Szczeniowski M, Mbudi PM, Vernette M. [Contagiousness of monkey pox for humans: results of an investigation of 2 outbreaks of the infection in Zaire]. *Zh Mikrobiol Epidemiol Immunobiol* 1985(8):38-43.
267. Jezek Z, Arita I, Mutombo M, Dunn C, Nakano JH, Szczeniowski M. Four generations of probable person-to-person transmission of human monkeypox. *Am J Epidemiol* 1986;123(6):1004-12.
268. Learned LA, Reynolds MG, Wassa DW, et al. Extended interhuman transmission of monkeypox in a hospital community in the Republic of the Congo, 2003. *Am J Trop Med Hyg* 2005;73(2):428-34.
269. Fleischauer AT, Kile JC, Davidson M, et al. Evaluation of human-to-human transmission of monkeypox from infected patients to health care workers. *Clin Infect Dis* 2005;40(5):689-94.
270. Likos AM, Sammons SA, Olson VA, et al. A tale of two clades: monkeypox viruses. *J Gen Virol* 2005;86(Pt 10):2661-72.
271. Fine PE, Jezek Z, Grab B, Dixon H. The transmission potential of monkeypox virus in human populations. *Int J Epidemiol* 1988;17(3):643-50.
272. Jezek Z, Grab B, Paluku KM, Szczeniowski MV. Human monkeypox: disease pattern, incidence and attack rates in a rural area of northern Zaire. *Trop Geogr Med* 1988;40(2):73-83.
273. CDC. "Norwalk-Like Viruses": Public Health Consequences and Outbreak Management. *MMWR - Morbidity & Mortality Weekly Report* 2001;50 (RR-09)(June):1-18.

274. Evans MR, Meldrum R, Lane W, et al. An outbreak of viral gastroenteritis following environmental contamination at a concert hall. *Epidemiol Infect* 2002;129(2):355-60.
275. Wu HM, Fornek M, Kellogg JS, et al. A Norovirus Outbreak at a Long-Term-Care Facility: The Role of Environmental Surface Contamination. *Infect Control Hosp Epidemiol* 2005;26(10):802-10.
276. Duizer E, Schwab KJ, Neill FH, Atmar RL, Koopmans MP, Estes MK. Laboratory efforts to cultivate noroviruses. *J Gen Virol* 2004;85(Pt 1):79-87.
277. Zingg W, Colombo C, Jucker T, Bossart W, Ruef C. Impact of an outbreak of norovirus infection on hospital resources. *Infect Control Hosp Epidemiol* 2005;26:263-7.
278. Calderon-Margalit R, Sheffer R, Halperin T, Orr N, Cohen D, Shohat T. A large-scale gastroenteritis outbreak associated with Norovirus in nursing homes. *Epidemiol Infect* 2005;133(1):35-40.
279. Marx A, Shay DK, Noel JS, et al. An outbreak of acute gastroenteritis in a geriatric long-term-care facility: combined application of epidemiological and molecular diagnostic methods. *Infect Control Hosp Epidemiol* 1999;20(5):306-11.
280. Gellert GA, Waterman SH, Ewert D, et al. An outbreak of acute gastroenteritis caused by a small round structured virus in a geriatric convalescent facility. *Infect Control Hosp Epidemiol* 1990;11(9):459-64.
281. Cooper E, Blamey S. A Norovirus Gastroenteritis Epidemic in a Long-Term-Care Facility. *Infect Control Hosp Epidemiol* 2005;26(3):256.
282. Navarro G, Sala RM, Segura F, et al. An Outbreak of Norovirus Infection in a Long-Term-Care. *Infect Control Hosp Epidemiol* 2005;26(3):259.
283. Green KY, Belliot G, Taylor JL, et al. A predominant role for Norwalk-like viruses as agents of epidemic gastroenteritis in Maryland nursing homes for the elderly. *J Infect Dis* 2002;185(2):133-46.
284. Widdowson MA, Cramer EH, Hadley L, et al. Outbreaks of acute gastroenteritis on cruise ships and on land: identification of a predominant circulating strain of norovirus--United States, 2002. *J Infect Dis* 2004;190(1):27-36.
285. CDC. Outbreaks of gastroenteritis associated with noroviruses on cruise ships--United States, 2002. *MMWR Morb Mortal Wkly Rep* 2002;51(49):1112-5.
286. CDC. Norovirus outbreak among evacuees from hurricane Katrina--Houston, Texas, September 2005. *MMWR Morb Mortal Wkly Rep* 2005;54(40):1016-8

287. Mattner F, Mattner L, Borck HU, Gastmeier P. Evaluation of the Impact of the Source (Patient Versus Staff) on Nosocomial Norovirus Outbreak Severity. *Infect Control Hosp Epidemiol* 2005;26(3):268-72.
288. Isakbaeva ET, Bulens SN, Beard RS, et al. Norovirus and child care: challenges in outbreak control. *Pediatr Infect Dis J* 2005;24(6):561-3.
289. Kapikian AZ, Estes MK, Chanock RM. Norwalk group of viruses. In: Fields BN, Knipe DM, Howley PM, eds. *Fields virology*. 3rd ed. Philadelphia, PA: Lippincott-Raven. 1996:783-810.
290. Duizer E, Bijkerk P, Rockx B, De Groot A, Twisk F, Koopmans M. Inactivation of caliciviruses. *Appl Environ Microbiol* 2004;70(8):4538-43.
291. Doultree JC, Druce JD, Birch CJ, Bowden DS, Marshall JA. Inactivation of feline calicivirus, a Norwalk virus surrogate. *J Hosp Infect* 1999;41(1):51-7.
292. Barker J, Vipond IB, Bloomfield SF. Effects of cleaning and disinfection in reducing the spread of Norovirus contamination via environmental surfaces. *J Hosp Infect* 2004;58(1):42-9.
293. Gulati BR, Allwood PB, Hedberg CW, Goyal SM. Efficacy of commonly used disinfectants for the inactivation of calicivirus on strawberry, lettuce, and a food-contact surface. *J Food Prot* 2001;64(9):1430-4.
294. Gehrke C, Steinmann J, Goroncy-Bermes P. Inactivation of feline calicivirus, a surrogate of norovirus (formerly Norwalk-like viruses), by different types of alcohol in vitro and in vivo. *J Hosp Infect* 2004;56(1):49-55.
295. Hutson AM, Atmar RL, Graham DY, Estes MK. Norwalk virus infection and disease is associated with ABO histo-blood group type. *J Infect Dis* 2002;185(9):1335-7.
296. National Center for Infectious Diseases - Division of Viral and Rickettsial Diseases. (www.cdc.gov/ncidod/dvrd/index.htm)
297. LeDuc JW. Epidemiology of hemorrhagic fever viruses. *Rev Infect Dis* 1989;11 Suppl 4:S730-5.
298. Roels TH, Bloom AS, Buffington J, et al. Ebola hemorrhagic fever, Kikwit, Democratic Republic of the Congo, 1995: risk factors for patients without a reported exposure. *J Infect Dis* 1999 Feb;179 Suppl 1:S92-7.
299. Suleiman MN, Muscat-Baron JM, Harries JR, et al. Congo/Crimean haemorrhagic fever in Dubai. An outbreak at the Rashid Hospital. *Lancet* 1980;2(8201):939-41.
300. Monath TP, Mertens PE, Patton R, et al. A hospital epidemic of Lassa fever in Zorzor, Liberia, March-April 1972. *Am J Trop Med Hyg* 2005;54(40):1016-9

301. Dowell SF, Mukunu R, Ksiazek TG, Khan AS, Rollin PE, Peters CJ. Transmission of Ebola hemorrhagic fever: a study of risk factors in family members, Kikwit, Democratic Republic of the Congo, 1995. *J Infect Dis* 1999 Feb;179 Suppl 1:S87-91.
302. Peters CJ. Marburg and Ebola--arming ourselves against the deadly filoviruses. *N Engl J Med* 2005;352(25):2571-3.
303. Ebola haemorrhagic fever in Zaire, 1976. *Bull World Health Organ* 1978;56(2):271-93.
304. Emond RT, Evans B, Bowen ET, Lloyd G. A case of Ebola virus infection. *Br Med J* 1977;2(6086):541-4.
305. Zaki SR, Shieh WJ, Greer PW, et al. A novel immunohistochemical assay for the detection of Ebola virus in skin: implications for diagnosis, spread, and surveillance of Ebola hemorrhagic fever. Commission de Lutte contre les Epidemies a Kikwit. *J Infect Dis* 1999;179 Suppl 1:S36-47.
306. Khan AS, Tshioko FK, Heymann DL, et al. The reemergence of Ebola hemorrhagic fever, Democratic Republic of the Congo, 1995. Commission de Lutte contre les Epidemies a Kikwit. *J Infect Dis* 1999;179 Suppl 1:S76-86.
307. Muyembe-Tamfum JJ, Kipasa M, Kiyungu C, Colebunders R. Ebola outbreak in Kikwit, Democratic Republic of the Congo: discovery and control measures. *J Infect Dis* 1999;179 Suppl 1:S259-62.
308. Haas WH, Breuer T, Pfaff G, et al. Imported Lassa fever in Germany: surveillance and management of contact persons. *Clin Infect Dis* 2003;36(10):1254-8.
309. Simpson DI. Marburg agent disease: in monkeys. *Trans R Soc Trop Med Hyg* 1969;63(3):303-9.
310. Jaax NK, Davis KJ, Geisbert TJ, et al. Lethal experimental infection of rhesus monkeys with Ebola-Zaire (Mayinga) virus by the oral and conjunctival route of exposure. *Arch Pathol Lab Med* 1996;120(2):140-55.
311. Stephenson EH, Larson EW, Dominik JW. Effect of environmental factors on aerosol-induced Lassa virus infection. *J Med Virol* 1984;14(4):295-303.
312. Johnson E, Jaax N, White J, Jahrling P. Lethal experimental infections of rhesus monkeys by aerosolized Ebola virus. *Int J Exp Pathol* 1995;76(4):227-36.
313. Jaax N, Jahrling P, Geisbert T, et al. Transmission of Ebola virus (Zaire strain) to uninfected control monkeys in a biocontainment laboratory. *Lancet* 1995;346(8991-8992):1669-71.
314. www.bt.cdc.gov/agent/vhf/.

315. Nguyen GT, Proctor SE, Sinkowitz-Cochran RL, Garrett DO, Jarvis WR. Status of infection surveillance and control programs in the United States, 1992-1996. Association for Professionals in Infection Control and Epidemiology, Inc. *Am J Infect Control* 2000;28(6):392-400.
316. Richards C, Emori TG, Edwards J, Fridkin S, Tolson J, Gaynes R. Characteristics of hospitals and infection control professionals participating in the National Nosocomial Infections Surveillance System 1999. *Am J Infect Control* 2001;29(6):400-3.
317. Wenzel RP, Thompson RL, Landry SM, et al. Hospital-acquired infections in intensive care unit patients: an overview with emphasis on epidemics. *Infect Control* 1983;4(5):371-5.
318. Wenzel RP, Gennings C. Bloodstream infections due to *Candida* species in the intensive care unit: identifying especially high-risk patients to determine prevention strategies. *Clin Infect Dis* 2005;41 Suppl 6:S389-93.
319. San Miguel LG, Cobo J, Otheo E, Sanchez-Sousa A, Abreira V, Moreno S. Secular trends of candidemia in a large tertiary-care hospital from 1988 to 2000: emergence of *Candida parapsilosis*. *Infect Control Hosp Epidemiol* 2005;26(6):548-52.
320. NNIS. National Nosocomial Infections Surveillance (NNIS) System Report, data summary from January 1992 through June 2004, issued October 2004. *Am J Infect Control* 2004;32(8):470-85.
321. Richards MJ, Edwards JR, Culver DH, Gaynes RP. Nosocomial infections in medical intensive care units in the United States. National Nosocomial Infections Surveillance System. *Crit Care Med* 1999;27(5):887-92.
322. Richards MJ, Edwards JR, Culver DH, Gaynes RP. Nosocomial infections in combined medical-surgical intensive care units in the United States. *Infect Control Hosp Epidemiol* 2000;21(8):510-5.
323. Hugonnet S, Eggimann P, Borst F, Maricot P, Chevrolet JC, Pittet D. Impact of ventilator-associated pneumonia on resource utilization and patient outcome. *Infect Control Hosp Epidemiol* 2004;25(12):1090-6.
324. O'Neill JM, Schutze GE, Heulitt MJ, Simpson PM, Taylor BJ. Nosocomial infections during extracorporeal membrane oxygenation. *Intensive Care Med* 2001;27(8):1247-53.
325. Villegas MV, Hartstein AI. *Acinetobacter* Outbreaks, 1977–2000. *Infect Control Hosp Epidemiol* 2003;24(4):284-95.
326. Gordon SM, Schmitt SK, Jacobs M, et al. Nosocomial bloodstream infections in patients with implantable left ventricular assist devices. *Ann Thorac Surg* 2001;72(3):725-30.
327. Giamarellou H. Nosocomial cardiac infections. *J Hosp Infect* 2002;50(2):91-105.

328. Fridkin SK. Increasing prevalence of antimicrobial resistance in intensive care units. *Crit Care Med* 2001;29(4 Suppl):N64-8.
329. Kollef MH, Fraser VJ. Antibiotic resistance in the intensive care unit. *Ann Intern Med* 2001;134(4):298-314.
330. Fridkin SK, Edwards JR, Courval JM, et al. The effect of vancomycin and third-generation cephalosporins on prevalence of vancomycin-resistant enterococci in 126 U.S. adult intensive care units. *Ann Intern Med* 2001;135(3):175-83.
331. Crnich CJ, Safdar N, Maki DG. The role of the intensive care unit environment in the pathogenesis and prevention of ventilator-associated pneumonia. *Respir Care* 2005;50(6):813-36; discussion 36-8.
332. Knaus WA, Wagner DP, Zimmerman JE, Draper EA. Variations in mortality and length of stay in intensive care units. *Ann Intern Med* 1993;118(10):753-61.
333. Villarino ME, Stevens LE, Schable B, et al. Risk factors for epidemic *Xanthomonas maltophilia* infection/colonization in intensive care unit patients. *Infect Control Hosp Epidemiol* 1992;13(4):201-6.
334. Sanchez V, Vazquez JA, Barth-Jones D, Dembry L, Sobel JD, Zervos MJ. Nosocomial acquisition of *Candida parapsilosis*: an epidemiologic study. *Am J Med* 1993;94(6):577-82.
335. Husni RN, Goldstein LS, Arroliga AC, et al. Risk factors for an outbreak of multi-drug-resistant *Acinetobacter* nosocomial pneumonia among intubated patients. *Chest* 1999;115(5):1378-82.
336. McDonald LC, Walker M, Carson L, et al. Outbreak of *Acinetobacter* spp. bloodstream infections in a nursery associated with contaminated aerosols and air conditioners. *Pediatr Infect Dis J* 1998;17(8):716-22.
337. Trick WE, Kioski CM, Howard KM, et al. Outbreak of *Pseudomonas aeruginosa* ventriculitis among patients in a neurosurgical intensive care unit. *Infect Control Hosp Epidemiol* 2000;21(3):204-8.
338. Guidry GG, Black-Payne CA, Payne DK, Jamison RM, George RB, Bocchini JA, Jr. Respiratory syncytial virus infection among intubated adults in a university medical intensive care unit. *Chest* 1991;100(5):1377-84.
339. Wurtz R, Karajovic M, Dacumos E, Jovanovic B, Hanumadass M. Nosocomial infections in a burn intensive care unit. *Burns* 1995;21(3):181-4.
340. Rodgers GL, Mortensen J, Fisher MC, Lo A, Cresswell A, Long SS. Predictors of infectious complications after burn injuries in children. *Pediatr Infect Dis J* 2000;19(10):990-5.
341. Pruitt BA, Jr., McManus AT, Kim SH, Goodwin CW. Burn wound infections: current status. *World J Surg* 1998;22(2):135-45.

342. Weber J, ed. *Epidemiology of Infections and Strategies for Control in Burn Care and Therapy*. St. Louis: Mosby, Inc.; 1998.
343. Heggors JP, McCoy L, Reisner B, Smith M, Edgar P, Ramirez RJ. Alternate antimicrobial therapy for vancomycin-resistant enterococci burn wound infections. *J Burn Care Rehabil* 1998;19(5):399-403.
344. Sheridan RL, Weber J, Benjamin J, Pasternack MS, Tompkins RG. Control of methicillin-resistant *Staphylococcus aureus* in a pediatric burn unit. *Am J Infect Control* 1994;22(6):340-5.
345. Matsumura H, Yoshizawa N, Narumi A, Harunari N, Sugamata A, Watanabe K. Effective control of methicillin-resistant *Staphylococcus aureus* in a burn unit. *Burns* 1996;22(4):283-6.
346. McGregor JC. Profile of the first four years of the Regional Burn Unit based at St. John's Hospital, West Lothian (1992-1996). *J R Coll Surg Edinb* 1998;43(1):45-8.
347. Desai MH, Rutan RL, Heggors JP, Herndon DN. Candida infection with and without nystatin prophylaxis. A 11-year experience with patients with burn injury. *Arch Surg* 1992;127(2):159-62.
348. Ekenna O, Sherertz RJ, Bingham H. Natural history of bloodstream infections in a burn patient population: the importance of candidemia. *Am J Infect Control* 1993;21(4):189-95.
349. Bowser-Wallace BH, Graves DB, Caldwell FT. An epidemiological profile and trend analysis of wound flora in burned children: 7 years' experience. *Burns Incl Therm Inj* 1984;11(1):16-25.
350. Tredget EE, Shankowsky HA, Rennie R, Burrell RE, Logsetty S. Pseudomonas infections in the thermally injured patient. *Burns* 2004;30(1):3-26.
351. Edgar P, Mlcak R, Desai M, Linares HA, Phillips LG, Heggors JP. Containment of a multiresistant *Serratia marcescens* outbreak. *Burns* 1997;23(1):15-8.
352. Embil JM, McLeod JA, Al-Barrak AM, et al. An outbreak of methicillin resistant *Staphylococcus aureus* on a burn unit: potential role of contaminated hydrotherapy equipment. *Burns* 2001;27(7):681-8.
353. Meier PA, Carter CD, Wallace SE, Hollis RJ, Pfaller MA, Herwaldt LA. A prolonged outbreak of methicillin-resistant *Staphylococcus aureus* in the burn unit of a tertiary medical center. *Infect Control Hosp Epidemiol* 1996;17(12):798-802.
354. Snyder LL, Wiebelhaus P, Boon SE, Morin RA, Goering R. Methicillin-resistant *Staphylococcus aureus* eradication in a burn center. *J Burn Care Rehabil* 1993;14(2 Pt 1):164-8.
355. May AK, Melton SM, McGwin G, Cross JM, Moser SA, Rue LW. Reduction of vancomycin-resistant enterococcal infections by limitation of

- broad-spectrum cephalosporin use in a trauma and burn intensive care unit. *Shock* 2000;14(3):259-64.
356. Sheridan RL, Weber JM, Budkevich LG, Tompkins RG. Candidemia in the pediatric patient with burns. *J Burn Care Rehabil* 1995;16(4):440-3.
357. Mayhall CG. The epidemiology of burn wound infections: then and now. *Clin Infect Dis* 2003;37(4):543-50.
358. McManus AT, Mason AD, Jr., McManus WF, Pruitt BA, Jr. A decade of reduced gram-negative infections and mortality associated with improved isolation of burned patients. *Arch Surg* 1994;129(12):1306-9.
359. Bryce EA, Walker M, Scharf S, et al. An outbreak of cutaneous aspergillosis in a tertiary-care hospital. *Infect Control Hosp Epidemiol* 1996;17(3):170-2.
360. Levenson C, Wohlford P, Djou J, Evans S, Zawacki B. Preventing postoperative burn wound aspergillosis. *J Burn Care Rehabil* 1991;12(2):132-5.
361. Tredget EE, Shankowsky HA, Joffe AM, et al. Epidemiology of infections with *Pseudomonas aeruginosa* in burn patients: the role of hydrotherapy. *Clin Infect Dis* 1992;15(6):941-9.
362. Wisplinghoff H, Perbix W, Seifert H. Risk factors for nosocomial bloodstream infections due to *Acinetobacter baumannii*: a case-control study of adult burn patients. *Clin Infect Dis* 1999;28(1):59-66.
363. Weber JM, Sheridan RL, Schulz JT, Tompkins RG, Ryan CM. Effectiveness of bacteria-controlled nursing units in preventing cross-colonization with resistant bacteria in severely burned children. *Infect Control Hosp Epidemiol* 2002;23(9):549-51.
364. Bayat A, Shaaban H, Dodgson A, Dunn KW. Implications for Burns Unit design following outbreak of multi-resistant *Acinetobacter* infection in ICU and Burns Unit. *Burns* 2003;29(4):303-6.
365. Lee JJ, Marvin JA, Heimbach DM, Grube BJ, Engrav LH. Infection control in a burn center. *J Burn Care Rehabil* 1990;11(6):575-80.
366. Raymond J, Aujard Y. Nosocomial infections in pediatric patients: a European, multicenter prospective study. European Study Group. *Infect Control Hosp Epidemiol* 2000;21(4):260-3.
367. Campins M, Vaque J, Rossello J, et al. Nosocomial infections in pediatric patients: a prevalence study in Spanish hospitals. EPINE Working Group. *Am J Infect Control* 1993;21(2):58-63.
368. Allen U, Ford-Jones EL. Nosocomial infections in the pediatric patient: an update. *Am J Infect Control* 1990;18(3):176-93.

369. Grohskopf LA, Sinkowitz-Cochran RL, Garrett DO, et al. A national point-prevalence survey of pediatric intensive care unit-acquired infections in the United States. *J Pediatr* 2002;140(4):432-8.
370. Sohn AH, Garrett DO, Sinkowitz-Cochran RL, et al. Prevalence of nosocomial infections in neonatal intensive care unit patients: Results from the first national point-prevalence survey. *J Pediatr* 2001;139(6):821-7.
371. Gaynes RP, Edwards JR, Jarvis WR, Culver DH, Tolson JS, Martone WJ. Nosocomial infections among neonates in high-risk nurseries in the United States. National Nosocomial Infections Surveillance System. *Pediatrics* 1996;98(3 Pt 1):357-61.
372. Richards MJ, Edwards JR, Culver DH, Gaynes RP. Nosocomial infections in pediatric intensive care units in the United States. National Nosocomial Infections Surveillance System. *Pediatrics* 1999;103(4):e39.
373. Maltezou HC, Drancourt M. Nosocomial influenza in children. *J Hosp Infect* 2003;55(2):83-91.
374. Moisiuk SE, Robson D, Klass L, et al. Outbreak of parainfluenza virus type 3 in an intermediate care neonatal nursery. *Pediatr Infect Dis J* 1998;17(1):49-53.(mj).
375. Mullins JA, Erdman DD, Weinberg GA, et al. Human metapneumovirus infection among children hospitalized with acute respiratory illness. *Emerg Infect Dis* 2004;10(4):700-5.
376. Hatherill M, Levin M, Lawrenson J, Hsiao NY, Reynolds L, Argent A. Evolution of an adenovirus outbreak in a multidisciplinary children's hospital. *J Paediatr Child Health* 2004;40(8):449-54.
377. Langley JM, Hanakowski M. Variation in risk for nosocomial chickenpox after inadvertent exposure. *J Hosp Infect* 2000;44(3):224-6.
378. Ratner AJ, Neu N, Jakob K, et al. Nosocomial rotavirus in a pediatric hospital. *Infect Control Hosp Epidemiol* 2001;22(5):299-301.
379. Avila-Aguero ML, German G, Paris MM, Herrera JF. Toys in a pediatric hospital: are they a bacterial source? *Am J Infect Control* 2004;32(5):287-90.
380. Nyqvist KH, Lutes LM. Co-bedding twins: a developmentally supportive care strategy. *J Obstet Gynecol Neonatal Nurs* 1998;27(4):450-6.
381. Feldman R, Eidelman AI, Sirota L, Weller A. Comparison of skin-to-skin (kangaroo) and traditional care: parenting outcomes and preterm infant development. *Pediatrics* 2002;110(1 Pt 1):16-26.
382. Conde-Agudelo A, Diaz-Rossello JL, Belizan JM. Kangaroo mother care to reduce morbidity and mortality in low birthweight infants. *Cochrane Database Syst Rev* 2003(2):CD002771.

383. Adcock PM, Pastor P, Medley F, Patterson JE, Murphy TV. Methicillin-resistant *Staphylococcus aureus* in two child care centers. *J Infect Dis* 1998;178(2):577-80.
384. Shahin R, Johnson IL, Jamieson F, McGeer A, Tolkin J, Ford-Jones EL. Methicillin-resistant *Staphylococcus aureus* carriage in a child care center following a case of disease. Toronto Child Care Center Study Group. *Arch Pediatr Adolesc Med* 1999;153(8):864-8.
385. Stover BH, Duff A, Adams G, Buck G, Hancock G, Rabalais G. Emergence and control of methicillin-resistant *Staphylococcus aureus* in a children's hospital and pediatric long-term care facility. *Am J Infect Control* 1992;20(5):248-55.
386. Herold BC, Immergluck LC, Maranan MC, et al. Community-acquired methicillin-resistant *Staphylococcus aureus* in children with no identified predisposing risk. *JAMA* 1998;279(8):593-8.
387. CDC. Four pediatric deaths from community-acquired methicillin-resistant *Staphylococcus aureus*--Minnesota and North Dakota, 1997-1999. *MMWR - Morbidity & Mortality Weekly Report* 1999;48:707-10.
388. Abi-Hanna P, Frank AL, Quinn JP, et al. Clonal features of community-acquired methicillin-resistant *Staphylococcus aureus* in children. *Clin Infect Dis* 2000;30(3):630-1.
389. Fergie JE, Purcell K. Community-acquired methicillin-resistant *Staphylococcus aureus* infections in south Texas children. *Pediatr Infect Dis J* 2001;20(9):860-3.
390. Sattler CA, Mason EO, Jr., Kaplan SL. Prospective comparison of risk factors and demographic and clinical characteristics of community-acquired, methicillin-resistant versus methicillin-susceptible *Staphylococcus aureus* infection in children. *Pediatr Infect Dis J* 2002;21(10):910-7.
391. Kaplan SL, Hulten KG, Gonzalez BE, et al. Three-year surveillance of community-acquired *Staphylococcus aureus* infections in children. *Clin Infect Dis* 2005;40(12):1785-91.
392. Jarvis WR. Infection control and changing health-care delivery systems. *Emerg Infect Dis* 2001;7(2):170-3.
393. Garibaldi RA. Residential care and the elderly: the burden of infection. *J Hosp Infect* 1999;43 Suppl:S9-18.
394. Hsu K, Harris JA. Control of infections in nonacute care pediatric settings. *Seminars Pedi Infect Dis* 2001;12(2):92-9.
395. Straus C, Singh L, Joseph G, et al. The 2000-2001 influenza pandemic in long-term care. *Staus C, Singh L, Joseph G, et al. The 2000-2001 influenza pandemic in long-term care.*

396. Centers for Medicare and Medicaid Services. Healthcare Industry Market Update. http://www.cms.hhs.gov/reports/hcimu/hcimu_05202003pdf May 2003.
397. Lee YL, Thrupp LD, Friis RH, Fine M, Maleki P, Cesario TC. Nosocomial infection and antibiotic utilization in geriatric patients: a pilot prospective surveillance program in skilled nursing facilities. *Gerontology* 1992;38(4):223-32.
398. Stevenson KB. Regional data set of infection rates for long-term care facilities: description of a valuable benchmarking tool. *Am J Infect Control* 1999;27(1):20-6.
399. Jackson MM, Fierer J, Barrett-Connor E, et al. Intensive surveillance for infections in a three-year study of nursing home patients. *Am J Epidemiol* 1992;135(6):685-96.
400. Darnowski SB, Gordon M, Simor AE. Two years of infection surveillance in a geriatric long-term care facility. *Am J Infect Control* 1991;19(4):185-90.
401. Hoffman N, Jenkins R, Putney K. Nosocomial infection rates during a one-year period in a nursing home care unit of a Veterans Administration hospital. *Am J Infect Control* 1990;18(2):55-63.
402. Tsan L, Hojlo C, Kearns MA, et al. Infection surveillance and control programs in the Department of Veterans Affairs nursing home care units: a preliminary assessment. *Am J Infect Control* 2006;34(2):80-3.
403. Kane RA, Caplan AL, Urv-Wong EK, Freeman IC, Aroskar MA, Finch M. Everyday matters in the lives of nursing home residents: wish for and perception of choice and control. *J Am Geriatr Soc* 1997;45:1086-93.
404. Libow LS, Starer P. Care of the nursing home patient. *N Engl J Med* 1989;321(2):93-6.
405. Perls TT, Herget M. Higher respiratory infection rates on an Alzheimer's special care unit and successful intervention. *J Am Geriatr Soc* 1995;43(12):1341-4.
406. Bradley SF. Issues in the management of resistant bacteria in long-term-care facilities. *Infect Control Hosp Epidemiol* 1999;20(5):362-6.
407. Crossley K. Vancomycin-resistant enterococci in long-term-care facilities. *Infect Control Hosp Epidemiol* 1998;19(7):521-5.
408. Strausbaugh LJ, Crossley KB, Nurse BA, Thrupp LD. Antimicrobial resistance in long-term-care facilities. *Infect Control Hosp Epidemiol* 1996;17(2):129-40.
409. Richards CL. Infections in Long-Term-Care Facilities: Screen or Clean? *Infect Control Hosp Epidemiol* 2005;26(10):800-01.

410. Drinka PJ, Krause P, Nest L, Goodman BM, Gravenstein S. Risk of acquiring influenza A in a nursing home from a culture-positive roommate. *Infect Control Hosp Epidemiol* 2003;24(11):872-4.
411. Falsey AR, Treanor JJ, Betts RF, Walsh EE. Viral respiratory infections in the institutionalized elderly: clinical and epidemiologic findings. *J Am Geriatr Soc* 1992;40(2):115-9.
412. Ellis SE, Coffey CS, Mitchel EF, Jr., Dittus RS, Griffin MR. Influenza- and respiratory syncytial virus-associated morbidity and mortality in the nursing home population. *J Am Geriatr Soc* 2003;51(6):761-7.
413. Louie JK, Yagi S, Nelson FA, et al. Rhinovirus outbreak in a long term care facility for elderly persons associated with unusually high mortality. *Clin Infect Dis* 2005;41(2):262-5.
414. Piednoir E, Bureau-Chalot F, Merle C, Gotzamanis A, Wuibout J, Bajolet O. Direct costs associated with a nosocomial outbreak of adenoviral conjunctivitis infection in a long-term care institution. *Am J Infect Control* 2002;30(7):407-10.
415. Addiss DG, Davis JP, Meade BD, et al. A pertussis outbreak in a Wisconsin nursing home. *J Infect Dis* 1991;164(4):704-10.
416. Gaynes R, Rimland D, Killum E, et al. Outbreak of *Clostridium difficile* infection in a long-term care facility: association with gatifloxacin use. *Clin Infect Dis* 2004;38(5):640-5.
417. High KP, Bradley S, Loeb M, Palmer R, Quagliarello V, Yoshikawa T. A new paradigm for clinical investigation of infectious syndromes in older adults: assessment of functional status as a risk factor and outcome measure. *Clin Infect Dis* 2005;40(1):114-22.
418. Loeb MB, Craven S, McGeer AJ, et al. Risk factors for resistance to antimicrobial agents among nursing home residents. *Am J Epidemiol* 2003;157(1):40-7.
419. Vergis EN, Brennen C, Wagener M, Muder RR. Pneumonia in long-term care: a prospective case-control study of risk factors and impact on survival. *Arch Intern Med* 2001;161(19):2378-81.
420. Loeb M, McGeer A, McArthur M, Walter S, Simor AE. Risk factors for pneumonia and other lower respiratory tract infections in elderly residents of long-term care facilities. *Arch Intern Med* 1999;159(17):2058-64.
421. Brandeis GH, Ooi WL, Hossain M, Morris JN, Lipsitz LA. A longitudinal study of risk factors associated with the formation of pressure ulcers in nursing homes. *J Am Geriatr Soc* 1994;42(4):388-93.
422. Allard JP, Aghdassi E, McArthur M, et al. Nutrition risk factors for survival in the elderly living in Canadian long-term care facilities. *J Am Geriatr Soc* 2004;52(1):59-65.

423. Pick N, McDonald A, Bennett N, et al. Pulmonary aspiration in a long-term care setting: clinical and laboratory observations and an analysis of risk factors. *J Am Geriatr Soc* 1996;44(7):763-8.
424. Nicolle LE. The chronic indwelling catheter and urinary infection in long-term-care facility residents. *Infect Control Hosp Epidemiol* 2001;22(5):316-21.
425. Pien EC, Hume KE, Pien FD. Gastrostomy tube infections in a community hospital. *Am J Infect Control* 1996;24(5):353-8.
426. Gomes GF, Pisani JC, Macedo ED, Campos AC. The nasogastric feeding tube as a risk factor for aspiration and aspiration pneumonia. *Curr Opin Clin Nutr Metab Care* 2003;6(3):327-33.
427. Bula CJ, Ghilardi G, Wietlisbach V, Petignat C, Francioli P. Infections and functional impairment in nursing home residents: a reciprocal relationship. *J Am Geriatr Soc* 2004;52(5):700-6.
428. Bradley SF, Terpenning MS, Ramsey MA, et al. Methicillin-resistant *Staphylococcus aureus*: colonization and infection in a long-term care facility. *Ann Intern Med* 1991;115(6):417-22.
429. Washio M, Nishisaka S, Kishikawa K, et al. Incidence of methicillin-resistant *Staphylococcus aureus* (MRSA) isolation in a skilled nursing home: a third report on the risk factors for the occurrence of MRSA infection in the elderly. *J Epidemiol* 1996;6(2):69-73.
430. Trick WE, Weinstein RA, DeMarais PL, et al. Colonization of skilled-care facility residents with antimicrobial-resistant pathogens. *J Am Geriatr Soc* 2001;49(3):270-6.
431. Nicolle LE, Garibaldi RA. Infection control in long-term-care facilities. *Infect Control Hosp Epidemiol* 1995;16(6):348-53.(s).
432. Crossley K. Long-term care facilities as sources of antibiotic-resistant nosocomial pathogens. *Curr Opin Infect Dis* 2001;14(4):455-9.
433. Smith PW, Rusnak PG. Infection prevention and control in the long-term-care facility. SHEA Long-Term-Care Committee and APIC Guidelines Committee. *Infect Control Hosp Epidemiol* 1997;18(12):831-49.(s).
434. Friedman C, Barnette M, Buck AS, et al. Requirements for infrastructure and essential activities of infection control and epidemiology in out-of-hospital settings: a consensus panel report. Association for Professionals in Infection Control and Epidemiology and Society for Healthcare Epidemiology of America. *Infect Control Hosp Epidemiol* 1999;20(10):695-705.
435. Nicolle LE. Infection control in long-term care facilities. *Clin Infect Dis* 2000;31(3):752-6.
436. Bradley SF. Methicillin-resistant *Staphylococcus aureus*: long-term care concerns. *Am J Med* 1999;106(5A):2S-10S; discussion 48S-52S.

437. www.cms.hhs.gov/manuals/Downloads/som107ap_pp_guidelines_ltcf
438. Mylotte JM, Goodnough S, Tayara A. Antibiotic-resistant organisms among long-term care facility residents on admission to an inpatient geriatrics unit: Retrospective and prospective surveillance. *Am J Infect Control* 2001;29(3):139-44.
439. Strausbaugh LJ, Jacobson C, Yost T. Methicillin-resistant *Staphylococcus aureus* in a nursing home and affiliated hospital: a four-year perspective. *Infect Control Hosp Epidemiol* 1993;14(6):331-6.
440. Wiener J, Quinn JP, Bradford PA, et al. Multiple antibiotic-resistant *Klebsiella* and *Escherichia coli* in nursing homes. *Jama* 1999;281(6):517-23.
441. Pop-Vicas AE, D'Agata EM. The rising influx of multidrug-resistant gram-negative bacilli into a tertiary care hospital. *Clin Infect Dis* 2005;40(12):1792-8.
442. Ly N, McCaig LF. National Hospital Ambulatory Medical Care Survey: 2000 outpatient department summary. *Adv Data* 2002(327):1-27.
443. Cherry DK, Woodwell DA. National Ambulatory Medical Care Survey: 2000 summary. *Adv Data* 2002(328):1-32.
444. Finelli L, Miller JT, Tokars JI, Alter MJ, Arduino MJ. National surveillance of dialysis-associated diseases in the United States, 2002. *Semin Dial* 2005;18(1):52-61.
445. D'Agata EM. Antimicrobial-resistant, Gram-positive bacteria among patients undergoing chronic hemodialysis. *Clin Infect Dis* 2002;35(10):1212-8.
446. Goodman RA, Solomon SL. Transmission of infectious diseases in outpatient health care settings. *JAMA* 1991;265(18):2377-81.
447. Nafziger DA, Lundstrom T, Chandra S, Massanari RM. Infection control in ambulatory care. *Infect Dis Clin North Am* 1997;11(2):279-96.
448. Herwaldt LA, Smith SD, Carter CD. Infection control in the outpatient setting. *Infect Control Hosp Epidemiol* 1998;19(1):41-74.
449. Hlady WG, Hopkins RS, Ogilby TE, Allen ST. Patient-to-patient transmission of hepatitis B in a dermatology practice. *Am J Public Health* 1993;83(12):1689-93.
450. Birnie GG, Quigley EM, Clements GB, Follet EA, Watkinson G. Endoscopic transmission of hepatitis B virus. *Gut* 1983;24(2):171-4.
451. Chant K, Lowe D, Rubin G, et al. Patient-to-patient transmission of HIV in private surgical consulting rooms. *Lancet* 1993;342(8886-8887):1548-9.
452. Chant K, Kociuba K, Munro R, et al. Investigation of Possible Patient-to-Patient Transmission of Hepatitis C in a Hospital. *NSW Public Health Bull* 1994;5(5):47-51.

453. CDC. Transmission of hepatitis B and C viruses in outpatient settings-- New York, Oklahoma, and Nebraska, 2000-2002. MMWR Morb Mortal Wkly Rep 2003;52(38):901-6.
454. Williams IT, Perz JF, Bell BP. Viral hepatitis transmission in ambulatory health care settings. Clin Infect Dis 2004;38(11):1592-8.
455. Couldwell DL, Dore GJ, Harkness JL, et al. Nosocomial outbreak of tuberculosis in an outpatient HIV treatment room. Aids 1996;10(5):521-5.
456. CDC. Mycobacterium tuberculosis Transmission in a Health Clinic -- Florida. MMWR 1989;38(15):256-8; 63-64.
457. Calder RA, Duclos P, Wilder MH, Pryor VL, Scheel WJ. Mycobacterium tuberculosis transmission in a health clinic. Bull Int Union Tuberc Lung Dis 1991;66(2-3):103-6.
458. Istre GR, McKee PA, West GR, et al. Measles spread in medical settings: an important focus of disease transmission? Pediatrics 1987;79(3):356-8.
459. Dawson C, Darrell R. Infections due to adenovirus type 8 in the United States. I. An outbreak of epidemic keratoconjunctivitis originating in a physician's office. N Engl J Med 1963;268:1031-4.
460. Montessori V, Scharf S, Holland S, Werker DH, Roberts FJ, Bryce E. Epidemic keratoconjunctivitis outbreak at a tertiary referral eye care clinic. Am J Infect Control 1998;26(4):399-405.
461. Jernigan JA, Lowry BS, Hayden FG, et al. Adenovirus type 8 epidemic keratoconjunctivitis in an eye clinic: risk factors and control. J Infect Dis 1993;167(6):1307-13.
462. Buehler JW, Finton RJ, Goodman RA, et al. Epidemic keratoconjunctivitis: report of an outbreak in an ophthalmology practice and recommendations for prevention. Infect Control 1984;5(8):390-4.
463. Johnston CP, Cooper L, Ruby W, Teeter T, al. E. Community-associated methicillin resistant *Staphylococcus aureus* skin infections among outpatient healthcare workers and its isolation in the clinic environment. Presented at the 15th Annual Scientific Meeting of the Society for Healthcare Epidemiology of America (SHEA), Los Angeles, California, 4/10/05. Abstract #132 2005.
464. Biddick R, Spilker T, Martin A, LiPuma JJ. Evidence of transmission of *Burkholderia cepacia*, *Burkholderia multivorans* and *Burkholderia dolosa* among persons with cystic fibrosis. FEMS Microbiol Lett 2003;228(1):57-62.
465. Griffiths AL, Jansen K, Carlin JB, et al. Effects of segregation on an epidemic *Pseudomonas aeruginosa* strain in a cystic fibrosis clinic. Am J Respir Crit Care Med 2005;171(9):1020-5.

466. Danzig LE, Short LJ, Collins K, et al. Bloodstream infections associated with a needleless intravenous infusion system in patients receiving home infusion therapy. *JAMA* 1995;273(23):1862-4.
467. Kellerman S, Shay DK, Howard J, et al. Bloodstream infections in home infusion patients: the influence of race and needleless intravascular access devices. *J Pediatr* 1996;129(5):711-7.
468. Do AN, Ray BJ, Banerjee SN, et al. Bloodstream infection associated with needleless device use and the importance of infection-control practices in the home health care setting. *J Infect Dis* 1999;179(2):442-8.
469. Tokars JI, Cookson ST, McArthur MA, Boyer CL, McGeer AJ, Jarvis WR. Prospective evaluation of risk factors for bloodstream infection in patients receiving home infusion therapy. *Ann Intern Med* 1999;131(5):340-7.
470. Manangan LP, Pearson ML, Tokars JI, Miller E, Jarvis WR. Feasibility of national surveillance of health-care-associated infections in home-care settings. *Emerg Infect Dis* 2002;8(3):233-6.
471. Shah SS, Manning ML, Leahy E, Magnusson M, Rheingold SR, Bell LM. Central venous catheter-associated bloodstream infections in pediatric oncology home care. *Infect Control Hosp Epidemiol* 2002;23(2):99-101.
472. Gorski LA. Central venous access device outcomes in a homecare agency: a 7-year study. *J Infus Nurs* 2004;27(2):104-11.
473. Rosenheimer L, Embry FC, Sanford J, Silver SR. Infection surveillance in home care: device-related incidence rates. *Am J Infect Control* 1998;26(3):359-63.
474. White MC, Ragland KE. Surveillance of intravenous catheter-related infections among home care clients. *Am J Infect Control* 1994;22(4):231-5.
475. Beltrami EM, McArthur MA, McGeer A, et al. The nature and frequency of blood contacts among home healthcare workers. *Infect Control Hosp Epidemiol* 2000;21(12):765-70.
476. Embry FC, Chinnes LF. Draft definitions for surveillance of infections in home health care. *Am J Infect Control* 2000;28(6):449-53.
477. Fraser TG, Stosor V, Wang Q, Allen A, Zembower TR. Vancomycin and home health care. *Emerg Infect Dis* 2005;11(10):1558-64.
478. Carrico RM, Niner S. Multidrug resistant organisms--VRE and MRSA: practical home care tips. *Home Healthc Nurse* 2002;20(1):23-8; quiz 8-9.
479. Friedman MM, Rhinehart E. Improving infection control in home care: from ritual to science-based practice. *Home Healthc Nurse* 2000;18(2):99-105; quiz 6.

480. Friedman MM, Rhinehart E. Putting infection control principles into practice in home care. *Nurs Clin North Am* 1999;34(2):463-82.
481. Davis PL, Madigan EA. Evidence-based practice and the home care nurse's bag. *Home Healthc Nurse* 1999;17(5):295-9.
482. Sitzman KL, Pett MA, Blosswick DS. An exploratory study of nurse bag use by home visiting nurses. *Home Healthc Nurse* 2002;20(4):237-43.
483. Anderson MA, Madigan EA, Helms LB. Nursing research in home health care: endangered species? *Home Care Provid* 2001;6(6):200-4.
484. White MC. Identifying infectious diseases in prisons: surveillance, protection, and intervention. *West J Med* 1999;170(3):177.
485. Puisis M. Update on public health in correctional facilities. *West J Med* 1998;169(6):374.
486. Levy MH, Lerwitworapong J. Issues facing TB control (3.1). Tuberculosis in prisons. *Scott Med J* 2000;45(5 Suppl):30-2; discussion 3.
487. Parece MS, Herrera GA, Voigt RF, Middlekauff SL, Irwin KL. STD testing policies and practices in U.S. city and county jails. *Sex Transm Dis* 1999;26(8):431-7.
488. Cieslak PR, Curtis MB, Coulombier DM, Hathcock AL, Bean NH, Tauxe RV. Preventable disease in correctional facilities. Desmoteriic foodborne outbreaks in the United States, 1974-1991. *Arch Intern Med* 1996;156(16):1883-8.
489. CDC. Public health dispatch: tuberculosis outbreak in a homeless population--Portland, Maine, 2002-2003. *MMWR Morb Mortal Wkly Rep* 2003;52(48):1184.
490. CDC. Public health dispatch: tuberculosis outbreak among homeless persons--King County, Washington, 2002-2003. *MMWR Morb Mortal Wkly Rep* 2003;52(49):1209-10.
491. CDC. Tuberculosis transmission in a homeless shelter population--New York, 2000-2003. *MMWR Morb Mortal Wkly Rep* 2005;54(6):149-52.
492. Baillargeon J, Kelley MF, Leach CT, Baillargeon G, Pollock BH. Methicillin-resistant *Staphylococcus aureus* infection in the Texas prison system. *Clin Infect Dis* 2004;38(9):e92-5.
493. Young DM, Harris HW, Charlebois ED, et al. An epidemic of methicillin-resistant *Staphylococcus aureus* soft tissue infections among medically underserved patients. *Arch Surg* 2004;139(9):947-51; discussion 51-3.
494. Pan ES, Diep BA, Carleton HA, et al. Increasing prevalence of methicillin-resistant *Staphylococcus aureus* infection in California jails. *Clin Infect Dis* 2003;37(10):1384-8.

495. CDC. Drug-susceptible tuberculosis outbreak in a state correctional facility housing HIV-infected inmates--South Carolina, 1999-2000. *MMWR Morb Mortal Wkly Rep* 2000;49(46):1041-4.
496. CDC. Methicillin-resistant *Staphylococcus aureus* skin or soft tissue infections in a state prison--Mississippi, 2000. *MMWR Morb Mortal Wkly Rep* 2001;50(42):919-22.
497. Mohle-Boetani JC, Miguelino V, Dewsnup DH, et al. Tuberculosis outbreak in a housing unit for human immunodeficiency virus-infected patients in a correctional facility: transmission risk factors and effective outbreak control. *Clin Infect Dis* 2002;34(5):668-76.
498. CDC. Prevention and Control of Tuberculosis in Correctional Facilities. Recommendations of the Advisory Council for the Elimination of Tuberculosis. *MMWR Recomm Rep* 1996;45(RR-8):1-37.
499. Whimbey E, Englund JA, Couch RB. Community respiratory virus infections in immunocompromised patients with cancer. *Am J Med* 1997;102(3A):10-8; discussion 25-6.
500. Zambon M, Bull T, Sadler CJ, Goldman JM, Ward KN. Molecular epidemiology of two consecutive outbreaks of parainfluenza 3 in a bone marrow transplant unit. *J Clin Microbiol* 1998;36(8):2289-93.
501. Gams AS, Howells WB, DeSwarte-Wallace J, Feusner JH, Buckley JD, Woods WG. Alpha hemolytic streptococcal infection during intensive treatment for acute myeloid leukemia: a report from the Children's cancer group study CCG-2891. *J Clin Oncol* 2000;18(9):1845-55.
502. Ek T, Mellander L, Andersson B, Abrahamsson J. Immune reconstitution after childhood acute lymphoblastic leukemia is most severely affected in the high risk group. *Pediatr Blood Cancer* 2005;44(5):461-8.
503. Pascual V, Allantaz F, Arce E, Punaro M, Banchereau J. Role of interleukin-1 (IL-1) in the pathogenesis of systemic onset juvenile idiopathic arthritis and clinical response to IL-1 blockade. *J Exp Med* 2005;201(9):1479-86.
504. Marchesoni A, Puttini PS, Gorla R, et al. Cyclosporine in addition to infliximab and methotrexate in refractory rheumatoid arthritis. *Clin Exp Rheumatol* 2005;23(6):916-7.
505. Isaacs KL, Lewis JD, Sandborn WJ, Sands BE, Targan SR. State of the art: IBD therapy and clinical trials in IBD. *Inflamm Bowel Dis* 2005;11 Suppl 1:S3-12.
506. CDC. Guidelines for preventing opportunistic infections among HIV-infected persons. *MMWR Morb Mortal Wkly Rep* 2002;51 (RR-8):1-52.
507. Kusne S, and Krystofak S. Infection control issues after solid organ transplantation in transplant infections (Second Edition), ed. Bowden RA,

Ljungman P, Paya CV. Lippincott, Williams and Wilkins. Philadelphia; 2003.

508. Anderson D, DeFor T, Burns L, et al. A comparison of related donor peripheral blood and bone marrow transplants: importance of late-onset chronic graft-versus-host disease and infections. *Biol Blood Marrow Transplant* 2003;9(1):52-9.
509. Pitchford KC, Corey M, Highsmith AK, et al. *Pseudomonas* species contamination of cystic fibrosis patients' home inhalation equipment. *J Pediatr* 1987;111(2):212-6.
510. Hamill RJ, Houston ED, Georghiou PR, et al. An outbreak of *Burkholderia* (formerly *Pseudomonas*) *cepacia* respiratory tract colonization and infection associated with nebulized albuterol therapy. *Ann Intern Med* 1995;122(10):762-6.
511. Hutchinson GR, Parker S, Pryor JA, et al. Home-use nebulizers: a potential primary source of *Burkholderia cepacia* and other colistin-resistant, gram-negative bacteria in patients with cystic fibrosis. *J Clin Microbiol* 1996;34(3):584-7.
512. Jakobsson BM, Onnered AB, Hjelte L, Nystrom B. Low bacterial contamination of nebulizers in home treatment of cystic fibrosis patients. *J Hosp Infect* 1997;36(3):201-7.
513. Rosenfeld M, Joy P, Nguyen CD, Krzewinski J, Burns JL. Cleaning home nebulizers used by patients with cystic fibrosis: is rinsing with tap water enough? *J Hosp Infect* 2001;49(3):229-30.
514. Govan JR. Infection control in cystic fibrosis: methicillin-resistant *Staphylococcus aureus*, *Pseudomonas aeruginosa* and the *Burkholderia cepacia* complex. *J R Soc Med* 2000;93(Suppl 38):40-5.
515. Frederiksen B, Koch C, Hoiby N. Changing epidemiology of *Pseudomonas aeruginosa* infection in Danish cystic fibrosis patients (1974-1995). *Pediatr Pulmonol* 1999;28(3):159-66.
516. Isles A, Maclusky I, Corey M, et al. *Pseudomonas cepacia* infection in cystic fibrosis: an emerging problem. *J Pediatr* 1984;104(2):206-10.
517. LiPuma JJ. *Burkholderia cepacia*. Management issues and new insights. *Clin Chest Med* 1998;19(3):473-86, vi.
518. Tablan OC, Chorba TL, Schidlow DV, et al. *Pseudomonas cepacia* colonization in patients with cystic fibrosis: risk factors and clinical outcome. *J Pediatr* 1985;107(3):382-7.
519. Hudson VL, Wielinski CL, Regelman WE. Prognostic implications of initial oropharyngeal bacterial flora in patients with cystic fibrosis diagnosed before the age of two years. *J Pediatr* 1993;122(6):854-60.

520. Farrell PM, Li Z, Kosorok MR, et al. Bronchopulmonary disease in children with cystic fibrosis after early or delayed diagnosis. *Am J Respir Crit Care Med* 2003;168(9):1100-8.
521. Smith DL, Gumery LB, Smith EG, Stableforth DE, Kaufmann ME, Pitt TL. Epidemic of *Pseudomonas cepacia* in an adult cystic fibrosis unit: evidence of person-to-person transmission. *J Clin Microbiol* 1993;31(11):3017-22.js.
522. Pegues DA, Schidlow DV, Tablan OC, Carson LA, Clark NC, Jarvis WR. Possible nosocomial transmission of *Pseudomonas cepacia* in patients with cystic fibrosis. *Arch Pediatr Adolesc Med* 1994;148(8):805-12.
523. Govan JR, Brown PH, Maddison J, et al. Evidence for transmission of *Pseudomonas cepacia* by social contact in cystic fibrosis. *Lancet* 1993;342(8862):15-9.
524. Pegues DA, Carson LA, Tablan OC, et al. Acquisition of *Pseudomonas cepacia* at summer camps for patients with cystic fibrosis. Summer Camp Study Group. *J Pediatr* 1994;124(5 Pt 1):694-702.
525. Tablan OC, Martone WJ, Doershuk CF, et al. Colonization of the respiratory tract with *Pseudomonas cepacia* in cystic fibrosis. Risk factors and outcomes. *Chest* 1987;91(4):527-32.
526. Thomassen MJ, Demko CA, Doershuk CF, Stern RC, Klinger JD. *Pseudomonas cepacia*: decrease in colonization in patients with cystic fibrosis. *Am Rev Respir Dis* 1986;134(4):669-71.js.
527. Weber DJ, Rutala WA. Gene therapy: a new challenge for infection control. *Infect Control Hosp Epidemiol* 1999;20(8):530-2.
528. Evans ME, Lesnaw JA. Infection control for gene therapy: a busy physician's primer. *Clin Infect Dis* 2002;35(5):597-605.
529. Strausbaugh LJ. Gene therapy and infection control: more light on the way. *Infect Control Hosp Epidemiol* 2000;21(10):630-2.
530. CDC. West Nile virus infections in organ transplant recipients--New York and Pennsylvania, August-September, 2005. *MMWR Morb Mortal Wkly Rep* 2005;54(40):1021-3.
531. Lawson CA. Cytomegalovirus after kidney transplantation: a case review. *Prog Transplant* 2005;15(2):157-60.
532. Tugwell BD, Patel PR, Williams IT, et al. Transmission of hepatitis C virus to several organ and tissue recipients from an antibody-negative donor. *Ann Intern Med* 2005;143(9):648-54.
533. Kainer MA, Linden JV, Whaley DN, et al. *Clostridium* infections associated with musculoskeletal-tissue allografts. *N Engl J Med* 2004;350(25):2564-71.

534. CDC. Invasive *Streptococcus pyogenes* after allograft implantation-- Colorado, 2003. *MMWR Morb Mortal Wkly Rep* 2003;52(48):1174-6.
535. Mungai M, Tegmeier G, Chamberland M, Parise M. Transfusion-transmitted malaria in the United States from 1963 through 1999. *N Engl J Med* 2001;344(26):1973-8.
536. Lux JZ, Weiss D, Linden JV, et al. Transfusion-associated babesiosis after heart transplant. *Emerg Infect Dis* 2003;9(1):116-9.
537. CDC. Chagas Disease After Organ Transplantation --- United States, 2001. *MMWR* 2002;51(10):210-2.
538. CDC. Lymphocytic choriomeningitis virus infection in organ transplant recipients--Massachusetts, Rhode Island, 2005. *MMWR Morb Mortal Wkly Rep* 2005;54(21):537-9.
539. Srinivasan A, Burton EC, Kuehnert MJ, et al. Transmission of rabies virus from an organ donor to four transplant recipients. *N Engl J Med* 2005;352(11):1103-11.
540. Gottesdiener KM. Transplanted infections: donor-to-host transmission with the allograft. *Ann Intern Med* 1989;110(12):1001-16.
541. Borie DC, Cramer DV, Phan-Thanh L, et al. Microbiological hazards related to xenotransplantation of porcine organs into man. *Infect Control Hosp Epidemiol* 1998;19(5):355-65.
542. CDC. U.S. Public Health Service Guideline on Infectious Disease Issues in Xenotransplantation. Centers for Disease Control and Prevention. *MMWR - Morbidity & Mortality Weekly Report* 2001;50 (RR-15)(August):1-46.
543. IOM. Institute of Medicine. To err is human: building a safer health system. Washington DC National Academy Press; 1999;<http://www.iom.edu/report.asp?id=5575>.
544. Gerberding JL. Hospital-onset infections: a patient safety issue. *Ann Intern Med* 2002;137(8):665-70.
545. Leape LL, Berwick DM, Bates DW. What practices will most improve safety? Evidence-based medicine meets patient safety. *JAMA* 2002;288(4):501-7.
546. Burke JP. Patient safety: infection control - a problem for patient safety. *N Engl J Med* 2003;348(7):651-6.
547. Shulman L, Ost D. Managing infection in the critical care unit: how can infection control make the ICU safe? *Crit Care Clin* 2005;21(1):111-28, ix.
548. Goldmann DA, Weinstein RA, Wenzel RP, et al. Strategies to Prevent and Control the Emergence and Spread of Antimicrobial-Resistant

- Microorganisms in Hospitals. A challenge to hospital leadership. *JAMA* 1996;275(3):234-40.
549. Scheckler WE, Brimhall D, Buck AS, et al. Requirements for infrastructure and essential activities of infection control and epidemiology in hospitals: a consensus panel report. Society for Healthcare Epidemiology of America. *Infect Control Hosp Epidemiol* 1998;19(2):114-24.
550. www.jointcommission.org/PatientSafety/NationalPatientSafetyGoals/
551. Jackson M, Chiarello LA, Gaynes RP, Gerberding JL. Nurse staffing and health care-associated infections: Proceedings from a working group meeting. *Am J Infect Control* 2002;30(4):199-206.
552. O'Boyle C, Jackson M, Henly SJ. Staffing requirements for infection control programs in US health care facilities: Delphi project. *Am J Infect Control* 2002;30(6):321-33.
553. Peterson LR, Hamilton JD, Baron EJ, et al. Role of clinical microbiology laboratories in the management and control of infectious diseases and the delivery of health care. *Clin Infect Dis* 2001;32(4):605-11.
554. McGowan JE, Jr., Tenover FC. Confronting bacterial resistance in healthcare settings: a crucial role for microbiologists. *Nat Rev Microbiol* 2004;2(3):251-8.
555. . (Accessed at
556. Curtis JR, Cook DJ, Wall RJ, et al. Intensive care unit quality improvement: a "how-to" guide for the interdisciplinary team. *Crit Care Med* 2006;34(1):211-8.
557. Pronovost PJ, Nolan T, Zeger S, Miller M, Rubin H. How can clinicians measure safety and quality in acute care? *Lancet* 2004;363(9414):1061-7.
558. Goldrick BA, Dingle DA, Gilmore GK, Curchoe RM, Plackner CL, Fabrey LJ. Practice analysis for infection control and epidemiology in the new millennium. *Am J Infect Control* 2002;30(8):437-48.
559. CDC. Guideline for Hand Hygiene in Health-Care Settings: Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force. *MMWR* 2002;51(16)(RR-16):1-44.
560. Bonomo RA, Rice LB. Emerging issues in antibiotic resistant infections in long-term care facilities. *J Gerontol A Biol Sci Med Sci* 1999;54(6):B260-7.
561. Larson EL, Early E, Cloonan P, Sugrue S, Parides M. An organizational climate intervention associated with increased handwashing and decreased nosocomial infections. *Behav Med* 2000;26(1):14-22.

562. Pittet D, Hugonnet S, Harbarth S, et al. Effectiveness of a hospital-wide programme to improve compliance with hand hygiene. *Infection Control Programme. Lancet* 2000;356(9238):1307-12.
563. Murthy R. Implementation of strategies to control antimicrobial resistance. *Chest* 2001;119(2 Suppl):405S-11S.
564. Rondeau KV, Wagar TH. Organizational learning and continuous quality improvement: examining the impact on nursing home performance. *Health Manage Forum* 2002;15(2):17-23.
565. Stelfox HT, Bates DW, Redelmeier DA. Safety of patients isolated for infection control. *JAMA* 2003;290(14):1899-905.
566. Haley RW, Culver DH, White JW, et al. The efficacy of infection surveillance and control programs in preventing nosocomial infections in US hospitals. *Am J Epidemiol* 1985;121(2):182-205.
567. McArthur BJ, Pugliese G, Weinstein S, et al. A national task analysis of infection control practitioners, 1982. Part One: methodology and demography. *Am J Infect Control* 1984;12(2):88-95.
568. Shannon R, McArthur BJ, Weinstein S, et al. A national task analysis of infection control practitioners, 1982. Part Two: Tasks, knowledge, and abilities for practice. *Am J Infect Control* 1984;12(3):187-96.
569. Pugliese G, McArthur BJ, Weinstein S, et al. A national task analysis of infection control practitioners, 1982. Part Three: The relationship between hospital size and tasks performed. *Am J Infect Control* 1984;12(4):221-7.
570. Larson E, Eisenberg R, Soule BM. Validating the certification process for infection control practice. *Am J Infect Control* 1988;16(5):198-205.
571. Bjerke NB, Fabrey LJ, Johnson CB, et al. Job analysis 1992: infection control practitioner. *Am J Infect Control* 1993;21(2):51-7.
572. Turner JG, Kolenc KM, Docken L. Job analysis 1996: Infection control professional. Certification Board in Infection Control and Epidemiology, Inc, 1996 Job Analysis Committee. *Am J Infect Control* 1999;27(2):145-57.
573. Health Canada. Nosocomial and Occupational Infections Section. Development of a resource model for infection prevention and control programs in acute, long term, and home care settings: conference proceedings of the Infection Prevention and Control Alliance. *AJIC* 2004;32:2-6.
574. Lee TH, Meyer GS, Brennan TA. A middle ground on public accountability. *N Engl J Med* 2004;350(23):2409-12.
575. Stevenson KB, Murphy CL, Samore MH, et al. Assessing the status of infection control programs in small rural hospitals in the western United States. *Am J Infect Control* 2004;32(5):255-61.

576. Simonds DN, Horan TC, Kelley R, Jarvis WR. Detecting pediatric nosocomial infections: how do infection control and quality assurance personnel compare? *Am J Infect Control* 1997;25(3):202-8.
577. Dawson SJ. The role of the infection control link nurse. *J Hosp Infect* 2003;54(4):251-7; quiz 320.
578. Wright J, Stover BH, Wilkerson S, Bratcher D. Expanding the infection control team: development of the infection control liaison position for the neonatal intensive care unit. *Am J Infect Control* 2002;30(3):174-8.
579. Teare EL, Peacock A. The development of an infection control link-nurse programme in a district general hospital. *J Hosp Infect* 1996;34(4):267-78.
580. Ching TY, Seto WH. Evaluating the efficacy of the infection control liaison nurse in the hospital. *J Adv Nurs* 1990;15(10):1128-31.
581. Amundsen J, Drennan DP. An infection control nurse-advisor program. *Am J Infect Control* 1983;11(1):20-3.
582. Ross KA. A program for infection surveillance utilizing an infection control liaison nurse. *Am J Infect Control* 1982;10(1):24-8.
583. Needleman J, Buerhaus P, Mattke S, Stewart M, Zelevinsky K. Nurse-staffing levels and the quality of care in hospitals. *N Engl J Med* 2002;346(22):1715-22.
584. Dimick JB, Swoboda SM, Pronovost PJ, Lipsett PA. Effect of nurse-to-patient ratio in the intensive care unit on pulmonary complications and resource use after hepatectomy. *Am J Crit Care* 2001;10(6):376-82.
585. Mayhall CG, Lamb VA, Gayle WE, Jr., Haynes BW, Jr. *Enterobacter cloacae* septicemia in a burn center: epidemiology and control of an outbreak. *J Infect Dis* 1979;139(2):166-71.
586. Goldmann DA, Durbin WA, Jr., Freeman J. Nosocomial infections in a neonatal intensive care unit. *J Infect Dis* 1981;144(5):449-59.(mj).
587. Arnow P, Allyn PA, Nichols EM, Hill DL, Pezzlo M, Bartlett RH. Control of methicillin-resistant *Staphylococcus aureus* in a burn unit: role of nurse staffing. *J Trauma* 1982;22(11):954-9.
588. Haley RW, Bregman DA. The role of understaffing and overcrowding in recurrent outbreaks of staphylococcal infection in a neonatal special-care unit. *J Infect Dis* 1982;145(6):875-85.
589. Fridkin SK, Pear SM, Williamson TH, Galgiani JN, Jarvis WR. The role of understaffing in central venous catheter-associated bloodstream infections. *Infect Control Hosp Epidemiol* 1996;17(3):150-8.
590. Robert J, Fridkin SK, Blumberg HM, et al. The influence of the composition of the nursing staff on primary bloodstream infection rates in a surgical intensive care unit. *Infect Control Hosp Epidemiol* 2000;21(1):12-7.(mj).

591. Li J, Birkhead GS, Strogatz DS, Coles FB. Impact of institution size, staffing patterns, and infection control practices on communicable disease outbreaks in New York State nursing homes. *Am J Epidemiol* 1996;143(10):1042-9.
592. Archibald LK, Manning ML, Bell LM, Banerjee S, Jarvis WR. Patient density, nurse-to-patient ratio and nosocomial infection risk in a pediatric cardiac intensive care unit. *Pediatr Infect Dis J* 1997;16(11):1045-8.
593. Harbarth S, Sudre P, Dharan S, Cadenas M, Pittet D. Outbreak of *Enterobacter cloacae* related to understaffing, overcrowding, and poor hygiene practices. *Infect Control Hosp Epidemiol* 1999;20(9):598-603.
594. Vicca AF. Nursing staff workload as a determinant of methicillin-resistant *Staphylococcus aureus* spread in an adult intensive therapy unit. *J Hosp Infect* 1999;43(2):109-13.
595. Stegenga J, Bell E, Matlow A. The role of nurse understaffing in nosocomial viral gastrointestinal infections on a general pediatrics ward. *Infect Control Hosp Epidemiol* 2002;23(3):133-6.
596. Alonso-Echanove J, Edwards JR, Richards MJ, et al. Effect of nurse staffing and antimicrobial-impregnated central venous catheters on the risk for bloodstream infections in intensive care units. *Infect Control Hosp Epidemiol* 2003;24(12):916-25.
597. Petrosillo N, Gilli P, Serraino D, et al. Prevalence of infected patients and understaffing have a role in hepatitis C virus transmission in dialysis. *Am J Kidney Dis* 2001;37(5):1004-10.
598. Pfaller MA, Herwaldt LA. The clinical microbiology laboratory and infection control: emerging pathogens, antimicrobial resistance, and new technology. *Clin Infect Dis* 1997;25(4):858-70.
599. Simor AE. The role of the laboratory in infection prevention and control programs in long-term-care facilities for the elderly. *Infect Control Hosp Epidemiol* 2001;22(7):459-63.
600. Weinstein RA, Mallison GF. The role of the microbiology laboratory in surveillance and control of nosocomial infections. *Am J Clin Pathol* 1978;69(2):130-6.
601. Kolmos HJ. Interaction between the microbiology laboratory and clinician: what the microbiologist can provide. *J Hosp Infect* 1999;43 Suppl:S285-91.
602. Clinical and Laboratory Standards Institute. www.clsi.org/
603. Ginocchio CC. Role of NCCLS in antimicrobial susceptibility testing and monitoring. *Am J Health Syst Pharm* 2002;59(8 Suppl 3):S7-11.
604. National Committee for Clinical Laboratory Standards. Performance standards for antimicrobial susceptibility testing; twelfth informational supplement. Document M100-S12. NCCLS, Wayne (PA) 2002.

605. NCCLS. (2002). Analysis and Presentation of Cumulative Antimicrobial Susceptibility Test Data. Approved Guideline. NCCLS document M39-A (ISBN 1-56238-422-9). Wayne: PA, NCCLS. 2002.
606. Halstead DC, Gomez N, McCarter YS. Reality of developing a community-wide antibiogram. *J Clin Microbiol* 2004;42(1):1-6.
607. Ernst EJ, Diekema DJ, BootsMiller BJ, et al. Are United States hospitals following national guidelines for the analysis and presentation of cumulative antimicrobial susceptibility data? *Diagn Microbiol Infect Dis* 2004;49(2):141-5.
608. Bergeron MG, Ouellette M. Preventing antibiotic resistance through rapid genotypic identification of bacteria and of their antibiotic resistance genes in the clinical microbiology laboratory. *J Clin Microbiol* 1998;36(8):2169-72.
609. Hacek DM, Suriano T, Noskin GA, Kruszynski J, Reisberg B, Peterson LR. Medical and economic benefit of a comprehensive infection control program that includes routine determination of microbial clonality. *Am J Clin Pathol* 1999;111(5):647-54.
610. Rodriguez WJ, Schwartz RH, Thorne MM. Evaluation of diagnostic tests for influenza in a pediatric practice. *Pediatr Infect Dis J* 2002;21(3):193-6.
611. CDC. Prevention and control of influenza. Recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep* 2005;54(RR-8):1-40.
612. Uyeki TM. Influenza diagnosis and treatment in children: a review of studies on clinically useful tests and antiviral treatment for influenza. *Pediatr Infect Dis J* 2003;22(2):164-77.
613. Chan EL, Antonishyn N, McDonald R, et al. The use of TaqMan PCR assay for detection of *Bordetella pertussis* infection from clinical specimens. *Arch Pathol Lab Med* 2002;126(2):173-6.
614. Barenfanger J, Drake C, Kacich G. Clinical and financial benefits of rapid bacterial identification and antimicrobial susceptibility testing. *J Clin Microbiol* 1999;37(5):1415-8.
615. Barenfanger J, Drake C, Leon N, Mueller T, Troutt T. Clinical and financial benefits of rapid detection of respiratory viruses: an outcomes study. *J Clin Microbiol* 2000;38(8):2824-8.
616. Ramers C, Billman G, Hartin M, Ho S, Sawyer MH. Impact of a diagnostic cerebrospinal fluid enterovirus polymerase chain reaction test on patient management. *JAMA* 2000;283(20):2680-5.
617. Mackie PL, Joannidis PA, Beattie J. Evaluation of an acute point-of-care system screening for respiratory syncytial virus infection. *J Hosp Infect* 2001;48(1):66-71.

618. Guillemot D, Courvalin P. Better control of antibiotic resistance. *Clin Infect Dis* 2001;33(4):542-7.
619. Paterson DL. The role of antimicrobial management programs in optimizing antibiotic prescribing within hospitals. *Clin Infect Dis* 2006;42 Suppl 2:S90-5.
620. Lundstrom T, Pugliese G, Bartley J, Cox J, Guither C. Organizational and environmental factors that affect worker health and safety and patient outcomes. *Am J Infect Control* 2002;30(2):93-106.
621. www.patientsafety.com/vision.html
622. Pronovost PJ, Jenckes MW, Dorman T, et al. Organizational characteristics of intensive care units related to outcomes of abdominal aortic surgery. *JAMA* 1999;281(14):1310-7.
623. Pronovost PJ, Angus DC, Dorman T, Robinson KA, Dremsizov TT, Young TL. Physician staffing patterns and clinical outcomes in critically ill patients: a systematic review. *JAMA* 2002;288(17):2151-62.
624. Pronovost PJ, Weast B, Holzmueller CG, et al. Evaluation of the culture of safety: survey of clinicians and managers in an academic medical center. *Qual Saf Health Care* 2003;12(6):405-10.
625. Nieva VF, Sorra J. Safety culture assessment: a tool for improving patient safety in healthcare organizations. *Qual Saf Health Care* 2003;12 Suppl 2:ii17-23.
626. Clarke SP, Rockett JL, Sloane DM, Aiken LH. Organizational climate, staffing, and safety equipment as predictors of needlestick injuries and near-misses in hospital nurses. *Am J Infect Control* 2002;30(4):207-16.
627. Rivers DL, Aday LA, Frankowski RF, Felknor S, White D, Nichols B. Predictors of nurses' acceptance of an intravenous catheter safety device. *Nurs Res* 2003;52(4):249-55.
628. Gershon RR, Karkashian CD, Grosch JW, et al. Hospital safety climate and its relationship with safe work practices and workplace exposure incidents. *Am J Infect Control* 2000;28(3):211-21.
629. Gershon RR, Vlahov D, Felknor SA, et al. Compliance with universal precautions among health care workers at three regional hospitals. *Am J Infect Control* 1995;23(4):225-36.
630. Michalsen A, Delclos GL, Felknor SA, et al. Compliance with universal precautions among physicians. *J Occup Environ Med* 1997;39(2):130-7.
631. Vaughn TE, McCoy KD, Beekmann SE, Woolson RE, Torner JC, Doebbeling BN. Factors promoting consistent adherence to safe needle precautions among hospital workers. *Infect Control Hosp Epidemiol* 2004;25(7):548-55.

632. Grosch JW, Gershon RR, Murphy LR, DeJoy DM. Safety climate dimensions associated with occupational exposure to blood-borne pathogens in nurses. *Am J Ind Med* 1999;Suppl 1:122-4.
633. Piotrowski MM, Hinshaw DB. The safety checklist program: creating a culture of safety in intensive care units. *Jt Comm J Qual Improv* 2002;28(6):306-15.
634. Weeks WB, Bagian JP. Developing a culture of safety in the Veterans Health Administration. *Eff Clin Pract* 2000;3(6):270-6.
635. Bagian JP, Gosbee JW. Developing a culture of patient safety at the VA. *Ambul Outreach* 2000:25-9.
636. Tokars JJ, McKinley GF, Otten J, et al. Use and efficacy of tuberculosis infection control practices at hospitals with previous outbreaks of multidrug-resistant tuberculosis. *Infect Control Hosp Epidemiol* 2001;22(7):449-55.
637. Maloney SA, Pearson ML, Gordon MT, Del Castillo R, Boyle JF, Jarvis WR. Efficacy of control measures in preventing nosocomial transmission of multidrug-resistant tuberculosis to patients and health care workers. *Ann Intern Med* 1995;122(2):90-5.
638. Montecalvo MA, Jarvis WR, Uman J, et al. Infection-control measures reduce transmission of vancomycin-resistant enterococci in an endemic setting. *Ann Intern Med* 1999;131(4):269-72.
639. Sherertz RJ, Ely EW, Westbrook DM, et al. Education of physicians-in-training can decrease the risk for vascular catheter infection. *Ann Intern Med* 2000;132(8):641-8.
640. Lynch P, Cummings MJ, Roberts PL, Herriott MJ, Yates B, Stamm WE. Implementing and evaluating a system of generic infection precautions: body substance isolation. *Am J Infect Control* 1990;18(1):1-12.
641. Kelen GD, DiGiovanna TA, Celentano DD, et al. Adherence to Universal (barrier) Precautions during interventions on critically ill and injured emergency department patients. *J Acquir Immune Defic Syndr* 1990;3(10):987-94.
642. Courington KR, Patterson SL, Howard RJ. Universal precautions are not universally followed. *Arch Surg* 1991;126(1):93-6.
643. Kaczmarek RG, Moore RM, Jr., McCrohan J, et al. Glove use by health care workers: results of a tristate investigation. *Am J Infect Control* 1991;19(5):228-32.
644. Freeman SW, Chambers CV. Compliance with universal precautions in a medical practice with a high rate of HIV infection. *J Am Board Fam Pract* 1992;5(3):313-8.

645. Friedland LR, Joffe M, Wiley JF, 2nd, Schapire A, Moore DF. Effect of educational program on compliance with glove use in a pediatric emergency department. *Am J Dis Child* 1992;146(11):1355-8.
646. Henry K, Campbell S, Maki M. A comparison of observed and self-reported compliance with universal precautions among emergency department personnel at a Minnesota public teaching hospital: implications for assessing infection control programs. *Ann Emerg Med* 1992;21(8):940-6.
647. Henry K, Campbell S, Collier P, Williams CO. Compliance with universal precautions and needle handling and disposal practices among emergency department staff at two community hospitals. *Am J Infect Control* 1994;22(3):129-37.
648. Eustis TC, Wright SW, Wrenn KD, Fowlie EJ, Slovis CM. Compliance with recommendations for universal precautions among prehospital providers. *Ann Emerg Med* 1995;25(4):512-5.
649. DiGiacomo JC, Hoff WS, Rotondo MF, et al. Barrier precautions in trauma resuscitation: real-time analysis utilizing videotape review. *Am J Emerg Med* 1997;15(1):34-9.
650. Thompson BL, Dwyer DM, Ussery XT, Denman S, Vacek P, Schwartz B. Handwashing and glove use in a long-term-care facility. *Infect Control Hosp Epidemiol* 1997;18(2):97-103.(s).
651. Helfgott AW, Taylor-Burton J, Garcini FJ, Eriksen NL, Grimes R. Compliance with universal precautions: knowledge and behavior of residents and students in a department of obstetrics and gynecology. *Infect Dis Obstet Gynecol* 1998;6(3):123-8.
652. Moore S, Goodwin H, Grossberg R, Toltzis P. Compliance with universal precautions among pediatric residents. *Arch Pediatr Adolesc Med* 1998;152(6):554-7.
653. Akduman D, Kim LE, Parks RL, et al. Use of personal protective equipment and operating room behaviors in four surgical subspecialties: personal protective equipment and behaviors in surgery. *Infect Control Hosp Epidemiol* 1999;20(2):110-4.
654. Brooks AJ, Phipson M, Potgieter A, Koertzen H, Boffard KD. Education of the trauma team: video evaluation of the compliance with universal barrier precautions in resuscitation. *Eur J Surg* 1999;165(12):1125-8.
655. Kidd F, Heitkemper P, Kressel AB. A comprehensive educational approach to improving patient isolation practice. *Clin Perform Qual Health Care* 1999;7(2):74-6.
656. Madan AK, Rentz DE, Wahle MJ, Flint LM. Noncompliance of health care workers with universal precautions during trauma resuscitations. *South Med J* 2001;94(3):277-80.

657. Madan AK, Raafat A, Hunt JP, Rentz D, Wahle MJ, Flint LM. Barrier precautions in trauma: is knowledge enough? *J Trauma* 2002;52(3):540-3.
658. Jeffe DB, Mutha S, Kim LE, Evanoff BA, Fraser VJ. Evaluation of a preclinical, educational and skills-training program to improve students' use of blood and body fluid precautions: one-year follow-up. *Prev Med* 1999;29(5):365-73.
659. Williams CO, Campbell S, Henry K, Collier P. Variables influencing worker compliance with universal precautions in the emergency department. *Am J Infect Control* 1994;22(3):138-48.
660. Larson E, McGeer A, Quraishi ZA, et al. Effect of an automated sink on handwashing practices and attitudes in high-risk units. *Infect Control Hosp Epidemiol* 1991;12(7):422-8.
661. Swoboda SM, Earsing K, Strauss K, Lane S, Lipsett PA. Electronic monitoring and voice prompts improve hand hygiene and decrease nosocomial infections in an intermediate care unit. *Crit Care Med* 2004;32(2):358-63.
662. Kretzer EK, Larson EL. Behavioral interventions to improve infection control practices. *Am J Infect Control* 1998;26(3):245-53.
663. CDC. Updated Guidelines for Evaluating Public Health Surveillance Systems. Recommendations from the Guidelines Working Group. *MMWR Recomm Rep* 2001;50(RR-13):1-35.
664. Semmelweis IP. Die aetiologie, der begriff und die prophylaxis des kindbettfiebers. Pest, Wein, und Leipzig: CA Harleben's Verlags-Expedition 1861.
665. Bratzler DW, Houck PM. Antimicrobial prophylaxis for surgery: an advisory statement from the National Surgical Infection Prevention Project. *Clin Infect Dis* 2004 Jun 15;38:1706-15.
666. Bloom BT, Craddock A, Delmore PM, et al. Reducing acquired infections in the NICU: observing and implementing meaningful differences in process between high and low acquired infection rate centers. *J Perinatol* 2003;23:489-92.
667. Braun BI, Kritchevsky SB, Wong ES, et al. Preventing central venous catheter-associated primary bloodstream infections: characteristics of practices among hospitals participating in the Evaluation of Processes and Indicators in Infection Control (EPIC) study. *Infect Control Hosp Epidemiol* 2003;24(12):926-35.
668. Baker OG. Process surveillance: an epidemiologic challenge for all health care organizations. *AJIC* 1997;25:96-101.
669. Loeb M, McGeer A, McArthur M, Peeling RW, Petric M, Simor AE. Surveillance for outbreaks of respiratory tract infections in nursing homes. *Cmaj* 2000;162(8):1133-7.

670. Nicolle LE. Preventing infections in non-hospital settings: long-term care. *Emerg Infect Dis* 2001;7(2):205-7.
671. Pottinger JM, Herwaldt LA, Perl TM. Basics of surveillance--an overview. *Infect Control Hosp Epidemiol* 1997;18(7):513-27.
672. Lee TB, Baker OG, Lee JT, Scheckler WE, Steele L, Laxton CE. Recommended practices for surveillance. Association for Professionals in Infection Control and Epidemiology, Inc. Surveillance Initiative working Group. *Am J Infect Control* 1998;26(3):277-88.
673. Haley RW. The scientific basis for using surveillance and risk factor data to reduce nosocomial infection rates. *J Hosp Infect* 1995;30 Suppl:3-14.
674. Benneyan JC, Lloyd RC, Plsek PE. Statistical process control as a tool for research and healthcare improvement. *Qual Saf Health Care* 2003;12(6):458-64.
675. Lemmen SW, Zolldann D, Gastmeier P, Lutticken R. Implementing and evaluating a rotating surveillance system and infection control guidelines in 4 intensive care units. *Am J Infect Control* 2001;29(2):89-93.
676. Gaynes R, Richards C, Edwards J, et al. Feeding back surveillance data to prevent hospital-acquired infections. *Emerg Infect Dis* 2001;7(2):295-8.
677. Tokars JI, Richards C, Andrus M, et al. The changing face of surveillance for health care-associated infections. *Clin Infect Dis* 2004;39:1347-52.
678. Sands KE, Yokoe DS, Hooper DC, et al. Detection of postoperative surgical-site infections: comparison of health plan-based surveillance with hospital-based programs. *Infect Control Hosp Epidemiol* 2003;24(10):741-3.
679. Jodra VM, Rodela AR, Martinez EM, Fresnena NL. Standardized infection ratios for three general surgery procedures: a comparison between Spanish hospitals and U.S. centers participating in the National Nosocomial Infections Surveillance System. *Infect Control Hosp Epidemiol* 2003;24(10):744-8.
680. McKibben L, Horan T, Tokars JI, et al. Guidance on public reporting of healthcare-associated infections: recommendations of the Healthcare Infection Control Practices Advisory Committee. *Am J Infect Control* 2005;33(4):217-26.
681. Gould D, Chamberlain A. The use of a ward-based educational teaching package to enhance nurses' compliance with infection control procedures. *J Clin Nurs* 1997;6(1):55-67.
682. Calabro K, Weltge A, Parnell S, Kouzekanani K, Ramirez E. Intervention for medical students: effective infection control. *Am J Infect Control* 1998;26(4):431-6.

683. Haiduven DJ, Hench CP, Simpkins SM, Stevens DA. Standardized management of patients and employees exposed to pertussis. *Infect Control Hosp Epidemiol* 1998;19(11):861-4.
684. Macartney KK, Gorelick MH, Manning ML, Hodinka RL, Bell LM. Nosocomial respiratory syncytial virus infections: the cost- effectiveness and cost-benefit of infection control. *Pediatrics* 2000;106(3):520-6.
685. Beekmann SE, Vaughn TE, McCoy KD, et al. Hospital bloodborne pathogens programs: program characteristics and blood and body fluid exposure rates. *Infect Control Hosp Epidemiol* 2001;22(2):73-82.
686. Sokas RK, Simmens S, Scott J. A training program in universal precautions for second-year medical students. *Acad Med* 1993;68(5):374-6.
687. Ostrowsky BE, Trick WE, Sohn AH, et al. Control of vancomycin-resistant enterococcus in health care facilities in a region. *N Engl J Med* 2001;344(19):1427-33.
688. Bonten MJ, Kollef MH, Hall JB. Risk factors for ventilator-associated pneumonia: from epidemiology to patient management. *Clin Infect Dis* 2004;38(8):1141-9.
689. Lau JT, Fung KS, Wong TW, et al. SARS transmission among hospital workers in Hong Kong. *Emerg Infect Dis* 2004;10(2):280-6.
690. Talbot TR, Bradley SE, Cosgrove SE, Ruef C, Siegel JD, Weber DJ. Influenza vaccination of healthcare workers and vaccine allocation for healthcare workers during vaccine shortages. *Infect Control Hosp Epidemiol* 2005;26(11):882-90.
691. Harbarth S, Siegrist CA, Schira JC, Wunderli W, Pittet D. Influenza immunization: improving compliance of healthcare workers. *Infect Control Hosp Epidemiol* 1998;19(5):337-42.
692. Bryant KA, Stover B, Cain L, Levine GL, Siegel J, Jarvis WR. Improving influenza immunization rates among healthcare workers caring for high-risk pediatric patients. *Infect Control Hosp Epidemiol* 2004;25(11):912-7.
693. Martinello RA, Jones L, Topal JE. Correlation between healthcare workers' knowledge of influenza vaccine and vaccine receipt. *Infect Control Hosp Epidemiol* 2003;24(11):845-7.
694. Goldrick B, Gruendemann B, Larson E. Learning styles and teaching/learning strategy preferences: implications for educating nurses in critical care, the operating room, and infection control. *Heart Lung* 1993;22(2):176-82.
695. Davis D, O'Brien MA, Freemantle N, Wolf FM, Mazmanian P, Taylor-Vaisey A. Impact of formal continuing medical education: do conferences, workshops, rounds, and other traditional continuing education activities

change physician behavior or health care outcomes? JAMA
1999;282(9):867-74.

696. Carr H, and Hinson P. Education and Training. ed. APIC Text of Infection Control and Epidemiology. 2nd edition. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc. (APIC); pp. 11-1; 2005.
697. Caffarella RS. Planning Programs for Adult Learners: A Practical Guide for Educators, Trainers, and Staff Developers, Second Edition. In. San Francisco: Jossey-Bass; 2001.
698. Sargeant J, Curran V, Jarvis-Selinger S, et al. Interactive on-line continuing medical education: physicians' perceptions and experiences. J Contin Educ Health Prof 2004;24(4):227-36.
699. Van Harrison R. Systems-based framework for continuing medical education and improvements in translating new knowledge into physicians' practices. J Contin Educ Health Prof 2004;24 Suppl 1:S50-62.
700. Cole TB, Glass RM. Learning associated with participation in journal-based continuing medical education. J Contin Educ Health Prof 2004;24(4):205-12.
701. Diekema DJ, Albanese MA, Schuldt SS, Doebbeling BN. Blood and body fluid exposures during clinical training: relation to knowledge of universal precautions. J Gen Intern Med 1996;11(2):109-11.
702. Diekema DJ, Schuldt SS, Albanese MA, Doebbeling BN. Universal precautions training of preclinical students: impact on knowledge, attitudes, and compliance. Prev Med 1995;24(6):580-5.
703. Warren DK, Zack JE, Cox MJ, Cohen MM, Fraser VJ. An educational intervention to prevent catheter-associated bloodstream infections in a nonteaching, community medical center. Crit Care Med 2003;31(7):1959-63.
704. Dubbert PM, Dolce J, Richter W, Miller M, Chapman SW. Increasing ICU staff handwashing: effects of education and group feedback. Infect Control Hosp Epidemiol 1990;11(4):191-3.
705. Avila-Aguero ML, Umana MA, Jimenez AL, Faingezicht I, Paris MM. Handwashing practices in a tertiary-care, pediatric hospital and the effect on an educational program. Clin Perform Qual Health Care 1998;6(2):70-2.
706. Lai KK, Fontecchio SA, Kelley AL, Melvin ZS. Knowledge of the transmission of tuberculosis and infection control measures for tuberculosis among healthcare workers. Infect Control Hosp Epidemiol 1996;17(3):168-70.
707. Koenig S, Chu J. Senior medical students' knowledge of universal precautions. Acad Med 1993;68(5):372-4.

708. Babcock HM, Zack JE, Garrison T, et al. An educational intervention to reduce ventilator-associated pneumonia in an integrated health system: a comparison of effects. *Chest* 2004;125(6):2224-31.
709. McGuckin M, Taylor A, Martin V, Porten L, Salcido R. Evaluation of a patient education model for increasing hand hygiene compliance in an inpatient rehabilitation unit. *Am J Infect Control* 2004;32(4):235-8.
710. Cirone N. Patient-education handbook. *Nursing* 1997;27(8):44-5.
711. Chase TM. Learning styles and teaching strategies: enhancing the patient education experience. *SCI Nurse* 2001;18:138-41.
712. Jarvis WR. Handwashing--the Semmelweis lesson forgotten? *Lancet* 1994;344(8933):1311-2.
713. Daniels IR, Rees BI. Handwashing: simple, but effective. *Ann R Coll Surg Engl* 1999;81:117-8.
714. Webster J, Faoagali JL, Cartwright D. Elimination of methicillin-resistant *Staphylococcus aureus* from a neonatal intensive care unit after hand washing with triclosan. *J Paediatr Child Health* 1994;30(1):59-64.
715. Zafar AB, Butler RC, Reese DJ, Gaydos LA, Mennonna PA. Use of 0.3% triclosan (Bacti-Stat) to eradicate an outbreak of methicillin-resistant *Staphylococcus aureus* in a neonatal nursery. *Am J Infect Control* 1995;23(3):200-8.
716. Malik RK, Montecalvo MA, Reale MR, et al. Epidemiology and control of vancomycin-resistant enterococci in a regional neonatal intensive care unit. *Pediatr Infect Dis J* 1999;18(4):352-6.
717. Pittet D, Boyce JM. Hand hygiene and patient care: pursuing the Semmelweis legacy. *Lancet Infect Dis* 2001:9-20.
718. Lin CM, Wu FM, Kim HK, Doyle MP, Michael BS, Williams LK. A comparison of hand washing techniques to remove *Escherichia coli* and caliciviruses under natural or artificial fingernails. *J Food Prot* 2003;66(12):2296-301.
719. Edel E, Houston S, Kennedy V, LaRocco M. Impact of a 5-minute scrub on the microbial flora found on artificial, polished, or natural fingernails of operating room personnel. *Nurs Res* 1998;47(1):54-9.
720. Pottinger J, Burns S, Manske C. Bacterial carriage by artificial versus natural nails. *Am J Infect Control* 1989;17(6):340-4.
721. Hedderwick SA, McNeil SA, Lyons MJ, Kauffman CA. Pathogenic organisms associated with artificial fingernails worn by healthcare workers. *Infect Control Hosp Epidemiol* 2000;21(8):505-9.
722. Passaro DJ, Waring L, Armstrong R, et al. Postoperative *Serratia marcescens* wound infections traced to an out-of-hospital source. *J Infect Dis* 1997;175(4):992-5.

723. Moolenaar RL, Crutcher JM, San Joaquin VH, et al. A prolonged outbreak of *Pseudomonas aeruginosa* in a neonatal intensive care unit: did staff fingernails play a role in disease transmission? *Infect Control Hosp Epidemiol* 2000;21(2):80-5.
724. Parry MF, Grant B, Yukna M, et al. *Candida* osteomyelitis and diskitis after spinal surgery: an outbreak that implicates artificial nail use. *Clin Infect Dis* 2001;32(3):352-7.
725. Boszczowski I, Nicoletti C, Puccini DM, et al. Outbreak of extended spectrum beta-lactamase-producing *Klebsiella pneumoniae* infection in a neonatal intensive care unit related to onychomycosis in a health care worker. *Pediatr Infect Dis J* 2005;24(7):648-50.
726. Trick WE, Vernon MO, Hayes RA, et al. Impact of ring wearing on hand contamination and comparison of hand hygiene agents in a hospital. *Clin Infect Dis* 2003;36(11):1383-90.
727. Pittet D, Dharan S, Touveneau S, Sauvan V, Perneger TV. Bacterial contamination of the hands of hospital staff during routine patient care. *Arch Intern Med* 1999;159(8):821-6.
728. Tenorio AR, Badri SM, Sahgal NB, et al. Effectiveness of gloves in the prevention of hand carriage of vancomycin-resistant enterococcus species by health care workers after patient care. *Clin Infect Dis* 2001;32(5):826-9.(s).
729. Mast ST, Woolwine JD, Gerberding JL. Efficacy of gloves in reducing blood volumes transferred during simulated needlestick injury. *J Infect Dis* 1993;168(6):1589-92.
730. Medical Glove Guidance Manual.
www.fda.gov/cdrh/dsma/gloveman/gloveman99.pdf
731. Korniewicz DM, El-Masri M, Broyles JM, Martin CD, O'Connell K P. Performance of latex and nonlatex medical examination gloves during simulated use. *Am J Infect Control* 2002;30(2):133-8.
732. Korniewicz DM, McLeskey SW. Latex allergy and gloving standards. *Semin Perioper Nurs* 1998;7(4):216-21.
733. Ranta PM, Ownby DR. A review of natural-rubber latex allergy in health care workers. *Clin Infect Dis* 2004;38(2):252-6.
734. Korniewicz DM, Kirwin M, Cresci K, et al. Barrier protection with examination gloves: double versus single. *Am J Infect Control* 1994;22(1):12-5.
735. Korniewicz DM, Kirwin M, Cresci K, Larson E. Leakage of latex and vinyl exam gloves in high and low risk clinical settings. *Am Ind Hyg Assoc J* 1993;54(1):22-6.
736. Rego A, Roley L. In-use barrier integrity of gloves: latex and nitrile superior to vinyl. *Am J Infect Control* 1999;27(5):405-10.

737. Kotilainen HR, Brinker JP, Avato JL, Gantz NM. Latex and vinyl examination gloves. Quality control procedures and implications for health care workers. *Arch Intern Med* 1989;149(12):2749-53.
738. Korniewicz DM, Laughon BE, Butz A, Larson E. Integrity of vinyl and latex procedure gloves. *Nurs Res* 1989;38(3):144-6.
739. OSHA. OSHA. Department of Labor: Occupational Safety and Health Administration. Occupational exposure to bloodborne pathogens: Final rule. 29 CFR Part 1910:1030 *Federal Register* 1991;56:64003-64182 Revised 2001 CFR 66 2001:5317-25.
740. CDC. Recommendations for preventing the spread of vancomycin resistance. Recommendations of the Hospital Infection Control Practices Advisory Committee (HICPAC). *MMWR Recomm Rep* 1995;44 (RR-12):1-13.
741. Olsen RJ, Lynch P, Coyle MB, Cummings J, Bokete T, Stamm WE. Examination gloves as barriers to hand contamination in clinical practice. *JAMA* 1993;270(3):350-3.
742. Doebbeling BN, Pfaller MA, Houston AK, Wenzel RP. Removal of nosocomial pathogens from the contaminated glove. Implications for glove reuse and handwashing. *Ann Intern Med* 1988;109(5):394-8.
743. Maki DG, McCormick RD, Zilz MA, et al. A MRSA outbreak in an SICU during universal precautions: new epidemiology for nosocomial MRSA. Abstract # 473 Presented at the 30th Annual Meeting of the Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC), Chicago, Illinois October 21-24, 1990.
744. Boyce JM, Jackson MM, Pugliese G, et al. Methicillin-resistant *Staphylococcus aureus* (MRSA): a briefing for acute care hospitals and nursing facilities. The AHA Technical Panel on Infections Within Hospitals. *Infect Control Hosp Epidemiol* 1994;15(2):105-15.
745. Boyce JM, Mermel LA, Zervos MJ, et al. Controlling vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 1995;16(11):634-7.
746. Gerding DN, Johnson S, Peterson LR, Mulligan ME, Silva J, Jr. *Clostridium difficile*-associated diarrhea and colitis. *Infect Control Hosp Epidemiol* 1995;16(8):459-77.
747. Cloney DL, Donowitz LG. Overgrown use for infection control in nurseries and neonatal intensive care units. *Am J Dis Child* 1986;140(7):680-3.
748. Pelke S, Ching D, Easa D, Melish ME. Gowning does not affect colonization or infection rates in a neonatal intensive care unit. *Arch Pediatr Adolesc Med* 1994;148(10):1016-20.
749. Slaughter S, Hayden MK, Nathan C, et al. A comparison of the effect of universal use of gloves and gowns with that of glove use alone on

- acquisition of vancomycin-resistant enterococci in a medical intensive care unit. *Ann Intern Med* 1996;125(6):448-56.
750. Duquette-Petersen L, Francis ME, Dohnalek L, Skinner R, Dudas P. The role of protective clothing in infection prevention in patients undergoing autologous bone marrow transplantation. *Oncol Nurs Forum* 1999;26(8):1319-24. <http://www.ons.org>.
751. Sartori M, La Terra G, Aglietta M, Manzin A, Navino C, Verzetti G. Transmission of hepatitis C via blood splash into conjunctiva. *Scand J Infect Dis* 1993;25(2):270-1.
752. Hosoglu S, Celen MK, Akalin S, Geyik MF, Soyoral Y, Kara IH. Transmission of hepatitis C by blood splash into conjunctiva in a nurse. *Am J Infect Control* 2003;31(8):502-4.
753. CDC. Update: human immunodeficiency virus infections in health-care workers exposed to blood of infected patients. *MMWR Morb Mortal Wkly Rep* 1987;36(19):285-9.
754. Keijman J, Tjhie J, Olde Damink S, Alink M. Unusual nosocomial transmission of *Mycobacterium tuberculosis*. *Eur J Clin Microbiol Infect Dis* 2001;20(11):808-9.
755. Weaver GH. Value of the face mask and other measures. *JAMA* 1918;70:76.
756. Weaver GH. Droplet infection and its prevention by the face mask. *J Infect Dis* 1919;24:218-30.
757. Davidson IR, Crisp AJ, Hinwood DC, Whitaker SC, Gregson RH. Eye splashes during invasive vascular procedures. *Br J Radiol* 1995;68(805):39-41.
758. Guidance for Industry and FDA Staff - Surgical Masks - Premarket Notification [510(k)] Submissions; Guidance for Industry and FDA. <http://www.fda.gov/cdrh/ode/guidance/094.html>
759. National Institute for Occupational Health and Safety - Eye Protection for Infection Control. <http://www.cdc.gov/niosh/topics/eye/eye-infectious.html>
760. Gala CL, Hall CB, Schnabel KC, et al. The use of eye-nose goggles to control nosocomial respiratory syncytial virus infection. *Jama* 1986;256(19):2706-8.
761. Agah R, Cherry JD, Garakian AJ, Chapin M. Respiratory syncytial virus (RSV) infection rate in personnel caring for children with RSV infections. Routine isolation procedure vs routine procedure supplemented by use of masks and goggles. *Am J Dis Child* 1987;141(6):695-7.
762. Thorburn K, Kerr S, Taylor N, van Saene HK. RSV outbreak in a paediatric intensive care unit. *J Hosp Infect* 2004;57(3):194-201.

763. <http://a257.g.akamaitech.net/7/257/2422/06jun20041800/edocket.access.gpo.gov/2004/04-25183.htm>
764. Occupational Safety & Health Administration - Respiratory Protection. www.osha.gov/dcsp/ote/trng-materials/respirators/respirators.html
765. Campbell DL, Coffey CC, Lenhart SW. Respiratory protection as a function of respirator fitting characteristics and fit-test accuracy. *Aihaj* 2001;62(1):36-44.
766. Lee K, Slavcev A, Nicas M. Respiratory protection against *Mycobacterium tuberculosis*: quantitative fit test outcomes for five type N95 filtering-facepiece respirators. *J Occup Environ Hyg* 2004;1(1):22-8.
767. Coffey CC, Campbell DL, Zhuang Z. Simulated workplace performance of N95 respirators. *Am Ind Hyg Assoc J* 1999;60(5):618-24.
768. Coffey CC, Lawrence RB, Zhuang Z, Campbell DL, Jensen PA, Myers WR. Comparison of five methods for fit-testing N95 filtering-facepiece respirators. *Appl Occup Environ Hyg* 2002;17(10):723-30.
769. National Personal Protective Technology Laboratory. www.cdc.gov/niosh/npptl/.
770. McGowan JE, Jr. Nosocomial tuberculosis: new progress in control and prevention. *Clin Infect Dis* 1995;21(3):489-505.
771. Jarvis WR. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*. *Am J Infect Control* 1995;23(2):146-51.
772. CDC. Emergency Preparedness & Response. www.btcdcgov 2003.
773. Anderson JD, Bonner M, Scheifele DW, Schneider BC. Lack of nosocomial spread of Varicella in a pediatric hospital with negative pressure ventilated patient rooms. *Infect Control* 1985;6(3):120-1.
774. Brunell PA, Wood D. Varicella serological status of healthcare workers as a guide to whom to test or immunize. *Infect Control Hosp Epidemiol* 1999;20(5):355-7.
775. Saiman L, LaRussa P, Steinberg SP, et al. Persistence of immunity to varicella-zoster virus after vaccination of healthcare workers. *Infect Control Hosp Epidemiol* 2001;22(5):279-83.
776. Willy ME, Koziol DE, Fleisher T, et al. Measles immunity in a population of healthcare workers. *Infect Control Hosp Epidemiol* 1994;15(1):12-7.
777. Wright LJ, Carlquist JF. Measles immunity in employees of a multihospital healthcare provider. *Infect Control Hosp Epidemiol* 1994;15(1):8-11.
778. CDC. Updated U.S. Public Health Service Guidelines for the Management of Occupational Exposures to HBV, HCV, and HIV. *MMWR Recomm Rep* 2001;50 (RR-11):1-52.

779. Do AN, Ciesielski CA, Metler RP, Hammett TA, Li J, Fleming PL. Occupationally acquired human immunodeficiency virus (HIV) infection: national case surveillance data during 20 years of the HIV epidemic in the United States. *Infect Control Hosp Epidemiol* 2003;24(2):86-96.
780. CDC. Update: universal precautions for prevention of transmission of human immunodeficiency virus, hepatitis B virus, and other bloodborne pathogens in health-care settings. *MMWR Morb Mortal Wkly Rep* 1988;37(24):377-82, 87-8.
781. Davis MS. Occupational hazards of operating: opportunities for improvement. *Infect Control Hosp Epidemiol* 1996;17(10):691-3.
782. Gerberding JL. Procedure-specific infection control for preventing intraoperative blood exposures. *Am J Infect Control* 1993;21(6):364-7.
783. Fry DE, Telford GL, Fecteau DL, Sperling RS, Meyer AA. Prevention of blood exposure. Body and facial protection. *Surg Clin North Am* 1995;75(6):1141-57.
784. Hansen ME. Bloodborne pathogens and procedure safety in interventional radiology. *Semin Ultrasound CT MR* 1998;19(2):209-14.
785. Holodnick CL, Barkauskas V. Reducing percutaneous injuries in the OR by educational methods. *Aorn J* 2000;72(3):461-4, 8-72, 75-6.
786. www.osha.gov/SLTC/bloodbornepathogens/index.html
787. www.cdc.gov/niosh/2000-108.html
788. National Institute for Occupational Health and Safety - Safer Medical Device Implementation in Health Care Facilities.
<http://www.cdc.gov/niosh/topics/bbp/safer/>
789. www.cdc.gov/sharpssafety/resources.html.
790. Catanzaro A. Nosocomial tuberculosis. *Am Rev Respir Dis* 1982;125(5):559-62.
791. Cepeda JA, Whitehouse T, Cooper B, et al. Isolation of patients in single rooms or cohorts to reduce spread of MRSA in intensive-care units: prospective two-centre study. *Lancet* 2005;365(9456):295-304.
792. Mulin B, Rouget C, Clement C, et al. Association of private isolation rooms with ventilator-associated *Acinetobacter baumannii* pneumonia in a surgical intensive-care unit. *Infect Control Hosp Epidemiol* 1997;18(7):499-503.
793. www.aia.org/aah_gd_hospcons.
794. Raad I, Abbas J, Whimbey E. Infection control of nosocomial respiratory viral disease in the immunocompromised host. *Am J Med* 1997;102(3A):48-52; discussion 3-4.

795. Isaacs D, Dickson H, O'Callaghan C, Sheaves R, Winter A, Moxon ER. Handwashing and cohorting in prevention of hospital acquired infections with respiratory syncytial virus. *Arch Dis Child* 1991;66(2):227-31.
796. Chang VT, Nelson K. The role of physical proximity in nosocomial diarrhea. *Clin Infect Dis* 2000;31(3):717-22.
797. Byers KE, Anglim AM, Anneski CJ, et al. A hospital epidemic of vancomycin-resistant *Enterococcus*: risk factors and control. *Infect Control Hosp Epidemiol* 2001;22(3):140-7.
798. Dassut B. The implementation of a commode cleaning and identification system. *Nurs Times* 2004;100(8):47.
799. Mayer RA, Geha RC, Helfand MS, Hoyen CK, Salata RA, Donskey CJ. Role of fecal incontinence in contamination of the environment with vancomycin-resistant enterococci. *Am J Infect Control* 2003;31(4):221-5.
800. Samore MH, Venkataraman L, DeGirolami PC, Arbeit RD, Karchmer AW. Clinical and molecular epidemiology of sporadic and clustered cases of nosocomial *Clostridium difficile* diarrhea. *Am J Med* 1996;100(1):32-40.
801. Clabots CR, Johnson S, Olson MM, Peterson LR, Gerding DN. Acquisition of *Clostridium difficile* by hospitalized patients: evidence for colonized new admissions as a source of infection. *J Infect Dis* 1992;166(3):561-7.
802. Samore MH. Epidemiology of nosocomial *clostridium difficile* diarrhoea. *J Hosp Infect* 1999;43 Suppl:S183-90.
803. Tokars JI, Satake S, Rimland D, et al. The prevalence of colonization with vancomycin-resistant *Enterococcus* at a Veterans' Affairs institution. *Infect Control Hosp Epidemiol* 1999;20(3):171-5.
804. Cone R, Mohan K, Thouless M, Corey L. Nosocomial transmission of rotavirus infection. *Pediatr Infect Dis J* 1988;7(2):103-9.
805. Bruce BB, Blass MA, Blumberg HM, Lennox JL, del Rio C, Horsburgh CR, Jr. Risk of *Cryptosporidium parvum* transmission between hospital roommates. *Clin Infect Dis* 2000;31(4):947-50.
806. Ford-Jones EL, Mindorff CM, Gold R, Petric M. The incidence of viral-associated diarrhea after admission to a pediatric hospital. *Am J Epidemiol* 1990;131(4):711-8.
807. Murray-Leisure KA, Geib S, Graceley D, et al. Control of epidemic methicillin-resistant *Staphylococcus aureus*. *Infect Control Hosp Epidemiol* 1990;11(7):343-50.
808. Jochimsen EM, Fish L, Manning K, et al. Control of vancomycin-resistant enterococci at a community hospital: efficacy of patient and staff cohorting. *Infect Control Hosp Epidemiol* 1999;20(2):106-9.

809. Sample ML, Gravel D, Oxley C, Toye B, Garber G, Ramotar K. An outbreak of vancomycin-resistant enterococci in a hematology-oncology unit: control by patient cohorting and terminal cleaning of the environment. *Infect Control Hosp Epidemiol* 2002;23(8):468-70.
810. Podnos YD, Cinat ME, Wilson SE, Cooke J, Gornick W, Thrupp LD. Eradication of multi-drug resistant *Acinetobacter* from an Intensive Care Unit. *Surgical Infections* 2001;2(2):297-301.
811. Graham PL, 3rd, Morel AS, Zhou J, et al. Epidemiology of methicillin-susceptible *Staphylococcus aureus* in the neonatal intensive care unit. *Infect Control Hosp Epidemiol* 2002;23(11):677-82.
812. Doherty JA, Brookfield DS, Gray J, McEwan RA. Cohorting of infants with respiratory syncytial virus. *J Hosp Infect* 1998;38(3):203-6.
813. Hall CB, Geiman JM, Douglas RG, Jr., Meagher MP. Control of nosocomial respiratory syncytial viral infections. *Pediatrics* 1978;62(5):728-32.
814. Buffington J, Chapman LE, Stobierski MG, et al. Epidemic keratoconjunctivitis in a chronic care facility: risk factors and measures for control. *J Am Geriatr Soc* 1993;41(11):1177-81.
815. Grehn M, Kunz J, Sigg P, Slongo R, Zbinden R. Nosocomial rotavirus infections in neonates: means of prevention and control. *J Perinat Med* 1990;18(5):369-74.
816. Tan YM, Chow PK, Tan BH, et al. Management of inpatients exposed to an outbreak of severe acute respiratory syndrome (SARS). *J Hosp Infect* 2004;58(3):210-5.
817. Talon D, Vichard P, Muller A, Bertin M, Jeunet L, Bertrand X. Modelling the usefulness of a dedicated cohort facility to prevent the dissemination of MRSA. *J Hosp Infect* 2003;54(1):57-62.
818. Hotchkiss JR, Strike DG, Simonson DA, Broccard AF, Crooke PS. An agent-based and spatially explicit model of pathogen dissemination in the intensive care unit. *Crit Care Med* 2005;33(1):168-76; discussion 253-4.
819. Austin DJ, Bonten MJ, Weinstein RA, Slaughter S, Anderson RM. Vancomycin-resistant enterococci in intensive-care hospital settings: transmission dynamics, persistence, and the impact of infection control programs. *Proc Natl Acad Sci U S A* 1999;96(12):6908-13.
820. Kovner CT, Harrington C. Counting nurses. Data show many nursing homes to be short staffed. *Am J Nurs* 2000;100(9):53-4.
821. Mueller C. Staffing problems in long-term care. Let's do something about it! *J Gerontol Nurs* 2003;29(3):3-4.
822. Stats & facts. Nursing staff shortages in long-term care facilities. *Manag Care Interface* 2000;13(11):46-7.

823. Mejias A, Chavez-Bueno S, Ramilo O. Human metapneumovirus: a not so new virus. *Pediatr Infect Dis J* 2004;23(1):1-7; quiz 8-10.
824. Iwane MK, Edwards KM, Szilagyi PG, et al. Population-based surveillance for hospitalizations associated with respiratory syncytial virus, influenza virus, and parainfluenza viruses among young children. *Pediatrics* 2004;113(6):1758-64.
825. Ong GM, Wyatt DE, O'Neill HJ, McCaughey C, Coyle PV. A comparison of nested polymerase chain reaction and immunofluorescence for the diagnosis of respiratory infections in children with bronchiolitis, and the implications for a cohorting strategy. *J Hosp Infect* 2001;49(2):122-8.
826. von Linstow ML, Larsen HH, Eugen-Olsen J, et al. Human metapneumovirus and respiratory syncytial virus in hospitalized danish children with acute respiratory tract infection. *Scan J Infect Dis* 2004;36:578-84.
827. Gehanno JF, Pestel-Caron M, Nouvellon M, Caillard JF. Nosocomial pertussis in healthcare workers from a pediatric emergency unit in France. *Infect Control Hosp Epidemiol* 1999;20(8):549-52.
828. www.cdc.gov/flu/professionals/infectioncontrol/resphygiene.htm.
829. Edlin BR, Tokars JI, Grieco MH, et al. An outbreak of multidrug-resistant tuberculosis among hospitalized patients with the acquired immunodeficiency syndrome. *N Engl J Med* 1992;326(23):1514-21.
830. CDC. Update: Severe Acute Respiratory Syndrome --- Toronto, Canada, 2003. *MMWR* 2003;52(23):547-50.
831. Starke JR. Transmission of *Mycobacterium tuberculosis* to and from children and adolescents. *Seminars Pedi Infect Dis* 2001;12:115-23.
832. Saiman L, Macdonald N, Burns JL, Hoiby N, Speert DP, Weber D. Infection control in cystic fibrosis: practical recommendations for the hospital, clinic, and social settings. *Am J Infect Control* 2000;28(5):381-5.(s).
833. COID. 2003 Report of the Committee on Infectious Diseases. In: Redbook. Elk Grove Village, IL: American Academy of Pediatrics; 2003.
834. Lau JT, Lau M, Kim JH, Tsui HY, Tsang T, Wong TW. Probable secondary infections in households of SARS patients in Hong Kong. *Emerg Infect Dis* 2004;10(2):235-43.
835. Hota B. Contamination, disinfection, and cross-colonization: are hospital surfaces reservoirs for nosocomial infection? *Clin Infect Dis* 2004;39(8):1182-9.
836. Rutala WA, Weber DJ. Disinfection and sterilization in health care facilities: what clinicians need to know. *Clin Infect Dis* 2004;39(5):702-9.

837. Boyce JM, Opal SM, Chow JW, et al. Outbreak of multidrug-resistant *Enterococcus faecium* with transferable vanB class vancomycin resistance. *J Clin Microbiol* 1994;32(5):1148-53.
838. Engelhart S, Krizek L, Glasmacher A, Fischnaller E, Marklein G, Exner M. *Pseudomonas aeruginosa* outbreak in a haematology-oncology unit associated with contaminated surface cleaning equipment. *J Hosp Infect* 2002;52(2):93-8.
839. Denton M, Wilcox MH, Parnell P, et al. Role of environmental cleaning in controlling an outbreak of *Acinetobacter baumannii* on a neurosurgical intensive care unit. *J Hosp Infect* 2004;56(2):106-10.
840. Hollyoak V, Allison D, Summers J. *Pseudomonas aeruginosa* wound infection associated with a nursing home's whirlpool bath. *Commun Dis Rep CDR Rev* 1995;5(7):R100-2.
841. Malik RE, Cooper RA, Griffith CJ. Use of audit tools to evaluate the efficacy of cleaning systems in hospitals. *Am J Infect Control* 2003;31(3):181-7.
842. Ansari SA, Springthorpe VS, Sattar SA. Survival and vehicular spread of human rotaviruses: possible relation to seasonality of outbreaks. *Rev Infect Dis* 1991;13(3):448-61.
843. Kaatz GW, Gitlin SD, Schaberg DR, et al. Acquisition of *Clostridium difficile* from the hospital environment. *Am J Epidemiol* 1988;127(6):1289-94.
844. Mayfield JL, Leet T, Miller J, Mundy LM. Environmental control to reduce transmission of *Clostridium difficile*. *Clin Infect Dis* 2000;31(4):995-1000.
845. Dennehy PH. Transmission of rotavirus and other enteric pathogens in the home. *Pediatr Infect Dis J* 2000;19(10 Suppl):S103-5.
846. Dennehy P. Rotavirus infections in infection control reference service. In: Abrutyn E, Goldmann D, Scheckler W, eds. Philadelphia: WE Saunders; 2001:821-3.
847. Wilcox MH, Fawley WN, Wigglesworth N, Parnell P, Verity P, Freeman J. Comparison of the effect of detergent versus hypochlorite cleaning on environmental contamination and incidence of *Clostridium difficile* infection. *J Hosp Infect* 2003;54(2):109-14.
848. Rutala WA, Weber DJ, Committee HICPA. Guideline for Disinfection and Sterilization in Health-Care Facilities 2007 (in press).
849. Bernards AT, Harinck HI, Dijkshoorn L, van der Reijden TJ, van den Broek PJ. Persistent *Acinetobacter baumannii*? Look inside your medical equipment. *Infect Control Hosp Epidemiol* 2004;25(11):1002-4.

850. Neely AN, Weber JM, Daviau P, et al. Computer equipment used in patient care within a multihospital system: recommendations for cleaning and disinfection. *Am J Infect Control* 2005;33(4):233-7.
851. Neely AN, Maley MP, Warden GD. Computer keyboards as reservoirs for *Acinetobacter baumannii* in a burn hospital. *Clin Infect Dis* 1999;29(5):1358-60.
852. Bures S, Fishbain JT, Uyehara CF, Parker JM, Berg BW. Computer keyboards and faucet handles as reservoirs of nosocomial pathogens in the intensive care unit. *Am J Infect Control* 2000;28(6):465-71.
853. Brooks S, Khan A, Stoica D, et al. Reduction in vancomycin-resistant *Enterococcus* and *Clostridium difficile* infections following change to tympanic thermometers. *Infect Control Hosp Epidemiol* 1998;19(5):333-6.
854. Jernigan JA, Siegman-Igra Y, Guerrant RC, Farr BM. A randomized crossover study of disposable thermometers for prevention of *Clostridium difficile* and other nosocomial infections. *Infect Control Hosp Epidemiol* 1998;19(7):494-9.
855. Weinstein SA, Gantz NM, Pelletier C, Hibert D. Bacterial surface contamination of patients' linen: isolation precautions versus standard care. *Am J Infect Control* 1989;17(5):264-7.
856. Pugliese G. Isolating and double-bagging laundry: is it really necessary? *Health Facil Manage* 1989;2(2):16, 8-21.
857. . (Accessed 2007, at
858. Kiehl E, Wallace R, Warren C. Tracking perinatal infection: is it safe to launder your scrubs at home? *MCN Am J Matern Child Nurs* 1997;22(4):195-7.
859. Jurkovich P. Home- versus hospital-laundered scrubs: a pilot study. *MCN Am J Matern Child Nurs* 2004;29(2):106-10.
860. United States Environmental Protection Agency - Medical Waste. www.epa.gov/epaoswer/other/medical/
861. www.cdc.gov/ncidod/dhqp/gl_enviroinfection.html
862. Maki DG, Alvarado C, Hassemer C. Double-bagging of items from isolation rooms is unnecessary as an infection control measure: a comparative study of surface contamination with single- and double-bagging. *Infect Control* 1986;7(11):535-7.
863. CDC. Recommended antimicrobial agents for the treatment and postexposure prophylaxis of pertussis: 2005 CDC Guidelines. *MMWR Recomm Rep* 2005;54(RR-14):1-16.
864. CDC. Prevention and control of meningococcal disease. Recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep* 2000;49(RR-7):1-10.

865. CDC. Notice to Readers: Additional options for preventive treatment for persons exposed to inhalational anthrax. *MMWR Morb Mortal Wkly Rep* 2001;50 (50):1142-51.
866. CDC. Updated U.S. Public Health Service guidelines for the management of occupational exposures to HIV and recommendations for postexposure prophylaxis. *MMWR Recomm Rep* 2005;54(RR-9):1-17.
867. Boyce JM. MRSA patients: proven methods to treat colonization and infection. *J Hosp Infect* 2001;48 Suppl A:S9-14.
868. Barrett FF, Mason EO, Jr., Fleming D. Brief clinical and laboratory observations. *J Pediatr* 1979;94(5):796-800.
869. American Academy of Pediatrics. Guidelines for Perinatal Care. American Academy of Obstetricians and Gynecologists, 2002. Elk Grove Village, IL; 2002.
870. Management of Multidrug-Resistant Organisms In Healthcare Settings, 2006. CDC, 2006. (Accessed 2007, at
871. Kallen AJ, Wilson CT, Larson RJ. Perioperative intranasal mupirocin for the prevention of surgical-site infections: systematic review of the literature and meta-analysis. *Infect Control Hosp Epidemiol* 2005;26(12):916-22.
872. Carrier M, Marchand R, Auger P, et al. Methicillin-resistant *Staphylococcus aureus* infection in a cardiac surgical unit. *J Thorac Cardiovasc Surg* 2002;123(1):40-4.
873. Tacconelli E, Carmeli Y, Aizer A, Ferreira G, Foreman MG, D'Agata EM. Mupirocin prophylaxis to prevent *Staphylococcus aureus* infection in patients undergoing dialysis: a meta-analysis. *Clin Infect Dis* 2003;37(12):1629-38.
874. CDC. Immunization of health-care workers: recommendations of the Advisory Committee on Immunization Practices (ACIP) and the Hospital Infection Control Practices Advisory Committee (HICPAC). *MMWR Recomm Rep* 1997;46(RR18):1-42.
875. Mahoney FJ, Stewart K, Hu H, Coleman P, Alter MJ. Progress toward the elimination of hepatitis B virus transmission among health care workers in the United States. *Arch Intern Med* 1997;157(22):2601-5.
876. Gladstone JL, Millian SJ. Rubella exposure in an obstetric clinic. *Obstet Gynecol* 1981;57(2):182-6.
877. Wilde JA, McMillan JA, Serwint J, Butta J, O'Riordan MA, Steinhoff MC. Effectiveness of influenza vaccine in health care professionals: a randomized trial. *JAMA* 1999;281(10):908-13.
878. Potter J, Stott DJ, Roberts MA, et al. Influenza vaccination of health care workers in long-term-care hospitals reduces the mortality of elderly patients. *J Infect Dis* 1997;175(1):1-6.

879. Pearson ML, Bridges CB, Harper SA. Influenza vaccination of health-care personnel: recommendations of the Healthcare Infection Control Practices Advisory Committee (HICPAC) and the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep* 2006;55(RR-2):1-16.
880. Wright SW, Decker MD, Edwards KM. Incidence of pertussis infection in healthcare workers. *Infect Control Hosp Epidemiol* 1999;20(2):120-3.
881. Calugar A, Ortega-Sanchez IR, Tiwari T, Oakes L, Jahre JA, Murphy TV. Nosocomial pertussis: costs of an outbreak and benefits of vaccinating health care workers. *Clin Infect Dis* 2006;42(7):981-8.
882. www.fda.gov
883. Campins-Marti M, Cheng HK, Forsyth K, et al. Recommendations are needed for adolescent and adult pertussis immunisation: rationale and strategies for consideration. *Vaccine* 2001;20(5-6):641-6.
884. www.cdc.gov/nip/recs/provisional_rec/default.htm
885. CDC. Recommended childhood and adolescent immunization schedule -- United States, 2006. *MMWR Morb Mortal Wkly Rep* 2006;54(Nos. 51 & 52):Q1-Q4.
886. Recommended childhood and adolescent immunization schedule--United States, 2006. *Pediatrics* 2006;117(1):239-40.
887. CDC. Recommended adult immunization schedule -- United States, October 2005-September 2006. *MMWR Morb Mortal Wkly Rep* 2005;54(40):Q1-Q4; October 14.
888. CDC. Prevention of Varicella Updated Recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 1999;48 (RR-6):1-5.
889. McKenney D, Pouliot KL, Wang Y, et al. Broadly protective vaccine for *Staphylococcus aureus* based on an in vivo-expressed antigen. *Science* 1999;284(5419):1523-7.
890. Shinefield H, Black S, Fattom A, et al. Use of a *Staphylococcus aureus* conjugate vaccine in patients receiving hemodialysis. *N Engl J Med* 2002;346(7):491-6.
891. Abadesso C, Almeida HI, Virella D, Carreiro MH, Machado MC. Use of palivizumab to control an outbreak of syncytial respiratory virus in a neonatal intensive care unit. *J Hosp Infect* 2004;58(1):38-41.
892. George RH, Gully PR, Gill ON, Innes JA, Bakhshi SS, Connolly M. An outbreak of tuberculosis in a children's hospital. *J Hosp Infect* 1986;8(2):129-42.

893. Simor AE, Lee M, Vearncombe M, et al. An outbreak due to multiresistant *Acinetobacter baumannii* in a burn unit: risk factors for acquisition and management. *Infect Control Hosp Epidemiol* 2002;23(5):261-7.
894. Puzniak LA, Leet T, Mayfield J, Kollef M, Mundy LM. To gown or not to gown: the effect on acquisition of vancomycin-resistant enterococci. *Clin Infect Dis* 2002;35(1):18-25.
895. Hanna H, Umphrey J, Tarrand J, Mendoza M, Raad I. Management of an outbreak of vancomycin-resistant enterococci in the medical intensive care unit of a cancer center. *Infect Control Hosp Epidemiol* 2001;22(4):217-9.
896. CDC. Recommendations for preventing transmission of infection with human T-lymphotropic virus type III/lymphadenopathy-associated virus in the workplace. *MMWR Morb Mortal Wkly Rep* 1985;34(450):681-6, 91-5.
897. CDC. Severe acute respiratory syndrome--Taiwan, 2003. *MMWR Morb Mortal Wkly Rep* 2003;52(20):461-6.
898. Capps JA. Measures for the prevention and control of respiratory infections in military camps. *JAMA* 1918;71:448-51.
899. Thomas C. Efficiency of surgical masks in use in hospital wards. Report to the Control of Infection Subcommittee. *Guys Hosp Rep* 1961;110:157-67.
900. Beck M, Antle BJ, Berlin D, et al. Wearing masks in a pediatric hospital: developing practical guidelines. *Can J Public Health* 2004;95(4):256-7.
901. Ryan MA, Christian RS, Wohlrabe J. Handwashing and respiratory illness among young adults in military training. *Am J Prev Med* 2001;21(2):79-83.
902. Roberts L, Smith W, Jorm L, Patel M, Douglas RM, McGilchrist C. Effect of infection control measures on the frequency of upper respiratory infection in child care: a randomized, controlled trial. *Pediatrics* 2000;105(4 Pt 1):738-42.js.
903. White C, Kolble R, Carlson R, et al. The effect of hand hygiene on illness rate among students in university residence halls. *Am J Infect Control* 2003;31(6):364-70.
904. Aiello AE, Larson EL. What is the evidence for a causal link between hygiene and infections? *Lancet Infect Dis* 2002;2(2):103-10.
905. www.aana.com/news.aspx?ucNavMenu_TSMMenuTargetID=171&ucNavMenu_TSMMenuTargetType=4&ucNavMenu_TSMMenuID=6&id=1613.
906. Watanakunakorn C, Stahl C. *Streptococcus salivarius* meningitis following myelography. *Infect Control Hosp Epidemiol* 1992;13(8):454.
907. Gelfand MS, Abolnik IZ. Streptococcal meningitis complicating diagnostic myelography: three cases and review. *Clin Infect Dis* 1995;20(3):582-7.

908. Schlesinger JJ, Salit IE, McCormack G. Streptococcal meningitis after myelography. *Arch Neurol* 1982;39(9):576-7.
909. Yaniv LG, Potasman I. Iatrogenic meningitis: an increasing role for resistant viridans streptococci? Case report and review of the last 20 years. *Scand J Infect Dis* 2000;32(6):693-6.
910. Schlegel L, Merlet C, Laroche JM, Fremaux A, Geslin P. Iatrogenic meningitis due to *Abiotrophia defectiva* after myelography. *Clin Infect Dis* 1999;28(1):155-6.
911. Schneeberger PM, Janssen M, Voss A. Alpha-hemolytic streptococci: a major pathogen of iatrogenic meningitis following lumbar puncture. Case reports and a review of the literature. *Infection* 1996;24(1):29-33.
912. Veringa E, van Belkum A, Schellekens H. Iatrogenic meningitis by *Streptococcus salivarius* following lumbar puncture. *J Hosp Infect* 1995;29(4):316-8.
913. Couzigou C, Vuong TK, Botharel AH, Aggoune M, Astagneau P. Iatrogenic *Streptococcus salivarius* meningitis after spinal anaesthesia: need for strict application of standard precautions. *J Hosp Infect* 2003;53(4):313-4.
914. Torres E, Alba D, Frank A, Diez-Tejedor E. Iatrogenic meningitis due to *Streptococcus salivarius* following a spinal tap. *Clin Infect Dis* 1993;17(3):525-6.
915. Trautmann M, Lepper PM, Schmitz FJ. Three cases of bacterial meningitis after spinal and epidural anesthesia. *Eur J Clin Microbiol Infect Dis* 2002;21(1):43-5.
916. Baer ET. Iatrogenic meningitis: the case for face masks. *Clin Infect Dis* 2000;31(2):519-21.
917. Black SR, Weinstein RA. The case for face masks-zorro or zero? *Clin Infect Dis* 2000;31(2):522-3.
918. Philips BJ, Fergusson S, Armstrong P, Anderson FM, Wildsmith JA. Surgical face masks are effective in reducing bacterial contamination caused by dispersal from the upper airway. *Br J Anaesth* 1992;69(4):407-8.
919. CDC. Guidelines for the Prevention of Intravascular Catheter-Related Infections. *MMWR* 2002;51(RR10)(10):1-26.
920. Catalano G, Houston SH, Catalano MC, et al. Anxiety and depression in hospitalized patients in resistant organism isolation. *South Med J* 2003;96(2):141-5.
921. Tarzi S, Kennedy P, Stone S, Evans M. Methicillin-resistant *Staphylococcus aureus*: psychological impact of hospitalization and isolation in an older adult population. *J Hosp Infect* 2001;49(4):250-4.

922. Kelly-Rossini L, Perlman DC, Mason DJ. The experience of respiratory isolation for HIV-infected persons with tuberculosis. *J Assoc Nurses AIDS Care* 1996;Jan-Feb; 7(1):29-36.
923. Knowles HE. The experience of infectious patients in isolation. *Nurs Times* 1993;89(30):53-6.
924. Evans HL, Shaffer MM, Hughes MG, et al. Contact isolation in surgical patients: a barrier to care? *Surgery* 2003;134(2):180-8.
925. Kirkland KB, Weinstein JM. Adverse effects of contact isolation. *Lancet* 1999;354(9185):1177-8.
926. Saint S, Higgins LA, Nallamotheu BK, Chenoweth C. Do physicians examine patients in contact isolation less frequently? A brief report. *Am J Infect Control* 2003;31(6):354-6.
927. Management of Multidrug-Resistant Organisms In Healthcare Settings, 2006. 2006. www.cdc.gov/ncidod/dhqp/pdf/ar/mdroGuideline2006.pdf
928. Hall CB, Powell KR, MacDonald NE, et al. Respiratory syncytial viral infection in children with compromised immune function. *N Engl J Med* 1986;315(2):77-81.
929. Lui SL, Luk WK, Cheung CY, Chan TM, Lai KN, Peiris JS. Nosocomial outbreak of parvovirus B19 infection in a renal transplant unit. *Transplantation* 2001;71(1):59-64.
930. Weinstock DM, Gubareva LV, Zuccotti G. Prolonged shedding of multidrug-resistant influenza A virus in an immunocompromised patient. *N Engl J Med* 2003;348(9):867-8.
931. van Tol MJ, Claas EC, Heemskerk B, et al. Adenovirus infection in children after allogeneic stem cell transplantation: diagnosis, treatment and immunity. *Bone Marrow Transplant* 2005;35 Suppl 1:S73-6.
932. Wood DJ, David TJ, Chrystie IL, Totterdell B. Chronic enteric virus infection in two T-cell immunodeficient children. *J Med Virol* 1988;24(4):435-44.
933. 933. Mori I, Matsumoto K, Sugimoto K, et al. Prolonged shedding of rotavirus in a geriatric inpatient. *J Med Virol* 2002;67(4):613-5.
934. Cederna JE, Terpenning MS, Ensberg M, Bradley SF, Kauffman CA. *Staphylococcus aureus* nasal colonization in a nursing home: eradication with mupirocin. *Infect Control Hosp Epidemiol* 1990;11(1):13-6.
935. Kauffman CA, Terpenning MS, He X, et al. Attempts to eradicate methicillin-resistant *Staphylococcus aureus* from a long-term-care facility with the use of mupirocin ointment. *Am J Med* 1993;94(4):371-8.
936. Montecalvo MA, de Lencastre H, Carraher M, et al. Natural history of colonization with vancomycin-resistant *Enterococcus faecium*. *Infect Control Hosp Epidemiol* 1995;16(12):680-5.

937. D'Agata EM, et al. High rate of false-negative results of the rectal swab culture method in detection of gastrointestinal colonization with vancomycin-resistant enterococci. *Clin Infect Dis* 2002;34(2):167-72.
938. Donskey CJ, Hoyer CK, Das SM, Helfand MS, Hecker MT. Recurrence of vancomycin-resistant *Enterococcus* stool colonization during antibiotic therapy. *Infect Control Hosp Epidemiol* 2002;23(8):436-40.
939. Scanvic A, Denic L, Gaillon S, Giry P, Andremont A, Lucet JC. Duration of colonization by methicillin-resistant *Staphylococcus aureus* after hospital discharge and risk factors for prolonged carriage. *Clin Infect Dis* 2001;32(10):1393-8.
940. Noskin GA, Bednarz P, Suriano T, Reiner S, Peterson LR. Persistent contamination of fabric-covered furniture by vancomycin-resistant enterococci: implications for upholstery selection in hospitals. *Am J Infect Control* 2000;28(4):311-3.
941. Gerson SL, Parker P, Jacobs MR, Creger R, Lazarus HM. Aspergillosis due to carpet contamination. *Infect Control Hosp Epidemiol* 1994;15(4 Pt 1):221-3.
942. Taplin D, Mertz PM. Flower vases in hospitals as reservoirs of pathogens. *Lancet* 1973;2(7841):1279-81.
943. Walsh TJ, Dixon DM. Nosocomial aspergillosis: environmental microbiology, hospital epidemiology, diagnosis and treatment. *Eur J Epidemiol* 1989;5(2):131-42.
944. Lass-Flörl C, Rath P, Niederwieser D, et al. *Aspergillus terreus* infections in haematological malignancies: molecular epidemiology suggests association with in-hospital plants. *J Hosp Infect* 2000;46(1):31-5.
945. Raad I, Hanna H, Osting C, et al. Masking of neutropenic patients on transport from hospital rooms is associated with a decrease in nosocomial aspergillosis during construction. *Infect Control Hosp Epidemiol* 2002;23(1):41-3.
946. www.cms.hhs.gov/CLIA.
947. Emori TG, Haley RW, Stanley RC. The infection control nurse in US hospitals, 1976-1977. Characteristics of the position and its occupant. *Am J Epidemiol* 1980;111(5):592-607.
948. Richet HM, Benbachir M, Brown DE, et al. Are there regional variations in the diagnosis, surveillance, and control of methicillin-resistant *Staphylococcus aureus*? *Infect Control Hosp Epidemiol* 2003;24(5):334-41.
949. Anderson DJ, Kirkland KB, McDonald JR, et al. Results of a survey of work duties of 56 infection control professionals (ICPs): Are new guidelines needed for the staffing of infection control (IC) programs?

- Abstract #146. In: 16th Annual Society for Healthcare Epidemiology of America. Chicago, Ill; 2006.
950. Harvey MA. Critical-care-unit bedside design and furnishing: impact on nosocomial infections. *Infect Control Hosp Epidemiol* 1998;19(8):597-601.
951. Srinivasan A, Beck C, Buckley T, et al. The ability of hospital ventilation systems to filter *Aspergillus* and other fungi following a building implosion. *Infect Control Hosp Epidemiol* 2002;23(9):520-4.
952. Maragakis LL, Bradley KL, Song X, et al. Increased catheter-related bloodstream infection rates after the introduction of a new mechanical valve intravenous access port. *Infect Control Hosp Epidemiol* 2006;27(1):67-70.
953. Organizations JCoAoH. *Comprehensive Accreditation Manual for Hospitals: The Official Handbook*. Oakbrook Terrace: JCAHO; 2007.
954. Peterson LR, Noskin GA. New technology for detecting multidrug-resistant pathogens in the clinical microbiology laboratory. *Emerg Infect Dis* 2001;7(2):306-11.
955. Diekema DJ, Doebbeling BN. Employee health and infection control. *Infect Control Hosp Epidemiol* 1995;16(5):292-301.
956. Rutala WA, Weber DJ, Healthcare Infection Control Practices Advisory Committee (HICPAC). *Guideline for Disinfection and Sterilization in Health-Care Facilities*. In preparation.
957. Weems JJ, Jr. Nosocomial outbreak of *Pseudomonas cepacia* associated with contamination of reusable electronic ventilator temperature probes. *Infect Control Hosp Epidemiol* 1993;14(10):583-6.
958. Berthelot P, Grattard F, Mahul P, et al. Ventilator temperature sensors: an unusual source of *Pseudomonas cepacia* in nosocomial infection. *J Hosp Infect* 1993;25(1):33-43.
959. 959. CDC. Bronchoscopy-related infections and pseudoinfections--New York, 1996 and 1998. *MMWR Morb Mortal Wkly Rep* 1999;48(26):557-60.
960. Heeg P, Roth K, Reichl R, Cogdill CP, Bond WW. Decontaminated single-use devices: an oxymoron that may be placing patients at risk for cross-contamination. *Infect Control Hosp Epidemiol* 2001;22(9):542-9.
961. www.fda.gov/cdrh/reprocessing/
962. CDC. *Prevention and Control of Influenza: Recommendations of the Advisory Committee on Immunization Practices (ACIP)*. *MMWR - Morbidity & Mortality Weekly Report* 2003;52(RR08):1-36.

963. Weinstock DM, Eagan J, Malak SA, et al. Control of influenza A on a bone marrow transplant unit. *Infect Control Hosp Epidemiol* 2000;21(11):730-2.
964. Cromer AL, Hutsell SO, Latham SC, et al. Impact of implementing a method of feedback and accountability related to contact precautions compliance. *Am J Infect Control* 2004;32(8):451-5.
965. Eveillard M, Eb F, Tramier B, et al. Evaluation of the contribution of isolation precautions in prevention and control of multi-resistant bacteria in a teaching hospital. *J Hosp Infect* 2001;47(2):116-24.
966. Pfeiffer J, Gilmore G. The Text as an Orientation Tool. In: Pfeiffer J, ed. *APIC Text of Infection Control and Epidemiology*. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc. (APIC); 2000:7/1 - 7/8.
967. Gaynes RP, Emori TG. Chapter 5: Surveillance for Nosocomial Infections. In: Abrutyn E, Goldmann DA, Scheckler WE, eds. *Saunders Infection Control Reference Service*. Philadelphia, PA: W.B. Saunders Company; 2001:40-4.
968. CDC. Monitoring hospital-acquired infections to promote patient safety - United States, 1990-1999. *MMWR Morb Mortal Wkly Rep* 2000;49(8):149-53.
969. 969. Curran ET, Benneyan JC, Hood J. Controlling methicillin-resistant *Staphylococcus aureus*: a feedback approach using annotated statistical process control charts. *Infect Control Hosp Epidemiol* 2002;23(1):13-8.
970. Lanotte P, Cantagrel S, Mereghetti L, et al. Spread of *Stenotrophomonas maltophilia* colonization in a pediatric intensive care unit detected by monitoring tracheal bacterial carriage and molecular typing. *Clin Microbiol Infect* 2003;9(11):1142-7.
971. Coopersmith CM, Zack JE, Ward MR, et al. The impact of bedside behavior on catheter-related bacteremia in the intensive care unit. *Arch Surg* 2004;139(2):131-6.
972. O'Brien KL, Beall B, Barrett NL, et al. Epidemiology of invasive group a streptococcus disease in the United States, 1995-1999. *Clin Infect Dis* 2002;35(3):268-76.
973. Nicolle LE, Dyck B, Thompson G, et al. Regional dissemination and control of epidemic methicillin-resistant *Staphylococcus aureus*. Manitoba Chapter of CHICA-Canada. *Infect Control Hosp Epidemiol* 1999;20(3):202-5.
974. Seybold U, Kourbatova EV, Johnson JG, et al. Emergence of community-associated methicillin-resistant *Staphylococcus aureus* USA300 genotype as a major cause of health care-associated blood stream infections. *Clin Infect Dis* 2006;42(5):647-56.

975. Bond WW, Favero MS, Petersen NJ, Gravelle CR, Ebert JW, Maynard JE. Survival of hepatitis B virus after drying and storage for one week. *Lancet* 1981;1(8219):550-1.
976. Ehrenkranz NJ, Alfonso BC. Failure of bland soap handwash to prevent hand transfer of patient bacteria to urethral catheters. *Infect Control Hosp Epidemiol* 1991;12(11):654-62.
977. Winnefeld M, Richard MA, Drancourt M, Grob JJ. Skin tolerance and effectiveness of two hand decontamination procedures in everyday hospital use. *Br J Dermatol* 2000;143(3):546-50.
978. Widmer AF. Replace hand washing with use of a waterless alcohol hand rub? *Clin Infect Dis* 2000;31(1):136-43.
979. Mortimer EA, Jr., Lipsitz PJ, Wolinsky E, Gonzaga AJ, Rammelkamp CH, Jr. Transmission of staphylococci between newborns. Importance of the hands to personnel. *Am J Dis Child* 1962;104:289-95.
980. Casewell M, Phillips I. Hands as route of transmission for *Klebsiella* species. *Br Med J* 1977;2(6098):1315-7.
981. Ojajarvi J. Effectiveness of hand washing and disinfection methods in removing transient bacteria after patient nursing. *J Hyg (Lond)* 1980;85(2):193-203.
982. Otter J, Havill N, Adams N, Joyce J. Extensive environmental contamination associated with patients with loose stools and MRSA colonization of the gastrointestinal tract. Abstract #159. In: the 16th annual scientific meeting of the Society for Healthcare Epidemiology of America. In. Chicago, Illinois; 2006.
983. Weber DJ, Sickbert-Bennett E, Gergen MF, Rutala WA. Efficacy of selected hand hygiene agents used to remove *Bacillus atrophaeus* (a surrogate of *Bacillus anthracis*) from contaminated hands. *Jama* 2003;289(10):1274-7.
984. Saiman L, Lerner A, Saal L, et al. Banning artificial nails from health care settings. *Am J Infect Control* 2002;30(4):252-4.
985. Johnson S, Gerding DN, Olson MM, et al. Prospective, controlled study of vinyl glove use to interrupt *Clostridium difficile* nosocomial transmission. *Am J Med* 1990;88(2):137-40.
986. Neal JG, Jackson EM, Suber F, Edlich RF. Latex glove penetration by pathogens: a review of the literature. *J Long Term Eff Med Implants* 1998;8(3-4):233-40.
987. Broyles JM, O'Connell KP, Korniewicz DM. PCR-based method for detecting viral penetration of medical exam gloves. *J Clin Microbiol* 2002;40(8):2725-8.

988. Patterson JE, Vecchio J, Pantelick EL, et al. Association of contaminated gloves with transmission of *Acinetobacter calcoaceticus* var. *anitratus* in an intensive care unit. *Am J Med* 1991;91(5):479-83.
989. Goldmann DA. Epidemiology and prevention of pediatric viral respiratory infections in health-care institutions. *Emerg Infect Dis* 2001;7(2):249-53.
990. Gaggero A, Avendano LF, Fernandez J, Spencer E. Nosocomial transmission of rotavirus from patients admitted with diarrhea. *J Clin Microbiol* 1992;30(12):3294-7.
991. Merritt K, Hitchins VM, Brown SA. Safety and cleaning of medical materials and devices. *J Biomed Mater Res* 2000;53(2):131-6.
992. Kampf G, Bloss R, Martiny H. Surface fixation of dried blood by glutaraldehyde and peracetic acid. *J Hosp Infect* 2004;57(2):139-43.
993. Weber DJ, Rutala WA. Role of environmental contamination in the transmission of vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 1997;18(5):306-9.
994. Byers KE, Durbin LJ, Simonton BM, Anglim AM, Adal KA, Farr BM. Disinfection of hospital rooms contaminated with vancomycin-resistant *Enterococcus faecium*. *Infect Control Hosp Epidemiol* 1998;19(4):261-4.
995. Martinez JA, Ruthazer R, Hansjosten K, Barefoot L, Snyderman DR. Role of environmental contamination as a risk factor for acquisition of vancomycin-resistant enterococci in patients treated in a medical intensive care unit. *Arch Intern Med* 2003;163(16):1905-12.
996. EPA. Federal Insecticide, Fungicide, and Rodenticidal Act 7 U.S.C. 136 et seq. In: Agency EP, ed.
997. Devine J, Cooke RP, Wright EP. Is methicillin-resistant *Staphylococcus aureus* (MRSA) contamination of ward-based computer terminals a surrogate marker for nosocomial MRSA transmission and handwashing compliance? *J Hosp Infect* 2001;48(1):72-5.
998. Sattar SA, Springthorpe S, Mani S, et al. Transfer of bacteria from fabrics to hands and other fabrics: development and application of a quantitative method using *Staphylococcus aureus* as a model. *J Appl Microbiol* 2001;90(6):962-70.
999. Shiomori T, Miyamoto H, Makishima K, et al. Evaluation of bedmaking-related airborne and surface methicillin-resistant *Staphylococcus aureus* contamination. *J Hosp Infect* 2002;50(1):30-5.
1000. Whyte W, Baird G, Annand R. Bacterial contamination on the surface of hospital linen chutes. *J Hyg (Lond)* 1969;67(3):427-35.
1001. Michaelsen GS. Designing Linen Chutes to Reduce Spread of Infectious Organisms. *Hospitals* 1965;39:116-9.

1002. Plott RT, Wagner RF, Jr., Tying SK. Iatrogenic contamination of multidose vials in simulated use. A reassessment of current patient injection technique. *Arch Dermatol* 1990;126(11):1441-4.
1003. Samandari T, Malakmadze N, Balter S, et al. A large outbreak of hepatitis B virus infections associated with frequent injections at a physician's office. *Infect Control Hosp Epidemiol* 2005;26(9):745-50.
1004. Comstock RD, Mallonee S, Fox JL, et al. A large nosocomial outbreak of hepatitis C and hepatitis B among patients receiving pain remediation treatments. *Infect Control Hosp Epidemiol* 2004;25(7):576-83.
1005. Germain JM, Carbonne A, Thiers V, et al. Patient-to-patient transmission of hepatitis C virus through the use of multidose vials during general anesthesia. *Infect Control Hosp Epidemiol* 2005;26(9):789-92.
1006. Macedo de Oliveira A, White KL, Leschinsky DP, et al. An outbreak of hepatitis C virus infections among outpatients at a hematology/oncology clinic. *Ann Intern Med* 2005;142(11):898-902.
1007. Hsu J, Jensen B, Arduino M, et al. Streptococcal Meningitis Following Myelogram Procedures. *Infect Control Hosp Epidemiol* 2007;28(5):614-17.
1008. Srinivasan A, Song X, Ross T, Merz W, Brower R, Perl TM. A prospective study to determine whether cover gowns in addition to gloves decrease nosocomial transmission of vancomycin-resistant enterococci in an intensive care unit. *Infect Control Hosp Epidemiol* 2002;23(8):424-8.
1009. Nichols WG, Corey L, Gooley T, Davis C, Boeckh M. Parainfluenza virus infections after hematopoietic stem cell transplantation: risk factors, response to antiviral therapy, and effect on transplant outcome. *Blood* 2001;98(3):573-8.
1010. Elizaga J, Olavarria E, Apperley J, Goldman J, Ward K. Parainfluenza virus 3 infection after stem cell transplant: relevance to outcome of rapid diagnosis and ribavirin treatment. *Clin Infect Dis* 2001;32(3):413-8.
1011. Oishi I, Kimura T, Murakami T, et al. Serial observations of chronic rotavirus infection in an immunodeficient child. *Microbiology and Immunology* 1991;35(11):953-61.
1012. Fierobe L, Lucet JC, Decre D, et al. An outbreak of imipenem-resistant *Acinetobacter baumannii* in critically ill surgical patients. *Infect Control Hosp Epidemiol* 2001;22(1):35-40.
1013. Montesinos I, Salido E, Delgado T, Lecuona M, Sierra A. Epidemiology of methicillin-resistant *Staphylococcus aureus* at a university hospital in the Canary Islands. *Infect Control Hosp Epidemiol* 2003;24(9):667-72.
1014. Poutanen SM, Vearncombe M, McGeer AJ, Gardam M, Large G, Simor AE. Nosocomial acquisition of methicillin-resistant *Staphylococcus*

- aureus during an outbreak of severe acute respiratory syndrome. *Infect Control Hosp Epidemiol* 2005;26(2):134-7.
1015. Yap FH, Gomersall CD, Fung KS, et al. Increase in methicillin-resistant *Staphylococcus aureus* acquisition rate and change in pathogen pattern associated with an outbreak of severe acute respiratory syndrome. *Clin Infect Dis* 2004;39(4):511-6.
1016. Layton MC, Perez M, Heald P, Patterson JE. An outbreak of mupirocin-resistant *Staphylococcus aureus* on a dermatology ward associated with an environmental reservoir. *Infect Control Hosp Epidemiol* 1993;14(7):369-75.
1017. Gilmore A, Stuart J, Andrews N. Risk of secondary meningococcal disease in health-care workers. *Lancet* 2000;356(9242):1654-5.
1018. [. www.cdc.gov/flu/avian/index.htm](http://www.cdc.gov/flu/avian/index.htm)
1019. www.hhs.gov/pandemicflu/plan/pdf/S04.pdf
1020. Ehresmann KR, Hedberg CW, Grimm MB, Norton CA, MacDonald KL, Osterholm MT. An outbreak of measles at an international sporting event with airborne transmission in a domed stadium. *J Infect Dis* 1995;171(3):679-83.
1021. Gustafson TL, Lavelly GB, Brawner ER, Jr., Hutcheson RH, Jr., Wright PF, Schaffner W. An outbreak of airborne nosocomial varicella. *Pediatrics* 1982;70(4):550-6.
1022. Hyams PJ, Stuewe MC, Heitzer V. Herpes zoster causing varicella (chickenpox) in hospital employees: cost of a casual attitude. *Am J Infect Control* 1984;12(1):2-5.
1023. Pavelchak N, DePersis RP, London M, et al. Identification of factors that disrupt negative air pressurization of respiratory isolation rooms. *Infect Control Hosp Epidemiol* 2000;21(3):191-5.
1024. Rice N, Streifel A, Vesley D. An evaluation of hospital special-ventilation-room pressures. *Infect Control Hosp Epidemiol* 2001;22(1):19-23.
1025. Hutton MD, Stead WW, Cauthen GM, Bloch AB, Ewing WM. Nosocomial transmission of tuberculosis associated with a draining abscess. *J Infect Dis* 1990;161(2):286-95.
1026. Frampton MW. An outbreak of tuberculosis among hospital personnel caring for a patient with a skin ulcer. *Ann Intern Med* 1992;117(4):312-3.
1027. Ammari LK, Bell LM, Hodinka RL. Secondary measles vaccine failure in healthcare workers exposed to infected patients. *Infect Control Hosp Epidemiol* 1993;14(2):81-6.

1028. Behrman A, Schmid DS, Crivaro A, Watson B. A cluster of primary varicella cases among healthcare workers with false-positive varicella zoster virus titers. *Infect Control Hosp Epidemiol* 2003;24(3):202-6.
1029. Josephson A, Gombert ME. Airborne transmission of nosocomial varicella from localized zoster. *J Infect Dis* 1988;158(1):238-41.
1030. Brodtkin RH. Zoster Causing Varicella. Current Dangers Of Contagion Without Isolation. *Arch Dermatol* 1963;88:322-4.
1031. Suzuki K, Yoshikawa T, Tomitaka A, Matsunaga K, Asano Y. Detection of aerosolized varicella-zoster virus DNA in patients with localized herpes zoster. *J Infect Dis* 2004;189(6):1009-12.
1032. Ruuskanen O, Salmi TT, Halonen P. Measles vaccination after exposure to natural measles. *J Pediatr* 1978;93(1):43-6.
1033. Berkovich S, Starr S. Use of live-measles-virus vaccine to abort an expected outbreak of measles within a closed population. *N Engl J Med* 1963;269:75-7.
1034. CDC. Measles, mumps, and rubella--vaccine use and strategies for elimination of measles, rubella, and congenital rubella syndrome and control of mumps: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep* 1998;47(RR-8):1-57.
1035. CDC. General recommendations on immunization: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 2006;55(RR-15):1-48.
1036. Watson B, Seward J, Yang A, et al. Postexposure effectiveness of varicella vaccine. *Pediatrics* 2000;105(1 Pt 1):84-8.
1037. Salzman MB, Garcia C. Postexposure varicella vaccination in siblings of children with active varicella. *Pediatr Infect Dis J* 1998;17(3):256-7.
1038. CDC. Vaccinia (smallpox) vaccine: recommendations of the Advisory Committee on Immunization Practices (ACIP), 2001. *MMWR Recomm Rep* 2001;50(RR-10):1-25; quiz CE1-7.
1039. Fulginiti VA, Papier A, Lane JM, Neff JM, Henderson DA. Smallpox vaccination: a review, part I. Background, vaccination technique, normal vaccination and revaccination, and expected normal reactions. *Clin Infect Dis* 2003;37(2):241-50.
1040. Dixon CW. Smallpox in Tripolitania, 1946: an epidemiological and clinical study of 500 cases, including trials of penicillin treatment. *J Hyg (Lond)* 1948;46:351-77.
1041. Murray WA, Streifel AJ, O'Dea TJ, Rhame FS. Ventilation for protection of immune compromised patients. *ASHRAE Transactions* 1988;94:1185.

1042. Rutala WA, Jones SM, Worthington JM, Reist PC, Weber DJ. Efficacy of portable filtration units in reducing aerosolized particles in the size range of *Mycobacterium tuberculosis*. *Infect Control Hosp Epidemiol* 1995;16(7):391-8.
1043. Mandell GL, Bennett JE, Dolin R. *Mandell, Douglas and Bennett's Principles and Practice of Infectious Diseases*. GL Mandell, JE Bennett, R Dolin, Eds. 5th edition. Churchill Livingstone, Philadelphia, 2000. 2000.
1044. *Control of Communicable Diseases Manual*. DL Heymann Ed. 18th edition, American Public Health Association, Washington, DC 2005. 2005.
1045. Vreden SG, Visser LG, Verweij JJ, et al. Outbreak of amebiasis in a family in The Netherlands. *Clin Infect Dis* 2000;31(4):1101-4.
1046. Thacker SB, Kimball AM, Wolfe M, Choi K, Gilmore L. Parasitic disease control in a residential facility for the mentally retarded: failure of selected isolation procedures. *Am J Public Health* 1981;71(3):303-5.
1047. Sampathkumar P. West Nile virus: epidemiology, clinical presentation, diagnosis, and prevention. *Mayo Clin Proc* 2003;78(9):1137-43; quiz 44.
1048. Ruben B, Band JD, Wong P, Colville J. Person-to-person transmission of *Brucella melitensis*. *Lancet* 1991;337(8732):14-5.
1049. Vandercam B, Zech F, de Cooman S, Bughin C, Gigi J, Wauters G. Isolation of *Brucella melitensis* from human sperm. *Eur J Clin Microbiol Infect Dis* 1990;9(4):303-4.
1050. Robichaud S, Libman M, Behr M, Rubin E. Prevention of laboratory-acquired brucellosis. *Clin Infect Dis* 2004;38(12):e119-22.
1051. Troy CJ, Peeling RW, Ellis AG, et al. *Chlamydia pneumoniae* as a new source of infectious outbreaks in nursing homes. *Jama* 1997;277(15):1214-8.
1052. Ekman MR, Grayston JT, Visakorpi R, Kleemola M, Kuo CC, Saikku P. An epidemic of infections due to *Chlamydia pneumoniae* in military conscripts. *Clin Infect Dis* 1993;17(3):420-5.
1053. Eickhoff TC. An outbreak of surgical wound infections due to *Clostridium perfringens*. *Surg Gynecol Obstet* 1962;114:102-8.
1054. Kohn GJ, Linne SR, Smith CM, Hoeprich PD. Acquisition of coccidioidomycosis at necropsy by inhalation of coccidioidal endospores. *Diagn Microbiol Infect Dis* 1992;15(6):527-30.
1055. Wright PW, Pappagianis D, Wilson M, et al. Donor-related coccidioidomycosis in organ transplant recipients. *Clin Infect Dis* 2003;37(9):1265-9.

1056. Maitreyi RS, Dar L, Muthukumar A, et al. Acute hemorrhagic conjunctivitis due to enterovirus 70 in India. *Emerg Infect Dis* 1999;5(2):267-9.
1057. CDC. Acute hemorrhagic conjunctivitis outbreak caused by Coxsackievirus A24--Puerto Rico, 2003. *MMWR Morb Mortal Wkly Rep* 2004;53(28):632-4.
1058. Faden H, Wynn RJ, Campagna L, Ryan RM. Outbreak of adenovirus type 30 in a neonatal intensive care unit. *J Pediatr* 2005;146(4):523-7.
1059. Chaberny IE, Schnitzler P, Geiss HK, Wendt C. An outbreak of epidemic keratoconjunctivitis in a pediatric unit due to adenovirus type 8. *Infect Control Hosp Epidemiol* 2003;24(7):514-9.
1060. Warren D, Nelson KE, Farrar JA, et al. A large outbreak of epidemic keratoconjunctivitis: problems in controlling nosocomial spread. *J Infect Dis* 1989;160(6):938-43.
1061. www.cdc.gov/ncidod/dvrd/cjd/qa_cjd_infection_control.htm
1062. Wang CY, Wu HD, Hsueh PR. Nosocomial transmission of cryptococcosis. *N Engl J Med* 2005;352(12):1271-2.
1063. Beyt BE, Jr., Waltman SR. Cryptococcal endophthalmitis after corneal transplantation. *N Engl J Med* 1978;298(15):825-6.
1064. Widdowson MA, Glass R, Monroe S, et al. Probable transmission of norovirus on an airplane. *Jama* 2005;293(15):1859-60.
1065. CDC. Prevention of Hepatitis A Through Active or Passive Immunization: Recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep* 1999;48(RR-12):1-37.
1066. Rosenblum LS, Villarino ME, Nainan OV, et al. Hepatitis A outbreak in a neonatal intensive care unit: risk factors for transmission and evidence of prolonged viral excretion among preterm infants. *J Infect Dis* 1991;164(3):476-82.
1067. Carl M, Kantor RJ, Webster HM, Fields HA, Maynard JE. Excretion of hepatitis A virus in the stools of hospitalized hepatitis patients. *J Med Virol* 1982;9(2):125-9.
1068. Robson SC, Adams S, Brink N, Woodruff B, Bradley D. Hospital outbreak of hepatitis E. *Lancet* 1992;339(8806):1424-5.
1069. Arvin A, Whitley R. Herpes Simplex virus infections in *Infectious Diseases of the Fetus and Newborn Infant*, ed. Remington JS and Klein JO. Fifth Edition. WB Saunders Co., Philadelphia, PA. 2001.
1070. Enright AM, Prober CG. Neonatal herpes infection: diagnosis, treatment and prevention. *Semin Neonatol* 2002;7(4):283-91.
1071. Esper F, Boucher D, Weibel C, Martinello RA, Kahn JS. Human metapneumovirus infection in the United States: clinical manifestations

- associated with a newly emerging respiratory infection in children. *Pediatrics* 2003;111(6 Pt 1):1407-10.
1072. Colodner R, Sakran W, Miron D, Teitler N, Khavalevsky E, Kopelowitz J. *Listeria monocytogenes* cross-contamination in a nursery. *Am J Infect Control* 2003;31(5):322-4.
1073. Farber JM, Peterkin PI, Carter AO, Varughese PV, Ashton FE, Ewan EP. Neonatal listeriosis due to cross-infection confirmed by isoenzyme typing and DNA fingerprinting. *J Infect Dis* 1991;163(4):927-8.
1074. Schuchat A, Lizano C, Broome CV, Swaminathan B, Kim C, Winn K. Outbreak of neonatal listeriosis associated with mineral oil. *Pediatr Infect Dis J* 1991;10(3):183-9.
1075. Pejaver RK, Watson AH, Mucklow ES. Neonatal cross-infection with *Listeria monocytogenes*. *J Infect* 1993;26(3):301-3.
1076. Jain SK, Persaud D, Perl TM, et al. Nosocomial malaria and saline flush. *Emerg Infect Dis* 2005;11(7):1097-9.
1077. Abulrahi HA, Bohlega EA, Fontaine RE, al-Seghayer SM, al-Ruwais AA. *Plasmodium falciparum* malaria transmitted in hospital through heparin locks. *Lancet* 1997;349(9044):23-5.
1078. Al-Saigul AM, Fontaine RE, Haddad Q. Nosocomial malaria from contamination of a multidose heparin container with blood. *Infect Control Hosp Epidemiol* 2000;21(5):329-30.
1079. Piro S, Sammud M, Badi S, Al Ssabi L. Hospital-acquired malaria transmitted by contaminated gloves. *J Hosp Infect* 2001;47(2):156-8.
1080. Book LS, Overall JC, Jr., Herbst JJ, Britt MR, Epstein B, Jung AL. Clustering of necrotizing enterocolitis. Interruption by infection-control measures. *N Engl J Med* 1977;297(18):984-6.
1081. Rotbart HA, Levin MJ. How contagious is necrotizing enterocolitis? *Pediatr Infect Dis* 1983;2(5):406-13.
1082. Rotbart HA, Levin MJ, Yolken RH, Manchester DK, Jantzen J. An outbreak of rotavirus-associated neonatal necrotizing enterocolitis. *J Pediatr* 1983;103(3):454-9.
1083. Gerber AR, Hopkins RS, Lauer BA, Curry-Kane AG, Rotbart HA. Increased risk of illness among nursery staff caring for neonates with necrotizing enterocolitis. *Pediatr Infect Dis* 1985;4(3):246-9.
1084. Sanchez MP, Erdman DD, Torok TJ, Freeman CJ, Matyas BT. Outbreak of adenovirus 35 pneumonia among adult residents and staff of a chronic care psychiatric facility. *J Infect Dis* 1997;176(3):760-3.(s).
1085. Singh-Naz N, Brown M, Ganeshanathan M. Nosocomial adenovirus infection: molecular epidemiology of an outbreak. *Pediatr Infect Dis J* 1993;12(11):922-5.

1086. Uemura T, Kawashita T, Ostuka Y, Tanaka Y, Kusubae R, Yoshinaga M. A recent outbreak of adenovirus type 7 infection in a chronic inpatient facility for the severely handicapped. *Infect Control Hosp Epidemiol* 2000;21(9):559-60.
1087. Nuorti JP, Butler JC, Crutcher JM, et al. An outbreak of multidrug-resistant pneumococcal pneumonia and bacteremia among unvaccinated nursing home residents. *N Engl J Med* 1998;338(26):1861-8.
1088. Houff SA, Burton RC, Wilson RW, et al. Human-to-human transmission of rabies virus by corneal transplant. *N Engl J Med* 1979;300(11):603-4.
1089. CDC. Human Rabies Prevention - United States, 1999 Recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR* 1999;48 (RR-1):1-21.
1090. Hayden FG. Rhinovirus and the lower respiratory tract. *Rev Med Virol* 2004;14(1):17-31.
1091. Valenti WM, Clarke TA, Hall CB, Menegus MA, Shapiro DL. Concurrent outbreaks of rhinovirus and respiratory syncytial virus in an intensive care nursery: epidemiology and associated risk factors. *J Pediatr* 1982;100(5):722-6.
1092. Chidekel AS, Rosen CL, Bazy AR. Rhinovirus infection associated with serious lower respiratory illness in patients with bronchopulmonary dysplasia. *Pediatr Infect Dis J* 1997;16(1):43-7.
1093. Drusin LM, Ross BG, Rhodes KH, Krauss AN, Scott RA. Nosocomial ringworm in a neonatal intensive care unit: a nurse and her cat. *Infect Control Hosp Epidemiol* 2000;21(9):605-7.
1094. Lewis SM, Lewis BG. Nosocomial transmission of *Trichophyton tonsurans* tinea corporis in a rehabilitation hospital. *Infect Control Hosp Epidemiol* 1997;18(5):322-5.
1095. Saiman L, Jakob K, Holmes KW, et al. Molecular epidemiology of staphylococcal scalded skin syndrome in premature infants. *Pediatr Infect Dis J* 1998;17(4):329-34.
1096. Ramage L, Green K, Pyskir D, Simor AE. An outbreak of fatal nosocomial infections due to group A streptococcus on a medical ward. *Infect Control Hosp Epidemiol* 1996;17(7):429-31.
1097. Kakis A, Gibbs L, Eguia J, et al. An outbreak of group A Streptococcal infection among health care workers. *Clin Infect Dis* 2002;35(11):1353-9.
1098. Schwartz B, Elliott JA, Butler JC, et al. Clusters of invasive group A streptococcal infections in family, hospital, and nursing home settings. *Clin Infect Dis* 1992;15(2):277-84.
1099. National Communicable Disease Center. Isolation Techniques for Use in Hospitals. 1st ed. Washington, DC: US Government Printing Office;. PHS publication no 2054 1970.

1100. CDC. Isolation Techniques for Use in Hospitals. 2nd ed. Washington, DC: US Government Printing Office;1975. HHS publication no. (CDC) 80-8314. 1975.
1101. Garner JS, Simmons BP. CDC Guideline for Isolation Precautions in Hospitals. Atlanta, GA: US Department of Health and Human Services, Public Health Service, Centers for Disease Control; 1983. HHS publication no. (CDC) 83-8314. Infect Control 1983;4:245-325.
1102. Lynch P, Jackson MM, Cummings MJ, Stamm WE. Rethinking the role of isolation practices in the prevention of nosocomial infections. Ann Intern Med 1987;107(2):243-6.



Management of Multidrug-Resistant Organisms In Healthcare Settings, 2006

Jane D. Siegel, MD; Emily Rhinehart, RN MPH CIC; Marguerite Jackson, PhD; Linda Chiarello, RN MS; the Healthcare Infection Control Practices Advisory Committee

Acknowledgement:

The authors and HICPAC gratefully acknowledge Dr. Larry Strausbaugh for his many contributions and valued guidance in the preparation of this guideline.



Healthcare Infection Control Practices Advisory Committee (HICPAC):

Chair

Patrick J. Brennan, MD
Professor of Medicine
Division of Infectious Diseases
University of Pennsylvania Medical School

Executive Secretary

Michael Bell, MD
Division of Healthcare Quality Promotion
National Center for Infectious Diseases
Centers for Disease Control and Prevention

Members

BRINSKO, Vicki L., RN, BA
Infection Control Coordinator
Vanderbilt University Medical Center

DELLINGER, E. Patchen., MD
Professor of Surgery
University of Washington School of
Medicine

ENGEL, Jeffrey, MD
Head General Communicable Disease Control
Branch
North Carolina State Epidemiologist

GORDON, Steven M., MD
Chairman, Department of Infections Diseases
Hospital Epidemiologist
Cleveland Clinic Foundation
Department of Infectious Disease

HARRELL, Lizzie J., PhD, D(ABMM)
Research Professor of Molecular Genetics,
Microbiology and Pathology
Associate Director, Clinical Microbiology
Duke University Medical Center
O'BOYLE, Carol, PhD, RN
Assistant Professor, School of Nursing
University of Minnesota

PEGUES, David Alexander, MD
Division of Infectious Diseases
David Geffen School of Medicine at UCLA

PERROTTA, Dennis M. PhD., CIC
Adjunct Associate Professor of Epidemiology
University of Texas School of Public Health
Texas A&M University School of Rural Public
Health

PITT, Harriett M., MS, CIC, RN
Director, Epidemiology
Long Beach Memorial Medical Center

RAMSEY, Keith M., MD
Professor of Medicine
Medical Director of Infection Control
The Brody School of Medicine at East Carolina
University

SINGH, Nalini, MD, MPH
Professor of Pediatrics
Epidemiology and International Health
The George Washington University Children's National
Medical Center

STEVENSON, Kurt Brown, MD, MPH
Division of Infectious Diseases
Department of Internal Medicine
The Ohio State University Medical Center

SMITH, Philip W., MD
Chief, Section of Infectious Diseases
Department of Internal Medicine
University of Nebraska Medical Center

HICPAC membership (past)

Robert A. Weinstein, MD (Chair)
Cook County Hospital
Chicago, IL

Jane D. Siegel, MD (Co-Chair)
University of Texas Southwestern Medical Center
Dallas, TX

Michele L. Pearson, MD
(Executive Secretary)
Centers for Disease Control and Prevention
Atlanta, GA

Raymond Y.W. Chinn, MD
Sharp Memorial Hospital
San Diego, CA

Alfred DeMaria, Jr, MD
Massachusetts Department of Public Health
Jamaica Plain, MA

James T. Lee, MD, PhD
University of Minnesota
Minneapolis, MN

William A. Rutala, PhD, MPH
University of North Carolina Health Care System
Chapel Hill, NC

William E. Scheckler, MD
University of Wisconsin
Madison, WI

Beth H. Stover, RN
Kosair Children's Hospital
Louisville, KY

Marjorie A. Underwood, RN, BSN CIC
Mt. Diablo Medical Center
Concord, CA

HICPAC Liaisons

William B. Baine, MD
Liaison to Agency for Healthcare Quality
Research

Joan Blanchard, RN, MSN, CNOR
Liaison to Association of periOperative
Registered Nurses

Patrick J. Brennan, MD
Liaison to Board of Scientific Counselors

Nancy Bjerke, RN, MPH, CIC
Liaison to Association of Professionals in
Infection Prevention and Control

Jeffrey P. Engel, MD
Liaison to Advisory Committee on Elimination of
Tuberculosis

David Henderson, MD
Liaison to National Institutes of Health

Lorine J. Jay MPH, RN, CPHQ
Liaison to Healthcare Resources Services
Administration

Stephen F. Jencks, MD, MPH
Liaison to Center for Medicare and Medicaid Services

Sheila A. Murphey, MD
Liaison to Food and Drug Administration

Mark Russi, MD, MPH
Liaison to American College of Occupational and
Environmental Medicine

Rachel L. Stricof, MPH
Liaison to Advisory Committee on Elimination of
Tuberculosis

Michael L. Tapper, MD
Liaison to Society for Healthcare Epidemiology of
America

Robert A. Wise, MD
Liaison to Joint Commission on the Accreditation of
Healthcare Organizations

Authors' Associations

Jane D. Siegel, MD
Professor of Pediatrics
Department of Pediatrics
University of Texas Southwestern Medical Center

Emily Rhinehart RN MPH CIC CPHQ
Vice President
AIG Consultants, Inc.

Marguerite Jackson, RN PhD CIC
Director, Administrative Unit, National Tuberculosis
Curriculum Consortium, Department of Medicine
University of California San Diego

Linda Chiarello, RN MS
Division of Healthcare Quality Promotion
National Center for Infectious Diseases, CDC

I. Introduction

Multidrug-resistant organisms (MDROs), including methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-resistant enterococci (VRE) and certain gram-negative bacilli (GNB) have important infection control implications that either have not been addressed or received only limited consideration in previous isolation guidelines. Increasing experience with these organisms is improving understanding of the routes of transmission and effective preventive measures. Although transmission of MDROs is most frequently documented in acute care facilities, all healthcare settings are affected by the emergence and transmission of antimicrobial-resistant microbes. The severity and extent of disease caused by these pathogens varies by the population(s) affected and by the institution(s) in which they are found. Institutions, in turn, vary widely in physical and functional characteristics, ranging from long-term care facilities (LTCF) to specialty units (e.g., intensive care units [ICU], burn units, neonatal ICUs [NICUs]) in tertiary care facilities. Because of this, the approaches to prevention and control of these pathogens need to be tailored to the specific needs of each population and individual institution. The prevention and control of MDROs is a national priority - one that requires that all healthcare facilities and agencies assume responsibility(1) (2). The following discussion and recommendations are provided to guide the implementation of strategies and practices to prevent the transmission of MRSA, VRE, and other MDROs. The administration of healthcare organizations and institutions should ensure that appropriate strategies are fully implemented, regularly evaluated for effectiveness, and adjusted such that there is a consistent decrease in the incidence of targeted MDROs. Successful prevention and control of MDROs requires administrative and scientific leadership and a financial and human resource commitment(3-5). Resources must be made available for infection prevention and control, including expert consultation, laboratory support, adherence monitoring, and data analysis. Infection prevention and control professionals have found that healthcare personnel (HCP) are more receptive and adherent to the recommended control measures when organizational leaders participate in efforts to reduce MDRO transmission(3).

II. Background

MDRO definition. For epidemiologic purposes, MDROs are defined as microorganisms, predominantly bacteria, that are resistant to one or more classes of antimicrobial agents (1). Although the names of certain MDROs describe resistance to only one agent (e.g., MRSA, VRE), these pathogens are frequently resistant to most available antimicrobial agents. These highly resistant organisms deserve special attention in healthcare facilities (2). In addition to MRSA and VRE, certain GNB, including those producing extended spectrum beta-lactamases (ESBLs) and others that are resistant to multiple classes of antimicrobial agents, are of particular concern.¹ In addition to *Escherichia coli* and *Klebsiella pneumoniae*, these include strains of *Acinetobacter baumannii* resistant to all antimicrobial agents, or all except imipenem,(6-12), and organisms such as *Stenotrophomonas maltophilia* (12-14), *Burkholderia cepacia* (15, 16), and *Ralstonia pickettii*(17) that are intrinsically resistant to the broadest-spectrum antimicrobial agents. In some residential settings (e.g., LTCFs), it is important to control multidrug-resistant *S. pneumoniae* (MDRSP) that are resistant to penicillin and other broad-spectrum agents such as macrolides and fluoroquinolones (18, 19). Strains of *S. aureus* that have intermediate susceptibility or are resistant to vancomycin (i.e., vancomycin-intermediate *S. aureus* [VISA], vancomycin-resistant *S. aureus* [VRSA]) (20-30) have affected specific populations, such as hemodialysis patients.

Clinical importance of MDROs. In most instances, MDRO infections have clinical manifestations that are similar to infections caused by susceptible pathogens. However, options for treating patients with these infections are often extremely limited. For example, until recently, only vancomycin provided effective therapy for potentially life-threatening MRSA infections and during the 1990's there were virtually no antimicrobial agents to treat infections caused by VRE. Although antimicrobials are now available for treatment of MRSA and VRE infections, resistance to each new agent has already emerged in clinical

¹ Multidrug-resistant strains of *M. tuberculosis* are not addressed in this document because of the markedly different patterns of transmission and spread of the pathogen and the very different control interventions that are needed for prevention of *M. tuberculosis* infection. Current recommendations for prevention and control of tuberculosis can be found at: <http://www.cdc.gov/mmwr/pdf/rr/rr5417.pdf>

isolates(31-37). Similarly, therapeutic options are limited for ESBL-producing isolates of gram-negative bacilli, strains of *A. baumannii* resistant to all antimicrobial agents except imipenem(8-11, 38) and intrinsically resistant *Stenotrophomonas* sp.(12-14, 39). These limitations may influence antibiotic usage patterns in ways that suppress normal flora and create a favorable environment for development of colonization when exposed to potential MDR pathogens (i.e., selective advantage)(40).

Increased lengths of stay, costs, and mortality also have been associated with MDROs (41-46). Two studies documented increased mortality, hospital lengths of stay, and hospital charges associated with multidrug-resistant gram-negative bacilli (MDR-GNBs), including an NICU outbreak of ESBL-producing *Klebsiella pneumoniae* (47) and the emergence of third-generation cephalosporin resistance in *Enterobacter* spp. in hospitalized adults (48). Vancomycin resistance has been reported to be an independent predictor of death from enterococcal bacteremia(44, 49-53). Furthermore, VRE was associated with increased mortality, length of hospital stay, admission to the ICU, surgical procedures, and costs when VRE patients were compared with a matched hospital population (54).

However, MRSA may behave differently from other MDROs. When patients with MRSA have been compared to patients with methicillin-susceptible *S. aureus* (MSSA), MRSA-colonized patients more frequently develop symptomatic infections(55, 56). Furthermore, higher case fatality rates have been observed for certain MRSA infections, including bacteremia(57-62), poststernotomy mediastinitis(63), and surgical site infections(64). These outcomes may be a result of delays in the administration of vancomycin, the relative decrease in the bactericidal activity of vancomycin(65), or persistent bacteremia associated with intrinsic characteristics of certain MRSA strains (66). Mortality may be increased further by *S. aureus* with reduced vancomycin susceptibility (VISA) (26, 67). Also some studies have reported an association between MRSA infections and increased length of stay, and healthcare costs(46, 61, 62), while others have not(64). Finally, some hospitals have observed an increase in the overall occurrence of staphylococcal infections following the introduction of MRSA into a hospital or special-care unit(68, 69).

III. Epidemiology of MDROs

Trends: Prevalence of MDROs varies temporally, geographically, and by healthcare setting(70, 71). For example, VRE emerged in the eastern United States in the early 1990s, but did not appear in the western United States until several years later, and MDRSP varies in prevalence by state(72). The type and level of care also influence the prevalence of MDROs. ICUs, especially those at tertiary care facilities, may have a higher prevalence of MDRO infections than do non-ICU settings (73, 74). Antimicrobial resistance rates are also strongly correlated with hospital size, tertiary-level care, and facility type (e.g., LTCF)(75, 76). The frequency of clinical infection caused by these pathogens is low in LTCFs(77, 78). Nonetheless, MDRO infections in LTCFs can cause serious disease and mortality, and colonized or infected LTCF residents may serve as reservoirs and vehicles for MDRO introduction into acute care facilities (78-88). Another example of population differences in prevalence of target MDROs is in the pediatric population. Point prevalence surveys conducted by the Pediatric Prevention Network (PPN) in eight U.S. PICUs and 7 U.S. NICUs in 2000 found $\leq 4\%$ of patients were colonized with MRSA or VRE compared with 10-24% were colonized with ceftazidime- or aminoglycoside-resistant gram-negative bacilli; $< 3\%$ were colonized with ESBL-producing gram negative bacilli. Despite some evidence that MDRO burden is greatest in adult hospital patients, MDRO require similar control efforts in pediatric populations as well(89).

During the last several decades, the prevalence of MDROs in U.S. hospitals and medical centers has increased steadily(90, 91). MRSA was first isolated in the United States in 1968. By the early 1990s, MRSA accounted for 20%-25% of *Staphylococcus aureus* isolates from hospitalized patients(92). In 1999, MRSA accounted for $>50\%$ of *S. aureus* isolates from patients in ICUs in the National Nosocomial Infection Surveillance (NNIS) system; in 2003, 59.5% of *S. aureus* isolates in NNIS ICUs were MRSA (93). A similar rise in prevalence has occurred with VRE (94). From 1990 to 1997, the prevalence of VRE in enterococcal isolates from hospitalized patients increased from $<1\%$ to approximately 15% (95). VRE accounted for almost 25% of enterococcus isolates in NNIS ICUs in 1999 (94), and 28.5% in 2003 (93).

GNB resistant to ESBLs, fluoroquinolones, carbapenems, and aminoglycosides also have increased in prevalence. For example, in 1997, the SENTRY Antimicrobial Surveillance Program found that among *K. pneumoniae* strains isolated in the United States, resistance rates to ceftazidime and other third-generation cephalosporins were 6.6%, 9.7%, 5.4%, and 3.6% for bloodstream, pneumonia, wound, and urinary tract infections, respectively (95). In 2003, 20.6% of all *K. pneumoniae* isolates from NNIS ICUs were resistant to these drugs ((93)). Similarly, between 1999 and 2003, *Pseudomonas aeruginosa* resistance to fluoroquinolone antibiotics increased from 23% to 29.5% in NNIS ICUs(74). Also, a 3-month survey of 15 Brooklyn hospitals in 1999 found that 53% of *A. baumannii* strains exhibited resistance to carbapenems and 24% of *P. aeruginosa* strains were resistant to imipenem (10). During 1994-2000, a national review of ICU patients in 43 states found that the overall susceptibility to ciprofloxacin decreased from 86% to 76% and was temporally associated with increased use of fluoroquinolones in the United States (96).

Lastly, an analysis of temporal trends of antimicrobial resistance in non-ICU patients in 23 U.S. hospitals during 1996-1997 and 1998-1999 (97) found significant increases in the prevalence of resistant isolates including MRSA, ciprofloxacin-resistant *P. aeruginosa*, and ciprofloxacin- or ofloxacin-resistant *E. coli*. Several factors may have contributed to these increases including: selective pressure exerted by exposure to antimicrobial agents, particularly fluoroquinolones, outside of the ICU and/or in the community(7, 96, 98); increasing rates of community-associated MRSA colonization and infection(99, 100); inadequate adherence to infection control practices; or a combination of these factors.

Important concepts in transmission. Once MDROs are introduced into a healthcare setting, transmission and persistence of the resistant strain is determined by the availability of vulnerable patients, selective pressure exerted by antimicrobial use, increased potential for transmission from larger numbers of colonized or infected patients (“colonization pressure”)(101, 102); and the impact of implementation and adherence to prevention efforts. Patients vulnerable to colonization and infection include those with severe disease, especially those with compromised host defenses from underlying medical conditions; recent surgery; or indwelling medical devices (e.g., urinary catheters or endotracheal

tubes(103, 104)). Hospitalized patients, especially ICU patients, tend to have more risk factors than non-hospitalized patients do, and have the highest infection rates. For example, the risk that an ICU patient will acquire VRE increases significantly once the proportion of ICU patients colonized with VRE exceeds 50%(101) or the number days of exposure to a VRE-patient exceeds 15 days(105). A similar effect of colonization pressure has been demonstrated for MRSA in a medical ICU(102). Increasing numbers of infections with MDROs also have been reported in non-ICU areas of hospitals(97).

There is ample epidemiologic evidence to suggest that MDROs are carried from one person to another via the hands of HCP(106-109). Hands are easily contaminated during the process of care-giving or from contact with environmental surfaces in close proximity to the patient(110-113). The latter is especially important when patients have diarrhea and the reservoir of the MDRO is the gastrointestinal tract(114-117). Without adherence to published recommendations for hand hygiene and glove use(111) HCP are more likely to transmit MDROs to patients. Thus, strategies to increase and monitor adherence are important components of MDRO control programs(106, 118).

Opportunities for transmission of MDROs beyond the acute care hospital results from patients receiving care at multiple healthcare facilities and moving between acute-care, ambulatory and/or chronic care, and LTC environments. System-wide surveillance at LDS Hospital in Salt Lake City, Utah, monitored patients identified as being infected or colonized with MRSA or VRE, and found that those patients subsequently received inpatient or outpatient care at as many as 62 different healthcare facilities in that system during a 5-year span(119).

Role of colonized HCP in MDRO transmission. Rarely, HCP may introduce an MDRO into a patient care unit(120-123). Occasionally, HCP can become persistently colonized with an MDRO, but these HCP have a limited role in transmission, unless other factors are present. Additional factors that can facilitate transmission, include chronic sinusitis(120), upper respiratory infection(123), and dermatitis(124).

Implications of community-associated MRSA (CA-MRSA). The emergence of new epidemic strains of MRSA in the community, among patients without established MRSA risk factors, may present new challenges to MRSA control in healthcare settings(125-128). Historically, genetic analyses of MRSA isolated from patients in hospitals worldwide revealed that a relatively small number of MRSA strains have unique qualities that facilitate their transmission from patient to patient within healthcare facilities over wide geographic areas, explaining the dramatic increases in HAIs caused by MRSA in the 1980s and early 1990s(129). To date, most MRSA strains isolated from patients with CA-MRSA infections have been microbiologically distinct from those endemic in healthcare settings, suggesting that some of these strains may have arisen *de novo* in the community via acquisition of methicillin resistance genes by established methicillin-susceptible *S. aureus* (MSSA) strains(130-132). Two pulsed-field types, termed USA300 and USA400 according to a typing scheme established at CDC, have accounted for the majority of CA-MRSA infections characterized in the United States, whereas pulsed-field types USA100 and USA200 are the predominant genotypes endemic in healthcare settings(133).

USA300 and USA400 genotypes almost always carry type IV of the staphylococcal chromosomal cassette (SCC) *mec*, the mobile genetic element that carries the *mecA* methicillin-resistance gene (133, 134). This genetic cassette is smaller than types I through III, the types typically found in healthcare associated MRSA strains, and is hypothesized to be more easily transferable between *S. aureus* strains.

CA-MRSA infection presents most commonly as relatively minor skin and soft tissue infections, but severe invasive disease, including necrotizing pneumonia, necrotizing fasciitis, severe osteomyelitis, and a sepsis syndrome with increased mortality have also been described in children and adults(134-136).

Transmission within hospitals of MRSA strains first described in the community (e.g. USA300 and USA400) are being reported with increasing frequency(137-140). Changing resistance patterns of MRSA in ICUs in the NNIS system from 1992 to 2003 provide additional evidence that the new epidemic MRSA strains are becoming established

healthcare-associated as well as community pathogens(90). Infections with these strains have most commonly presented as skin disease in community settings. However, intrinsic virulence characteristics of the organisms can result in clinical manifestations similar to or potentially more severe than traditional healthcare-associated MRSA infections among hospitalized patients. The prevalence of MRSA colonization and infection in the surrounding community may therefore affect the selection of strategies for MRSA control in healthcare settings.

IV. MDRO Prevention and Control

Prevention of Infections. Preventing infections will reduce the burden of MDROs in healthcare settings. Prevention of antimicrobial resistance depends on appropriate clinical practices that should be incorporated into all routine patient care. These include optimal management of vascular and urinary catheters, prevention of lower respiratory tract infection in intubated patients, accurate diagnosis of infectious etiologies, and judicious antimicrobial selection and utilization. Guidance for these preventive practices include the Campaign to Reduce Antimicrobial Resistance in Healthcare Settings (www.cdc.gov/drugresistance/healthcare/default.htm), a multifaceted, evidence-based approach with four parallel strategies: infection prevention; accurate and prompt diagnosis and treatment; prudent use of antimicrobials; and prevention of transmission. Campaign materials are available for acute care hospitals, surgical settings, dialysis units, LTCFs and pediatric acute care units.

To reduce rates of central-venous-line associated bloodstream infections(CVL-BSIs) and ventilator-associated pneumonia (VAP), a group of bundled evidence-based clinical practices have been implemented in many U.S. healthcare facilities(118, 141-144). One report demonstrated a sustained effect on the reduction in CVL-BSI rates with this approach(145). Although the specific effect on MDRO infection and colonization rates have not been reported, it is logical that decreasing these and other healthcare-associated infections will in turn reduce antimicrobial use and decrease opportunities for emergence and transmission of MDROs.

Prevention and Control of MDRO transmission

Overview of the MDRO control literature. Successful control of MDROs has been documented in the United States and abroad using a variety of combined interventions. These include improvements in hand hygiene, use of Contact Precautions until patients are culture-negative for a target MDRO, active surveillance cultures (ASC), education, enhanced environmental cleaning, and improvements in communication about patients with MDROs within and between healthcare facilities.

Representative studies include:

- Reduced rates of MRSA transmission in The Netherlands, Belgium, Denmark, and other Scandinavian countries after the implementation of aggressive and sustained infection control interventions (i.e., ASC; preemptive use of Contact Precautions upon admission until proven culture negative; and, in some instances, closure of units to new admissions). MRSA generally accounts for a very small proportion of *S. aureus* clinical isolates in these countries(146-150).
- Reduced rates of VRE transmission in healthcare facilities in the three-state Siouland region (Iowa, Nebraska, and South Dakota) following formation of a coalition and development of an effective region-wide infection control intervention that included ASC and isolation of infected patients. The overall prevalence rate of VRE in the 30 participating facilities decreased from 2.2% in 1997 to 0.5% in 1999(151).
- Eradication of endemic MRSA infections from two NICUs. The first NICU included implementation of ASC, Contact Precautions, use of triple dye on the umbilical cord, and systems changes to improve surveillance and adherence to recommended practices and to reduce overcrowding(152). The second NICU used ASC and Contact Precautions; surgical masks were included in the barriers used for Contact Precautions(153).
- Control of an outbreak and eventual eradication of VRE from a burn unit over a 13-month period with implementation of aggressive culturing, environmental cleaning, and barrier isolation(154).
- Control of an outbreak of VRE in a NICU over a 3-year period with implementation of ASC, other infection control measures such as use of a waterless hand disinfectant, and mandatory in-service education(155).

- Eradication of MDR-strains of *A. baumannii* from a burn unit over a 16-month period with implementation of strategies to improve adherence to hand hygiene, isolation, environmental cleaning, and temporary unit closure(38).
- In addition, more than 100 reports published during 1982-2005 support the efficacy of combinations of various control interventions to reduce the burden of MRSA, VRE, and MDR-GNBs (Tables 1 and 2). Case-rate reduction or pathogen eradication was reported in a majority of studies.
- VRE was eradicated in seven special-care units(154, 156-160), two hospitals(161, 162), and one LTCF(163).
- MRSA was eradicated from nine special-care units(89, 152, 153, 164-169), two hospitals(170), one LTCF(167), and one Finnish district(171). Furthermore, four MRSA reports described continuing success in sustaining low endemic MDRO rates for over 5 years(68, 166, 172, 173).
- An MDR-GNB was eradicated from 13 special-care units(8, 9, 38, 174-180) and two hospitals (11, 181).

These success stories testify to the importance of having dedicated and knowledgeable teams of healthcare professionals who are willing to persist for years, if necessary, to control MDROs. Eradication and control of MDROs, such as those reported, frequently required periodic reassessment and the addition of new and more stringent interventions over time (tiered strategy). For example, interventions were added in a stepwise fashion during a 3-year effort that eventually eradicated MRSA from an NICU(152). A series of interventions was adopted throughout the course of a year to eradicate VRE from a burn unit(154). Similarly, eradication of carbapenem-resistant strains of *A. baumannii* from a hospital required multiple and progressively more intense interventions over several years(11).

Nearly all studies reporting successful MDRO control employed a median of 7 to 8 different interventions concurrently or sequentially (Table 1). These figures may underestimate the actual number of control measures used, because authors of these reports may have considered their earliest efforts routine (e.g., added emphasis on handwashing), and did not include them as interventions, and some "single measures" are, in fact, a complex

combination of several interventions. The use of multiple concurrent control measures in these reports underscores the need for a comprehensive approach for controlling MDROs.

Several factors affect the ability to generalize the results of the various studies reviewed, including differences in definition, study design, endpoints and variables measured, and period of follow-up. Two-thirds of the reports cited in Tables 1 and 2 involved perceived outbreaks, and one-third described efforts to reduce endemic transmission. Few reports described preemptive efforts or prospective studies to control MDROs before they had reached high levels within a unit or facility.

With these and other factors, it has not been possible to determine the effectiveness of individual interventions, or a specific combination of interventions, that would be appropriate for all healthcare facilities to implement in order to control their target MDROs. Randomized controlled trials are necessary to acquire this level of evidence. An NIH-sponsored, randomized controlled trial on the prevention of MRSA and VRE transmission in adult ICUs is ongoing and may provide further insight into optimal control measures (<http://clinicaltrials.gov/ct/show/NCT00100386?order=1>). This trial compares the use of education (to improve adherence to hand hygiene) and Standard Precautions to the use of ASC and Contact Precautions.

Control Interventions. The various types of interventions used to control or eradicate MDROs may be grouped into seven categories. These include administrative support, judicious use of antimicrobials, surveillance (routine and enhanced), Standard and Contact Precautions, environmental measures, education and decolonization. These interventions provide the basis for the recommendations for control of MDROs in healthcare settings that follow this review and as summarized in Table 3. In the studies reviewed, these interventions were applied in various combinations and degrees of intensity, with differences in outcome.

- 1. Administrative support.** In several reports, administrative support and involvement were important for the successful control of the target MDRO(3, 152, 182-185), and authorities in infection control have strongly recommended such support(2, 106, 107,

186). There are several examples of MDRO control interventions that require administrative commitment of fiscal and human resources. One is the use of ASC(8, 38, 68, 107, 114, 151, 152, 167, 168, 183, 184, 187-192). Other interventions that require administrative support include: 1) implementing system changes to ensure prompt and effective communications e.g., computer alerts to identify patients previously known to be colonized/infected with MDROs(184, 189, 193, 194); 2), providing the necessary number and appropriate placement of hand washing sinks and alcohol-containing hand rub dispensers in the facility(106, 195); 3) maintaining staffing levels appropriate to the intensity of care required(152, 196-202); and 4) enforcing adherence to recommended infection control practices (e.g., hand hygiene, Standard and Contact Precautions) for MDRO control. Other measures that have been associated with a positive impact on prevention efforts, that require administrative support, are direct observation with feedback to HCP on adherence to recommended precautions and keeping HCP informed about changes in transmission rates(3, 152, 182, 203-205). A “How-to guide” for implementing change in ICUs, including analysis of structure, process, and outcomes when designing interventions, can assist in identification of needed administrative interventions(195). Lastly, participation in existing, or the creation of new, city-wide, state-wide, regional or national coalitions, to combat emerging or growing MDRO problems is an effective strategy that requires administrative support(146, 151, 167, 188, 206, 207).

2. Education. Facility-wide, unit-targeted, and informal, educational interventions were included in several successful studies(3, 189, 193, 208-211). The focus of the interventions was to encourage a behavior change through improved understanding of the problem MDRO that the facility was trying to control. Whether the desired change involved hand hygiene, antimicrobial prescribing patterns, or other outcomes, enhancing understanding and creating a culture that supported and promoted the desired behavior, were viewed as essential to the success of the intervention. Educational campaigns to enhance adherence to hand hygiene practices in conjunction with other control measures have been associated temporally with decreases in MDRO transmission in various healthcare settings(3, 106, 163).

3. *Judicious use of antimicrobial agents.* While a comprehensive review of antimicrobial stewardship is beyond the scope of this guideline, recommendations for control of MDROs must include attention to judicious antimicrobial use. A temporal association between formulary changes and decreased occurrence of a target MDRO was found in several studies, especially in those that focused on MDR-GNBs(98, 177, 209, 212-218). Occurrence of *C. difficile*-associated disease has also been associated with changes in antimicrobial use(219). Although some MRSA and VRE control efforts have attempted to limit antimicrobial use, the relative importance of this measure for controlling these MDROs remains unclear(193, 220). Limiting antimicrobial use alone may fail to control resistance due to a combination of factors; including 1) the relative effect of antimicrobials on providing initial selective pressure, compared to perpetuating resistance once it has emerged; 2) inadequate limits on usage; or 3) insufficient time to observe the impact of this intervention. With the intent of addressing #2 and #3 above in the study design, one study demonstrated a decrease in the prevalence of VRE associated with a formulary switch from ticarcillin-clavulanate to piperacillin-tazobactam(221).

The CDC Campaign to Prevent Antimicrobial Resistance that was launched in 2002 provides evidence-based principles for judicious use of antimicrobials and tools for implementation(222) www.cdc.gov/drugresistance/healthcare. This effort targets all healthcare settings and focuses on effective antimicrobial treatment of infections, use of narrow spectrum agents, treatment of infections and not contaminants, avoiding excessive duration of therapy, and restricting use of broad-spectrum or more potent antimicrobials to treatment of serious infections when the pathogen is not known or when other effective agents are unavailable. Achieving these objectives would likely diminish the selective pressure that favors proliferation of MDROs. Strategies for influencing antimicrobial prescribing patterns within healthcare facilities include education; formulary restriction; prior-approval programs, including pre-approved indications; automatic stop orders; academic interventions to counteract pharmaceutical influences on prescribing patterns; antimicrobial cycling(223-226);

computer-assisted management programs(227-229); and active efforts to remove redundant antimicrobial combinations(230). A systematic review of controlled studies identified several successful practices. These include social marketing (i.e. consumer education), practice guidelines, authorization systems, formulary restriction, mandatory consultation, and peer review and feedback. It further suggested that online systems that provide clinical information, structured order entry, and decision support are promising strategies(231). These changes are best accomplished through an organizational, multidisciplinary, antimicrobial management program(232).

- 4. MDRO surveillance.** Surveillance is a critically important component of any MDRO control program, allowing detection of newly emerging pathogens, monitoring epidemiologic trends, and measuring the effectiveness of interventions. Multiple MDRO surveillance strategies have been employed, ranging from surveillance of clinical microbiology laboratory results obtained as part of routine clinical care, to use of ASC to detect asymptomatic colonization.

Surveillance for MDROs isolated from routine clinical cultures.

Antibiograms. The simplest form of MDRO surveillance is monitoring of clinical microbiology isolates resulting from tests ordered as part of routine clinical care. This method is particularly useful to detect emergence of new MDROs not previously detected, either within an individual healthcare facility or community-wide. In addition, this information can be used to prepare facility- or unit-specific summary antimicrobial susceptibility reports that describe pathogen-specific prevalence of resistance among clinical isolates. Such reports may be useful to monitor for changes in known resistance patterns that might signal emergence or transmission of MDROs, and also to provide clinicians with information to guide antimicrobial prescribing practices(233-235).

MDRO Incidence Based on Clinical Culture Results. Some investigators have used clinical microbiology results to calculate measures of incidence of MDRO isolates in specific populations or patient care locations (e.g. new MDRO

isolates/1,000 patient days, new MDRO isolates per month)(205, 236, 237). Such measures may be useful for monitoring MDRO trends and assessing the impact of prevention programs, although they have limitations. Because they are based solely on positive culture results without accompanying clinical information, they do not distinguish colonization from infection, and may not fully demonstrate the burden of MDRO-associated disease. Furthermore, these measures do not precisely measure acquisition of MDRO colonization in a given population or location. Isolating an MDRO from a clinical culture obtained from a patient several days after admission to a given unit or facility does not establish that the patient acquired colonization in that unit. On the other hand, patients who acquire MDRO colonization may remain undetected by clinical cultures(107). Despite these limitations, incidence measures based on clinical culture results may be highly correlated with actual MDRO transmission rates derived from information using ASC, as demonstrated in a recent multicenter study(237). These results suggest that incidence measures based on clinical cultures alone might be useful surrogates for monitoring changes in MDRO transmission rates.

MDRO Infection Rates. Clinical cultures can also be used to identify targeted MDRO infections in certain patient populations or units(238, 239). This strategy requires investigation of clinical circumstances surrounding a positive culture to distinguish colonization from infection, but it can be particularly helpful in defining the clinical impact of MDROs within a facility.

Molecular typing of MDRO isolates. Many investigators have used molecular typing of selected isolates to confirm clonal transmission to enhance understanding of MDRO transmission and the effect of interventions within their facility(38, 68, 89, 92, 138, 152, 190, 193, 236, 240).

Surveillance for MDROs by Detecting Asymptomatic Colonization

Another form of MDRO surveillance is the use of active surveillance cultures (ASC) to identify patients who are colonized with a targeted MDRO(38, 107, 241). This

approach is based upon the observation that, for some MDROs, detection of colonization may be delayed or missed completely if culture results obtained in the course of routine clinical care are the primary means of identifying colonized patients(8, 38, 107, 114, 151, 153, 167, 168, 183, 184, 187, 189, 191-193, 242-244). Several authors report having used ASC when new pathogens emerge in order to define the epidemiology of the particular agent(22, 23, 107, 190). In addition, the authors of several reports have concluded that ASC, in combination with use of Contact Precautions for colonized patients, contributed directly to the decline or eradication of the target MDRO(38, 68, 107, 151, 153, 184, 217, 242). However, not all studies have reached the same conclusion. Poor control of MRSA despite use of ASC has been described(245). A recent study failed to identify cross-transmission of MRSA or MSSA in a MICU during a 10 week period when ASC were obtained, despite the fact that culture results were not reported to the staff(246). The investigators suggest that the degree of cohorting and adherence to Standard Precautions might have been the important determinants of transmission prevention, rather than the use of ASC and Contact Precautions for MRSA-colonized patients. The authors of a systematic review of the literature on the use of isolation measures to control healthcare-associated MRSA concluded that there is evidence that concerted efforts that include ASC and isolation can reduce MRSA even in endemic settings. However, the authors also noted that methodological weaknesses and inadequate reporting in published research make it difficult to rule out plausible alternative explanations for reductions in MRSA acquisition associated with these interventions, and therefore concluded that the precise contribution of active surveillance and isolation alone is difficult to assess(247).

Mathematical modeling studies have been used to estimate the impact of ASC use in control of MDROs. One such study evaluating interventions to decrease VRE transmission indicated that use of ASC (versus no cultures) could potentially decrease transmission 39% and that with pre-emptive isolation plus ASC, transmission could be decreased 65%(248). Another mathematical model examining the use of ASC and isolation for control of MRSA predicted that isolating colonized or

infected patients on the basis of clinical culture results is unlikely to be successful at controlling MRSA, whereas use of active surveillance and isolation can lead to successful control, even in settings where MRSA is highly endemic.(249) There is less literature on the use of ASC in controlling MDR-GNBs. Active surveillance cultures have been used as part of efforts to successful control of MDR-GNBs in outbreak settings. The experience with ASC as part of successful control efforts in endemic settings is mixed. One study reported successful reduction of extended-spectrum beta-lactamase –producing Enterobacteriaceae over a six year period using a multifaceted control program that included use of ASC(245). Other reports suggest that use of ASC is not necessary to control endemic MDR-GNBs.(250, 251).

More research is needed to determine the circumstances under which ASC are most beneficial(252), but their use should be considered in some settings, especially if other control measures have been ineffective. When use of ASC is incorporated into MDRO prevention programs, the following should be considered:

- The decision to use ASC as part of an infection prevention and control program requires additional support for successful implementation, including: 1) personnel to obtain the appropriate cultures, 2) microbiology laboratory personnel to process the cultures, 3) mechanism for communicating results to caregivers, 4) concurrent decisions about use of additional isolation measures triggered by a positive culture (e.g. Contact Precautions) and 5) mechanism for assuring adherence to the additional isolation measures.
- The populations targeted for ASC are not well defined and vary among published reports. Some investigators have chosen to target specific patient populations considered at high risk for MDRO colonization based on factors such as location (e.g. ICU with high MDRO rates), antibiotic exposure history, presence of underlying diseases, prolonged duration of stay, exposure to other MDRO-colonized patients, patients transferred from other facilities known to have a high prevalence of MDRO carriage, or having a history of recent hospital or nursing home stays(107, 151, 253). A more commonly employed strategy involves obtaining surveillance cultures from all patients admitted to units experiencing

high rates of colonization/infection with the MDROs of interest, unless they are already known to be MDRO carriers(153, 184, 242, 254). In an effort to better define target populations for active surveillance, investigators have attempted to create prediction rules to identify subpopulations of patients at high risk for colonization on hospital admission(255, 256). Decisions about which populations should be targeted for active surveillance should be made in the context of local determinations of the incidence and prevalence of MDRO colonization within the intervention facility as well as other facilities with whom patients are frequently exchanged(257).

- Optimal timing and interval of ASC are not well defined. In many reports, cultures were obtained at the time of admission to the hospital or intervention unit or at the time of transfer to or from designated units (e.g., ICU)(107). In addition, some hospitals have chosen to obtain cultures on a periodic basis [e.g., weekly(8, 153, 159) to detect silent transmission. Others have based follow-up cultures on the presence of certain risk factors for MDRO colonization, such as antibiotic exposure, exposure to other MDRO colonized patients, or prolonged duration of stay in a high risk unit(253).
- Methods for obtaining ASC must be carefully considered, and may vary depending upon the MDRO of interest.
 - MRSA: Studies suggest that cultures of the nares identify most patients with MRSA and perirectal and wound cultures can identify additional carriers(152, 258-261).
 - VRE: Stool, rectal, or perirectal swabs are generally considered a sensitive method for detection of VRE. While one study suggested that rectal swabs may identify only 60% of individuals harboring VRE, and may be affected by VRE stool density(262), this observation has not been reported elsewhere in the literature.
 - MDR-GNBs: Several methods for detection of MDR-GNBs have been employed, including use of peri-rectal or rectal swabs alone or in combination with oro-pharyngeal, endotracheal, inguinal, or wound cultures. The absence of standardized screening media for many gram-

negative bacilli can make the process of isolating a specific MDR-GNB a relatively labor-intensive process(38, 190, 241, 250).

- Rapid detection methods: Using conventional culture methods for active surveillance can result in a delay of 2-3 days before results are available. If the infection control precautions (e.g., Contact Precautions) are withheld until the results are available, the desired infection control measures could be delayed. If empiric precautions are used pending negative surveillance culture results, precautions may be unnecessarily implemented for many, if not most, patients. For this reason, investigators have sought methods for decreasing the time necessary to obtain a result from ASC. Commercially available media containing chromogenic enzyme substrates (CHROMagar MRSA(263, 264) has been shown to have high sensitivity and specificity for identification of MRSA and facilitate detection of MRSA colonies in screening cultures as early as 16 hours after inoculation. In addition, real-time PCR-based tests for rapid detection of MRSA directly from culture swabs (< 1-2 hours) are now commercially available(265-267), as well as PCR-based tests for detection of vanA and van B genes from rectal swabs(268). The impact of rapid testing on the effectiveness of active surveillance as a prevention strategy, however, has not been fully determined. Rapid identification of MRSA in one study was associated with a significant reduction in MRSA infections acquired in the medical ICU, but not the surgical ICU(265). A mathematical model characterizing MRSA transmission dynamics predicted that, in comparison to conventional culture methods, the use of rapid detection tests may decrease isolation needs in settings of low-endemicity and result in more rapid reduction in prevalence in highly-endemic settings(249).
- Some MDRO control reports described surveillance cultures of healthcare personnel during outbreaks, but colonized or infected healthcare personnel are rarely the source of ongoing transmission, and this strategy should be reserved for settings in which specific healthcare personnel have been epidemiologically implicated in the transmission of MDROs(38, 92, 152-154, 188).

5. Infection Control Precautions. Since 1996 CDC has recommended the use of Standard and Contact Precautions for MDROs “judged by an infection control program...to be of special clinical and epidemiologic significance.” This recommendation was based on general consensus and was not necessarily evidence-based. No studies have directly compared the efficacy of Standard Precautions alone versus Standard Precautions and Contact Precautions, with or without ASC, for control of MDROs. Some reports mention the use of one or both sets of precautions as part of successful MDRO control efforts; however, the precautions were not the primary focus of the study intervention(164, 190, 205, 269-271). The NIH-sponsored study mentioned earlier (Section: *Overview of the MDRO control literature*) may provide some answers, <http://clinicaltrials.gov/ct/show/NCT00100386?order=1>).

Standard Precautions have an essential role in preventing MDRO transmission, even in facilities that use Contact Precautions for patients with an identified MDRO. Colonization with MDROs is frequently undetected; even surveillance cultures may fail to identify colonized persons due to lack of sensitivity, laboratory deficiencies, or intermittent colonization due to antimicrobial therapy(262). Therefore, Standard Precautions must be used in order to prevent transmission from potentially colonized patients. Hand hygiene is an important component of Standard Precautions. The authors of the *Guideline for Hand Hygiene in Healthcare Settings*(106) cited nine studies that demonstrated a temporal relationship between improved adherence to recommended hand hygiene practices and control of MDROs. It is noteworthy that in one report the frequency of hand hygiene did not improve with use of Contact Precautions but did improve when gloves were used (per Standard Precautions) for contact with MDRO patients(272).

MDRO control efforts frequently involved changes in isolation practices, especially during outbreaks. In the majority of reports, Contact Precautions were implemented for all patients found to be colonized or infected with the target MDRO (See Table 2).

Some facilities also preemptively used Contact Precautions, in conjunction with ASC, for all new admissions or for all patients admitted to a specific unit, until a negative screening culture for the target MDRO was reported(30, 184, 273).

Contact Precautions are intended to prevent transmission of infectious agents, including epidemiologically important microorganisms, which are transmitted by direct or indirect contact with the patient or the patient's environment. A single-patient room is preferred for patients who require Contact Precautions. When a single-patient room is not available, consultation with infection control is necessary to assess the various risks associated with other patient placement options (e.g., cohorting, keeping the patient with an existing roommate). HCP caring for patients on Contact Precautions should wear a gown and gloves for all interactions that may involve contact with the patient or potentially contaminated areas in the patient's environment. Donning gown and gloves upon room entry and discarding before exiting the patient room is done to contain pathogens, especially those that have been implicated in transmission through environmental contamination (e.g., VRE, *C. difficile*, noroviruses and other intestinal tract agents; RSV)(109, 111, 274-277).

Cohorting and other MDRO control strategies. In several reports, cohorting of patients(152, 153, 167, 183, 184, 188, 189, 217, 242), cohorting of staff(184, 217, 242, 278), use of designated beds or units(183, 184), and even unit closure(38, 146, 159, 161, 279, 280) were necessary to control transmission. Some authors indicated that implementation of the latter two strategies were the turning points in their control efforts; however, these measures usually followed many other actions to prevent transmission. In one, two-center study, moving MRSA-positive patients into single rooms or cohorting these patients in designated bays failed to reduce transmission in ICUs. However, in this study adherence to recommendations for hand hygiene between patient contacts was only 21%(281). Other published studies, including one commissioned by the American Institute of Architects and the Facility Guidelines Institute (www.aia.org/aah_gd_hospcons), have documented a beneficial relationship between private rooms and reduction in risk of acquiring MDROs(282). Additional

studies are needed to define the specific contribution of using single-patient rooms and/or cohorting on preventing transmission of MDROs.

Duration of Contact Precautions. The necessary duration of Contact Precautions for patients treated for infection with an MDRO, but who may continue to be colonized with the organism at one or more body sites, remains an unresolved issue. Patients may remain colonized with MDROs for prolonged periods; shedding of these organisms may be intermittent, and surveillance cultures may fail to detect their presence(84, 250, 283). The 1995 HICPAC guideline for preventing the transmission of VRE suggested three negative stool/perianal cultures obtained at weekly intervals as a criterion for discontinuation of Contact Precautions(274). One study found these criteria generally reliable(284). However, this and other studies have noted a recurrence of VRE positive cultures in persons who subsequently receive antimicrobial therapy and persistent or intermittent carriage of VRE for more than 1 year has been reported(284-286). Similarly, colonization with MRSA can be prolonged(287, 288). Studies demonstrating initial clearance of MRSA following decolonization therapy have reported a high frequency of subsequent carriage(289, 290). There is a paucity of information in the literature on when to discontinue Contact Precautions for patients colonized with a MDR-GNB, possibly because infection and colonization with these MDROs are often associated with outbreaks. Despite the uncertainty about when to discontinue Contact Precautions, the studies offer some guidance. In the context of an outbreak, prudence would dictate that Contact Precautions be used indefinitely for all previously infected and known colonized patients. Likewise, if ASC are used to detect and isolate patients colonized with MRSA or VRE, and there is no decolonization of these patients, it is logical to assume that Contact Precautions would be used for the duration of stay in the setting where they were first implemented. In general, it seems reasonable to discontinue Contact Precautions when three or more surveillance cultures for the target MDRO are repeatedly negative over the course of a week or two in a patient who has not received antimicrobial therapy for several weeks, especially in the absence of a

draining wound, profuse respiratory secretions, or evidence implicating the specific patient in ongoing transmission of the MDRO within the facility.

Barriers used for contact with patients infected or colonized with MDROs.

Three studies evaluated the use of gloves with or without gowns for all patient contacts to prevent VRE acquisition in ICU settings(30, 105, 273). Two of the studies showed that use of both gloves and gowns reduced VRE transmission(30, 105) while the third showed no difference in transmission based on the barriers used(273). One study in a LTCF compared the use of gloves only, with gloves plus contact isolation, for patients with four MDROs, including VRE and MRSA, and found no difference(86). However, patients on contact isolation were more likely to acquire MDR-*K. pneumoniae* strains that were prevalent in the facility; reasons for this were not specifically known. In addition to differences in outcome, differing methodologies make comparisons difficult. Specifically, HCP adherence to the recommended protocol, the influence of added precautions on the number of HCP-patient interactions, and colonization pressure were not consistently assessed.

Impact of Contact Precautions on patient care and well-being. There are limited data regarding the impact of Contact Precautions on patients. Two studies found that HCP, including attending physicians, were half as likely to enter the rooms of(291), or examine(292), patients on Contact Precautions. Other investigators have reported similar observations on surgical wards(293). Two studies reported that patients in private rooms and on barrier precautions for an MDRO had increased anxiety and depression scores(294, 295). Another study found that patients placed on Contact Precautions for MRSA had significantly more preventable adverse events, expressed greater dissatisfaction with their treatment, and had less documented care than control patients who were not in isolation(296). Therefore, when patients are placed on Contact Precautions, efforts must be made by the healthcare team to counteract these potential adverse effects.

6. Environmental measures. The potential role of environmental reservoirs, such as surfaces and medical equipment, in the transmission of VRE and other MDROs has been the subject of several reports(109-111, 297, 298). While environmental cultures are not routinely recommended(299), environmental cultures were used in several studies to document contamination, and led to interventions that included the use of dedicated noncritical medical equipment(217, 300), assignment of dedicated cleaning personnel to the affected patient care unit(154), and increased cleaning and disinfection of frequently-touched surfaces (e.g., bedrails, charts, bedside commodes, doorknobs). A common reason given for finding environmental contamination with an MDRO was the lack of adherence to facility procedures for cleaning and disinfection. In an educational and observational intervention, which targeted a defined group of housekeeping personnel, there was a persistent decrease in the acquisition of VRE in a medical ICU(301). Therefore, monitoring for adherence to recommended environmental cleaning practices is an important determinant for success in controlling transmission of MDROs and other pathogens in the environment(274, 302).

In the MDRO reports reviewed, enhanced environmental cleaning was frequently undertaken when there was evidence of environmental contamination and ongoing transmission. Rarely, control of the target MDRO required vacating a patient care unit for complete environmental cleaning and assessment(175, 279).

7. Decolonization. Decolonization entails treatment of persons colonized with a specific MDRO, usually MRSA, to eradicate carriage of that organism. Although some investigators have attempted to decolonize patients harboring VRE(220), few have achieved success. However, decolonization of persons carrying MRSA in their nares has proved possible with several regimens that include topical mupirocin alone or in combination with orally administered antibiotics (e.g., rifampin in combination with trimethoprim- sulfamethoxazole or ciprofloxacin) plus the use of an antimicrobial soap for bathing(303). In one report, a 3-day regimen of baths with povidone-iodine and nasal therapy with mupirocin resulted in eradication of nasal MRSA

colonization(304). These and other methods of MRSA decolonization have been thoroughly reviewed.(303, 305-307).

Decolonization regimens are not sufficiently effective to warrant routine use. Therefore, most healthcare facilities have limited the use of decolonization to MRSA outbreaks, or other high prevalence situations, especially those affecting special-care units. Several factors limit the utility of this control measure on a widespread basis: 1) identification of candidates for decolonization requires surveillance cultures; 2) candidates receiving decolonization treatment must receive follow-up cultures to ensure eradication; and 3) recolonization with the same strain, initial colonization with a mupirocin-resistant strain, and emergence of resistance to mupirocin during treatment can occur(289, 303, 308-310). HCP implicated in transmission of MRSA are candidates for decolonization and should be treated and culture negative before returning to direct patient care. In contrast, HCP who are colonized with MRSA, but are asymptomatic, and have not been linked epidemiologically to transmission, do not require decolonization.

IV. Discussion

This review demonstrates the depth of published science on the prevention and control of MDROs. Using a combination of interventions, MDROs in endemic, outbreak, and non-endemic settings have been brought under control. However, despite the volume of literature, an appropriate set of evidence-based control measures that can be universally applied in all healthcare settings has not been definitively established. This is due in part to differences in study methodology and outcome measures, including an absence of randomized, controlled trials comparing one MDRO control measure or strategy with another. Additionally, the data are largely descriptive and quasi-experimental in design(311). Few reports described preemptive efforts or prospective studies to control MDROs before they had reached high levels within a unit or facility. Furthermore, small hospitals and LTCFs are infrequently represented in the literature.

A number of questions remain and are discussed below.

Impact on other MDROs from interventions targeted to one MDRO Only one report described control efforts directed at more than one MDRO, i.e., MDR-GNB and MRSA(312). Several reports have shown either decreases or increases in other pathogens with efforts to control one MDRO. For example, two reports on VRE control efforts demonstrated an increase in MRSA following the prioritization of VRE patients to private rooms and cohort beds(161). Similarly an outbreak of *Serratia marcescens* was temporally associated with a concurrent, but unrelated, outbreak of MRSA in an NICU(313). In contrast, Wright and colleagues reported a decrease in MRSA and VRE acquisition in an ICU during and after their successful effort to eradicate an MDR-strain of *A. baumannii* from the unit(210).

Colonization with multiple MDROs appears to be common(314, 315). One study found that nearly 50% of residents in a skilled-care unit in a LTCF were colonized with a target MDRO and that 26% were co-colonized with >1 MDRO; a detailed analysis showed that risk factors for colonization varied by pathogen(316). One review of the literature(317) reported that patient risk factors associated with colonization with MRSA, VRE, MDR-GNB, *C. difficile* and *Candida sp* were the same. This review concluded that control programs that focus on only one organism or one antimicrobial drug are unlikely to succeed because vulnerable patients will continue to serve as a magnet for other MDROs.

Costs. Several authors have provided evidence for the cost-effectiveness of approaches that use ASC(153, 191, 253, 318, 319). However, the supportive evidence often relied on assumptions, projections, and estimated attributable costs of MDRO infections. Similar limitations apply to a study suggesting that gown use yields a cost benefit in controlling transmission of VRE in ICUs(320). To date, no studies have directly compared the benefits and costs associated with different MDRO control strategies.

Feasibility. The subject of feasibility, as it applies to the extrapolation of results to other healthcare settings, has not been addressed. For example, smaller hospitals and LTCFs may lack the on-site laboratory services needed to obtain ASC in a timely manner. This factor could limit the applicability of an aggressive program based on obtaining ASC and preemptive placement of patients on Contact Precautions in these settings. However, with

the growing problem of antimicrobial resistance, and the recognized role of all healthcare settings for control of this problem, it is imperative that appropriate human and fiscal resources be invested to increase the feasibility of recommended control strategies in every setting.

Factors that influence selection of MDRO control measures. Although some common principles apply, the preceding literature review indicates that no single approach to the control of MDROs is appropriate for all healthcare facilities. Many factors influence the choice of interventions to be applied within an institution, including:

- **Type and significance of problem MDROs within the institution.** Many facilities have an MRSA problem while others have ESBL-producing *K. pneumoniae*. Some facilities have no VRE colonization or disease; others have high rates of VRE colonization without disease; and still others have ongoing VRE outbreaks. The magnitude of the problem also varies. Healthcare facilities may have very low numbers of cases, e.g., with a newly introduced strain, or may have prolonged, extensive outbreaks or colonization in the population. Between these extremes, facilities may have low or high levels of endemic colonization and variable levels of infection.
- **Population and healthcare-settings.** The presence of high-risk patients (e.g., transplant, hematopoietic stem-cell transplant) and special-care units (e.g. adult, pediatric, and neonatal ICUs; burn; hemodialysis) will influence surveillance needs and could limit the areas of a facility targeted for MDRO control interventions. Although it appears that MDRO transmission seldom occurs in ambulatory and outpatient settings, some patient populations (e.g., hemodialysis, cystic fibrosis) and patients receiving chemotherapeutic agents are at risk for colonization and infection with MDROs. Furthermore, the emergence of VRSA within the outpatient setting(22, 23, 25) demonstrates that even these settings need to make MDRO prevention a priority.

Differences of opinion on the optimal strategy to control MDROs. Published guidance on the control of MDROs reflects areas of ongoing debate on optimal control strategies. A key issue is the use of ASC in control efforts and preemptive use of Contact Precautions pending negative surveillance culture results(107, 321, 322). The various guidelines currently available exhibit a spectrum of approaches, which their authors deem to be evidence-based. One guideline for control of MRSA and VRE, the Society for Healthcare Epidemiology of America (SHEA) guideline from 2003(107), emphasizes routine use of ASC and Contact Precautions. That position paper does not address control of MDR-GNBs. The salient features of SHEA recommendations for MRSA and VRE control and the recommendations in this guideline for control of MDROs, including MRSA and VRE, have been compared(323); recommended interventions are similar. Other guidelines for VRE and MRSA, e.g., those proffered by the Michigan Society for Infection Control (www.msic-online.org/resource_sections/aro_guidelines), emphasize consistent practice of Standard Precautions and tailoring the use of ASC and Contact Precautions to local conditions, the specific MDROs that are prevalent and being transmitted, and the presence of risk factors for transmission. A variety of approaches have reduced MDRO rates(3, 164, 165, 209, 214, 240, 269, 324). Therefore, selection of interventions for controlling MDRO transmission should be based on assessments of the local problem, the prevalence of various MDRO and feasibility. Individual facilities should seek appropriate guidance and adopt effective measures that fit their circumstances and needs. Most studies have been in acute care settings; for non-acute care settings (e.g., LCTF, small rural hospitals), the optimal approach is not well defined.

Two-Tiered Approach for Control of MDROs. Reports describing successful control of MDRO transmission in healthcare facilities have included seven categories of interventions (Table 3). As a rule, these reports indicate that facilities confronted with an MDRO problem selected a combination of control measures, implemented them, and reassessed their impact. In some cases, new measures were added serially to further enhance control efforts. This evidence indicates that the control of MDROs is a dynamic process that requires a systematic approach tailored to the problem and healthcare setting. The nature of this evidence gave rise to the two-tiered approach to MDRO control

recommended in this guideline. This approach provides the flexibility needed to prevent and control MDRO transmission in every kind of facility addressed by this guideline. Detailed recommendations for MDRO control in all healthcare settings follow and are summarized in Table 3. Table 3, which applies to all healthcare settings, contains two tiers of activities. In the first tier are the baseline level of MDRO control activities designed to ensure recognition of MDROs as a problem, involvement of healthcare administrators, and provision of safeguards for managing unidentified carriers of MDROs.

With the emergence of an MDRO problem that cannot be controlled with the basic set of infection control measures, additional control measures should be selected from the second tier of interventions presented in Table 3. Decisions to intensify MDRO control activity arise from surveillance observations and assessments of the risk to patients in various settings. Circumstances that may trigger these decisions include:

- Identification of an MDRO from even one patient in a facility or special unit with a highly vulnerable patient population (e.g., an ICU, NICU, burn unit) that had previously not encountered that MDRO.
- Failure to decrease the prevalence or incidence of a specific MDRO (e.g., incidence of resistant clinical isolates) despite infection control efforts to stop its transmission. (Statistical process control charts or other validated methods that account for normal variation can be used to track rates of targeted MDROs)(205, 325, 326).

The combination of new or increased frequency of MDRO isolates and patients at risk necessitates escalation of efforts to achieve or re-establish control, i.e., to reduce rates of transmission to the lowest possible level. Intensification of MDRO control activities should begin with an assessment of the problem and evaluation of the effectiveness of measures in current use. Once the problem is defined, appropriate additional control measures should be selected from the second tier of Table 3. A knowledgeable infection prevention and control professional or healthcare epidemiologist should make this determination. This approach requires support from the governing body and medical staff of the facility. Once interventions are implemented, ongoing surveillance should be used to determine whether selected control measures are effective and if additional measures or consultation are

indicated. The result of this process should be to decrease MDRO rates to minimum levels. Healthcare facilities must not accept ongoing MDRO outbreaks or high endemic rates as the status quo. With selection of infection control measures appropriate to their situation, all facilities *can achieve* the desired goal and reduce the MDRO burden substantially.

V. Prevention of transmission of Multidrug Resistant Organisms (Table 3)

The CDC/HICPAC system for categorizing recommendations is as follows:

Category IA Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiologic studies.

Category IB Strongly recommended for implementation and supported by some experimental, clinical, or epidemiologic studies and a strong theoretical rationale.

Category IC Required for implementation, as mandated by federal and/or state regulation or standard.

Category II Suggested for implementation and supported by suggestive clinical or epidemiologic studies or a theoretical rationale.

No recommendation Unresolved issue. Practices for which insufficient evidence or no consensus regarding efficacy exists.

V.A. General recommendations for all healthcare settings independent of the prevalence of multidrug resistant organism (MDRO) infections or the population served.

V.A.1. Administrative measures

V.A.1.a. Make MDRO prevention and control an organizational patient safety priority.(3, 146, 151, 154, 182, 185, 194, 205, 208, 210, 242, 327, 328)

Category IB

V.A.1.b. Provide administrative support, and both fiscal and human resources, to prevent and control MDRO transmission within the healthcare organization (3, 9, 146, 152, 182-184, 208, 328, 329) *Category IB*

V.A.1.c. In healthcare facilities without expertise for analyzing epidemiologic data, recognizing MDRO problems, or devising effective control strategies (e.g., small or rural hospitals, rehabilitation centers, long-term care facilities [LTCFs], freestanding ambulatory centers), identify experts who can provide consultation as needed.(151, 188) *Category II*

V.A.1.d. Implement systems to communicate information about reportable MDROs [e.g., VRSA, VISA, MRSA, Penicillin resistant *S. pneumoniae*(PRSP)] to administrative personnel and as required by state and local health

- authorities (www.cdc.gov/epo/dphsi/nndsshis.htm). Refer to websites for updated requirements of local and state health departments. *Category II/IC*
- V.A.1.e. Implement a multidisciplinary process to monitor and improve healthcare personnel (HCP) adherence to recommended practices for Standard and Contact Precautions(3, 105, 182, 184, 189, 242, 273, 312, 330). *Category IB*
 - V.A.1.f. Implement systems to designate patients known to be colonized or infected with a targeted MDRO and to notify receiving healthcare facilities and personnel prior to transfer of such patients within or between facilities.(87, 151) *Category IB*
 - V.A.1.g. Support participation of the facility or healthcare system in local, regional, and national coalitions to combat emerging or growing MDRO problems.(41, 146, 151, 167, 188, 206, 207, 211, 331). *Category IB*
 - V.A.1.h. Provide updated feedback at least annually to healthcare providers and administrators on facility and patient-care-unit trends in MDRO infections. Include information on changes in prevalence or incidence of infection, results of assessments for system failures, and action plans to improve adherence to and effectiveness of recommended infection control practices to prevent MDRO transmission.(152, 154, 159, 184, 204, 205, 242, 312, 332) *Category IB*
 - V.A.2. Education and training of healthcare personnel
 - V.A.2.a. Provide education and training on risks and prevention of MDRO transmission during orientation and periodic educational updates for healthcare personnel; include information on organizational experience with MDROs and prevention strategies.(38, 152, 154, 173, 176, 189, 190, 203, 204, 217, 242, 330, 333, 334) *Category IB*
 - V.A.3. Judicious use of antimicrobial agents. The goal of the following recommendations is to ensure that systems are in place to promote optimal treatment of infections and appropriate antimicrobial use.
 - V.A.3.a. In hospitals and LTCFs, ensure that a multidisciplinary process is in place to review antimicrobial utilization, local susceptibility patterns

(antibiograms), and antimicrobial agents included in the formulary to foster appropriate antimicrobial use.(209, 212, 214, 215, 217, 242, 254, 334-339)

Category IB

V.A.3.b. Implement systems (e.g., computerized physician order entry, comment in microbiology susceptibility report, notification from a clinical pharmacist or unit director) to prompt clinicians to use the appropriate antimicrobial agent and regimen for the given clinical situation.(156, 157, 161, 166, 174, 175, 212, 214, 218, 254, 334, 335, 337, 340-346) *Category IB*

V.A.3.b.i. Provide clinicians with antimicrobial susceptibility reports and analysis of current trends, updated at least annually, to guide antimicrobial prescribing practices.(342, 347) *Category IB*

V.A.3.b.ii. In settings that administer antimicrobial agents but have limited electronic communication system infrastructures to implement physician prompts (e.g., LTCFs, home care and infusion companies), implement a process for appropriate review of prescribed antimicrobials. Prepare and distribute reports to prescribers that summarize findings and provide suggestions for improving antimicrobial use. (342, 348, 349) *Category II*

V.A.4. Surveillance

V.A.4.a. In microbiology laboratories, use standardized laboratory methods and follow published guidance for determining antimicrobial susceptibility of targeted (e.g., MRSA, VRE, MDR-ESBLs) and emerging (e.g., VRSA, MDR-*Acinetobacter baumannii*) MDROs.(8, 154, 177, 190, 193, 209, 254, 347, 350-353) *Category IB*

V.A.4.b. In all healthcare organizations, establish systems to ensure that clinical microbiology laboratories (in-house and out-sourced) promptly notify infection control staff or a medical director/ designee when a novel resistance pattern for that facility is detected.(9, 22, 154, 162, 169) *Category IB*

V.A.4.c. In hospitals and LTCFs, develop and implement laboratory protocols for storing isolates of selected MDROs for molecular typing when needed to

confirm transmission or delineate the epidemiology of the MDRO within the healthcare setting.(7, 8, 38, 140, 153, 154, 187, 190, 208, 217, 354, 355)

Category IB

- V.A.4.d. Prepare facility-specific antimicrobial susceptibility reports as recommended by the Clinical and Laboratory Standards Institute (CLSI) (www.phppo.cdc.gov/dls/master/default.aspx); monitor these reports for evidence of changing resistance patterns that may indicate the emergence or transmission of MDROs.(347, 351, 356, 357) *Category IB/IC*
 - V.A.4.d.i. In hospitals and LTCFs with special-care units (e.g., ventilator-dependent, ICU, or oncology units), develop and monitor unit-specific antimicrobial susceptibility reports.(358-361) *Category IB*
 - V.A.4.d.ii. Establish a frequency for preparing summary reports based on volume of clinical isolates, with updates at least annually.(347, 362) *Category II/IC*
 - V.A.4.d.iii. In healthcare organizations that outsource microbiology laboratory services (e.g., ambulatory care, home care, LTCFs, smaller acute care hospitals), specify by contract that the laboratory provide either facility-specific susceptibility data or local or regional aggregate susceptibility data in order to identify prevalent MDROs and trends in the geographic area served.(363) *Category II*
- V.A.4.e. Monitor trends in the incidence of target MDROs in the facility over time using appropriate statistical methods to determine whether MDRO rates are decreasing and whether additional interventions are needed.(152, 154, 183, 193, 205, 209, 217, 242, 300, 325, 326, 364, 365) *Category IA*
 - V.A.4.e.i. Specify isolate origin (i.e., location and clinical service) in MDRO monitoring protocols in hospitals and other large multi-unit facilities with high-risk patients.(8, 38, 152-154, 217, 358, 361) *Category IB*
 - V.A.4.e.ii. Establish a baseline (e.g., incidence) for targeted MDRO isolates by reviewing results of clinical cultures; if more timely or localized information is needed, perform baseline point prevalence studies of colonization in high-risk units. When possible, distinguish

colonization from infection in analysis of these data.(152, 153, 183, 184, 189, 190, 193, 205, 242, 365) *Category IB*

V.A.5. Infection control precautions to prevent transmission of MDROs

V.A.5.a. Follow Standard Precautions during all patient encounters in all settings in which healthcare is delivered.(119, 164, 255, 315, 316) *Category IB*

V.A.5.b. Use masks according to Standard Precautions when performing splash-generating procedures (e.g., wound irrigation, oral suctioning, intubation); when caring for patients with open tracheostomies and the potential for projectile secretions; and in circumstances where there is evidence of transmission from heavily colonized sources (e.g., burn wounds). Masks are not otherwise recommended for prevention of MDRO transmission from patients to healthcare personnel during routine care (e.g., upon room entry).(8, 22, 151, 152, 154, 189, 190, 193, 208, 240, 366) *Category IB*

V.A.5.c. Use of Contact Precautions

V.A.5.c.i. In *acute-care hospitals*, implement Contact Precautions routinely for all patients infected with target MDROs and for patients that have been previously identified as being colonized with target MDROs (e.g., patients transferred from other units or facilities who are known to be colonized). (11, 38, 68, 114, 151, 183, 188, 204, 217, 242, 304) *Category IB*

V.A.5.c.ii. In LTCFs, consider the individual patient's clinical situation and prevalence or incidence of MDRO in the facility when deciding whether to implement or modify Contact Precautions in addition to Standard Precautions for a patient infected or colonized with a target MDRO. *Category II*

V.A.5.c.ii.1. For relatively healthy residents (e.g., mainly independent) follow Standard Precautions, making sure that gloves and gowns are used for contact with uncontrolled secretions, pressure ulcers, draining wounds, stool incontinence, and ostomy tubes/bags. (78-80, 85, 151, 367, 368) *Category II*

- V.A.5.c.ii.2. For ill residents (e.g., those totally dependent upon healthcare personnel for healthcare and activities of daily living, ventilator-dependent) and for those residents whose infected secretions or drainage cannot be contained, use Contact Precautions in addition to Standard Precautions.(316, 369, 370) *Category II*
- V.A.5.c.iii. For MDRO colonized or infected patients without draining wounds, diarrhea, or uncontrolled secretions, establish ranges of permitted ambulation, socialization, and use of common areas based on their risk to other patients and on the ability of the colonized or infected patients to observe proper hand hygiene and other recommended precautions to contain secretions and excretions.(151, 163, 371) *Category II*
- V.A.5.d. In *ambulatory settings*, use Standard Precautions for patients known to be infected or colonized with target MDROs, making sure that gloves and gowns are used for contact with uncontrolled secretions, pressure ulcers, draining wounds, stool incontinence, and ostomy tubes and bags. *Category II*
- V.A.5.e. In *home care settings*
- Follow Standard Precautions making sure to use gowns and gloves for contact with uncontrolled secretions, pressure ulcers, draining wounds, stool incontinence, and ostomy tubes and bags. *Category II*
 - Limit the amount of reusable patient-care equipment that is brought into the home of patients infected or colonized with MDROs. When possible, leave patient-care equipment in the home until the patient is discharged from home care services. *Category II*
 - If noncritical patient-care equipment (e.g., stethoscopes) cannot remain in the home, clean and disinfect items before removing them from the home, using a low to intermediate level disinfectant, or place reusable items in a plastic bag for transport

to another site for subsequent cleaning and disinfection.

Category II

V.A.5.e.i. No recommendation is made for routine use of gloves, gowns, or both to prevent MDRO transmission in ambulatory or home care settings. *Unresolved issue*

V.A.5.e.ii. In *hemodialysis units*, follow the “Recommendations to Prevent Transmission of Infections in Chronic Hemodialysis Patients”(372)(www.cms.hhs.gov/home/regsguidance.asp).

Category IC

V.A.5.f. Discontinuation of Contact Precautions. No recommendation can be made regarding when to discontinue Contact Precautions. *Unresolved issue* (See Background for discussion of options)

V.A.5.g. Patient placement in hospitals and LTCFs

V.A.5.g.i. When single-patient rooms are available, assign priority for these rooms to patients with known or suspected MDRO colonization or infection. Give highest priority to those patients who have conditions that may facilitate transmission, e.g., uncontained secretions or excretions.(8, 38, 110, 151, 188, 208, 240, 304) *Category IB*

V.A.5.g.ii. When single-patient rooms are not available, cohort patients with the same MDRO in the same room or patient-care area.(8, 38, 92, 151-153, 162, 183, 184, 188, 217, 242, 304) *Category IB*

V.A.5.g.iii. When cohorting patients with the same MDRO is not possible, place MDRO patients in rooms with patients who are at low risk for acquisition of MDROs and associated adverse outcomes from infection and are likely to have short lengths of stay. *Category II*

V.A.6. Environmental measures

V.A.6.a. Clean and disinfect surfaces and equipment that may be contaminated with pathogens, including those that are in close proximity to the patient (e.g., bed rails, over bed tables) and frequently-touched surfaces in the patient care environment (e.g., door knobs, surfaces in and surrounding toilets in patients’ rooms) on a more frequent schedule compared to that for minimal

touch surfaces (e.g., horizontal surfaces in waiting rooms).(111, 297, 373)
Category IB

V.A.6.b. Dedicate noncritical medical items to use on individual patients known to be infected or colonized with MDROs.(38, 217, 324, 374, 375) *Category IB*

V.A.6.c. Prioritize room cleaning of patients on Contact Precautions. Focus on cleaning and disinfecting frequently touched surfaces (e.g., bedrails, bedside commodes, bathroom fixtures in the patient's room, doorknobs) and equipment in the immediate vicinity of the patient.(109, 110, 114-117, 297, 301, 373, 376, 377) *Category IB*

V.B. Intensified interventions to prevent MDRO transmission

The interventions presented below have been utilized in various combinations to reduce transmission of MDROs in healthcare facilities. Neither the effectiveness of individual components nor that of specific combinations of control measures has been assessed in controlled trials. Nevertheless, various combinations of control elements selected under the guidance of knowledgeable content experts have repeatedly reduced MDRO transmission rates in a variety of healthcare settings.

V.B.1. Indications and approach

V.B.1.a. Indications for intensified MDRO control efforts (VII.B.1.a.i and VII.B.1.a.ii) should result in selection and implementation of one or more of the interventions described in VII.B.2 to VII.B.8 below. Individualize the selection of control measures according to local considerations(8, 11, 38, 68, 114, 152-154, 183-185, 189, 190, 193, 194, 209, 217, 242, 312, 364, 365). *Category IB*

V.B.1.a.i. When incidence or prevalence of MDROs are not decreasing despite implementation of and correct adherence to the routine control measures described above, intensify MDRO control efforts by adopting one or more of the interventions described below.(92, 152, 183, 184, 193, 365) *Category IB*

V.B.1.a.ii. When the *first* case or outbreak of an epidemiologically important MDRO (e.g., VRE, MRSA, VISA, VRSA, MDR-GNB) is identified

within a healthcare facility or unit.(22, 23, 25, 68, 170, 172, 184, 240, 242, 378) *Category IB*

V.B.1.b. Continue to monitor the incidence of target MDRO infection and colonization after additional interventions are implemented. If rates do not decrease, implement more interventions as needed to reduce MDRO transmission.(11, 38, 68, 92, 152, 175, 184, 365) *Category IB*

V.B.2. Administrative measures

V.B.2.a. Identify persons with experience in infection control and the epidemiology of MDRO, either in house or through outside consultation, for assessment of the local MDRO problem and for the design, implementation, and evaluation of appropriate control measures (3, 68, 146, 151-154, 167, 184, 190, 193, 242, 328, 377). *Category IB*

V.B.2.b. Provide necessary leadership, funding, and day-to-day oversight to implement interventions selected. Involve the governing body and leadership of the healthcare facility or system that have organizational responsibility for this and other infection control efforts.(8, 38, 152, 154, 184, 189, 190, 208) *Category IB*

V.B.2.c. Evaluate healthcare system factors for their role in creating or perpetuating transmission of MDROs, including: staffing levels, education and training, availability of consumable and durable resources, communication processes, policies and procedures, and adherence to recommended infection control measures (e.g., hand hygiene and Standard or Contact Precautions). Develop, implement, and monitor action plans to correct system failures.(3, 8, 38, 152, 154, 172, 173, 175, 188, 196, 198, 199, 208, 217, 280, 324, 379, 380) *Category IB*

V.B.2.d. During the process, update healthcare providers and administrators on the progress and effectiveness of the intensified interventions. Include information on changes in prevalence, rates of infection and colonization; results of assessments and corrective actions for system failures; degrees of adherence to recommended practices; and action plans to improve

adherence to recommended infection control practices to prevent MDRO transmission.(152, 154, 159, 184, 204, 205, 312, 332, 381) *Category IB*

V.B.3. Educational interventions

Intensify the frequency of MDRO educational programs for healthcare personnel, especially those who work in areas in which MDRO rates are not decreasing. Provide individual or unit-specific feedback when available.(3, 38, 152, 154, 159, 170, 182, 183, 189, 190, 193, 194, 204, 205, 209, 215, 218, 312) *Category IB*

V.B.4. Judicious use of antimicrobial agents

Review the role of antimicrobial use in perpetuating the MDRO problem targeted for intensified intervention. Control and improve antimicrobial use as indicated. Antimicrobial agents that may be targeted include vancomycin, third-generation cephalosporins, and anti-anaerobic agents for VRE(217); third-generation cephalosporins for ESBLs(212, 214, 215); and quinolones and carbapenems(80, 156, 166, 174, 175, 209, 218, 242, 254, 329, 334, 335, 337, 341). *Category IB*

V.B.5. Surveillance

V.B.5.a. Calculate and analyze prevalence and incidence rates of targeted MDRO infection and colonization in populations at risk; when possible, distinguish colonization from infection(152, 153, 183, 184, 189, 190, 193, 205, 215, 242, 365). *Category IB*

V.B.5.a.i. Include only one isolate per patient, not multiple isolates from the same patient, when calculating rates(347, 382). *Category II*

V.B.5.a.ii. Increase the frequency of compiling and monitoring antimicrobial susceptibility summary reports for a targeted MDRO as indicated by an increase in incidence of infection or colonization with that MDRO. *Category II*

V.B.5.b. Develop and implement protocols to obtain active surveillance cultures (ASC) for targeted MDROs from patients in populations at risk (e.g., patients in intensive care, burn, bone marrow/stem cell transplant, and oncology units; patients transferred from facilities known to have high

MDRO prevalence rates; roommates of colonized or infected persons; and patients known to have been previously infected or colonized with an MDRO).(8, 38, 68, 114, 151-154, 167, 168, 183, 184, 187-190, 192, 193, 217, 242) *Category IB*

- V.B.5.b.i. Obtain ASC from areas of skin breakdown and draining wounds. In addition, include the following sites according to target MDROs:
 - V.B.5.b.i.1. For MRSA: Sampling the anterior nares is usually sufficient; throat, endotracheal tube aspirate, percutaneous gastrostomy sites, and perirectal or perineal cultures may be added to increase the yield. Swabs from several sites may be placed in the same selective broth tube prior to transport.(117, 383, 384) *Category IB*
 - V.B.5.b.i.2. For VRE: Stool, rectal, or perirectal samples should be collected.(154, 193, 217, 242)
Category IB
 - V.B.5.b.i.3. For MDR-GNB: Endotracheal tube aspirates or sputum should be cultured if a respiratory tract reservoir is suspected, (e.g., *Acinetobacter* spp., *Burkholderia* spp.).(385, 386) *Category IB*.
- V.B.5.b.ii. Obtain surveillance cultures for the target MDRO from patients at the time of admission to high-risk areas, e.g., ICUs, and at periodic intervals as needed to assess MDRO transmission.(8, 151, 154, 159, 184, 208, 215, 242, 387) *Category IB*
- V.B.5.c. Conduct culture surveys to assess the efficacy of the enhanced MDRO control interventions.
 - V.B.5.c.i. Conduct serial (e.g., weekly, until transmission has ceased and then decreasing frequency) unit-specific point prevalence culture surveys of the target MDRO to determine if transmission has decreased or ceased.(107, 167, 175, 184, 188, 218, 339) *Category IB*
 - V.B.5.c.ii. Repeat point-prevalence culture surveys at routine intervals or at time of patient discharge or transfer until transmission has ceased.(8, 152-154, 168, 178, 190, 215, 218, 242, 388) *Category IB*

- V.B.5.c.iii. If indicated by assessment of the MDRO problem, collect cultures to assess the colonization status of roommates and other patients with substantial exposure to patients with known MDRO infection or colonization.(25, 68, 167, 193) *Category IB*
- V.B.5.d. Obtain cultures of healthcare personnel for target MDRO when there is epidemiologic evidence implicating the healthcare staff member as a source of ongoing transmission.(153, 365) *Category IB*
- V.B.6. Enhanced infection control precautions
 - V.B.6.a. Use of Contact Precautions
 - V.B.6.a.i. Implement Contact Precautions routinely for all patients colonized or infected with a target MDRO.(8, 11, 38, 68, 114, 151, 154, 183, 188, 189, 217, 242, 304) *Category IA*
 - V.B.6.a.ii. Because environmental surfaces and medical equipment, especially those in close proximity to the patient, may be contaminated, don gowns and gloves *before or upon entry* to the patient's room or cubicle.(38, 68, 154, 187, 189, 242) *Category IB*
 - V.B.6.a.iii. In LTCFs, modify Contact Precautions to allow MDRO-colonized/infected patients whose site of colonization or infection can be appropriately contained and who can observe good hand hygiene practices to enter common areas and participate in group activities.(78, 86, 151, 367) *Category IB*
 - V.B.6.b. When ASC are obtained as part of an intensified MDRO control program, implement Contact Precautions until the surveillance culture is reported negative for the target MDRO.(8, 30, 153, 389, 390) *Category IB*
 - V.B.6.c. No recommendation is made regarding universal use of gloves, gowns, or both in high-risk units in acute-care hospitals.(153, 273, 312, 320, 391)
Unresolved issue
- V.B.7. Implement policies for patient admission and placement as needed to prevent transmission of a problem MDRO.(183, 184, 189, 193, 242, 339, 392)
Category IB

- V.B.7.a.i. Place MDRO patients in single-patient rooms.(6, 151, 158, 160, 166, 170, 187, 208, 240, 282, 393-395) *Category IB*
 - V.B.7.a.ii. Cohort patients with the same MDRO in designated areas (e.g., rooms, bays, patient care areas).(8, 151, 152, 159, 161, 176, 181, 183, 184, 188, 208, 217, 242, 280, 339, 344) *Category IB*
 - V.B.7.a.iii. When transmission continues despite adherence to Standard and Contact Precautions and cohorting patients, assign dedicated nursing and ancillary service staff to the care of MDRO patients only. Some facilities may consider this option when intensified measures are first implemented.(184, 217, 242, 278) *Category IB*
 - V.B.7.a.iv. Stop new admissions to the unit of facility if transmission continues despite the implementation of the enhanced control measures described above. (Refer to state or local regulations that may apply upon closure of hospital units or services.).(9, 38, 146, 159, 161, 168, 175, 205, 279, 280, 332, 339, 396) *Category IB*
- V.B.8. Enhanced environmental measures
- V.B.8.a. Implement patient-dedicated or single-use disposable noncritical equipment (e.g., blood pressure cuff, stethoscope) and instruments and devices.(38, 104, 151, 156, 159, 163, 181, 217, 324, 329, 367, 389, 390, 394) *Category IB*
 - V.B.8.b. Intensify and reinforce training of environmental staff who work in areas targeted for intensified MDRO control and monitor adherence to environmental cleaning policies. Some facilities may choose to assign dedicated staff to targeted patient care areas to enhance consistency of proper environmental cleaning and disinfection services.(38, 154, 159, 165, 172, 173, 175, 178-181, 193, 205, 208, 217, 279, 301, 327, 339, 397) *Category IB*
 - V.B.8.c. Monitor (i.e., supervise and inspect) cleaning performance to ensure consistent cleaning and disinfection of surfaces in close proximity to the patient and those likely to be touched by the patient and HCP (e.g.,

- bedrails, carts, bedside commodes, doorknobs, faucet handles).(8, 38, 109, 111, 154, 169, 180, 208, 217, 301, 333, 398) *Category IB*
- V.B.8.d. Obtain environmental cultures (e.g., surfaces, shared medical equipment) when there is epidemiologic evidence that an environmental source is associated with ongoing transmission of the targeted MDRO.(399-402) *Category IB*
- V.B.8.e. Vacate units for environmental assessment and intensive cleaning when previous efforts to eliminate environmental reservoirs have failed.(175, 205, 279, 339, 403) *Category II*
- V.B.9. Decolonization
- V.B.9.a. Consult with physicians with expertise in infectious diseases and/or healthcare epidemiology on a case-by-case basis regarding the appropriate use of decolonization therapy for patients or staff during limited periods of time, as a component of an intensified MRSA control program).(152, 168, 170, 172, 183, 194, 304) *Category II*
- V.B.9.b. When decolonization for MRSA is used, perform susceptibility testing for the decolonizing agent against the target organism in the individual being treated or the MDRO strain that is epidemiologically implicated in transmission. Monitor susceptibility to detect emergence of resistance to the decolonizing agent. Consult with a microbiologist for appropriate testing for mupirocin resistance, since standards have not been established.(289, 290, 304, 308) *Category IB*
- V.B.9.b.i. Because mupirocin-resistant strains may emerge and because it is unusual to eradicate MRSA when multiple body sites are colonized, do not use topical mupirocin *routinely* for MRSA decolonization of patients as a component of MRSA control programs in any healthcare setting.(289, 404) *Category IB*
- V.B.9.b.ii. Limit decolonization of HCP found to be colonized with MRSA to persons who have been epidemiologically linked as a likely source of ongoing transmission to patients. Consider reassignment of HCP

if decolonization is not successful and ongoing transmission to patients persists.(120, 122, 168) *Category IB*

- V.B.9.c. No recommendation can be made for decolonizing patients with VRE or MDR-GNB. Regimens and efficacy of decolonization protocols for VRE and MDR-GNB have not been established.(284, 286, 288, 307, 387, 405)
Unresolved issue

Glossary - Multidrug-Resistant Organisms

Ambulatory care settings. Facilities that provide health care to patients who do not remain overnight (e.g., hospital-based outpatient clinics, nonhospital-based clinics and physician offices, urgent care centers, surgicenters, free-standing dialysis centers, public health clinics, imaging centers, ambulatory behavioral health and substance abuse clinics, physical therapy and rehabilitation centers, and dental practices).

Cohorting. In the context of this guideline, this term applies to the practice of grouping patients infected or colonized with the same infectious agent together to confine their care to one area and prevent contact with susceptible patients (cohorting patients). During outbreaks, healthcare personnel may be assigned to a cohort of patients to further limit opportunities for transmission (cohorting staff).

Contact Precautions. Contact Precautions are a set of practices used to prevent transmission of infectious agents that are spread by direct or indirect contact with the patient or the patient's environment. Contact Precautions also apply where the presence of excessive wound drainage, fecal incontinence, or other discharges from the body suggest an increased transmission risk. A single patient room is preferred for patients who require Contact Precautions. When a single patient room is not available, consultation with infection control is helpful to assess the various risks associated with other patient placement options (e.g., cohorting, keeping the patient with an existing roommate). In multi-patient rooms, ≥ 3 feet spatial separation of between beds is advised to reduce the opportunities for inadvertent sharing of items between the infected/colonized patient and other patients. Healthcare personnel caring for patients on Contact Precautions wear a gown and gloves for all interactions that may involve contact with the patient or potentially contaminated areas in the patient's environment. Donning of gown and gloves upon room entry, removal before exiting the patient room and performance of hand hygiene immediately upon exiting are done to contain pathogens.

Epidemiologically important pathogens. Infectious agents that have one or more of the following characteristics: 1) A propensity for transmission within healthcare facilities based on published reports and the occurrence of temporal or geographic clusters of ≥ 2 patients, (e.g., VRE, MRSA and MSSA, *Clostridium difficile*, norovirus, RSV, influenza, rotavirus, *Enterobacter* spp; *Serratia* spp., group A streptococcus). However, for group A streptococcus, most experts consider a single case of healthcare-associated disease a trigger for investigation and enhanced control measures because of the devastating outcomes associated with HAI group A streptococcus infections. For susceptible bacteria that are known to be associated with asymptomatic colonization, isolation from normally sterile body fluids in patients with significant clinical disease would be the trigger to consider the organism as epidemiologically important. 2) Antimicrobial resistance implications:

- Resistance to first-line therapies (e.g., MRSA, VRE, VISA, VRSA, ESBL-producing organisms).
- Unusual or usual agents with unusual patterns of resistance within a facility, (e.g., the first isolate of *Burkholderia cepacia* complex or *Ralstonia* spp. in non-CF patients or a quinolone-resistant strain of *Pseudomonas* in a facility).
- Difficult to treat because of innate or acquired resistance to multiple classes of antimicrobial agents (e.g., *Stenotrophomonas maltophilia*, *Acinetobacter* spp.).

3) Associated with serious clinical disease, increased morbidity and mortality (e.g., MRSA and MSSA, group A streptococcus); or 4) A newly discovered or reemerging pathogen. The strategies described for MDROs may be applied for control of epidemiologically important organisms other than MDROs.

Hand hygiene. A general term that applies to any one of the following: 1) handwashing with plain (nonantimicrobial) soap and water); 2) antiseptic hand wash (soap containing antiseptic agents and water); 3) antiseptic hand rub (waterless antiseptic product, most often alcohol-based, rubbed on all surfaces of hands); or 4) surgical hand antisepsis

(antiseptic hand wash or antiseptic hand rub performed preoperatively by surgical personnel to eliminate transient hand flora and reduce resident hand flora).

Healthcare-associated infection (HAI). An infection that develops in a patient who is cared for in any setting where healthcare is delivered (e.g., acute care hospital, chronic care facility, ambulatory clinic, dialysis center, surgicenter, home) and is related to receiving health care (i.e., was not incubating or present at the time healthcare was provided). In ambulatory and home settings, HAI would apply to any infection that is associated with a medical or surgical intervention performed in those settings.

Healthcare epidemiologist A person whose primary training is medical (M.D., D.O.) and/or masters or doctorate-level epidemiology who has received advanced training in healthcare epidemiology. Typically these professionals direct or provide consultation to an infection prevention and control program in a hospital, long term care facility (LTCF), or healthcare delivery system (also see infection prevention and control professional).

Healthcare personnel (HCP). All paid and unpaid persons who work in a healthcare setting, also known as healthcare workers (e.g. any person who has professional or technical training in a healthcare-related field and provides patient care in a healthcare setting or any person who provides services that support the delivery of healthcare such as dietary, housekeeping, engineering, maintenance personnel).

Home care. A wide-range of medical, nursing, rehabilitation, hospice, and social services delivered to patients in their place of residence (e.g., private residence, senior living center, assisted living facility). Home health-care services include care provided by home health aides and skilled nurses, respiratory therapists, dietitians, physicians, chaplains, and volunteers; provision of durable medical equipment; home infusion therapy; and physical, speech, and occupational therapy.

Infection prevention and control professional (ICP). A person whose primary training is in either nursing, medical technology, microbiology, or epidemiology and who has acquired

specialized training in infection control. Responsibilities may include collection, analysis, and feedback of infection data and trends to healthcare providers; consultation on infection risk assessment, prevention and control strategies; performance of education and training activities; implementation of evidence-based infection control practices or those mandated by regulatory and licensing agencies; application of epidemiologic principles to improve patient outcomes; participation in planning renovation and construction projects (e.g., to ensure appropriate containment of construction dust); evaluation of new products or procedures on patient outcomes; oversight of employee health services related to infection prevention; implementation of preparedness plans; communication within the healthcare setting, with local and state health departments, and with the community at large concerning infection control issues; and participation in research.

Infection prevention and control program. A multidisciplinary program that includes a group of activities to ensure that recommended practices for the prevention of healthcare-associated infections are implemented and followed by healthcare personnel, making the healthcare setting safe from infection for patients and healthcare personnel. The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) requires the following five components of an infection prevention and control program for accreditation: 1) *surveillance*: monitoring patients and healthcare personnel for acquisition of infection and/or colonization; 2) *investigation*: identification and analysis of infection problems or undesirable trends; 3) *prevention*: implementation of measures to prevent transmission of infectious agents and to reduce risks for device- and procedure-related infections; 4) *control*: evaluation and management of outbreaks; and 5) *reporting*: provision of information to external agencies as required by state and federal law and regulation (www.jcaho.org). The infection prevention and control program staff has the ultimate authority to determine infection control policies for a healthcare organization with the approval of the organization's governing body.

Long-term care facilities (LTCFs). An array of residential and outpatient facilities designed to meet the bio-psychosocial needs of persons with sustained self-care deficits. These include skilled nursing facilities, chronic disease hospitals, nursing homes, foster and group homes, institutions for the developmentally disabled, residential care facilities, assisted

living facilities, retirement homes, adult day health care facilities, rehabilitation centers, and long-term psychiatric hospitals.

Mask. A term that applies collectively to items used to cover the nose and mouth and includes both procedure masks and surgical masks (www.fda.gov/cdrh/ode/guidance/094.html#4).

Multidrug-resistant organisms (MDROs). In general, bacteria (excluding *M. tuberculosis*) that are resistant to one or more classes of antimicrobial agents and usually are resistant to all but one or two commercially available antimicrobial agents (e.g., MRSA, VRE, extended spectrum beta-lactamase [ESBL]-producing or intrinsically resistant gram-negative bacilli).

Nosocomial infection. Derived from two Greek words “nosos” (disease) and “komeion” (to take care of). Refers to any infection that develops during or as a result of an admission to an acute care facility (hospital) and was not incubating at the time of admission.

Standard Precautions. A group of infection prevention practices that apply to all patients, regardless of suspected or confirmed diagnosis or presumed infection status. Standard Precautions are a combination and expansion of Universal Precautions and Body Substance Isolation. Standard Precautions are based on the principle that all blood, body fluids, secretions, excretions except sweat, nonintact skin, and mucous membranes may contain transmissible infectious agents. Standard Precautions includes hand hygiene, and depending on the anticipated exposure, use of gloves, gown, mask, eye protection, or face shield. Also, equipment or items in the patient environment likely to have been contaminated with infectious fluids must be handled in a manner to prevent transmission of infectious agents, (e.g. wear gloves for handling, contain heavily soiled equipment, properly clean and disinfect or sterilize reusable equipment before use on another patient).

1. IOM (1998), eds. Harrison, P. F. & Lederberg, J. (National Academy Press, Washington, DC), pp. 8-74.
2. Shlaes, D. M., Gerding, D. N., John, J. F., Jr., Craig, W. A., Bornstein, D. L., Duncan, R. A., Eckman, M. R., Farrer, W. E., Greene, W. H., Lorian, V., *et al.* (1997) *Infect Control Hosp Epidemiol* **18**, 275-291.
3. Larson, E. L., Early, E., Cloonan, P., Sugrue, S., & Parides, M. (2000) *Behav Med* **26**, 14-22.
4. Goldmann, D. A., Weinstein, R. A., Wenzel, R. P., Tablan, O. C., Duma, R. J., Gaynes, R. P., Schlosser, J., & Martone, W. J. (1996) *JAMA* **275**, 234-240.
5. Murthy, R. (2001) *Chest* **119**, 405S-411S.
6. Mahgoub, S., Ahmed, J., & Glatt, A. E. (2002) *Infect Control Hosp Epidemiol* **23**, 477-479.
7. Fournier, P. E. & Richet, H. (2006) *Clin Infect Dis* **42**, 692-699.
8. Fierobe, L., Lucet, J. C., Decre, D., Muller-Serieys, C., Deleuze, A., Joly-Guillou, M. L., Mantz, J., & Desmonts, J. M. (2001) *Infect Control Hosp Epidemiol* **22**, 35-40.
9. Ling, M. L., Ang, A., Wee, M., & Wang, G. C. (2001) *Infect Control Hosp Epidemiol* **22**, 48-49.
10. Landman, D., Quale, J. M., Mayorga, D., Adedeji, A., Vangala, K., Ravishankar, J., Flores, C., & Brooks, S. (2002) *Arch Intern Med* **162**, 1515-1520.
11. Urban, C., Segal-Maurer, S., & Rahal, J. J. (2003) *Clin Infect Dis* **36**, 1268-1274.
12. Gales, A. C., Jones, R. N., Forward, K. R., Linares, J., Sader, H. S., & Verhoef, J. (2001) *Clin Infect Dis* **32 Suppl 2**, S104-113.
13. del Toro, M. D., Rodriguez-Bano, J., Herrero, M., Rivero, A., Garcia-Ordenez, M. A., Corzo, J., & Perez-Cano, R. (2002) *Medicine (Baltimore)* **81**, 228-239.
14. Hanes, S. D., Demirkan, K., Tolley, E., Boucher, B. A., Croce, M. A., Wood, G. C., & Fabian, T. C. (2002) *Clin Infect Dis* **35**, 228-235.
15. Saiman, L. & Siegel, J. (2003) *Infect Control Hosp Epidemiol* **24**, S6-52.
16. Loukil, C., Saizou, C., Doit, C., Bidet, P., Mariani-Kurkdjian, P., Aujard, Y., Beaufils, F., & Bingen, E. (2003) *Infect Control Hosp Epidemiol* **24**, 707-710.
17. Ryan, M. P., Pembroke, J. T., & Adley, C. C. (2006) *J Hosp Infect* **62**, 278-284.
18. Fry, A. M., Udeagu, C. C., Soriano-Gabarro, M., Fridkin, S., Musinski, D., LaClaire, L., Elliott, J., Cook, D. J., Kornblum, J., Layton, M., *et al.* (2005) *Infect Control Hosp Epidemiol* **26**, 239-247.
19. Carter, R. J., Sorenson, G., Heffernan, R., Kiehlbauch, J. A., Kornblum, J. S., Leggiadro, R. J., Nixon, L. J., Wertheim, W. A., Whitney, C. G., & Layton, M. (2005) *Infect Control Hosp Epidemiol* **26**, 248-255.
20. Whitener, C. J., Park, S. Y., Browne, F. A., Parent, L. J., Julian, K., Bozdogan, B., Appelbaum, P. C., Chaitram, J., Weigel, L. M., Jernigan, J., *et al.* (2004) *Clin Infect Dis* **38**, 1049-1055.
21. CDC (1997) *MMWR Morb Mortal Wkly Rep* **46 (33)**, 765-766.
22. CDC (2002) *MMWR Morb Mortal Wkly Rep* **51 (26)**, 565-567.
23. CDC (2002) *MMWR - Morbidity & Mortality Weekly Report* **51(40)**, 902.
24. CDC (2004) *MMWR Morb Mortal Wkly Rep* **53**, 322-323.
25. Chang, S., Sievert, D. M., Hageman, J. C., Boulton, M. L., Tenover, F. C., Downes, F. P., Shah, S., Rudrik, J. T., Pupp, G. R., Brown, W. J., *et al.* (2003) *N Engl J Med* **348**, 1342-1347.

26. Fridkin, S. K., Hageman, J., McDougal, L. K., Mohammed, J., Jarvis, W. R., Perl, T. M., & Tenover, F. C. (2003) *Clin Infect Dis* **36**, 429-439.
27. Hageman, J. C., Fridkin, S. K., Mohammed, J. M., Steward, C. D., Gaynes, R. P., & Tenover, F. C. (2003) *Infect Control Hosp Epidemiol* **24**, 356-361.
28. Rotun, S. S., McMath, V., Schoonmaker, D. J., Maupin, P. S., Tenover, F. C., Hill, B. C., & Ackman, D. M. (1999) *Emerg Infect Dis* **5**, 147-149.
29. Smith, T. L., Pearson, M. L., Wilcox, K. R., Cruz, C., Lancaster, M. V., Robinson-Dunn, B., Tenover, F. C., Zervos, M. J., Band, J. D., White, E., *et al.* (1999) *N Engl J Med* **340**, 493-501.
30. Srinivasan, A., Dick, J. D., & Perl, T. M. (2002) *Clin Microbiol Rev* **15**, 430-438.
31. Gonzales, R. D., Schreckenberger, P. C., Graham, M. B., Kelkar, S., DenBesten, K., & Quinn, J. P. (2001) *Lancet* **357**, 1179.
32. Soltani, M., Beighton, D., Philpott-Howard, J., & Woodford, N. (2001) *Antimicrob Agents Chemother* **45**, 645-646.
33. Pai, M. P., Rodvold, K. A., Schreckenberger, P. C., Gonzales, R. D., Petrolatti, J. M., & Quinn, J. P. (2002) *Clin Infect Dis* **35**, 1269-1272.
34. Pillai, S. K., Sakoulas, G., Wennersten, C., Eliopoulos, G. M., Moellering, R. C., Jr., Ferraro, M. J., & Gold, H. S. (2002) *J Infect Dis* **186**, 1603-1607.
35. Hershberger, E., Donabedian, S., Konstantinou, K., & Zervos, M. J. (2004) *Clin Infect Dis* **38**, 92-98.
36. Mangili, A., Bica, I., Snyderman, D. R., & Hamer, D. H. (2005) *Clin Infect Dis* **40**, 1058-1060.
37. Sabol, K., Patterson, J. E., Lewis, J. S., 2nd, Owens, A., Cadena, J., & Jorgensen, J. H. (2005) *Antimicrob Agents Chemother* **49**, 1664-1665.
38. Simor, A. E., Lee, M., Vearncombe, M., Jones-Paul, L., Barry, C., Gomez, M., Fish, J. S., Cartotto, R. C., Palmer, R., & Louie, M. (2002) *Infect Control Hosp Epidemiol* **23**, 261-267.
39. Clarke, N. M., Patterson, J., & Lynch, I. J. (2003) *Curr Opin Crit Care* **9**, 413-423.
40. Martone, W. J. (1998) *Infect Control Hosp Epidemiol* **19**, 539-545.
41. The Brooklyn Antibiotic Task Force (2002) *Infect Control Hosp Epidemiol* **23**, 106-108.
42. Wilson, S. J., Knipe, C. J., Zieger, M. J., Gabehart, K. M., Goodman, J. E., Volk, H. M., & Sood, R. (2004) *Am J Infect Control* **32**, 342-344.
43. Qavi, A., Segal-Maurer, S., Mariano, N., Urban, C., Rosenberg, C., Burns, J., Chiang, T., Maurer, J., & Rahal, J. J. (2005) *Infect Control Hosp Epidemiol* **26**, 63-68.
44. Song, X., Srinivasan, A., Plaut, D., & Perl, T. M. (2003) *Infect Control Hosp Epidemiol* **24**, 251-256.
45. Aloush, V., Navon-Venezia, S., Seigman-Igra, Y., Cabili, S., & Carmeli, Y. (2006) *Antimicrob Agents Chemother* **50**, 43-48.
46. Cosgrove, S. E. (2006) *Clin Infect Dis* **42 Suppl 2**, S82-89.
47. Stone, P. W., Gupta, A., Loughrey, M., Della-Latta, P., Cimiotti, J., Larson, E., Rubenstein, D., & Saiman, L. (2003) *Infect Control Hosp Epidemiol* **24**, 601-606.
48. Cosgrove, S. E., Kaye, K. S., Eliopoulos, G. M., & Carmeli, Y. (2002) *Arch Intern Med* **162**, 185-190.
49. Linden, P. K., Pasculle, A. W., Manez, R., Kramer, D. J., Fung, J. J., Pinna, A. D., & Kusne, S. (1996) *Clin Infect Dis* **22**, 663-670.
50. Vergis, E. N., Hayden, M. K., Chow, J. W., Snyderman, D. R., Zervos, M. J., Linden, P. K., Wagener, M. M., Schmitt, B., & Muder, R. R. (2001) *Ann Intern Med* **135**, 484-492.
51. Salgado, C. D. & Farr, B. M. (2003) *Infect Control Hosp Epidemiol* **24**, 690-698.

52. DiazGranados, C. A. & Jernigan, J. A. (2005) *J Infect Dis* **191**, 588-595.
53. DiazGranados, C. A., Zimmer, S. M., Klein, M., & Jernigan, J. A. (2005) *Clin Infect Dis* **41**, 327-333.
54. Carmeli, Y., Eliopoulos, G., Mozaffari, E., & Samore, M. (2002) *Arch Intern Med* **162**, 2223-2228.
55. Davis, K. A., Stewart, J. J., Crouch, H. K., Florez, C. E., & Hospenthal, D. R. (2004) *Clin Infect Dis* **39**, 776-782.
56. Muder, R. R., Brennen, C., Wagener, M. M., Vickers, R. M., Rihs, J. D., Hancock, G. A., Yee, Y. C., Miller, J. M., & Yu, V. L. (1991) *Ann Intern Med* **114**, 107-112.
57. Cosgrove, S. E., Sakoulas, G., Perencevich, E. N., Schwaber, M. J., Karchmer, A. W., & Carmeli, Y. (2003) *Clin Infect Dis* **36**, 53-59.
58. Melzer, M., Eykyn, S. J., Gransden, W. R., & Chinn, S. (2003) *Clin Infect Dis* **37**, 1453-1460.
59. Selvey, L. A., Whitby, M., & Johnson, B. (2000) *Infect Control Hosp Epidemiol* **21**, 645-648.(s).
60. Romero-Vivas, J., Rubio, M., Fernandez, C., & Picazo, J. J. (1995) *Clin Infect Dis* **21**, 1417-1423.
61. Blot, S. I., Vandewoude, K. H., Hoste, E. A., & Colardyn, F. A. (2002) *Arch Intern Med* **162**, 2229-2235.
62. Reed, S. D., Friedman, J. Y., Engemann, J. J., Griffiths, R. I., Anstrom, K. J., Kaye, K. S., & al., e. (2005) *Infect Control Hosp Epidemiol* **26**, 175-183.
63. Mekontso-Dessap, A., Kirsch, M., Brun-Buisson, C., & Loisanche, D. (2001) *Clin Infect Dis* **32**, 877-883.
64. Engemann, J. J., Carmeli, Y., Cosgrove, S. E., Fowler, V. G., Bronstein, M. Z., Trivette, S. L., Briggs, J. P., Sexton, D. J., & Kaye, K. S. (2003) *Clin Infect Dis* **36**, 592-598.
65. Jones, R. N. (2006) *Clin Infect Dis* **42 Suppl 1**, S13-24.
66. Fowler, V. G., Jr., Sakoulas, G., McIntyre, L. M., Meka, V. G., Arbeit, R. D., Cabell, C. H., Stryjewski, M. E., Eliopoulos, G. M., Reller, L. B., Corey, G. R., *et al.* (2004) *J Infect Dis* **190**, 1140-1149.
67. Woods, C. W., Cheng, A. C., Fowler, V. G., Jr., Moorefield, M., Frederick, J., Sakoulas, G., Meka, V. G., Tenover, F. C., Zwadyk, P., & Wilson, K. H. (2004) *Clin Infect Dis* **38**, 1188-1191.
68. Jernigan, J. A., Clemence, M. A., Stott, G. A., Titus, M. G., Alexander, C. H., Palumbo, C. M., & Farr, B. M. (1995) *Infect Control Hosp Epidemiol* **16**, 686-696.
69. Stamm, A. M., Long, M. N., & Belcher, B. (1993) *Am J Infect Control* **21**, 70-74.
70. Harbarth, S., Albrich, W., Goldmann, D. A., & Huebner, J. (2001) *Lancet Infect Dis* **1**, 251-261.
71. Zinn, C. S., Westh, H., & Rosdahl, V. T. (2004) *Microb Drug Resist* **10**, 160-168.
72. Whitney, C. G., Farley, M. M., Hadler, J., Harrison, L. H., Lexau, C., Reingold, A., Lefkowitz, L., Cieslak, P. R., Cetron, M., Zell, E. R., *et al.* (2000) *N Engl J Med* **343**, 1917-1924.
73. Kollef, M. H. & Fraser, V. J. (2001) *Ann Intern Med* **134**, 298-314.
74. Fridkin, S. K. (2001) *Crit Care Med* **29**, N64-68.
75. Diekema, D. J., BootsMiller, B. J., Vaughn, T. E., Woolson, R. F., Yankey, J. W., Ernst, E. J., Flach, S. D., Ward, M. M., Franciscus, C. L., Pfaller, M. A., *et al.* (2004) *Clin Infect Dis* **38**, 78-85.

76. Polgreen, P. M., Beekmann, S. E., Chen, Y. Y., Doern, G. V., Pfaller, M. A., Brueggemann, A. B., Herwaldt, L. A., & Diekema, D. J. (2006) *Infect Control Hosp Epidemiol* **27**, 252-256.
77. Bradley, S. F., Terpenning, M. S., Ramsey, M. A., Zarins, L. T., Jorgensen, K. A., Sottile, W. S., Schaberg, D. R., & Kauffman, C. A. (1991) *Ann Intern Med* **115**, 417-422.
78. Brennen, C., Wagener, M. M., & Muder, R. R. (1998) *J Am Geriatr Soc* **46**, 157-160.
79. Strausbaugh, L. J., Crossley, K. B., Nurse, B. A., & Thrupp, L. D. (1996) *Infect Control Hosp Epidemiol* **17**, 129-140.
80. Bradley, S. F. (1999) *Infect Control Hosp Epidemiol* **20**, 362-366.
81. Bradley, S. F. (1999) *Am J Med* **106**, 2S-10S; discussion 48S-52S.
82. Wiener, J., Quinn, J. P., Bradford, P. A., Goering, R. V., Nathan, C., Bush, K., & Weinstein, R. A. (1999) *Jama* **281**, 517-523.
83. McNeil, S. A., Mody, L., & Bradley, S. F. (2002) *Geriatrics* **57**, 16-18, 21-14, 27.
84. Pacio, G. A., Visintainer, P., Maguire, G., Wormser, G. P., Raffalli, J., & Montecalvo, M. A. (2003) *Infect Control Hosp Epidemiol* **24**, 246-250.
85. Rahimi, A. R. (1998) *J Am Geriatr Soc* **46**, 1555-1557.
86. Trick, W. E., Weinstein, R. A., DeMarais, P. L., Tomaska, W., Nathan, C., McAllister, S. K., Hageman, J. C., Rice, T. W., Westbrook, G., & Jarvis, W. R. (2004) *J Am Geriatr Soc* **52**, 2003-2009.
87. Ben-Ami, R., Schwaber, M. J., Navon-Venezia, S., Schwartz, D., Giladi, M., Chmelnitsky, I., Leavitt, A., & Carmeli, Y. (2006) *Clin Infect Dis* **42**, 925-934.
88. Elizaga, M. L., Weinstein, R. A., & Hayden, M. K. (2002) *Clin Infect Dis* **34**, 441-446.
89. Saiman, L., Cronquist, A., Wu, F., Zhou, J., Rubenstein, D., Eisner, W., Kreiswirth, B. N., & Della-Latta, P. (2003) *Infect Control Hosp Epidemiol* **24**, 317-321.
90. Klevens, R. M., Edwards, J. R., Tenover, F. C., McDonald, L. C., Horan, T., & Gaynes, R. (2006) *Clin Infect Dis* **42**, 389-391.
91. Gaynes, R. & Edwards, J. R. (2005) *Clin Infect Dis* **41**, 848-854.
92. Boyce, J. M., Jackson, M. M., Pugliese, G., Batt, M. D., Fleming, D., Garner, J. S., Hartstein, A. I., Kauffman, C. A., Simmons, M., Weinstein, R., *et al.* (1994) *Infect Control Hosp Epidemiol* **15**, 105-115.
93. NNIS (2003) *Am J Infect Control* **31**, 481-498.
94. Fridkin, S. K., Edwards, J. R., Courval, J. M., Hill, H., Tenover, F. C., Lawton, R., Gaynes, R. P., & McGowan, J. E., Jr. (2001) *Ann Intern Med* **135**, 175-183.
95. Jones, R. N. (2001) *Chest* **119**, 397S-404S.
96. Neuhauser, M. M., Weinstein, R. A., Rydman, R., Danziger, L. H., Karam, G., & Quinn, J. P. (2003) *JAMA* **289**, 885-888.
97. Fridkin, S. K., Hill, H. A., Volkova, N. V., Edwards, J. R., Lawton, R. M., Gaynes, R. P., & McGowan, J. E., Jr. (2002) *Emerg Infect Dis* **8**, 697-701.
98. Madaras-Kelly, K. J., Remington, R. E., Lewis, P. G., & Stevens, D. L. (2006) *Infect Control Hosp Epidemiol* **27**, 155-169.
99. Fridkin, S. K., Hageman, J. C., Morrison, M., Sanza, L. T., Como-Sabetti, K., Jernigan, J. A., Harriman, K., Harrison, L. H., Lynfield, R., & Farley, M. M. (2005) *N Engl J Med* **352**, 1436-1444.
100. Kuehnert, M. J., Kruszon-Moran, D., Hill, H. A., McQuillan, G., McAllister, S. K., Fosheim, G., McDougal, L. K., Chaitram, J., Jensen, B., Fridkin, S. K., *et al.* (2006) *J Infect Dis* **193**, 172-179.

101. Bonten, M. J., Slaughter, S., Ambergen, A. W., Hayden, M. K., van Voorhis, J., Nathan, C., & Weinstein, R. A. (1998) *Arch Intern Med* **158**, 1127-1132.
102. Merrer, J., Santoli, F., Appere de Vecchi, C., Tran, B., De Jonghe, B., & Outin, H. (2000) *Infect Control Hosp Epidemiol* **21**, 718-723.
103. Lautenbach, E., Patel, J. B., Bilker, W. B., Edelstein, P. H., & Fishman, N. O. (2001) *Clin Infect Dis* **32**, 1162-1171.
104. Goetz, A. M., Rihs, J. D., Wagener, M. M., & Muder, R. R. (1998) *Am J Infect Control* **26**, 558-562.
105. Puzniak, L. A., Leet, T., Mayfield, J., Kollef, M., & Mundy, L. M. (2002) *Clin Infect Dis* **35**, 18-25.
106. CDC (2002) *MMWR* **51(16)**, 1-44.
107. Muto, C. A., Jernigan, J. A., Ostrowsky, B. E., Richet, H. M., Jarvis, W. R., Boyce, J. M., & Farr, B. M. (2003) *Infect Control Hosp Epidemiol* **24**, 362-386.
108. Almuneef, M. A., Baltimore, R. S., Farrel, P. A., Reagan-Cirincione, P., & Dembry, L. M. (2001) *Clin Infect Dis* **32**, 220-227.
109. Duckro, A. N., Blom, D. W., Lyle, E. A., Weinstein, R. A., & Hayden, M. K. (2005) *Arch Intern Med* **165**, 302-307.
110. Boyce, J. M., Potter-Bynoe, G., Chenevert, C., & King, T. (1997) *Infect Control Hosp Epidemiol* **18**, 622-627.(mj).
111. Bhalla, A., Pultz, N. J., Gries, D. M., Ray, A. J., Eckstein, E. C., Aron, D. C., & Donskey, C. J. (2004) *Infect Control Hosp Epidemiol* **25**, 164-167.
112. Larson, E. L., Cimiotti, J. P., Haas, J., Nesin, M., Allen, A., Della-Latta, P., & Saiman, L. (2005) *Pediatr Crit Care Med* **6**, 457-461.
113. Lee, Y. L., Cesario, T., Lee, R., Nothvogel, S., Nassar, J., Farsad, N., & Thrupp, L. (1994) *Am J Infect Control* **22**, 346-351.
114. Boyce, J. M., Opal, S. M., Chow, J. W., Zervos, M. J., Potter-Bynoe, G., Sherman, C. B., Romulo, R. L., Fortna, S., & Medeiros, A. A. (1994) *J Clin Microbiol* **32**, 1148-1153.
115. Gerding, D. N., Johnson, S., Peterson, L. R., Mulligan, M. E., & Silva, J., Jr. (1995) *Infect Control Hosp Epidemiol* **16**, 459-477.
116. Donskey, C. J. (2004) *Clin Infect Dis* **39**, 219-226.
117. Boyce, J. M., Havill, N. L., & Maria, B. (2005) *J Clin Microbiol* **43**, 5992-5995.
118. www.ihl.org/IHI/Programs/Campaign.
119. Evans, R., Lloyd, J. F., Abouzelof, R. H., Taylor, C. W., Anderson, V. R., & Samore, M. H. (2004) *Medinfo* **2004**, 212-216.
120. Boyce, J. M., Opal, S. M., Potter-Bynoe, G., & Medeiros, A. A. (1993) *Clin Infect Dis* **17**, 496-504.
121. Zawacki, A., O'Rourke, E., Potter-Bynoe, G., Macone, A., Harbarth, S., & Goldmann, D. (2004) *Infect Control Hosp Epidemiol* **25**, 1083-1089.
122. Faibis, F., Laporte, C., Fiacre, A., Delisse, C., Lina, G., Demachy, M.-C., & Botterel, F. (2005) *Infect Control Hosp Epidemiol* **26**, 213-215.
123. Sheretz, R. J., Reagan, D. R., Hampton, K. D., Robertson, K. L., Streed, S. A., Hoen, H. M., Thomas, R., & Gwaltney, J. M., Jr. (1996) *Ann Intern Med* **124**, 539-547.
124. Wang, J. T., Chang, S. C., Ko, W. J., Chang, Y. Y., Chen, M. L., Pan, H. J., & Luh, K. T. (2001) *J Hosp Infect* **47**, 104-109.
125. Herold, B. C., Immergluck, L. C., Maranan, M. C., Lauderdale, D. S., Gaskin, R. E., Boyle-Vavra, S., Leitch, C. D., & Daum, R. S. (1998) *JAMA* **279**, 593-598.

126. CDC (1999) *MMWR - Morbidity & Mortality Weekly Report* **48**, 707-710.
127. Fergie, J. E. & Purcell, K. (2001) *Pediatr Infect Dis J* **20**, 860-863.
128. Sattler, C. A., Mason, E. O., Jr., & Kaplan, S. L. (2002) *Pediatr Infect Dis J* **21**, 910-917.
129. Enright, M. C., Robinson, D. A., Randle, G., Feil, E. J., Grundmann, H., & Spratt, B. G. (2002) *Proc Natl Acad Sci U S A* **99**, 7687-7692.
130. Pan, E. S., Diep, B. A., Carleton, H. A., Charlebois, E. D., Sensabaugh, G. F., Haller, B. L., & Perdreau-Remington, F. (2003) *Clin Infect Dis* **37**, 1384-1388.
131. Daum, R. S., Ito, T., Hiramatsu, K., Hussain, F., Mongkolrattanothai, K., Jamklang, M., & Boyle-Vavra, S. (2002) *J Infect Dis* **186**, 1344-1347.
132. Said-Salim, B., Mathema, B., & Kreiswirth, B. N. (2003) *Infect Control Hosp Epidemiol* **24**, 451-455.
133. McDougal, L. K., Steward, C. D., Killgore, G. E., Chaitram, J. M., McAllister, S. K., & Tenover, F. C. (2003) *J Clin Microbiol* **41**, 5113-5120.
134. Zetola, N., Francis, J. S., Nuermberger, E. L., & Bishai, W. R. (2005) *Lancet Infect Dis* **5**, 275-286.
135. Adem, P. V., Montgomery, C. P., Husain, A. N., Koogler, T. K., Arangelovich, V., Humilier, M., Boyle-Vavra, S., & Daum, R. S. (2005) *N Engl J Med* **353**, 1245-1251.
136. Bocchini, C. E., Hulten, K. G., Mason, E. O., Jr., Gonzalez, B. E., Hammerman, W. A., & Kaplan, S. L. (2006) *Pediatrics* **117**, 433-440.
137. Healy, C. M., Hulten, K. G., Palazzi, D. L., Campbell, J. R., & Baker, C. J. (2004) *Clin Infect Dis* **39**, 1460-1466.
138. Saiman, L., O'keefe, M., Graham, P. L., Wu, F., Said-Salim, B., Kreiswirth, B., LaSala, A., Schlievert, P. M., & Della Latta, P. (2003) *Clin Infect Dis* **37**, 1313-1319.
139. Eckhardt, C., Halvosa, J. S., Ray, S. M., & Blumberg, H. M. (2003) *Infect Control Hosp Epidemiol* **24**, 460-461.
140. Seybold, U., Kourbatova, E. V., Johnson, J. G., Halvosa, S. J., Wang, Y. F., King, M. D., Ray, S. M., & Blumberg, H. M. (2006) *Clin Infect Dis* **42**, 647-656.
141. Berenholtz, S. M., Pronovost, P. J., Lipsett, P. A., Hobson, D., Earsing, K., Farley, J. E., Milanovich, S., Garrett-Mayer, E., Winters, B. D., Rubin, H. R., *et al.* (2004) *Crit Care Med* **32**, 2014-2020.
142. Coopersmith, C. M., Rebmann, T. L., Zack, J. E., Ward, M. R., Corcoran, R. M., Schallom, M. E., Sona, C. S., Buchman, T. G., Boyle, W. A., Polish, L. B., *et al.* (2002) *Crit Care Med* **30**, 59-64.
143. Babcock, H. M., Zack, J. E., Garrison, T., Trovillion, E., Jones, M., Fraser, V. J., & Kollef, M. H. (2004) *Chest* **125**, 2224-2231.
144. Warren, D. K., Zack, J. E., Cox, M. J., Cohen, M. M., & Fraser, V. J. (2003) *Crit Care Med* **31**, 1959-1963.
145. Eggimann, P., Hugonnet, S., Sax, H., Harbarth, S., Chevrolet, J. C., & Pittet, D. (2005) *Ann Intern Med* **142**, 875-876.
146. Verhoef, J., Beaujean, D., Blok, H., Baars, A., Meyler, A., van der Werken, C., & Weersink, A. (1999) *Eur J Clin Microbiol Infect Dis* **18**, 461-466.
147. Salmenlinna, S., Lyytikäinen, O., Kotilainen, P., Scotford, R., Siren, E., & Vuopio-Varkila, J. (2000) *Eur J Clin Microbiol Infect Dis* **19**, 101-107.
148. Struelens, M. J., Ronveaux, O., Jans, B., & Mertens, R. (1996) *Infect Control Hosp Epidemiol* **17**, 503-508.

149. Voss, A., Milatovic, D., Wallrauch-Schwarz, C., Rosdahl, V. T., & Braveny, I. (1994) *Eur J Clin Microbiol Infect Dis* **13**, 50-55.
150. Rosdahl, V. T. & Knudsen, A. M. (1991) *Infect Control Hosp Epidemiol* **12**, 83-88.
151. Ostrowsky, B. E., Trick, W. E., Sohn, A. H., Quirk, S. B., Holt, S., Carson, L. A., Hill, B. C., Arduino, M. J., Kuehnert, M. J., & Jarvis, W. R. (2001) *N Engl J Med* **344**, 1427-1433.
152. Haley, R. W., Cushion, N. B., Tenover, F. C., Bannerman, T. L., Dryer, D., Ross, J., Sanchez, P. J., & Siegel, J. D. (1995) *J Infect Dis* **171**, 614-624.
153. Jernigan, J. A., Titus, M. G., Groschel, D. H., Getchell-White, S., & Farr, B. M. (1996) *Am J Epidemiol* **143**, 496-504.
154. Falk, P. S., Winnike, J., Woodmansee, C., Desai, M., & Mayhall, C. G. (2000) *Infect Control Hosp Epidemiol* **21**, 575-582.
155. Sherer, C. R., Sprague, B. M., Campos, J. M., Nambiar, S., Temple, R., Short, B., & Singh, N. (2005) *Emerg Infect Dis* **11**, 1470-1472.
156. Nourse, C., Byrne, C., Murphy, H., Kaufmann, M. E., Clarke, A., & Butler, K. (2000) *Epidemiol Infect* **124**, 53-59.
157. Rubin, L. G., Tucci, V., Cercenado, E., Eliopoulos, G., & Isenberg, H. D. (1992) *Infect Control Hosp Epidemiol* **13**, 700-705.
158. Karanfil, L. V., Murphy, M., Josephson, A., Gaynes, R., Mandel, L., Hill, B. C., & Swenson, J. M. (1992) *Infect Control Hosp Epidemiol* **13**, 195-200.
159. Hanna, H., Umphrey, J., Tarrand, J., Mendoza, M., & Raad, I. (2001) *Infect Control Hosp Epidemiol* **22**, 217-219.
160. Dembry, L. M., Uzokwe, K., & Zervos, M. J. (1996) *Infect Control Hosp Epidemiol* **17**, 286-292.
161. Bartley, P. B., Schooneveldt, J. M., Looke, D. F., Morton, A., Johnson, D. W., & Nimmo, G. R. (2001) *J Hosp Infect* **48**, 43-54.
162. Christiansen, K. J., Tibbett, P. A., Beresford, W., Pearman, J. W., Lee, R. C., Coombs, G. W., Kay, I. D., O'Brien, F. G., Palladino, S., Douglas, C. R., *et al.* (2004) *Infect Control Hosp Epidemiol* **25**, 384-390.
163. Armstrong-Evans, M., Litt, M., McArthur, M. A., Willey, B., Cann, D., Liska, S., Nusinowitz, S., Gould, R., Blacklock, A., Low, D. E., *et al.* (1999) *Infect Control Hosp Epidemiol* **20**, 312-317.
164. Webster, J., Faoagali, J. L., & Cartwright, D. (1994) *J Paediatr Child Health* **30**, 59-64.
165. Zafar, A. B., Butler, R. C., Reese, D. J., Gaydos, L. A., & Mennonna, P. A. (1995) *Am J Infect Control* **23**, 200-208.
166. Carrier, M., Marchand, R., Auger, P., Hebert, Y., Pellerin, M., Perrault, L. P., Cartier, R., Bouchard, D., Poirier, N., & Page, P. (2002) *J Thorac Cardiovasc Surg* **123**, 40-44.
167. Kotilainen, P., Routamaa, M., Peltonen, R., Evesti, P., Eerola, E., Salmenlinna, S., Vuopio-Varkila, J., & Rossi, T. (2001) *Arch Intern Med* **161**, 859-863.
168. Back, N. A., Linnemann, C. C., Jr., Staneck, J. L., & Kotagal, U. R. (1996) *Infect Control Hosp Epidemiol* **17**, 227-231.
169. Embil, J. M., McLeod, J. A., Al-Barrak, A. M., Thompson, G. M., Aoki, F. Y., Witwicki, E. J., Stranc, M. F., Kabani, A. M., Nicoll, D. R., & Nicolle, L. E. (2001) *Burns* **27**, 681-688.
170. Rao, N., Jacobs, S., & Joyce, L. (1988) *Infect Control Hosp Epidemiol* **9**, 255-260.
171. Kotilainen, P., Routamaa, M., Peltonen, R., Oksi, J., Rintala, E., Meurman, O., Lehtonen, O. P., Eerola, E., Salmenlinna, S., Vuopio-Varkila, J., *et al.* (2003) *Emerg Infect Dis* **9**, 169-175.
172. Cohen, S. H., Morita, M. M., & Bradford, M. (1991) *Am J Med* **91**, 233S-237S.

173. Adeyemi-Doro, F. A., Scheel, O., Lyon, D. J., & Cheng, A. F. (1997) *Infect Control Hosp Epidemiol* **18**, 765-767.
174. van der Zwet, W. C., Parlevliet, G. A., Savelkoul, P. H., Stoof, J., Kaiser, A. M., Koeleman, J. G., & Vandenbroucke-Grauls, C. M. (1999) *J Hosp Infect* **42**, 295-302.
175. Macrae, M. B., Shannon, K. P., Rayner, D. M., Kaiser, A. M., Hoffman, P. N., & French, G. L. (2001) *J Hosp Infect* **49**, 183-192.
176. Villari, P., Crispino, M., Salvadori, A., & Scarcella, A. (2001) *Infect Control Hosp Epidemiol* **22**, 630-634.
177. Paterson, D. L., Singh, N., Rihs, J. D., Squier, C., Rihs, B. L., & Muder, R. R. (2001) *Clin Infect Dis* **33**, 126-128.
178. Bukholm, G., Tannaes, T., Kjelsberg, A. B., & Smith-Erichsen, N. (2002) *Infect Control Hosp Epidemiol* **23**, 441-446.
179. Roberts, S. A., Findlay, R., & Lang, S. D. (2001) *J Hosp Infect* **48**, 228-232.
180. Hollander, R., Ebke, M., Barck, H., & von Pritzbuier, E. (2001) *J Hosp Infect* **48**, 207-213.
181. Podnos, Y. D., Cinat, M. E., Wilson, S. E., Cooke, J., Gornick, W., & Thrupp, L. D. (2001) *Surgical Infections* **2**, 297-301.
182. Pittet, D., Hugonnet, S., Harbarth, S., Mourouga, P., Sauvan, V., Touveneau, S., & Perneger, T. V. (2000) *Lancet* **356**, 1307-1312.
183. Murray-Leisure, K. A., Geib, S., Graceley, D., Rubin-Slutsky, A. B., Saxena, N., Muller, H. A., & Hamory, B. H. (1990) *Infect Control Hosp Epidemiol* **11**, 343-350.
184. Jochimsen, E. M., Fish, L., Manning, K., Young, S., Singer, D. A., Baker, R., & Jarvis, W. R. (1999) *Infect Control Hosp Epidemiol* **20**, 106-109.
185. Calfee, D. P. & Farr, B. M. (2002) *Infect Control Hosp Epidemiol* **23**, 407-410.
186. Scheckler, W. E., Brimhall, D., Buck, A. S., Farr, B. M., Friedman, C., Garibaldi, R. A., Gross, P. A., Harris, J. A., Hierholzer, W. J., Jr., Martone, W. J., *et al.* (1998) *Infect Control Hosp Epidemiol* **19**, 114-124.
187. Boyce, J. M., Mermel, L. A., Zervos, M. J., Rice, L. B., Potter-Bynoe, G., Giorgio, C., & Medeiros, A. A. (1995) *Infect Control Hosp Epidemiol* **16**, 634-637.
188. Nicolle, L. E., Dyck, B., Thompson, G., Roman, S., Kabani, A., Plourde, P., Fast, M., & Embil, J. (1999) *Infect Control Hosp Epidemiol* **20**, 202-205.
189. Lucet, J. C., Decre, D., Fichelle, A., Joly-Guillou, M. L., Pernet, M., Deblangy, C., Kosmann, M. J., & Regnier, B. (1999) *Clin Infect Dis* **29**, 1411-1418.
190. D'Agata, E. M., Thayer, V., & Schaffner, W. (2000) *Infect Control Hosp Epidemiol* **21**, 588-591.
191. Papia, G., Louie, M., Tralla, A., Johnson, C., Collins, V., & Simor, A. E. (1999) *Infect Control Hosp Epidemiol* **20**, 473-477.
192. Siddiqui, A. H., Harris, A. D., Hebden, J., Wilson, P. D., Morris, J. G., Jr., & Roghmann, M. C. (2002) *Am J Infect Control* **30**, 40-43.
193. Byers, K. E., Anglim, A. M., Anneski, C. J., Germanson, T. P., Gold, H. S., Durbin, L. J., Simonton, B. M., & Farr, B. M. (2001) *Infect Control Hosp Epidemiol* **22**, 140-147.
194. Harbarth, S., Martin, Y., Rohner, P., Henry, N., Auckenthaler, R., & Pittet, D. (2000) *J Hosp Infect* **46**, 43-49.
195. Curtis, J. R., Cook, D. J., Wall, R. J., Angus, D. C., Bion, J., Kacmarek, R., Kane-Gill, S. L., Kirchhoff, K. T., Levy, M., Mitchell, P. H., *et al.* (2006) *Crit Care Med* **34**, 211-218.
196. Arnow, P., Allyn, P. A., Nichols, E. M., Hill, D. L., Pezzlo, M., & Bartlett, R. H. (1982) *J Trauma* **22**, 954-959.

197. Fridkin, S. K., Pear, S. M., Williamson, T. H., Galgiani, J. N., & Jarvis, W. R. (1996) *Infect Control Hosp Epidemiol* **17**, 150-158.
198. Harbarth, S., Sudre, P., Dharan, S., Cadenas, M., & Pittet, D. (1999) *Infect Control Hosp Epidemiol* **20**, 598-603.
199. Vicca, A. F. (1999) *J Hosp Infect* **43**, 109-113.
200. Robert, J., Fridkin, S. K., Blumberg, H. M., Anderson, B., White, N., Ray, S. M., Chan, J., & Jarvis, W. R. (2000) *Infect Control Hosp Epidemiol* **21**, 12-17.(mj).
201. Jackson, M., Chiarello, L. A., Gaynes, R. P., & Gerberding, J. L. (2002) *Am J Infect Control* **30**, 199-206.
202. Grundmann, H., Hori, S., Winter, B., Tami, A., & Austin, D. J. (2002) *J Infect Dis* **185**, 481-488.
203. Dubbert, P. M., Dolce, J., Richter, W., Miller, M., & Chapman, S. W. (1990) *Infect Control Hosp Epidemiol* **11**, 191-193.
204. Nettleman, M. D., Trilla, A., Fredrickson, M., & Pfaller, M. (1991) *Am J Med* **91**, 228S-232S.
205. Curran, E. T., Benneyan, J. C., & Hood, J. (2002) *Infect Control Hosp Epidemiol* **23**, 13-18.
206. Gerber, S. I., Jones, R. C., Scott, M. V., Price, J. S., Dworkin, M. S., Filippell, M. B., Rearick, T., Pur, S. L., McAuley, J. B., Lavin, M. A., *et al.* (2006) *Infect Control Hosp Epidemiol* **27**, 139-145.
207. Chicago Antimicrobial Resistance Project.
208. Rampling, A., Wiseman, S., Davis, L., Hyett, A. P., Walbridge, A. N., Payne, G. C., & Cornaby, A. J. (2001) *J Hosp Infect* **49**, 109-116.
209. Rice, L. B., Eckstein, E. C., DeVente, J., & Shlaes, D. M. (1996) *Clin Infect Dis* **23**, 118-124.
210. Wright, M. O., Hebden, J. N., Harris, A. D., Shanholtz, C. B., Standiford, H. C., Furuno, J. P., & Perencevich, E. N. (2004) *Infect Control Hosp Epidemiol* **25**, 167-168.
211. Smith, D. L., Dushoff, J., Perencevich, E. N., Harris, A. D., & Levin, S. A. (2004) *Proc Natl Acad Sci U S A* **101**, 3709-3714.
212. Rahal, J. J., Urban, C., Horn, D., Freeman, K., Segal-Maurer, S., Maurer, J., Mariano, N., Marks, S., Burns, J. M., Dominick, D., *et al.* (1998) *JAMA* **280**, 1233-1237.
213. Rahal, J. J., Urban, C., & Segal-Maurer, S. (2002) *Clin Infect Dis* **34**, 499-503.
214. Meyer, K. S., Urban, C., Eagan, J. A., Berger, B. J., & Rahal, J. J. (1993) *Ann Intern Med* **119**, 353-358.
215. Pena, C., Pujol, M., Ardanuy, C., Ricart, A., Pallares, R., Linares, J., Ariza, J., & Gudiol, F. (1998) *Antimicrob Agents Chemother* **42**, 53-58.
216. Quale, J. M., Landman, D., Bradford, P. A., Visalli, M., Ravishankar, J., Flores, C., Mayorga, D., Vangala, K., & Adedeji, A. (2002) *Clin Infect Dis* **35**, 834-841.
217. Rupp, M. E., Marion, N., Fey, P. D., Bolam, D. L., Iwen, P. C., Overfelt, C. M., & Chapman, L. (2001) *Infect Control Hosp Epidemiol* **22**, 301-303.
218. Calil, R., Marba, S. T., von Nowakonski, A., & Tresoldi, A. T. (2001) *Am J Infect Control* **29**, 133-138.
219. McDonald, L. C. (2005) *Infect Control Hosp Epidemiol* **26**, 672-675.
220. Harbarth, S., Cosgrove, S., & Carmeli, Y. (2002) *Antimicrob Agents Chemother* **46**, 1619-1628.
221. Winston, L. G., Charlebois, E. D., Pang, S., Bangsberg, D. R., Perdreau-Remington, F., & Chambers, H. F. (2004) *Am J Infect Control* **32**, 462-469.
222. Brinsley, K., Srinivasan, A., Sinkowitz-Cochran, R., Lawton, R., McIntyre, R., Kravitz, G., Burke, B., Shadowen, R., & Cardo, D. (2005) *Am J Infect Control* **33**, 53-54.

223. Bruno-Murtha, L. A., Bruschi, J., Bor, D., Li, W., & Zucker, D. (2005) *Infect Control Hosp Epidemiol* **26**, 81-87.
224. Fridkin, S. K. (2003) *Clin Infect Dis* **36**, 1438-1444.
225. John, J. F., Jr. (2000) *Infect Control Hosp Epidemiol* **21**, 9-11.
226. McGowan, J. E., Jr. (2000) *Infect Control Hosp Epidemiol* **21**, S36-43.
227. Evans, R. S., Pestotnik, S. L., Classen, D. C., Clemmer, T. P., Weaver, L. K., Orme, J. F., Jr., Lloyd, J. F., & Burke, J. P. (1998) *N Engl J Med* **338**, 232-238.
228. Huskins, W. C. (2001) *Semin Pediatr Infect Dis* **12**, 138-146.
229. Mullett, C. J., Evans, R. S., Christenson, J. C., & Dean, J. M. (2001) *Pediatrics* **108**, E75.
230. Glowacki, R. C., Schwartz, D. N., Itokazu, G. S., Wisniewski, M. F., Kieszkowski, P., & Weinstein, R. A. (2003) *Clin Infect Dis* **37**, 59-64.
231. Parrino, T. A. (2005) *Pharmacotherapy* **25**, 289-298.
232. Paterson, D. L. (2006) *Clin Infect Dis* **42 Suppl 2**, S90-95.
233. Binkley, S., Fishman, N. O., LaRosa, L. A., Marr, A. M., Nachamkin, I., Wordell, D., Bilker, W. B., & Lautenbach, E. (2006) *Infect Control Hosp Epidemiol* **27**, 682-687.
234. McGowan, J. E., Jr. & Tenover, F. C. (2004) *Nat Rev Microbiol* **2**, 251-258.
235. Fridkin, S. K., Edwards, J. R., Tenover, F. C., Gaynes, R. P., & McGowan, J. E., Jr. (2001) *Clin Infect Dis* **33**, 324-330.
236. Foca, M., Jakob, K., Whittier, S., Della Latta, P., Factor, S., Rubenstein, D., & Saiman, L. (2000) *N Engl J Med* **343**, 695-700.
237. Huang (In press) *J Infect Dis*.
238. Gaynes, R. P. & Emori, T. G. (2001) in *Saunders Infection Control Reference Service*, eds. Abrutyn, E., Goldmann, D. A., & Scheckler, W. E. (W.B. Saunders Company, Philadelphia, PA), pp. 40-44.
239. Pottinger, J. M., Herwaldt, L. A., & Perl, T. M. (1997) *Infect Control Hosp Epidemiol* **18**, 513-527.
240. Hartstein, A. I., LeMonte, A. M., & Iwamoto, P. K. (1997) *Infect Control Hosp Epidemiol* **18**, 42-48.
241. Piagnerelli, M., Kennes, B., Brogniez, Y., Deplano, A., & Govaerts, D. (2000) *Infect Control Hosp Epidemiol* **21**, 651-653.
242. Montecalvo, M. A., Jarvis, W. R., Uman, J., Shay, D. K., Petrullo, C., Rodney, K., Gedris, C., Horowitz, H. W., & Wormser, G. P. (1999) *Ann Intern Med* **131**, 269-272.
243. Talon, D. R. & Bertrand, X. (2001) *Infect Control Hosp Epidemiol* **22**, 505-509.
244. Lucet, J. C., Grenet, K., Armand-Lefevre, L., Harnal, M., Bouvet, E., Regnier, B., & al., e. (2005) *Infect Control Hosp Epidemiol* **26**.
245. Troche, G., Joly, L. M., Guibert, M., & Zazzo, J. F. (2005) *Infect Control Hosp Epidemiol* **26**, 161-165.
246. Nijssen, S., Bonten, M. J., & Weinstein, R. A. (2005) *Clin Infect Dis* **40**, 405-409.
247. Cooper, B. S., Stone, S. P., Kibbler, C. C., Cookson, B. D., Roberts, J. A., Medley, G. F., Duckworth, G., Lai, R., & Ebrahim, S. (2004) *Bmj* **329**, 533.
248. Perencevich, E. N., Fisman, D. N., Lipsitch, M., Harris, A. D., Morris, J. G., Jr., & Smith, D. L. (2004) *Clin Infect Dis* **38**, 1108-1115.
249. Bootsma, M. C., Diekmann, O., & Bonten, M. J. (2006) *Proc Natl Acad Sci U S A* **103**, 5620-5625.
250. Gardam, M. A., Burrows, L. L., Kus, J. V., Brunton, J., Low, D. E., Conly, J. M., & Humar, A. (2002) *J Infect Dis* **186**, 1754-1760.

251. Thouverez, M., Talon, D., & Bertrand, X. (2004) *Infect Control Hosp Epidemiol* **25**, 838-841.
252. Armeanu, E. & Bonten, M. J. (2005) *Clin Infect Dis* **41**, 210-216.
253. Muto, C. A., Giannetta, E. T., Durbin, L. J., Simonton, B. M., & Farr, B. M. (2002) *Infect Control Hosp Epidemiol* **23**, 429-435.
254. Morris, J. G., Jr., Shay, D. K., Hebden, J. N., McCarter, R. J., Jr., Perdue, B. E., Jarvis, W., Johnson, J. A., Dowling, T. C., Polish, L. B., & Schwalbe, R. S. (1995) *Ann Intern Med* **123**, 250-259.
255. Furuno, J. P., McGregor, J. C., Harris, A. D., Johnson, J. A., Johnson, J. K., Langenberg, P., Venezia, R. A., Finkelstein, J., Smith, D. L., Strauss, S. M., et al. (2006) *Arch Intern Med* **166**, 580-585.
256. Harbarth, S., Sax, H., Fankhauser-Rodriguez, C., Schrenzel, J., Agostinho, A., & Pittet, D. (2006) *Am J Med* **119**, 275 e215-223.
257. Lee, T. A., Hacek, D. M., Stroupe, K. T., Collins, S. M., & Peterson, L. R. (2005) *Infect Control Hosp Epidemiol* **26**, 39-46.
258. Manian, F. A., Senkel, D., Zack, J., & Meyer, L. (2002) *Infect Control Hosp Epidemiol* **23**, 516-519.
259. Troillet, N., Carmeli, Y., Samore, M. H., Dakos, J., Eichelberger, K., DeGirolami, P. C., & Karchmer, A. W. (1998) *Infect Control Hosp Epidemiol* **19**, 181-185.
260. Sanford, M. D., Widmer, A. F., Bale, M. J., Jones, R. N., & Wenzel, R. P. (1994) *Clin Infect Dis* **19**, 1123-1128.
261. Lucet, J. C., Chevret, S., Durand-Zaleski, I., Chastang, C., & Regnier, B. (2003) *Arch Intern Med* **163**, 181-188.
262. D'Agata, E. M., et al. (2002) *Clin Infect Dis* **34**, 167-172.
263. Flayhart, D., Hindler, J. F., Bruckner, D. A., Hall, G., Shrestha, R. K., Vogel, S. A., Richter, S. S., Howard, W., Walther, R., & Carroll, K. C. (2005) *J Clin Microbiol* **43**, 5536-5540.
264. Perry, J. D., Davies, A., Butterworth, L. A., Hopley, A. L., Nicholson, A., & Gould, F. K. (2004) *J Clin Microbiol* **42**, 4519-4523.
265. Harbarth, S., Masuet-Aumatell, C., Schrenzel, J., Francois, P., Akakpo, C., Renzi, G., Pugin, J., Ricou, B., & Pittet, D. (2006) *Crit Care* **10**, R25.
266. Huletsky, A., Lebel, P., Picard, F. J., Bernier, M., Gagnon, M., Boucher, N., & Bergeron, M. G. (2005) *Clin Infect Dis* **40**, 976-981.
267. Warren, D. K., Liao, R. S., Merz, L. R., Eveland, M., & Dunne, W. M., Jr. (2004) *J Clin Microbiol* **42**, 5578-5581.
268. Palladino, S., Kay, I. D., Flexman, J. P., Boehm, I., Costa, A. M., Lambert, E. J., & Christiansen, K. J. (2003) *J Clin Microbiol* **41**, 2483-2486.
269. Fazal, B. A., Telzak, E. E., Blum, S., Turett, G. S., Petersen-Fitzpatrick, F. E., & Lorian, V. (1996) *Infect Control Hosp Epidemiol* **17**, 372-374.
270. Toltzis, P., Hoyen, C., & et al. (1999) *Pediatrics* **103** (4 Pt1), 719-723.
271. Weinstein, R. A. & Kabins, S. A. (1981) *Am J Med* **70**, 449-454.
272. Kim, P. W., Roghmann, M. C., Perencevich, E. N., & Harris, A. D. (2003) *Am J Infect Control* **31**, 97-103.
273. Slaughter, S., Hayden, M. K., Nathan, C., Hu, T. C., Rice, T., Van Voorhis, J., Matushek, M., Franklin, C., & Weinstein, R. A. (1996) *Ann Intern Med* **125**, 448-456.
274. CDC (1995) *MMWR Recomm Rep* **44** (RR-12), 1-13.
275. Evans, M. R., Meldrum, R., Lane, W., Gardner, D., Ribeiro, C. D., Gallimore, C. I., & Westmoreland, D. (2002) *Epidemiol Infect* **129**, 355-360.

276. Hall, C. B., Douglas, R. G., Jr., Schnabel, K. C., & Geiman, J. M. (1981) *Infect Immun* **33**, 779-783.
277. Wu, H. M., Fornek, M., Kellogg, J. S., Chapin, A. R., Gibson, K., Schwab, E., Spencer, C., & Henning, K. (2005) *Infect Control Hosp Epidemiol* **26**, 802-810.
278. Austin, D. J., Bonten, M. J., Weinstein, R. A., Slaughter, S., & Anderson, R. M. (1999) *Proc Natl Acad Sci U S A* **96**, 6908-6913.
279. Law, M. R., Gill, O. N., & Turner, A. (1988) *Epidemiol Infect* **101**, 301-309.
280. Ruchel, R., Mergeryan, H., Boger, O., Langefeld, C., & Witte, W. (1999) *Infect Control Hosp Epidemiol* **20**, 353-355.
281. Cepeda, J. A., Whitehouse, T., Cooper, B., Hails, J., Jones, K., Kwaku, F., Taylor, L., Hayman, S., Cookson, B., Shaw, S., *et al.* (2005) *Lancet* **365**, 295-304.
282. Mulin, B., Rouget, C., Clement, C., Bailly, P., Julliot, M. C., Viel, J. F., Thouverez, M., Vieille, I., Barale, F., & Talon, D. (1997) *Infect Control Hosp Epidemiol* **18**, 499-503.
283. Nouwen, J. L., Ott, A., Kluytmans-Vandenbergh, M. F., Boelens, H. A., Hofman, A., van Belkum, A., & Verbrugh, H. A. (2004) *Clin Infect Dis* **39**, 806-811.
284. Byers, K. E., Anglim, A. M., Anneski, C. J., & Farr, B. M. (2002) *Infect Control Hosp Epidemiol* **23**, 207-211.
285. Baden, L. R., Thiemke, W., Skolnik, A., Chambers, R., Strymish, J., Gold, H. S., Moellering, R. C., Jr., & Eliopoulos, G. M. (2001) *Clin Infect Dis* **33**, 1654-1660.
286. Donskey, C. J., Huyen, C. K., Das, S. M., Helfand, M. S., & Hecker, M. T. (2002) *Infect Control Hosp Epidemiol* **23**, 436-440.
287. Ridenour, G. A., Wong, E. S., Call, M. A., & Climo, M. W. (2006) *Infect Control Hosp Epidemiol* **27**, 271-278.
288. Scanvic, A., Denic, L., Gaillon, S., Giry, P., Andremont, A., & Lucet, J. C. (2001) *Clin Infect Dis* **32**, 1393-1398.
289. Kauffman, C. A., Terpenning, M. S., He, X., Zarins, L. T., Ramsey, M. A., Jorgensen, K. A., Sottile, W. S., & Bradley, S. F. (1993) *Am J Med* **94**, 371-378.
290. Strausbaugh, L. J., Jacobson, C., Sewell, D. L., Potter, S., & Ward, T. T. (1992) *Infect Control Hosp Epidemiol* **13**, 151-159.
291. Kirkland, K. B. & Weinstein, J. M. (1999) *Lancet* **354**, 1177-1178.
292. Saint, S., Higgins, L. A., Nallamotheu, B. K., & Chenoweth, C. (2003) *Am J Infect Control* **31**, 354-356.
293. Evans, H. L., Shaffer, M. M., Hughes, M. G., Smith, R. L., Chong, T. W., Raymond, D. P., Pelletier, S. J., Pruett, T. L., & Sawyer, R. G. (2003) *Surgery* **134**, 180-188.
294. Catalano, G., Houston, S. H., Catalano, M. C., Butera, A. S., Jennings, S. M., Hakala, S. M., Burrows, S. L., Hickey, M. G., Duss, C. V., Skelton, D. N., *et al.* (2003) *South Med J* **96**, 141-145.
295. Tarzi, S., Kennedy, P., Stone, S., & Evans, M. (2001) *J Hosp Infect* **49**, 250-254.
296. Stelfox, H. T., Bates, D. W., & Redelmeier, D. A. (2003) *JAMA* **290**, 1899-1905.
297. Hota, B. (2004) *Clin Infect Dis* **39**, 1182-1189.
298. Martinez, J. A., Ruthazer, R., Hansjosten, K., Barefoot, L., & Snyderman, D. R. (2003) *Arch Intern Med* **163**, 1905-1912.
299. CDC (2003) *MMWR* **52(RR10);1-42**.
300. Simor, A. E. (2001) *Infect Control Hosp Epidemiol* **22**, 459-463.
301. Hayden, M. K., Bonten, M. J., Blom, D. W., Lyle, E. A., van de Vijver, D. A., & Weinstein, R. A. (2006) *Clin Infect Dis* **42**, 1552-1560.

302. Lai, K. K., Kelley, A. L., Melvin, Z. S., Belliveau, P. P., & Fontecchio, S. A. (1998) *Infect Control Hosp Epidemiol* **19**, 647-652.
303. Boyce, J. M. (2001) *J Hosp Infect* **48 Suppl A**, S9-14.
304. Montesinos, I., Salido, E., Delgado, T., Lecuona, M., & Sierra, A. (2003) *Infect Control Hosp Epidemiol* **24**, 667-672.
305. Chen, S. F. (2005) *Pediatr Infect Dis J* **24**, 79-80.
306. Kaplan, S. L. (2005) *Pediatr Infect Dis J* **24**, 457-458.
307. Loeb, M., Main, C., Walker-Dilks, C., & Eady, A. (2003) *Cochrane Database Syst Rev*, CD003340.
308. Deshpande, L. M., Fix, A. M., Pfaller, M. A., & Jones, R. N. (2002) *Diagn Microbiol Infect Dis* **42**, 283-290.
309. Mody, L., Kauffman, C. A., McNeil, S. A., Galecki, A. T., & Bradley, S. F. (2003) *Clin Infect Dis* **37**, 1467-1474.
310. Walker, E. S., Vasquez, J. E., Dula, R., Bullock, H., & Sarubbi, F. A. (2003) *Infect Control Hosp Epidemiol* **24**, 342-346.
311. Harris, A. D., Bradham, D. D., Baumgarten, M., Zuckerman, I. H., Fink, J. C., & Perencevich, E. N. (2004) *Clin Infect Dis* **38**, 1586-1591.
312. Eveillard, M., Eb, F., Tramier, B., Schmit, J. L., Lescure, F. X., Biendo, M., Canarelli, B., Daoudi, F., Laurans, G., Rousseau, F., *et al.* (2001) *J Hosp Infect* **47**, 116-124.
313. Campbell, J. R., Zaccaria, E., Mason, E. O., Jr., & Baker, C. J. (1998) *Infect Control Hosp Epidemiol* **19**, 924-928.
314. Harris, A. D., Nemoy, L., Johnson, J. A., Martin-Carnahan, A., Smith, D. L., Standiford, H., & Perencevich, E. N. (2004) *Infect Control Hosp Epidemiol* **25**, 105-108.
315. Warren, D. K., Nitin, A., Hill, C., Fraser, V. J., & Kollef, M. H. (2004) *Infect Control Hosp Epidemiol* **25**, 99-104.
316. Trick, W. E., Weinstein, R. A., DeMarais, P. L., Kuehnert, M. J., Tomaska, W., Nathan, C., Rice, T. W., McAllister, S. K., Carson, L. A., & Jarvis, W. R. (2001) *J Am Geriatr Soc* **49**, 270-276.
317. Safdar, N. & Maki, D. G. (2002) *Ann Intern Med* **136**, 834-844.
318. Montecalvo, M. A., Jarvis, W. R., Uman, J., Shay, D. K., Petrullo, C., Horowitz, H. W., & Wormser, G. P. (2001) *Infect Control Hosp Epidemiol* **22**, 437-442.
319. Rubinovitch, B. & Pittet, D. (2001) *J Hosp Infect* **47**, 9-18.
320. Puzniak, L. A., Gillespie, K. N., Leet, T., Kollef, M., & Mundy, L. M. (2004) *Infect Control Hosp Epidemiol* **25**, 418-424.
321. Cookson, B. (1997) *Bmj* **314**, 664-665.
322. Farr, B. M. & Jarvis, W. R. (2002) *Infect Control Hosp Epidemiol* **23**, 65-68.
323. Strausbaugh, L. J., Siegel, J. D., & Weinstein, R. A. (2006) *Clin Infect Dis* **42**, 828-835.
324. Brooks, S., Khan, A., Stoica, D., Griffith, J., Friedeman, L., Mukherji, R., Hameed, R., & Schupf, N. (1998) *Infect Control Hosp Epidemiol* **19**, 333-336.
325. Benneyan, J. C., Lloyd, R. C., & Plsek, P. E. (2003) *Qual Saf Health Care* **12**, 458-464.
326. Gustafson, T. L. (2000) *Am J Infect Control* **28**, 406-414.
327. Aubry-Damon, H., Legrand, P., Brun-Buisson, C., Astier, A., Soussy, C. J., & Leclercq, R. (1997) *Clin Infect Dis* **25**, 647-653.
328. Cooper, B. S., Medley, G. F., Stone, S. P., Kibbler, C. C., Cookson, B. D., Roberts, J. A., Duckworth, G., Lai, R., & Ebrahim, S. (2004) *Proc Natl Acad Sci U S A* **101**, 10223-10228.

329. Brown, A. R., Amyes, S. G., Paton, R., Plant, W. D., Stevenson, G. M., Winney, R. J., & Miles, R. S. (1998) *J Hosp Infect* **40**, 115-124.
330. Cromer, A. L., Hutsell, S. O., Latham, S. C., Bryant, K. G., Wacker, B. B., Smith, S. A., Bendyk, H. A., Valainis, G. T., & Carney, M. C. (2004) *Am J Infect Control* **32**, 451-455.
331. Pittsburgh Regional Project.
332. Assadian, O., Berger, A., Aspöck, C., Mustafa, S., Kohlhauser, C., & Hirschl, A. M. (2002) *Infect Control Hosp Epidemiol* **23**, 457-461.
333. Byers, K. E., Durbin, L. J., Simonton, B. M., Anglim, A. M., Adal, K. A., & Farr, B. M. (1998) *Infect Control Hosp Epidemiol* **19**, 261-264.
334. Patterson, J. E., Hardin, T. C., Kelly, C. A., Garcia, R. C., & Jorgensen, J. H. (2000) *Infect Control Hosp Epidemiol* **21**, 455-458.
335. Bantar, C., Sartori, B., Vesco, E., Heft, C., Saul, M., Salamone, F., & Oliva, M. E. (2003) *Clin Infect Dis* **37**, 180-186.
336. Bisson, G., Fishman, N. O., Patel, J. B., Edelstein, P. H., & Lautenbach, E. (2002) *Infect Control Hosp Epidemiol* **23**, 254-260.
337. Carling, P., Fung, T., Killion, A., Terrin, N., & Barza, M. (2003) *Infect Control Hosp Epidemiol* **24**, 699-706.
338. Quale, J., Landman, D., Saurina, G., Atwood, E., DiTore, V., & Patel, K. (1996) *Clin Infect Dis* **23**, 1020-1025.
339. Sample, M. L., Gravel, D., Oxley, C., Toye, B., Garber, G., & Ramotar, K. (2002) *Infect Control Hosp Epidemiol* **23**, 468-470.
340. Burke, J. P. & Pestotnik, S. L. (1999) *J Chemother* **11**, 530-535.
341. Cooper, E., Paull, A., & O'Reilly, M. (2002) *Infect Control Hosp Epidemiol* **23**, 151-153.
342. Lagerlov, P., Loeb, M., Andrew, M., & Hjortdahl, P. (2000) *Qual Health Care* **9**, 159-165.
343. Lemmen, S. W., Zolldann, D., Gastmeier, P., & Lutticken, R. (2001) *Am J Infect Control* **29**, 89-93.
344. Liu, S. C., Leu, H. S., Yen, M. Y., Lee, P. I., & Chou, M. C. (2002) *Am J Infect Control* **30**, 381-385.
345. Monnet, D. L. (1998) *Infect Control Hosp Epidemiol* **19**, 552-559.
346. Pestotnik, S. L., Classen, D. C., Evans, R. S., & Burke, J. P. (1996) *Ann Intern Med* **124**, 884-890.
347. NCCLS (2002).
348. Kupronis, B. A., Richards, C. L., & Whitney, C. G. (2003) *J Am Geriatr Soc* **51**, 1520-1525.
349. Viray, M., Linkin, D., Maslow, J. N., Stieritz, D. D., Carson, L. S., Bilker, W. B., & Lautenbach, E. (2005) *Infect Control Hosp Epidemiol* **26**, 56-62.
350. Chaitram, J. M., Jevitt, L. A., Lary, S., & Tenover, F. C. (2003) *J Clin Microbiol* **41**, 2372-2377.
351. Ernst, E. J., Diekema, D. J., BootsMiller, B. J., Vaughn, T., Yankey, J. W., Flach, S. D., Ward, M. M., Franciscus, C. L., Acosta, E., Pfaller, M. A., *et al.* (2004) *Diagn Microbiol Infect Dis* **49**, 141-145.
352. Ginocchio, C. C. (2002) *Am J Health Syst Pharm* **59**, S7-11.
353. Stevenson, K. B., Samore, M., Barbera, J., Moore, J. W., Hannah, E., Houck, P., Tenover, F. C., & Gerberding, J. L. (2003) *Diagn Microbiol Infect Dis* **47**, 303-311.
354. Gupta, A., Della-Latta, P., Todd, B., San Gabriel, P., Haas, J., Wu, F., Rubenstein, D., & Saiman, L. (2004) *Infect Control Hosp Epidemiol* **25**, 210-215.

355. Rodriguez-Bano, J., Navarro, M. D., Romero, L., Muniain, M. A., Perea, E. J., Perez-Cano, R., Hernandez, J. R., & Pascual, A. (2006) *Clin Infect Dis* **42**, 37-45.
356. Bhavnani, S. M., Hammel, J. P., Forrest, A., Jones, R. N., & Ambrose, P. G. (2003) *Clin Infect Dis* **37**, 344-350.
357. Halstead, D. C., Gomez, N., & McCarter, Y. S. (2004) *J Clin Microbiol* **42**, 1-6.
358. Fridkin, S. K., Steward, C. D., Edwards, J. R., Pryor, E. R., McGowan, J. E., Jr., Archibald, L. K., Gaynes, R. P., & Tenover, F. C. (1999) *Clin Infect Dis* **29**, 245-252.
359. Lang, A., De Fina, G., Meyer, R., Aschbacher, R., Rizza, F., Mayr, O., & Casini, M. (2001) *Eur J Clin Microbiol Infect Dis* **20**, 657-660.
360. White, R. L., Friedrich, L. V., Mihm, L. B., & Bosso, J. A. (2000) *Clin Infect Dis* **31**, 16-23.
361. Zoutman, D. E. & Ford, B. D. (2005) *Am J Infect Control* **33**, 1-5.
362. cms.
363. Peterson, L. R., Hamilton, J. D., Baron, E. J., Tompkins, L. S., Miller, J. M., Wilfert, C. M., Tenover, F. C., & Thomson Jr, R. B., Jr. (2001) *Clin Infect Dis* **32**, 605-611.
364. Calfee, D. P., Giannetta, E. T., Durbin, L. J., Germanson, T. P., & Farr, B. M. (2003) *Clin Infect Dis* **37**, 326-332.
365. Thompson, R. L., Cabezudo, I., & Wenzel, R. P. (1982) *Ann Intern Med* **97**, 309-317.
366. Lacey, S., Flaxman, D., Scales, J., & Wilson, A. (2001) *J Hosp Infect* **48**, 308-311.
367. Greenaway, C. A. & Miller, M. A. (1999) *Infect Control Hosp Epidemiol* **20**, 341-343.
368. Spindel, S. J., Strausbaugh, L. J., & Jacobson, C. (1995) *Infect Control Hosp Epidemiol* **16**, 217-223.
369. Bula, C. J., Ghilardi, G., Wietlisbach, V., Petignat, C., & Francioli, P. (2004) *J Am Geriatr Soc* **52**, 700-706.
370. High, K. P., Bradley, S., Loeb, M., Palmer, R., Quagliarello, V., & Yoshikawa, T. (2005) *Clin Infect Dis* **40**, 114-122.
371. Silverblatt, F. J., Tibert, C., Mikolich, D., Blazek-D'Arezzo, J., Alves, J., Tack, M., & Agatiello, P. (2000) *J Am Geriatr Soc* **48**, 1211-1215.
372. CDC (2001) *MMWR* **50(RR05)**, 1-43.
373. Samore, M. H., Venkataraman, L., DeGirolami, P. C., Arbeit, R. D., & Karchmer, A. W. (1996) *Am J Med* **100**, 32-40.
374. Brooks, S. E., Veal, R. O., Kramer, M., Dore, L., Schupf, N., & Adachi, M. (1992) *Infect Control Hosp Epidemiol* **13**, 98-103.
375. Jernigan, J. A., Siegman-Igra, Y., Guerrant, R. C., & Farr, B. M. (1998) *Infect Control Hosp Epidemiol* **19**, 494-499.
376. Chang, V. T. & Nelson, K. (2000) *Clin Infect Dis* **31**, 717-722.
377. Nicolle, L. E. (2000) *Clin Infect Dis* **31**, 752-756.
378. Bonten, M. J., Slaughter, S., Hayden, M. K., Nathan, C., van Voorhis, J., & Weinstein, R. A. (1998) *Crit Care Med* **26**, 2001-2004.
379. Loeb, M. B., Craven, S., McGeer, A. J., Simor, A. E., Bradley, S. F., Low, D. E., Armstrong-Evans, M., Moss, L. A., & Walter, S. D. (2003) *Am J Epidemiol* **157**, 40-47.
380. McDonald, L. C., Banerjee, S. N., & Jarvis, W. R. (1998) *Infect Control Hosp Epidemiol* **19**, 772-777.
381. Montecalvo, M. A., de Lencastre, H., Carraher, M., Gedris, C., Chung, M., VanHorn, K., & Wormser, G. P. (1995) *Infect Control Hosp Epidemiol* **16**, 680-685.
382. Shannon, K. P. & French, G. L. (2002) *J Antimicrob Chemother* **50**, 965-969.

383. Singh, K., Gavin, P. J., Vescio, T., Thomson Jr, R. B., Jr., Deddish, R. B., Fisher, A., Noskin, G. A., & Peterson, L. R. (2003) *J Clin Microbiol* **41**, 2755-2757.
384. Grmek-Kosnik, I., Ihan, A., Dermota, U., Rems, M., Kosnik, M., & Jorn Kolmos, H. (2005) *J Hosp Infect* **61**, 155-161.
385. Villegas, M. V. & Hartstein, A. I. (2003) *Infect Control Hosp Epidemiol* **24**, 284-295.
386. Ramsey, A. H., Skonieczny, P., Coolidge, D. T., Kurzynski, T. A., Proctor, M. E., & Davis, J. P. (2001) *Infect Control Hosp Epidemiol* **22**, 423-426.
387. Harbarth, S., Liassine, N., Dharan, S., Herrault, P., Auckenthaler, R., & Pittet, D. (2000) *Clin Infect Dis* **31**, 1380-1385.
388. Lucet, J. C., Chevret, S., Decre, D., Vanjak, D., Macrez, A., Bedos, J. P., Wolff, M., & Regnier, B. (1996) *Clin Infect Dis* **22**, 430-436.
389. Malik, R. K., Montecalvo, M. A., Reale, M. R., Li, K., Maw, M., Munoz, J. L., Gedris, C., van Horn, K., Carnevale, K. A., Levi, M. H., *et al.* (1999) *Pediatr Infect Dis J* **18**, 352-356.
390. Stosor, V., Kruszynski, J., Suriano, T., Noskin, G. A., & Peterson, L. R. (1999) *Infect Control Hosp Epidemiol* **20**, 653-659.
391. Srinivasan, A., Song, X., Ross, T., Merz, W., Brower, R., & Perl, T. M. (2002) *Infect Control Hosp Epidemiol* **23**, 424-428.
392. Rumbak, M. J. & Cancio, M. R. (1995) *Crit Care Med* **23**, 1200-1203.
393. Quale, J., Landman, D., Atwood, E., Kreiswirth, B., Willey, B. M., Ditore, V., Zaman, M., Patel, K., Saurina, G., Huang, W., *et al.* (1996) *Am J Infect Control* **24**, 372-379.
394. Livornese, L. L., Jr., Dias, S., Samel, C., Romanowski, B., Taylor, S., May, P., Pitsakis, P., Woods, G., Kaye, D., Levison, M. E., *et al.* (1992) *Ann Intern Med* **117**, 112-116.
395. Gastmeier, P., Schwab, F., Geffers, C., & Ruden, H. (2004) *Infect Control Hosp Epidemiol* **25**, 109-113.
396. Ridwan, B., Mascini, E., van Der Reijden, N., Verhoef, J., & Bonten, M. (2002) *Bmj* **324**, 666-668.
397. Hitomi, S., Kubota, M., Mori, N., Baba, S., Yano, H., Okuzumi, K., & Kimura, S. (2000) *J Hosp Infect* **46**, 123-129.
398. Weber, D. J. & Rutala, W. A. (1997) *Infect Control Hosp Epidemiol* **18**, 306-309.
399. Schelenz, S. & French, G. (2000) *J Hosp Infect* **46**, 23-30.
400. Kirschke, D. L., Jones, T. F., Craig, A. S., Chu, P. S., Mayernick, G. G., Patel, J. A., & Schaffner, W. (2003) *N Engl J Med* **348**, 214-220.
401. Srinivasan, A., Wolfenden, L. L., Song, X., Mackie, K., Hartsell, T. L., Jones, H. D., Diette, G. B., Orens, J. B., Yung, R. C., Ross, T. L., *et al.* (2003) *N Engl J Med* **348**, 221-227.
402. Mangram, A. & Jarvis, W. R. (1996) *Infect Control Hosp Epidemiol* **17**, 718-720.
403. Vriens, M. R., Fluit, A. C., Troelstra, A., Verhoef, J., & van der Werken, C. (2002) *Infect Control Hosp Epidemiol* **23**, 491-494.
404. Cederna, J. E., Terpenning, M. S., Ensberg, M., Bradley, S. F., & Kauffman, C. A. (1990) *Infect Control Hosp Epidemiol* **11**, 13-16.
405. Hachem, R. & Raad, I. (2002) *Infect Control Hosp Epidemiol* **23**, 43-44.
406. Lui, S. L., Luk, W. K., Cheung, C. Y., Chan, T. M., Lai, K. N., & Peiris, J. S. (2001) *Transplantation* **71**, 59-64.
407. Zafar, A. B., Sylvester, L. K., & Beidas, S. O. (2002) *Am J Infect Control* **30**, 425-429.
408. Darouiche, R., Wright, C., Hamill, R., Koza, M., Lewis, D., & Markowski, J. (1991) *Antimicrob Agents Chemother* **35**, 1612-1615.

409. Goetz, M. B., Mulligan, M. E., Kwok, R., O'Brien, H., Caballes, C., & Garcia, J. P. (1992) *Am J Med* **92**, 607-614.
410. Pan, A., Carnevale, G., Catenazzi, P., Colombini, P., Crema, L., Dolcetti, L., Ferrari, L., Mondello, P., Signorini, L., Tinelli, C., *et al.* (2005) *Infect Control Hosp Epidemiol* **26**, 127-133.
411. Silvestri, L., Milanese, M., Oblach, L., Fontana, F., Gregori, D., Guerra, R., & van Saene, H. K. (2002) *Am J Infect Control* **30**, 391-399.
412. Weber, J. M., Sheridan, R. L., Schulz, J. T., Tompkins, R. G., & Ryan, C. M. (2002) *Infect Control Hosp Epidemiol* **23**, 549-551.

Table 1. Categorization of Reports about Control of MDROs in Healthcare Settings, 1982-2005

MDRO	MDR-GNB	MRSA	VRE
No. of Studies Reviewed/category	30	35	39
Types of Healthcare Facilities from which Study or Report Arose			
No. (%) from academic facilities ^α	30 (100)	28 (80)	33 (85)
No. (%) from other hospitals	0	4 (11)	3 (8)
No. (%) from LTCFs	0	1 (3)	2 (5)
No. (%) from multiple facilities in a region	0	2 (6)	1 (2)
Unit of Study for MDRO Control Efforts			
Special unit ^β	20	13	19
Hospital	10	19	17
LTCF	0	1	2
Region	0	2	1
Nature of Study or Report on MDRO Control^χ			
Outbreak	22	19	28
Non-outbreak	8	16	11
Total Period of Observation after Interventions Introduced			
Less than 1 year	17	14	25
1-2 years	6	6	6
2-5 years	5	11	8
Greater than 5 years	2	4	
Numbers of Control Measures Employed in Outbreaks/Studies			
Range	2-12	0-11	1-12
Median	7	7	8
Mode	8	7	9

^α Variably described as university hospitals, medical school affiliated hospitals, VA teaching hospitals, and, to a much lesser extent, community teaching hospitals

^β Includes intensive care units, burn units, dialysis units, hematology/oncology units, neonatal units, neonatal intensive care units, and, in a few instances, individual wards of a hospital

^χ Based on authors' description – if they called their experience an outbreak or not; authors vary in use of term so there is probable overlap between two categories

Table 2. Control Measures for MDROs Employed in Studies Performed in Healthcare Settings, 1982-2005

Focus of MDRO (No. of Studies)	MDR-GNB (n=30)	MRSA (n=35)	VRE (n=39)
No. (%) of Studies Using Control Measure			
Education of staff, patients or visitors	19 (63)	11 (31)	20 (53)
Emphasis on handwashing	16 (53)	21 (60)	9 (23)
Use of antiseptics for handwashing	8 (30)	12 (36)	16 (41)
Contact Precautions or glove use ^α	20 (67)	27 (77)	34 (87)
Private Rooms	4 (15)	10 (28)	10 (27)
Segregation of cases	4 (15)	3 (9)	5 (14)
Cohorting of Patients	11 (37)	12 (34)	14 (36)
Cohorting of Staff	2 (7)	6 (17)	9 (23)
Change in Antimicrobial Use	12 (41)	1 (3)	17 (44)
Surveillance cultures of patients	19 (63)	34 (97)	36 (92)
Surveillance cultures of staff	9 (31)	8 (23)	7 (19)
Environmental cultures	15 (50)	14 (42)	15 (38)
Extra cleaning & disinfection	11 (37)	7 (21)	20 (51)
Dedicated Equipment	5 (17)	0	12 (32)
Decolonization	3 (10)	25 (71)	4 (11)
Ward closure to new admission or to all patients	6 (21)	4 (12)	5 (14)
Other miscellaneous measures	6 (22) ^β	9 (27) ^χ	17 (44) ^δ

^α Contact Precautions mentioned specifically, use of gloves with gowns or aprons mentioned, barrier precautions, strict isolation, all included under this heading

^β includes signage, record flagging, unannounced inspections, selective decontamination, and peer compliance monitoring (1 to 4 studies employing any of these measures)

^χ includes requirements for masks, signage, record tracking, alerts, early discharge, and preventive isolation of new admissions pending results of screening cultures (1 to 4 studies employing any of these measures)

^δ includes computer flags, signage, requirement for mask, one-to-one nursing, changing type of thermometer used, and change in rounding sequence (1 to 7 studies employing any of these measures)

References for Tables 1 and 2

MDR-GNBs: (6, 8, 9, 11, 16, 38, 174, 175, 180, 209, 210, 213-215, 218, 334, 388, 406, 407)

MRSA: (68, 89, 152, 153, 165-173, 183, 188, 194, 204, 205, 208, 240, 269, 279, 280, 289, 304, 312, 327, 365, 392, 397, 408-412)

Table 3.

Tier 1. General Recommendations for Routine Prevention and Control of MDROs in Healthcare Settings						
Administrative Measures/Adherence Monitoring	MDRO Education	Judicious Antimicrobial Use	Surveillance	Infection Control Precautions to Prevent Transmission	Environmental Measures	Decolonization
<p>Make MDRO prevention/control an organizational priority. Provide administrative support and both fiscal and human resources to prevent and control MDRO transmission. <i>(IB)</i></p> <p>Identify experts who can provide consultation and expertise for analyzing epidemiologic data, recognizing MDRO problems, or devising effective control strategies, as needed. <i>(II)</i></p> <p>Implement systems to communicate information about reportable MDROs to administrative personnel and state/local health departments. <i>(II)</i></p> <p>Implement a multi-disciplinary process to monitor and improve HCP adherence to recommended practices for Standard and Contact Precautions. <i>(IB)</i></p> <p>Implement systems to designate patients known to be colonized or infected with a targeted MDRO and to notify receiving healthcare facilities or personnel prior to transfer of such patients within or between facilities. <i>(IB)</i></p> <p>Support participation in local, regional and/or national coalitions to combat emerging or growing MDRO problems. <i>(IB)</i></p> <p>Provide updated feedback at least annually to healthcare providers and administrators on facility and patient-care unit MDRO infections. Include information on changes in prevalence and incidence, problem assessment and performance improvement plans. <i>(IB)</i></p>	<p>Provide education and training on risks and prevention of MDRO transmission during orientation and periodic educational updates for HCP; include information on organizational experience with MDROs and prevention strategies. <i>(IB)</i></p>	<p>In hospitals and LTCFs, ensure that a multi-disciplinary process is in place to review local susceptibility patterns (antibiograms), and antimicrobial agents included in the formulary, to foster appropriate antimicrobial use. <i>(IB)</i></p> <p>Implement systems (e.g., CPOE, susceptibility report comment, pharmacy or unit director notification) to prompt clinicians to use the appropriate agent and regimen for the given clinical situation. <i>(IB)</i></p> <p>Provide clinicians with antimicrobial susceptibility reports and analysis of current trends, updated at least annually, to guide antimicrobial prescribing practices. <i>(IB)</i></p> <p>In settings with limited electronic communication system infrastructures to implement physician prompts, etc., at a minimum implement a process to review antibiotic use. Prepare and distribute reports to providers. <i>(II)</i></p>	<p>Use standardized laboratory methods and follow published guidelines for determining antimicrobial susceptibilities of targeted and emerging MDROs.</p> <p>Establish systems to ensure that clinical micro labs (in-house and outsourced) promptly notify infection control or a medical director/designee when a novel resistance pattern for that facility is detected. <i>(IB)</i></p> <p>In hospitals and LTCFs:</p> <p>...develop and implement laboratory protocols for storing isolates of selected MDROs for molecular typing when needed to confirm transmission or delineate epidemiology of MDRO in facility. <i>(IB)</i></p> <p>...establish laboratory-based systems to detect and communicate evidence of MDROs in clinical isolates <i>(IB)</i></p> <p>...prepare facility-specific antimicrobial susceptibility reports as recommended by CLSI; monitor reports for evidence of changing resistance that may indicate emergence or transmission of MDROs <i>(IA/IC)</i></p> <p>...develop and monitor special-care unit-specific antimicrobial susceptibility reports (e.g., ventilator-dependent units, ICUs, oncology units). <i>(IB)</i></p> <p>...monitor trends in incidence of target MDROs in the facility over time to determine if MDRO rates are decreasing or if additional interventions are needed. <i>(IA)</i></p>	<p>Follow Standard Precautions in all healthcare settings. <i>(IB)</i></p> <p>Use of Contact Precautions (CP):</p> <p>--- In <u>acute care settings</u>: Implement CP for all patients known to be colonized/infected with target MDROs. <i>(IB)</i></p> <p>--- In <u>LTCFs</u>: Consider the individual patient's clinical situation and facility resources in deciding whether to implement CP <i>(II)</i></p> <p>--- In <u>ambulatory and home care settings</u>, follow Standard Precautions <i>(II)</i></p> <p>---In <u>hemodialysis units</u>: Follow dialysis specific guidelines <i>(IC)</i></p> <p>No recommendation can be made regarding when to discontinue CP. <i>(Unresolved issue)</i></p> <p>Masks are not recommended for routine use to prevent transmission of MDROs from patients to HCWs. Use masks according to Standard Precautions when performing splash-generating procedures, caring for patients with open tracheostomies with potential for projectile secretions, and when there is evidence for transmission from heavily colonized sources (e.g., burn wounds).</p> <p>Patient placement in hospitals and LTCFs:</p> <p>When single-patient rooms are available, assign priority for these rooms to patients with known or suspected MDRO colonization or infection. Give highest priority to those patients who have conditions that may facilitate transmission, e.g., uncontained secretions or excretions. When single-patient rooms are not available, cohort patients with the same MDRO in the same room or patient-care area. <i>(IB)</i></p> <p>When cohorting patients with the same MDRO is not possible, place MDRO patients in rooms with patients who are at low risk for acquisition of MDROs and associated adverse outcomes from infection and are likely to have short lengths of stay. <i>(II)</i></p>	<p>Follow recommended cleaning, disinfection and sterilization guidelines for maintaining patient care areas and equipment.</p> <p>Dedicate non-critical medical items to use on individual patients known to be infected or colonized with an MDRO. Prioritize room cleaning of patients on Contact Precautions. Focus on cleaning and disinfecting frequently touched surfaces (e.g., bed rails, bedside commodes, bathroom fixtures in patient room, doorknobs) and equipment in immediate vicinity of patient.</p>	<p>Not recommended routinely</p>

Tier 2. Recommendations for Intensified MDRO control efforts

Institute one or more of the interventions described below when 1) incidence or prevalence of MDROs are not decreasing despite the use of routine control measures; or 2) the *first* case or outbreak of an epidemiologically important MDRO (e.g., VRE, MRSA, VISA, VRSA, MDR-GNB) is identified within a healthcare facility or unit *(IB)* Continue to monitor the incidence of target MDRO infection and colonization; if rates do not decrease, implement additional interventions as needed to reduce MDRO transmission.

Administrative Measures/Adherence Monitoring	MDRO Education	Judicious Antimicrobial Use	Surveillance	Infection Control Precautions to Prevent Transmission	Environmental Measures	Decolonization
<p>Obtain expert consultation from persons with experience in infection control and the epidemiology of MDROs, either in-house or through outside consultation, for assessment of the local MDRO problem and guidance in the design, implementation and evaluation of appropriate control measures. <i>(IB)</i></p> <p>Provide necessary leadership, funding and day-to-day oversight to implement interventions selected. <i>(IB)</i></p> <p>Evaluate healthcare system factors for role in creating or perpetuating MDRO transmission, including staffing levels, education and training, availability of consumable and durable resources; communication processes, and adherence to infection control measures. <i>(IB)</i></p> <p>Update healthcare providers and administrators on the progress and effectiveness of the intensified interventions. <i>(IB)</i></p>	<p>Intensify the frequency of educational programs for healthcare personnel, especially for those who work in areas where MDRO rates are not decreasing. Provide individual or unit-specific feedback when available. <i>(IB)</i></p>	<p>Review the role of antimicrobial use in perpetuating the MDRO problem targeted for intensified intervention. Control and improve antimicrobial use as indicated. Antimicrobial agents that may be targeted include vancomycin, third-^d generation cephalosporins, anti-anaerobic agents for VRE; third generation cephalosporins for ESBLs; and quinolones and carbapenems. <i>(IB)</i></p>	<p>Calculate and analyze incidence rates of target MDROs (single isolates/patient; location-, service-specific) <i>(IB)</i></p> <p>Increase frequency of compiling, monitoring antimicrobial susceptibility summary reports <i>(II)</i></p> <p>Implement laboratory protocols for storing isolates of selected MDROs for molecular typing; perform typing if needed <i>(IB)</i></p> <p>Develop and implement protocols to obtain active surveillance cultures from patients in populations at risk. <i>(IB)</i> (See recommendations for appropriate body sites and culturing methods.)</p> <p>Conduct culture surveys to assess efficacy of intensified MDRO control interventions.</p> <p>Conduct serial (e.g., weekly) unit-specific point prevalence culture surveys of the target MDRO to determine if transmission has decreased or ceased. <i>(IB)</i></p> <p>Repeat point-prevalence culture-surveys at routine intervals and at time of patient discharge or transfer until transmission has ceased. <i>(IB)</i></p> <p>If indicated by assessment of the MDRO problem, collect cultures to assess the colonization status of roommates and other patients with substantial exposure to patients with known MDRO infection or colonization. <i>(IB)</i></p> <p>Obtain cultures from HCP for target MDROs when there is epidemiologic evidence implicating the staff member as a source of ongoing transmission. <i>(IB)</i></p>	<p>Use of Contact Precautions: Implement Contact Precautions (CP) routinely for all patients colonized or infected with a target MDRO. <i>(IA)</i> Don gowns and gloves before or upon entry to the patient’s room or cubicle. <i>(IB)</i> In LTCFs, modify CP to allow MDRO-colonized/infected patients whose site of colonization or infection can be appropriately contained and who can observe good hand hygiene practices to enter common areas and participate in group activities When active surveillance cultures are obtained as part of an intensified MDRO control program, implement CP until the surveillance culture is reported negative for the target MDRO <i>(IB)</i></p> <p>No recommendation is made for universal use of gloves and/or gowns. <i>(Unresolved issue)</i></p> <p>Implement policies for patient admission and placement as needed to prevent transmission of the problem MDRO. <i>(IB)</i></p> <p>When single-patient rooms are available, assign priority for these rooms to patients with known or suspected MDRO colonization or infection. Give highest priority to those patients who have conditions that may facilitate transmission, e.g., uncontained secretions or excretions. When single-patient rooms are not available, cohort patients with the same MDRO in the same room or patient-care area. <i>(IB)</i></p> <p>When cohorting patients with the same MDRO is not possible, place MDRO patients in rooms with patients who are at low risk for acquisition of MDROs and associated adverse outcomes from infection and are likely to have short lengths of stay. <i>(II)</i></p> <p>Stop new admissions to the unit or facility if transmission continues despite the implementation of the intensified control measures. <i>(IB)</i></p>	<p>Implement patient.-dedicated use of non-critical equipment <i>(IB)</i></p> <p>Intensify and reinforce training of environmental staff who work in areas targeted for intensified MDRO control. Some facilities may choose to assign dedicated staff to targeted patient care areas to enhance consistency of proper environmental cleaning and disinfection services <i>(IB)</i></p> <p>Monitor cleaning performance to ensure consistent cleaning and disinfection of surfaces in close proximity to the patient and those likely to be touched by the patient and HCWs (e.g., bedrails, carts, bedside commodes, doorknobs, faucet handles) <i>(IB)</i>.</p> <p>Obtain environmental cultures (e.g., surfaces, shared equipment) only when epidemiologically implicated in transmission <i>(IB)</i></p> <p>Vacate units for environmental assessment and intensive cleaning when previous efforts to control environmental transmission have failed <i>(II)</i></p>	<p>Consult with experts on a case-by-case basis regarding the appropriate use of decolonization therapy for patients or staff during limited period of time as a component of an intensified MRSA control program <i>(II)</i></p> <p>When decolonization for MRSA is used, perform susceptibility testing for the decolonizing agent against the target organism or the MDRO strain epidemiologically implicated in transmission. Monitor susceptibility to detect emergence of resistance to the decolonizing agent. Consult with microbiologists for appropriate testing for mupirocin resistance, since standards have not been established.</p> <p>Do not use topical mupirocin routinely for MRSA decolonization of patients as a component of MRSA control programs in any healthcare setting. <i>(IB)</i></p> <p>Limit decolonization to HCP found to be colonized with MRSA who have been epidemiologically implicated in ongoing transmission of MRSA to patients. <i>(IB)</i></p> <p>No recommendation can be made for decolonization of patients who carry VRE or MDR-GNB.</p>



Methicillin-Resistant *Staphylococcus aureus* (MRSA) Infections

Activity C: ELC Prevention Collaboratives

John Jernigan, MD, MS

Alex Kallen, MD, MPH

Division of Healthcare Quality Promotion

Centers for Disease Control and Prevention

Draft – 1/19/10 ---- Disclaimer: The findings and conclusions in this presentation are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.



Outline



- **Background**
 - Impact
 - HHS Prevention Targets
 - Pathogenesis
 - Epidemiology
- **Prevention Strategies**
 - Core
 - Supplemental
- **Measurement**
 - Process
 - Outcome
- **Tools for Implementation/Resources/References**



Background: Impact



Staphylococcus aureus is a common cause of healthcare-associated infections

- Second most common overall cause of healthcare-associated infections reported to the National Healthcare Safety Network (NHSN)
 - Coagulase-negative staphylococci (15%), *S. aureus* (14%)
 - Most common cause of surgical site infections(30%) and ventilator associated pneumonia (24%)
- Methicillin-resistance in *S. aureus* was first identified in the 1960s primarily among hospitalized patients
- Since that time, methicillin-resistant *S. aureus* (MRSA) has become a predominant cause of *S. aureus* infections in both healthcare and community settings
 - Primarily due to transmission of relatively few ancestral clones rather than the de novo development of methicillin-resistance among susceptible strains

Hidron et al. Infect Control Hosp Epidemiol 2008;29:996-1011



Background: Impact



- Current estimates suggest that 49-65% of healthcare-associated *S. aureus* infections reported to NHSN are caused by methicillin-resistant strains
- National population-based estimates of invasive MRSA infections
 - 94,360 invasive MRSA infections annually in the US
 - Associated 18,650 deaths each year
 - 86% of all invasive MRSA infections are healthcare-associated

Hidron et al. Infect Control Hosp Epidemiol 2008;29:996-1011

Klevens et al. JAMA 2007;298:1763-71



Background: Impact



Why the Emergence of MRSA is a Healthcare Pathogen is Important (1)

- MRSA has emerged as one of the predominant pathogens in healthcare-associated infections
- Treatment options for MRSA are limited and less effective than options available for susceptible *S. aureus* infections and result in higher morbidity and mortality
- High prevalence influences unfavorable antibiotic prescribing, which contributes to further spread of resistance
 - prevalent MRSA → more vancomycin use → more vancomycin resistance (VRE and VRSA)
more linezolid/daptomycin use → more resistance



Background: Impact



Why the Emergence of MRSA is a Healthcare Pathogen is Important (2)

- MRSA adds to overall *S. aureus* infection burden
 - Preventing MRSA infections reduces overall burden of *S. aureus* infections
- MRSA is a marker for ability to contain transmission of important pathogens in the healthcare setting
 - Programs that successfully prevent MRSA transmission are likely to have benefit when applied to other epidemiologically important healthcare pathogens that spread by patient-to-patient transmission



Background: HHS Prevention Targets



- Population-based surveillance
 - 50% reduction in incidence rate of all healthcare-associated invasive MRSA infections
- National Healthcare Safety Network
 - 50% reduction in incidence rate of hospital-onset MRSA bacteremia

HHS Action Plan to Prevent HAI

(<http://www.hhs.gov/ophs/initiatives/hai/infection.html>)



Background: Pathogenesis



- For MRSA, colonization generally precedes infection
- In addition, colonization can be long-lasting -- months or years in some subpopulations
- In general, MRSA is transmitted person to person; the “*de novo*” generation of resistance in *S. aureus* is very rare
- Transmission of MRSA from the environment to people, although it can occur, is less common than transmission from person to person



Background: Epidemiology



- Once acquired, MRSA colonization can be long-lasting -- months or years in some subpopulations
 - A patient acquiring MRSA colonization during a hospital stay has increased risk for MRSA infections following discharge, or during subsequent acute and long-term care admissions
- MRSA carriers also serve as reservoirs for further transmission as they move through and across healthcare facilities
- Healthcare facilities that share patients are interdependent upon one another with regard to their MRSA experience
 - The quality of MRSA control in one facility may influence the MRSA experience in others
 - There may be advantages to coordinated multicenter control programs involving facilities that share patients with one another



Background: Epidemiology



- Successful MRSA prevention is possible
 - Single and multi-center studies have demonstrated that MRSA prevention programs can be effective
 - Reductions in incidence of MRSA disease by up to 70% have been documented in acute-care facilities
 - Significant intervention-associated reductions in the proportion of *S. aureus* infections caused by MRSA have also been documented in these studies

Ellingson K et al. Presented at SHEA 2009, Abstract 512.

Huang et al. Clin Infect Dis 2006; 43:971-88.

Robicsek et al. Ann Intern Med 2008; 148:409-18.



Epidemiology

- Successful MRSA prevention is possible
 - According to NSHN data, rates of central line-associated BSI (CLABSI) caused by MRSA have declined by nearly 50% in the past decade
 - This observation may be primarily attributable to successful CLABSI prevention efforts
 - The proportion of all *S. aureus* CLABSI caused by MRSA has continued to increase during the same time period
- Population-based estimates suggest the incidence of invasive healthcare-associated MRSA disease decreased by 11-17% in the US between 2005-2007

Burton et al. JAMA 2009; 301:727-36

Kallen AJ, et al. Presented at SHEA 2009 Abstract 49



Prevention Strategies

- **Core Strategies**

- High levels of scientific evidence
- Demonstrated feasibility

- **Supplemental Strategies**

- Some scientific evidence
- Variable levels of feasibility

*The Collaborative should at a minimum include core prevention strategies. Supplemental prevention strategies also may be used. Hospitals should not be excluded from participation if they already have ongoing interventions using supplemental prevention strategies. Project coordinators should carefully track which prevention strategies are being used by participating facilities.



Prevention Strategies: Basic Rationale



- Because MRSA colonization generally precedes infection with this organism, MRSA interventions primarily have targeted two broad areas:
 - Preventing transmission from colonized to uncolonized persons – a focus of most of the interventions in this toolkit
 - Preventing infection in colonized individuals:
 - Not MRSA-specific: Strategies aimed at preventing device and procedure-associated infections (e.g., ventilator associated pneumonias, central line associated bloodstream infections, etc), not necessarily MRSA-specific
 - MRSA-specific: MRSA decolonization strategies



Core Prevention Strategies



- Assessing hand hygiene practices
- Implementing Contact Precautions
- Recognizing previously colonized patients
- Rapidly reporting MRSA lab results
- Providing MRSA education for healthcare providers



Core Prevention Strategies: Hand Hygiene

- Hand hygiene should be a cornerstone of prevention efforts
 - Prevents transmission of pathogens via hands of healthcare personnel
- As part of a hand hygiene intervention, consider:
 - Ensuring easy access to soap and water/alcohol-based hand gels
 - Education for healthcare personnel and patients
 - Observation of practices - particularly around high-risk procedures (before and after contact with colonized or infected patients)
 - Feedback – “Just in time” feedback if failure to perform hand hygiene observed



Core Prevention Strategies: Contact Precautions



- Involves use of gown and gloves for patient care
 - Don equipment prior to room entry
 - Remove prior to room exit
- Single room (preferred) or cohorting for MRSA colonized/infected patients
- Use of dedicated non-essential items may help decrease transmission due to contact with these fomites
 - Blood pressure cuffs
 - Stethoscopes
 - IV poles and pumps



Core Prevention Strategies: Recognizing Previously Colonized

- Patients can be colonized with MRSA for months
- There is no single 'best' strategy for discontinuation of isolation precautions for MRSA patients
- Being able to recognize previously colonized or infected patients who have not met criteria for discontinuing isolation allows them to be subject to interventions in a timely fashion



Core Prevention Strategies: Laboratory Reporting



- Facilities should have a mechanism for rapidly communicating positive MRSA results from laboratory to clinical area
- Allows for rapid institution of interventions on newly identified MRSA patients



Core Prevention Strategies: Education



- To improve adherence to hand hygiene
- To improve adherence to interventions (e.g., Contact Precautions)
- Encourage behavioral change through a better understanding of the problem



Core Prevention Strategies: Device and Procedure-Associated Prevention Measures

- In addition to measures designed to prevent MRSA transmission, healthcare facilities should routinely implement strategies for preventing device- and procedure-associated infections
 - Central line-associated bloodstream infections
 - Surgical site infections
 - Catheter-associated urinary tract infections
 - Ventilator-associated pneumonia



Supplemental Prevention Strategies

- Active surveillance testing – screening of patients to detect colonization even if no evidence of infection
 - Widely used and even recommended as a core prevention strategy by some, but precise role remains controversial
- Other novel strategies
 - Decolonization
 - Chlorhexidine bathing



Supplemental Prevention Strategies: Active Surveillance Testing (AST)



- When clinical culture results alone are used to identify MRSA carriers, more than half of all MRSA-colonized patients remain unrecognized*
 - The rationale for active surveillance testing is to identify all colonized patients so that additional precautions can be applied (e.g. Contact Precautions)
- To date, results of studies evaluating AST have had mixed results
 - Huang et al. Clin Infect Dis 2006; 43:971-978
 - Observational study
 - Found largest decrease in MRSA bacteremia associated with institution of active surveillance
 - Robicsek et al. Ann Intern Med 2008; 148:409-418
 - Observational study
 - Found significant decrease in MRSA disease with universal institution of AST combined with decolonization regimens
 - Harbarth et al. JAMA 2008; 299:1149-1157
 - Observational study
 - No significant decrease in MRSA disease with institution of rapid AST
- Several successful MRSA prevention collaboratives have used AST as one of their interventions

*Salgado CD, Farr BM. Infect Control Hosp Epidemiol 2006; 27:116-121.



Supplemental Prevention Strategies: Active Surveillance Testing (2)



Testing methods:

- Culture
 - Pros
 - Generally less costly
 - A common practice most labs are used to
 - Cons
 - May take 72 hours to identify MRSA colonized patients. If pre-emptive isolation not employed, may allow for transmission prior to recognizing patient as positive
- Polymerase chain reaction
 - Pros
 - Rapid results
 - Cons
 - Expensive
 - Technically more challenging



Supplemental Prevention Strategies: Active Surveillance Testing (3)



Unknowns:

- **Which body sites should be tested?**
 - Nares most common
 - Other potential sites include wounds, axillae, groin
 - Adding more sites increases yield of testing; contribution to goal of decreasing transmission unclear
- **Frequency of testing?**
 - Generally done at time of admission, sometimes repeated weekly
 - Including discharge AST allows for identification of transmission events that occurred during hospitalization
- **Who should be tested?**
 - One commonly employed strategy: focusing on patients in high-risk areas (e.g., ICUs)
 - Some employ facility-wide AST



Supplemental Prevention Strategies: Decolonization Therapy for MRSA Carriers

- Decolonization is use of topical and/or systemic agents to suppress or eliminate colonization
- May reduce risk of subsequent infections in MRSA carriers
- May help decrease MRSA spread by reducing reservoir of transmission
- No data yet to definitively support its routine use in general patient care settings
 - Robicsek and Harbarth studies used decolonization in addition to AST with mixed results
 - Growing evidence suggests that pre-operative *S. aureus* decolonization regimens decrease risk of subsequent *S. aureus* infection in some surgical populations



Supplemental Prevention Strategies: Decolonization Therapy for MRSA Carriers (2)

Unknowns:

- Which body sites should be targeted?
 - just nares or whole body
- Which decolonization regimen?
 - Intranasal mupirocin, chlorhexidine baths
 - May be advantageous to use combination of mupirocin and chlorhexidine
 - Other agents (oral agents)
- Will emergence of mupirocin resistance be a limiting factor?
 - Also potential cross-resistance to other therapeutic agents



Supplemental Prevention Strategies: Universal use of Chlorhexidine Bathing in High-Risk Patient Populations

- Use of daily chlorhexidine baths in ICU populations may decrease overall rates of bloodstream infections and MRSA acquisition, but effect on MRSA infections less clear
- Does not require AST since applied to all patients in the target population

Climo MW, et al. Crit Care Med 2009; 37:1858-65



Summary of Prevention Strategies



Core Measures

- Assessing hand hygiene practices
- Implementing Contact Precautions
- Recognizing previously colonized patients
- Rapidly reporting MRSA lab results
- Providing MRSA education for healthcare providers

Supplemental Measures

- Active surveillance testing
- Decolonization
- Chlorhexidine bathing



Measurement: Outcome Using NHSN to support MRSA Prevention Collaboratives

- NHSN provides a module designed to facilitate prevention of healthcare-associated MRSA and other multidrug-resistant organisms
 - Provides methods and reporting mechanisms for both outcome and process measures

<http://www.cdc.gov/nhsn>



Measurement: Outcome

MRSA Outcome Measures



- MRSA Infection Incidence Rate
 - Numerator = Number of MRSA infections*
 - Denominator = Number of patient-days (stratified by time and location)

**per current NHSN definitions for healthcare associated infection*

<http://www.cdc.gov/nhsn>



Measurement: Outcome NHSN



- **Laboratory Identified MRSA Events**
 - Proxy Measure for MDRO Healthcare Acquisition
 - Numerator = Number of 1st MRSA isolates per patient (infection or colonization) identified from a clinical culture (i.e. not from AST) among those with no documented prior evidence of infection or colonization
 - Denominator = number of patient days for the location or facility
 - Proxy Measure for MDRO Bloodstream Infection
 - Numerator = Total number of patients with MRSA blood isolate and no prior positive blood culture in ≤ 2 weeks
 - Denominator = Number of patient-days for same period

Note : isolates of MRSA are generally attributed to the location or facility under surveillance if they come from cultures collected more than 3 calendar days after admission (if day of admission is day 1)

<http://www.cdc.gov/nhsn>



Measurement: Outcome

Other Potential Measures Available in NHSN



- **Measures Based on Active Surveillance Testing**
 - Admission prevalence rate
 - Incidence of MRSA colonization
- **Other Laboratory Identified MRSA Events**
 - Admission prevalence rate (community-onset MRSA)
 - Overall prevalence rate (community-onset plus healthcare facility-onset)
 - MRSA bloodstream infection admission prevalence rate
 - Proportion of *S. aureus* resistant to methicillin

<http://www.cdc.gov/nhsn>



Measurement: Process

MRSA Process Measures



- As part of the MDRO module, NHSN allows facilities to track adherence to:
 - Active surveillance testing
 - Contact Precautions
 - Hand hygiene

<http://www.cdc.gov/nhsn>



Evaluation Considerations

- **Assess baseline policies and procedures**
- **Areas to consider**
 - Surveillance
 - Prevention strategies
 - Measurement
- **Coordinator should track new policies/practices implemented during collaboration**



References

- Burton DC, Edwards JR, Horan TC, et al. Methicillin-resistant *Staphylococcus aureus* central line-associated bloodstream infections in US intensive care units. JAMA 2009;301:727-36.
- Calfee D, Salgado CD, Classen D, et al. SHEA Compendium: Strategies to Prevent MRSA Transmission in Acute Care Hospitals Infect Control Hosp Epidemiol 2008; 29:S62-S80.



References

- Climo MW, Sepkowitz KA, Zuccotti G, et al. The effectiveness of daily bathing with chlorhexidine on the acquisition of methicillin-resistant *Staphylococcus aureus*, vancomycin-resistant *Enterococcus*, and healthcare-associated bloodstream infections: results of a quasi-experimental multicenter trial. *Crit Care Med* 2009;37:1858-65.
- Cohen AI, Calfee D, Fridkin SK, et al. Recommendations for metrics for multidrug-resistant organisms in healthcare settings: SHEA/HICPAC Position Paper. *Infect Control Hosp Epidemiol* 2008; 29:901-13.



References

- Ellingson K, Iversen N, Zuckerman JM, et al. A successful multi-center intervention to prevent transmission of methicillin-resistant *Staphylococcus aureus*. Presented at SHEA 2009, Abstract 512.
- HICPAC – Management of Multidrug Resistant Organisms in Healthcare Settings, 2006
<http://www.cdc.gov/ncidod/dhqp/pdf/ar/MDROGuideline2006.pdf>
- Hidron AL, Edwards JR, Patel J, et al. NHSN annual update: antimicrobial-resistant pathogens associated with healthcare-associated infections: annual summary of data reported to the National Healthcare Safety Network at the Centers for Disease Control and Prevention, 2006-2007. *Infect Control Hosp Epidemiol*; 2008;29:966-1011.



References

- Huang SS, Yokoe, DS, Hinrichsen VL, et al. Impact of routine intensive care unit surveillance cultures and resultant barrier precautions on hospital-wide methicillin-resistant *Staphylococcus aureus* bacteremia. *CLin Infect Dis* 2006;43:971-8.
- Kallen AJ, Yi Mu, Bulens SN, et al. Changes in the incidence of healthcare-associated invasive MRSA infections and concurrent MRSA control practices in the US, 2005 to 2007 Presented at SHEA 2009. Abstract 49.
- Klevens, RM, Morrison MA, Nadle J, et al. Invasive methicillin-resistant *Staphylococcus aureus* infections in the United States. *JAMA*;2007:298:1763-71.



References

- Robicsek A, Beaumont JL, Paule SM, et al. Universal surveillance for methicillin-resistant *Staphylococcus aureus* in 3 affiliated hospitals. *Ann Intern Med* 2008;148:409-18.
- Harbarth S, Fankhauser C, Srenzel J, et al. Universal screening for methicillin-resistant *Staphylococcus aureus* at hospital admission and nosocomial infection in surgical patients. *JAMA* 2008;229:1149-57.



Additional Resources

- HHS Action Plan to Prevent Healthcare Associated Infections. June 2009 <http://www.hhs.gov/ophs/initiatives/hai/infection.html>
- Overview of Methicillin-Resistant *Staphylococcus aureus* Surveillance through the National Healthcare Safety Network
http://www.cdc.gov/nhsn/PDFs/Overview_MRSA_Surveillance_Final12_08.pdf
- Multidrug-Resistant Organism & *Clostridium difficile*-Associated Disease (MDRO/CDAD) Module
http://www.cdc.gov/nhsn/PDFs/pscManual/12pscMDRO_CDA_Dcurrent.pdf
- NHSN Web site – www.cdc.gov/nhsn



Additional Reference Slides



- The following slides may be used for presentations on MRSA
- Explanations are available in the notes sections of the slides



Distribution and Rank Order of 9 Most Common Pathogens Reported for 28,502 HAIs, NHSN 2006-2007



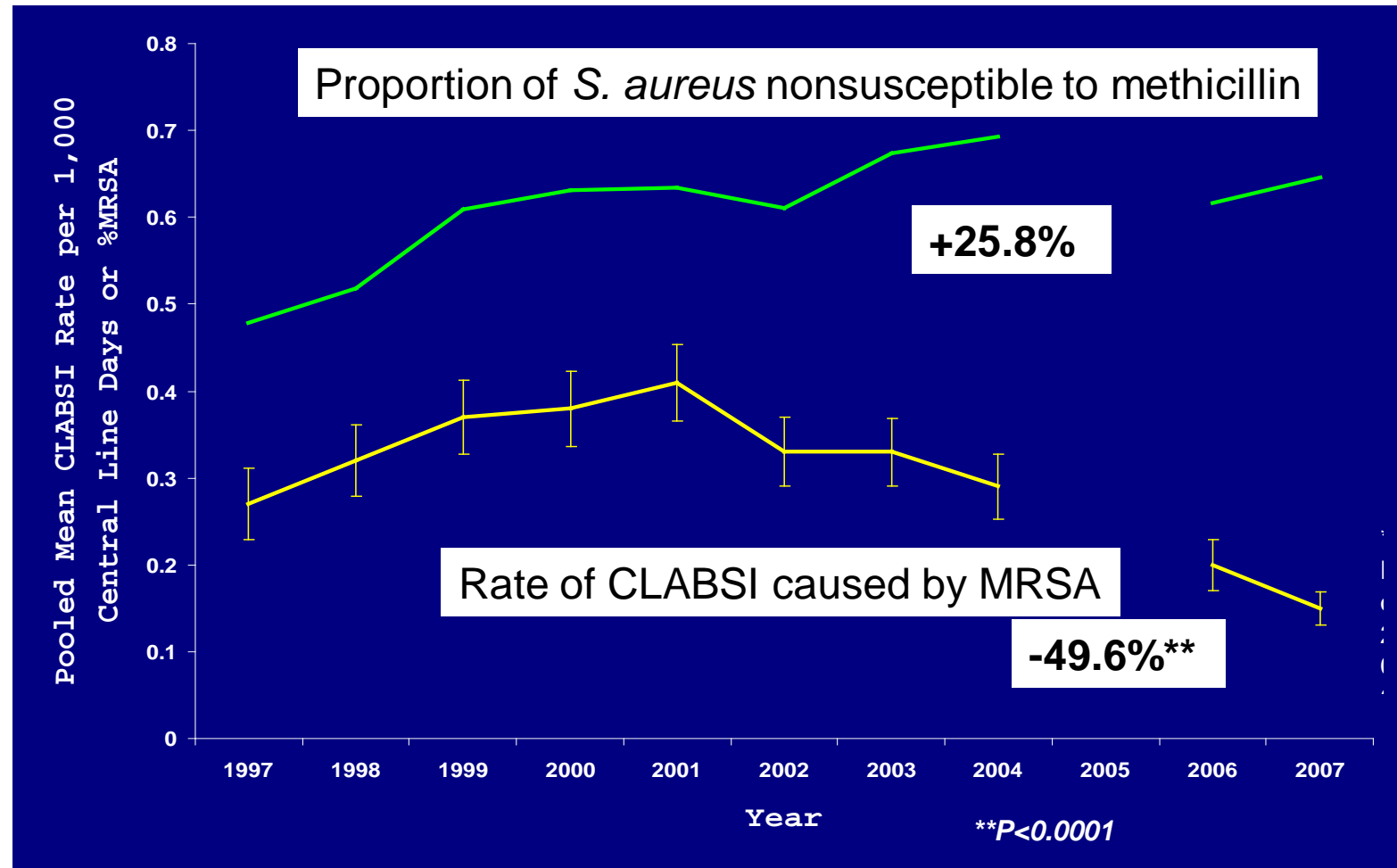
Pathogen	Column %				Total*
	CLABSI 11,428	CAUTI 9,377	VAP 5,960	SSI 7,025	
CoNS	34	3	1	14	15
<i>S. aureus</i>	10	2	24	30	14
<i>Enterococcus</i> spp.	15	15	1	11	12
<i>Candida</i> spp.	12	21	<1	2	11
<i>E. coli</i>	3	22	5	10	10
<i>P. aeruginosa</i>	3	10	16	5	8
<i>K. pneumoniae</i>	5	8	7	3	6
<i>Enterobacter</i> spp.	4	4	8	4	5
<i>A. baumannii</i>	2	1	8	1	3

15.6% of healthcare-associated infections had >1 pathogen (polymicrobial)

Hidron et al. Infect Control Hosp Epidemiol 2008;29:996-1011



Trends in % MRSA and Rates of MRSA Central Line-Associated Bloodstream Infections (CLABSI) — United States, 1997-2007



Burton et al. JAMA 2009; 301:727-36



Modeled Incidence and Percent Change for All Invasive Hospital-Onset and Healthcare-Associated, Community-Onset MRSA infections, 2005-2007



<i>Year</i>	<i>Modeled incidence per 100,000 population</i>	<i>Modeled percent change from previous year</i>	<i>Total modeled percent change</i>	<i>P-value</i>
<i>Hospital-onset</i>				
2005	9.95			
2006	8.96	-9.97%		
2007	8.24	-8.08%	-17.2%	0.01
<i>Healthcare-associated, community-onset</i>				
2005	22.13			
2006	21.11	-4.59%		
2007	19.70	-6.71%	-11.0%	0.04

Kallen AJ, et al, SHEA 2009, Abstract 49



Norovirus Gastroenteritis:

Management of Outbreaks in Healthcare Settings



U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

Norovirus



- ❑ The most common cause of cases of acute gastroenteritis and gastroenteritis outbreaks
- ❑ Can affect nearly everyone in the population (from children to the elderly and everyone in between!) particularly because there is no long term immunity to the virus
- ❑ Causes acute but self-limited diarrhea, often with vomiting, abdominal cramping, fever, and fatigue
 - Most individuals recover from acute symptoms with 2-3 days , but can be more severe in vulnerable populations

Burden of Norovirus Infection



- ❑ #1 cause of acute gastroenteritis in U.S.
 - 21 million cases annually
 - 1 in 14 Americans become ill each year
 - 71,000 hospitalized annually in U.S.
 - 80 deaths annually among elderly in U.K.
 - 91,000 emergency room visits overall in the U.S.

- ❑ Occurs year round with peak activity during the winter months

- ❑ Cases occur in all settings, across the globe

Norovirus in Healthcare Facilities

- ❑ Norovirus is a recognized cause of gastroenteritis outbreaks in institutions.
- ❑ Healthcare facilities are the most commonly reported settings of norovirus gastroenteritis outbreaks in the US and other industrialized countries.
- ❑ Outbreaks of gastroenteritis in healthcare settings pose a risk to patients, healthcare personnel, and to the efficient provision of healthcare services.



Norovirus Activity in Healthcare

- Incidence of norovirus outbreaks in acute care facilities and community hospitals within the United States remains unclear.
- This is in contrast with the established high burden of acute care hospital outbreaks reported in many other industrialized countries.

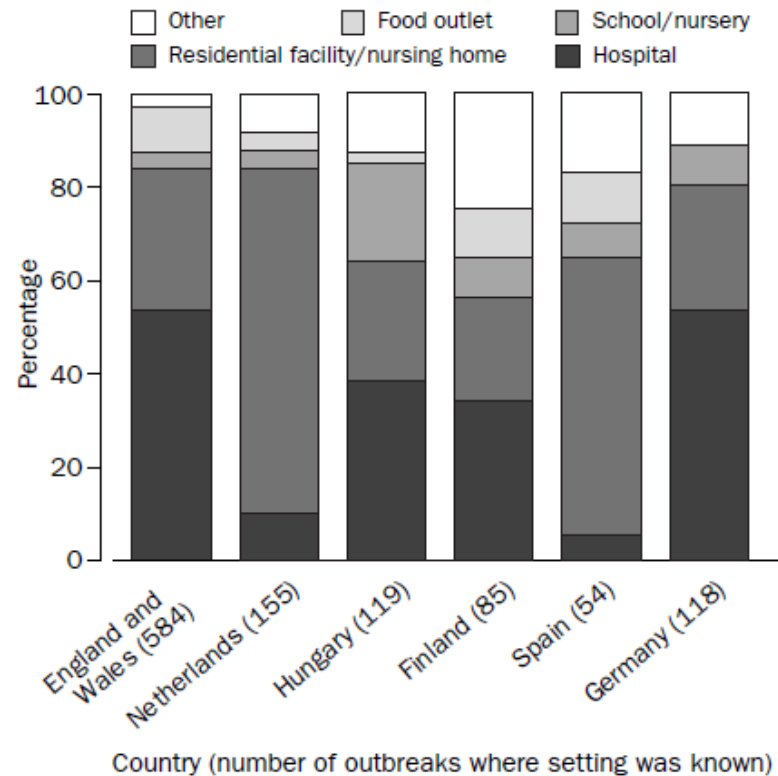
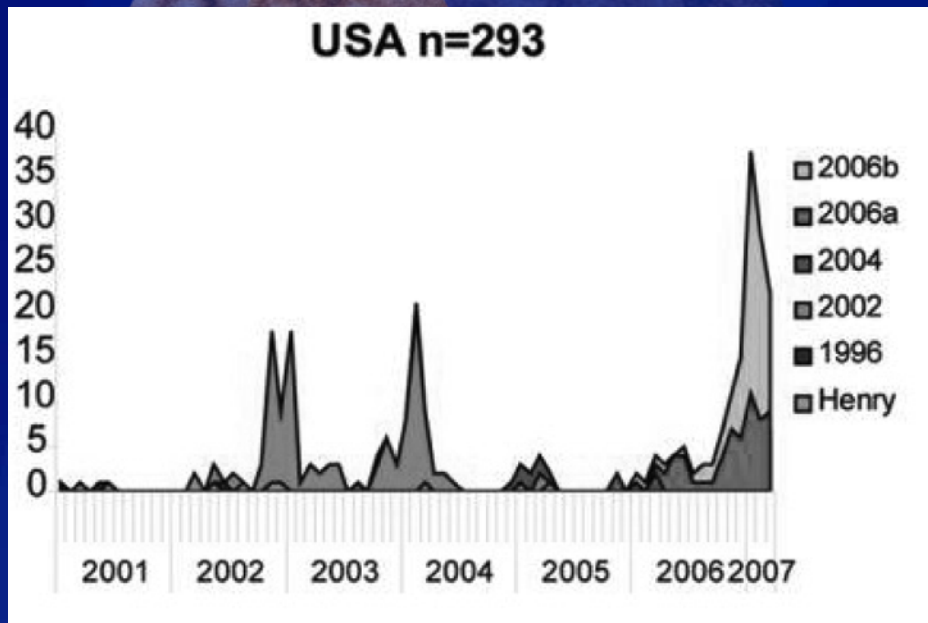


Figure 4: **Setting of norovirus outbreak in 2002 for six European regions**

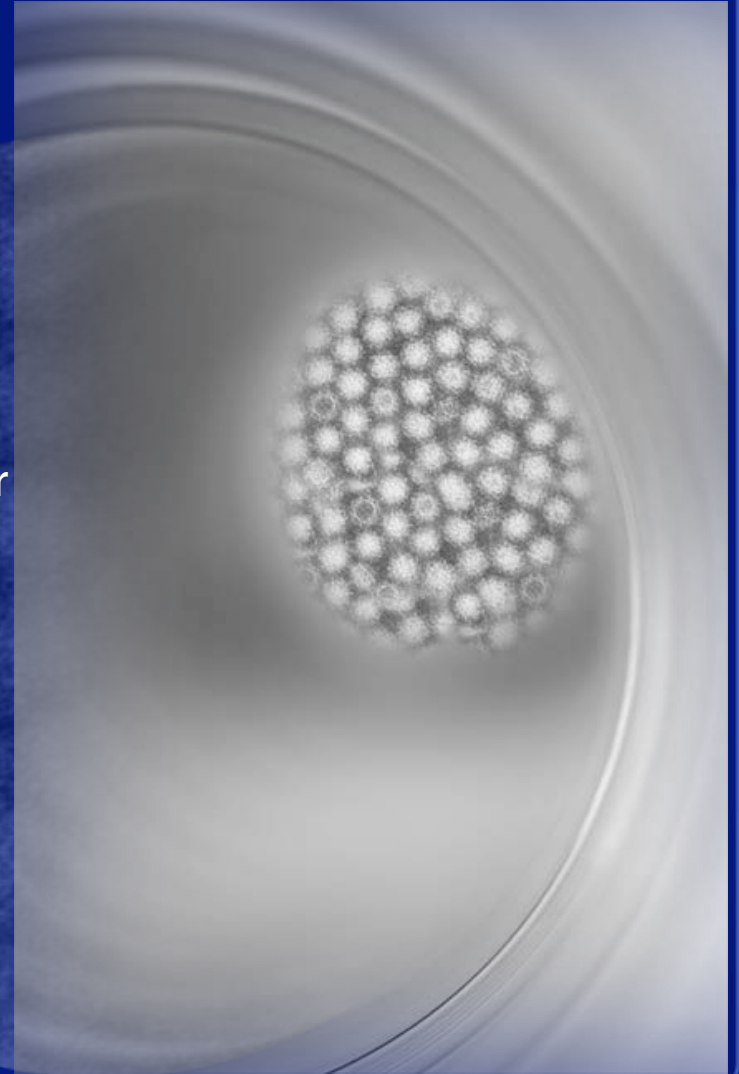
Dynamic Nature of Norovirus in the US



- Genogroup II type 4 (GII.4) noroviruses cause >75% of outbreaks worldwide
- New strains of GI.4 emerge every 3-5 years
- The periodic emergence of new strains is associated with heightened norovirus activity
- New strains in the 2002/03 and 2006/07 winters caused a surge in outbreaks

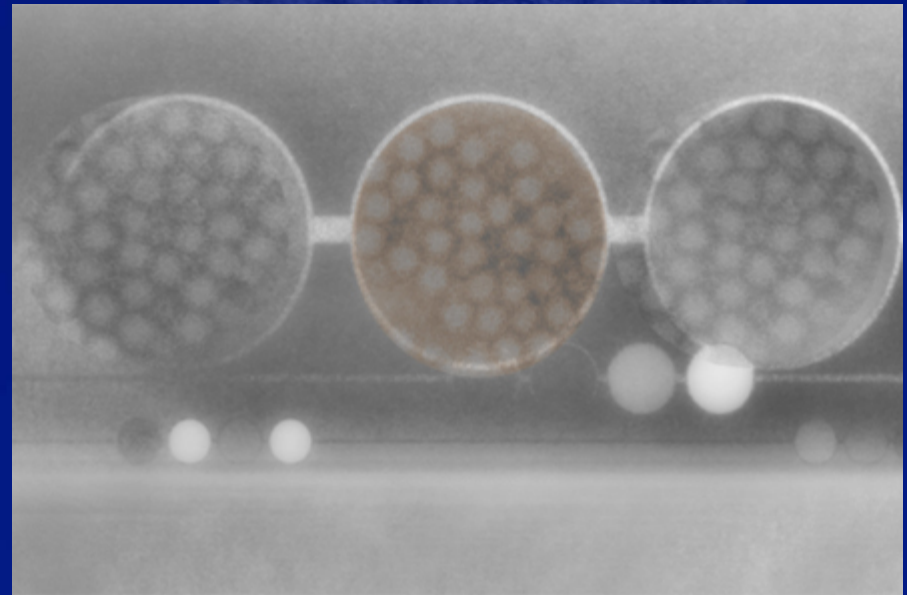
Clinical Disease

- ❑ Infectious dose: 18-1000 viral particles
- ❑ Incubation period: 12-48 hours
- ❑ Acute-onset vomiting and/or diarrhea
 - Watery, non-bloody stools
 - Abdominal cramps, nausea, low-grade fever
 - 30% infections asymptomatic
- ❑ Most recover after 12-72 hours
 - Up to 10% seek medical attention; some require hospitalization and fluid therapy
 - More severe illness and death possible in elderly and those with other illnesses



Viral Shedding

- ❑ Primarily in stool, but can also be present in vomitus
- ❑ Shedding peaks 4 days after exposure
- ❑ In some individuals, shedding may occur for at least 2-3 weeks
- ❑ $\sim 10^{12}$ viral copies/gram feces
- ❑ May occur after resolution of symptoms
- ❑ Infectivity of shed virus in environment unknown
- ❑ Shedding in asymptomatic individuals is common but their role in transmission is not known

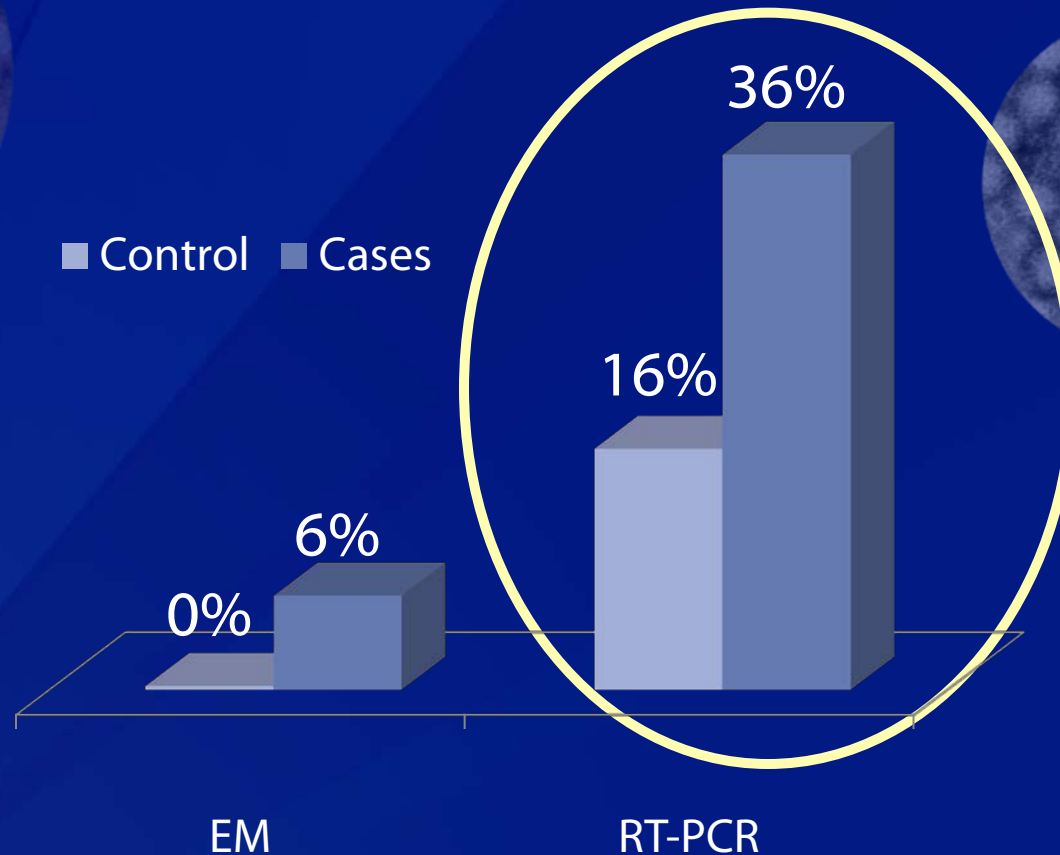


Immunity to Norovirus

- ❑ Short-term immunity after infection
- ❑ There is little cross protective immunity (against different genotypes)
- ❑ No long-term immunity
 - Protection believed to last less than one year, and in some studies, protection may only last a few months
- ❑ Genetic susceptibility
 - A portion of the population may be genetically resistant to norovirus infection
 - Currently no commercially available test to identify those who might carry genes conferring resistance to norovirus infection



Norovirus Prevalence in the Community



Using sensitive PCR diagnostics, norovirus is frequently detected in stools of both infected individuals (cases) and healthy asymptomatic individuals (controls)

Transmission of Disease

- ❑ Person to person
 - Direct fecal-oral
 - Ingestion of aerosolized vomitus
 - Indirect via fomites or contaminated environment

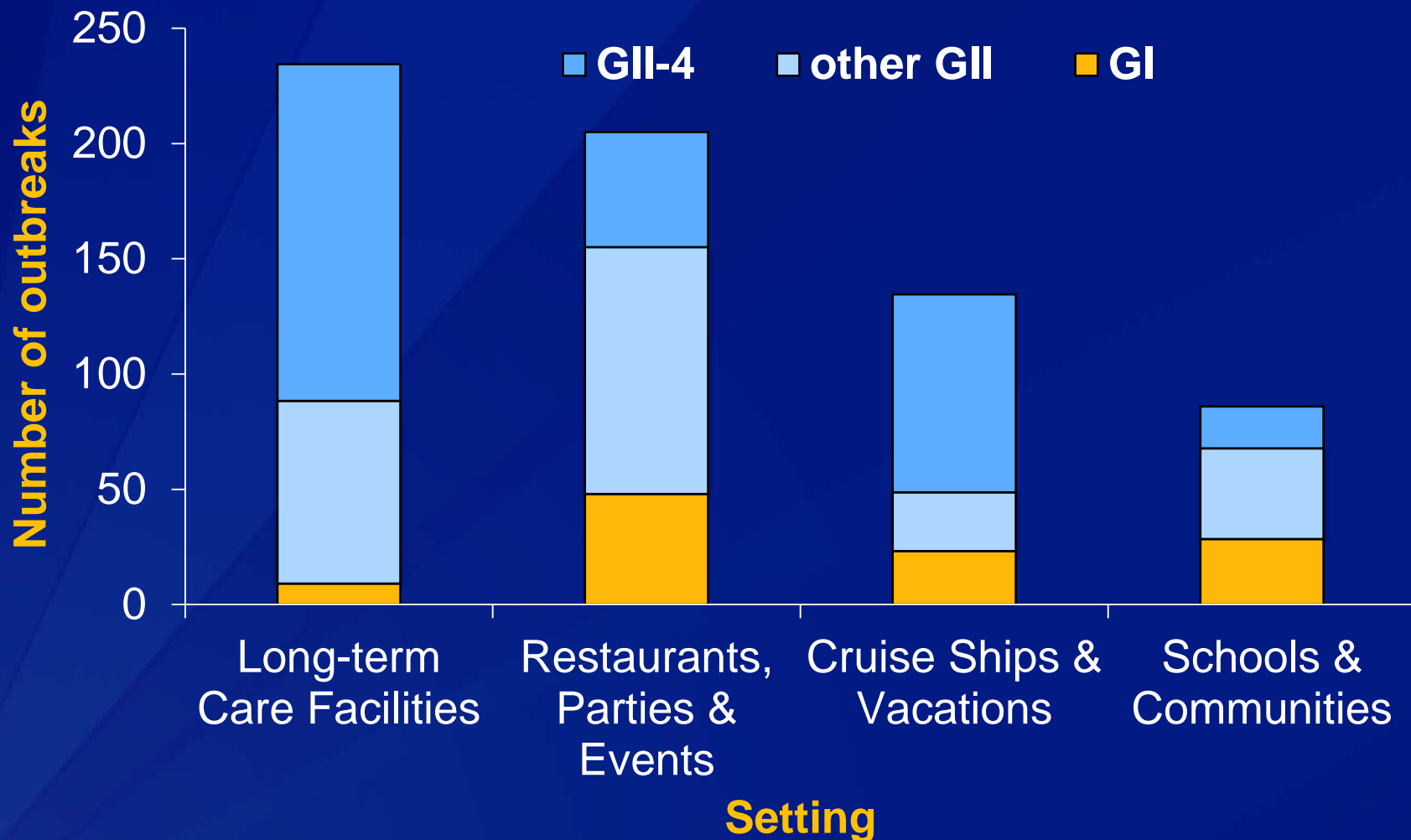
- ❑ Food
 - Contamination by infected food handlers
 - Point of service or source (e.g., raspberries, oysters)

- ❑ Recreational and Drinking Water
 - Well contamination from septic tank
 - Chlorination system breakdown

- ★ In healthcare, the most likely and common modes of transmission are through direct contact with infected persons or contaminated equipment

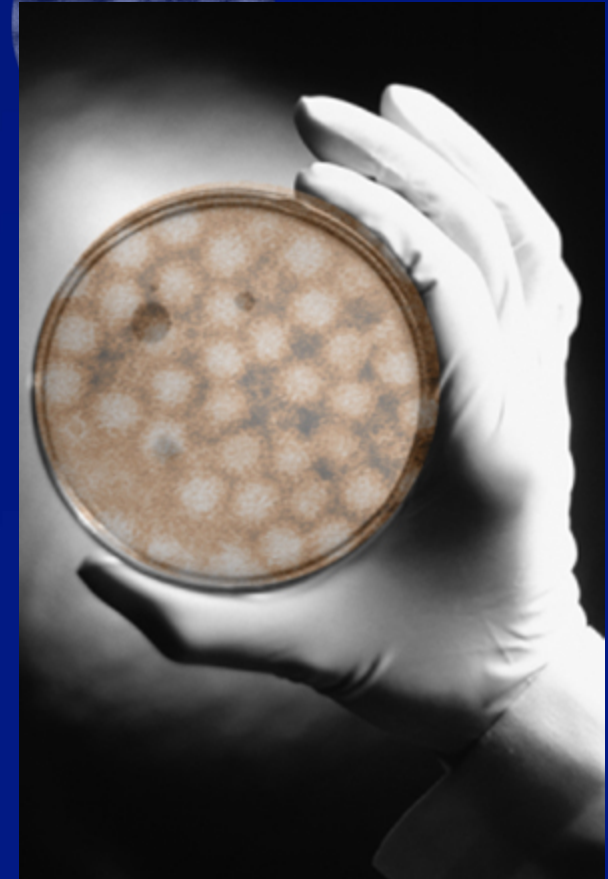


Setting of Norovirus Outbreaks Reported to CDC, United States 1994-2006



Laboratory Confirmation of Norovirus

- ❑ Where available, reverse transcription polymerase chain reaction (RT-PCR) confirmation is the preferred diagnostic for norovirus
- ❑ State public health laboratories may be able to provide RT-PCR diagnostics to confirm norovirus
- ❑ Typically, state laboratories require a minimum number of stool samples from a subset of symptomatic patients before initiating confirmatory testing



Submitting Clinical Samples for Norovirus Testing

- ❑ Consult with receiving clinical, local or state health labs prior to submitting samples for norovirus identification
 - Depending on laboratory policies, may need multiple suspect cases before specimen testing can be performed
- ❑ Stool specimens should be collected from individuals during acute phase of illness
 - Virus may be able to be detected in specimens taken later in the course of illness, but sensitivity is reduced
- ❑ Submit stool specimens as early as possible during a potential outbreak or cluster
- ❑ While not ideal, vomitus may be submitted for testing to some labs
- ❑ Both staff and patient cases can be tested

What should clinical staff do when they suspect norovirus?

- Key Infection Control Activities
 - Rapid identification and isolation of suspected cases of norovirus gastroenteritis
 - Communicating the presence of suspected cases to Infection Preventionists
 - Promoting increased adherence to hand hygiene, particularly the use of soap and water after contact with symptomatic patients
 - Enhanced environmental cleaning and disinfection

- Promptly initiate investigations
 - Collection of clinical and epidemiological information
 - Obtain clinical samples

Infection Control: Patient Isolation or Cohorting



- ❑ In healthcare settings where risk of transmission is high, use of isolation precautions is often the most effective means of interrupting transmission
- ❑ CONTACT PRECAUTIONS – single occupancy room with a dedicated bathroom, strict adherence to hand hygiene, wear gloves and gown upon room entry
 - Use Contact Precautions for a minimum of 48 hours after the resolution of symptoms
 - Symptomatic patients may be cohorted together
 - Exclude ill staff members and food handlers in healthcare facilities for a minimum of 48 hours following resolution of their symptoms
 - Exclude non-essential personnel and visitors

Infection Control: Hand Hygiene

- ❑ Wash with soap and water after contact with symptomatic patients
 - For all other indications, refer to the 2002 Guideline for Hand Hygiene*
- ❑ Alcohol-based hand sanitizers
 - Currently available products appear to be relatively ineffective against norovirus
 - Consider using FDA-compliant alcohol-based hand sanitizers for other indications (e.g., before contact with NV patient)*



*CDC HICPAC Guideline for Hand Hygiene in Health-Care Settings:
<http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf>

Infection Control: Environmental Cleaning and Disinfection

- ❑ The use of chemical cleaning and disinfecting agents are key in interrupting norovirus spread from contaminated environmental surfaces.
- ❑ Increase the frequency of cleaning and disinfection of patient care areas and frequently touched surfaces
e.g., increase ward/unit level cleaning to twice daily, with frequently touched surfaces cleaned and disinfected three times daily
- ❑ Use commercial cleaning and disinfection products registered with the U.S. Environmental Protection Agency (e.g., sodium hypochlorite (bleach) solution, hydrogen peroxide products, etc.)
http://www.epa.gov/pesticides/antimicrobials/list_g_norovirus.pdf
- ❑ It is critical to follow manufacturer instructions for methods of application, amount, dilution, and contact time

Infection Control: Other Considerations

- ❑ To reduce transmission, and depending on the magnitude of the outbreak, cohort staff to care for patients who are
 - asymptomatic unexposed
 - asymptomatic, potentially exposed
 - symptomatic
- ❑ Remove communal or shared food items for staff or patients for the duration of the outbreak
- ❑ Group activities for patients may need to be suspended; minimize patient movements within a patient care area to help control transmission



Surveillance for Norovirus Cases

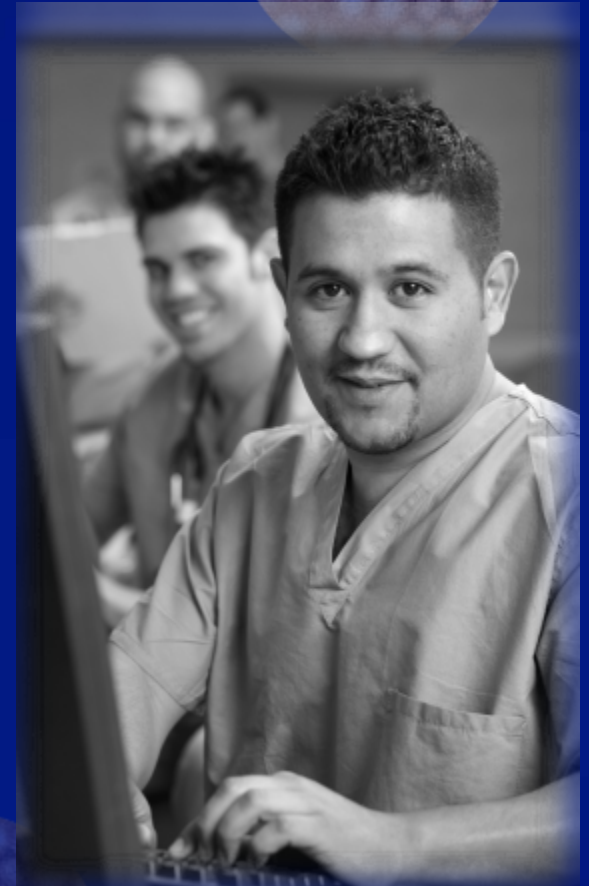
- ❑ Units can use a “line list” to track symptomatic staff and patients
- ❑ During an outbreak, collect key information to assist with controlling the outbreak and to inform local/state health departments on outbreak details
- ❑ Suggested line list elements
 - Case (staff/patient) identifier
 - Case location
 - Symptoms
 - Outcome / Date of Resolution
 - Diagnostics submitted



Reporting Outbreaks

Internal Communication

- ❑ Report gastroenteritis outbreaks (e.g., 2 or more suspected or confirmed cases among staff or patients) to infection control units
- ❑ Outbreaks should also be reported to clinical management
- ❑ Important to include communications, laboratory, environmental services, admitting, occupational health departments



Reporting Outbreaks

External Reporting

- ❑ Report norovirus outbreaks to your local, county, or state health department
- ❑ In most states, all outbreaks of public health significance are reportable to the state health department
- ❑ Health departments enter norovirus outbreak data (among other pathogens) into National Outbreak Reporting System (NORS) → Centers for Disease Control and Prevention (CDC)



Summary: Management of Norovirus Outbreaks

- ❑ Create awareness of concurrent norovirus outbreaks in the community/ other local healthcare facilities
- ❑ Detect and confirm suspected norovirus cases rapidly
- ❑ During outbreaks, implement
 - Contact Precautions,
 - enhanced hand hygiene,
 - environmental infection control measures,
 - exclusion of ill staff from work for a minimum of 48 hrs after symptom resolution
 - surveillance for new and resolving cases,
- ❑ Develop a communication plan during outbreaks to include key departments and services
- ❑ Consult with and report outbreak to local/state health departments

Additional Resources

- ❑ **Norovirus in healthcare settings**

<http://www.cdc.gov/HAI/organisms/norovirus.html>

- ❑ **CDC HICPAC Guideline for the Prevention and Control of Norovirus Gastroenteritis Outbreaks in Healthcare Settings**

<http://www.cdc.gov/hicpac/pdf/norovirus/Norovirus-Guideline-2011.pdf>

- ❑ **Updated Norovirus Outbreak Management and Disease Prevention Guidelines**

http://www.cdc.gov/mmwr/preview/mmwrhtml/rr6003a1.htm?s_cid=rr6003a1_e

- ❑ **General information on norovirus**

<http://www.cdc.gov/ncidod/dvrd/revb/gastro/norovirus.htm>

For more information please contact Centers for Disease Control and Prevention

1600 Clifton Road NE, Atlanta, GA 30333

Telephone: 1-800-CDC-INFO (232-4636)/TTY: 1-888-232-6348

E-mail: cdcinfo@cdc.gov

Web: <http://www.cdc.gov>

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.



U.S. Department of Health and Human Services

Centers for Disease Control and Prevention

GUIDELINE FOR THE PREVENTION AND CONTROL OF NOROVIRUS GASTROENTERITIS OUTBREAKS IN HEALTHCARE SETTINGS

Taranisia MacCannell, PhD, MSc¹; Craig A. Umscheid, MD, MSCE²; Rajender K. Agarwal, MD, MPH²; Ingi Lee, MD, MSCE²; Gretchen Kuntz, MSW, MSLIS²; Kurt B. Stevenson, MD, MPH³ and the Healthcare Infection Control Practices Advisory Committee (HICPAC)⁴

¹ Division of Healthcare Quality Promotion
Centers for Disease Control and Prevention
Atlanta, GA

² Center for Evidence-based Practice
University of Pennsylvania Health System
Philadelphia, PA

³ Division of Infectious Diseases
The Ohio State University,
Columbus, OH



4 Healthcare Infection Control Practices Advisory Committee (HICPAC)

CHAIRMAN

BRENNAN, Patrick J., MD
Chief Medical Officer
Division of Infectious Diseases
University of Pennsylvania Health System
Philadelphia, PA

MEMBERS

BRATZLER, Dale, DO, MPH
President and CEO
Oklahoma Foundation for Medical Quality
Oklahoma City, OK

BURNS, Lillian A., MT, MPH
Director Infection Control/Epidemiology
Staten Island University Hospital
Staten Island, NY

ELWARD, Alexis, MD
Assistant Professor, Pediatrics Infectious
Diseases
Washington University School of Medicine
St. Louis, MO

HUANG, Susan, MD, MPH
UC Irvine School of Medicine
Division of Infectious Diseases
Orange, CA

LUNDSTROM, Tammy, MD, JD
Chief Medical Officer
Providence Hospital
Southfield, MI

MCCARTER, Yvette S., PhD
Director, Clinical Microbiology
Laboratory
Department of Pathology
University of Florida Health Science
Center
Jacksonville, FL

MURPHY, Denise M. MPH, RN, CIC
Vice President, Quality and Patient Safety
Main Line Health System
Bryn Mawr, PA

EXECUTIVE SECRETARY

BELL, Michael R., MD
Deputy Director
Division of Healthcare Quality Promotion
Centers for Disease Control and Prevention
Atlanta, GA

OLMSTED, Russell N. MPH
Epidemiologist
Infection Control Services
St. Joseph Mercy Health System
Ann Arbor, MI

OSTROFF, Stephen, M.D.
Director, Bureau of Epidemiology
Pennsylvania Department of Health
Harrisburg, PA

PEGUES, David Alexander, MD
Professor of Medicine, Hospital Epidemiologist
David Geffen School of Medicine at UCLA
Los Angeles, CA

PRONOVOST, Peter J. M.D., Ph.D., F.C.C.M.
Director, Johns Hopkins Quality and Safety
Research Group
Department of Anesthesiology & Critical Care
Medicine
Johns Hopkins University
Baltimore, Maryland

SCHECTER, William, P., MD
Professor of Surgery
Department of Surgery
San Francisco General Hospital
San Francisco, CA

SOULE, Barbara M., RN, MPA, CIC
Practice Leader
Infection Prevention and Control Services
Joint Commission Resources/Joint
Commission International
Olympia, Washington

EX-OFFICIO MEMBERS

Agency for Healthcare Research and Quality (AHRQ)

BAINE, William B., MD
Senior Medical Advisor
Center for Outcomes and Evidence
Rockville, MD

Center for Medicare & Medicaid Services (CMS)

MILLER, Jeannie, RN, MPH
Deputy Director, Clinical Standards Group
Baltimore, MD

Food and Drug Administration (FDA)

MURPHEY, Sheila A., MD
Infection Control Devices Branch
Division of Anesthesiology, General Hospital
Infection Control Dental Devices
Center for Devices and Radiology Health
Rockville, MD

Health Resources and Services Administration (HRSA)

MOORE, Paul D., Ph.D
Senior Health Policy Advisor
Health Resources and Services Administration
U.S. Department of Health and Human Services
Office of Rural Health Policy
Rockville, MD

National Institutes of Health (NIH)

HENDERSON, David, MD
Deputy Director for Clinical Care
Associate Director for Hospital Epidemiology
and Quality Improvement
NIH Clinical Center
Bethesda, MD

Department of Veterans Affairs (VA)

ROSELLE, Gary A., MD
National Program Director, Infectious Diseases
VA Central Office
Cincinnati VA Medical Center
Cincinnati, OH

LIAISONS

Advisory Council for the Elimination of Tuberculosis (ACET)

STRICOF, Rachel L., MPH
Albany, New York

American College of Occupational and Environmental Medicine

RUSSI, Mark, MD, MPH
Professor of Medicine
Yale University School of Medicine
Director, Occupational Health
Yale-New Haven Hospital
New Haven, CT

American Health Care Assn (AHCA)

FITZLER, Sandra L., RN
Senior Director of Clinical Services
Washington, DC

American Hospital Association (AHA)

SCHULMAN, Roslyne, MHA, MBA
Senior Associate Director, Policy Development
Washington, DC

Association of periOperative Registered Nurses (AORN)

BLANCHARD, Joan C., RN, BSN, MSS, CNOR, CIC
Denver, CO

Association of Professionals of Infection Control and Epidemiology, Inc. (APIC)

DeBAUN, Barbara, MSN, RN, CIC
San Francisco, CA

Council of State and Territorial Epidemiologists (CSTE)

KAINER, Marion MD, MPH
Director, Hospital Infections and Antimicrobial Resistance Program
Tennessee Department Health
Nashville, TN

Consumers Union

MCGIFFERT, Lisa
Senior Policy Analyst on Health Issues
Project Director, Stop Hospital Infections Organization
Yonkers, NY

Infectious Diseases Society of America (IDSA)

HUSKINS, W. Charles M.D., M.Sc.
Division of Pediatric Infectious Diseases
Assistant Professor of Pediatrics
College of Medicine
Rochester, MN

Public Health Agency of Canada

PATON, Shirley, RN, MN
Senior Technical Advisor Healthcare Acquired Infections
Center for Communicable Diseases and Infection Control
Ottawa, Ontario

Society for Healthcare Epidemiology of America (SHEA)

MARAGAKIS, Lisa, MD
Assistant Professor of Medicine
John Hopkins Medical Institutions
Baltimore, MD

Society of Critical Care Medicine

William A. Brock, MD, FCCM, FCCP, FACP
Mount Prospect, IL

Society of Hospital Medicine

SAINT, Sanjay, MD, MPH
Director, Ann Arbor VA Medical Center/University of Michigan Patient Safety Enhancement Program
Ann Arbor VA Medical Center
Ann Arbor, Michigan

The Joint Commission

WISE, Robert A., MD
Vice President
Division of Standards & Survey Methods
Oakbrook Terrace, IL

Acknowledgement

HICPAC thanks the following members who served on the HICPAC Norovirus Gastroenteritis Guideline writing group during the guideline development process: Alexis Elward and Keith M. Ramsey.

HICPAC thanks the following experts for reviewing a draft of this guideline: John Boyce, Hospital of St. Raphael, New Haven, CT; Aron Hall, NCIRD, CDC; L. Clifford McDonald, DHQP, CDC; and Umesh Parashar, NCIRD, CDC. The opinions of the reviewers might not be reflected in all of the recommendations contained in this document.

Disclosure of Financial Interests and Relationships

The authors T.M., C.A.U., R.K.A., I.L., G.K., and K.B.S. report no actual or potential conflicts of interest. C.A.U., R.K.A. and I.L. received funding from the CDC to support the guideline development process.

Table of Contents

Abbreviations	7
I. Executive Summary	10
II. Summary of Recommendations.....	11
III. Implementation and Audit	18
IV. Recommendations for Further Research	19
V. Background	20
VI. Scope and Purpose.....	22
VII. Methods	22
VIII. Evidence Review.....	27
References	43

Abbreviations

AIDS	Acquired immune deficiency syndrome
BAS	Basic science study
°C	Celsius
CaCV	Calicivirus
CCU	Cardiac/coronary care unit
CDC	Centers for Disease Control and Prevention
CI	Confidence interval
CICU	Cardiac/coronary intensive care unit
CSTE	Council of State and Territorial Epidemiologists
DES	Descriptive study
DHQP	Division of Healthcare Quality Promotion
DIAG	Diagnostic study
DNA	Deoxyribonucleic acid
ECL	Electrochemiluminescence
EFORS	Electronic Foodborne Outbreak Reporting System
EIA	Enzyme immunoassay
ELISA	Enzyme-linked immunoassay
EM	Electron microscopy
EPA	Environmental Protection Agency
FBDSS	Foodborne Disease Outbreak Surveillance System
FCV	Feline calicivirus
FDA	Food and Drug Administration
FN	False negative
FP	False positive
GRADE	Grading of Recommendations Assessment, Development, and Evaluation
HBGA	Histo-blood group antigen

HICPAC	Healthcare Infection Control Practices Advisory Committee
HIV	Human immunodeficiency virus
Km	Kilometer
LUX	Light-upon-extension
MI	Milliliter
MMWR	Morbidity and Mortality Weekly Report
MNV	Murine norovirus
N/A	Not applicable
NASBA	Nucleic acid sequence-based amplification
NCIRD	National Center for Immunization and Respiratory Diseases
NIH	National Institutes of Health
NLV	Norwalk-like virus
No	Number
NORS	National Outbreak Reporting System
NPV	Negative predictive value
OBS	Observational study
OR	Odds ratio
ORF	Open reading frame
P	P value
PCR	Polymerase chain reaction
PPE	Personal protective equipment
PPM	Part per million
PPV	Positive predictive value
RCT	Randomized controlled trial
RHD	Rapid humidifying device
RIA	Radioimmunoassay
RF	Reduction factor

RR	Relative risk
RT	Room temperature
RT-LAMP	Reverse transcription loop-mediated amplification assay
RT-PCR	Reverse transcriptase polymerase chain reaction
SD	Standard deviation
SPIEM	Solid-phase immune electron microscopy
SR	Systematic review
SRFV	Small round featureless virus
SRSV	Small round structured virus
TCID	Tissue culture infective dose
TE	Transcriptional enhancement
TEM	Transmission electron microscopy
TN	True negative
TP	True positive
UV	Ultraviolet
Vs	Versus

I. Executive Summary

Norovirus gastroenteritis infections and outbreaks have been increasingly described and reported in both non-healthcare and healthcare settings during the past several years. In response, several states have developed guidelines to assist both healthcare institutions and communities on preventing the transmission of norovirus infections and helped develop the themes and key questions to answer through an evidence-based review. This guideline addresses prevention and control of norovirus gastroenteritis outbreaks in healthcare settings. The guideline also includes specific recommendations for implementation, performance measurement, and surveillance. Recommendations for further research are provided to address knowledge gaps identified during the literature review in the prevention and control of norovirus gastroenteritis outbreaks. Guidance for norovirus outbreak management and disease prevention in non-healthcare settings can be found at <http://www.cdc.gov/mmwr/pdf/rr/rr6003.pdf>.

This document is intended for use by infection prevention staff, physicians, healthcare epidemiologists, healthcare administrators, nurses, other healthcare providers, and persons responsible for developing, implementing, and evaluating infection prevention and control programs for healthcare settings across the continuum of care. The guideline can also be used as a resource for societies or organizations that wish to develop more detailed implementation guidance for prevention and control of norovirus gastroenteritis outbreaks for specialized settings or populations.

To evaluate the evidence on preventing and controlling norovirus gastroenteritis outbreaks in healthcare settings, published material addressing three key questions were examined:

1. What host, viral, or environmental characteristics increase or decrease the risk of norovirus infection in healthcare settings?
2. What are the best methods to identify an outbreak of norovirus gastroenteritis in a healthcare setting?
3. What interventions best prevent or contain outbreaks of norovirus gastroenteritis in the healthcare setting?

Explicit links between the evidence and recommendations are available in the [Evidence Review](#) in the body of the guideline and [Evidence Tables](#) and [GRADE Tables](#) in the [Appendices](#). **It is important to note that the Category I recommendations are all considered strong and should be implemented;** it is only the *quality* of the evidence underlying the recommendation that distinguishes between levels A and B. Category IC recommendations are required by state or federal regulation and may have any level of supporting evidence. The categorization scheme used in this guideline is presented in Table 1: [Summary of Recommendations](#) and described further in the [Methods](#) section. The [Implementation and Audit](#) section includes a prioritization of recommendations (i.e., high-priority recommendations that are essential for every healthcare facility) in order to provide facilities more guidance on implementation of these guidelines. A list of recommended performance measures that can potentially be used for reporting purposes is also included.

Evidence-based recommendations were cross-checked with those from other guidelines identified in an initial systematic search. Recommendations from other guidelines on topics not directly addressed by this systematic review of the evidence were included in the [Summary of Recommendations](#) if they were deemed critical to the target users of this guideline. Unlike recommendations informed by the search of primary studies, these recommendations are stated independently of a key question.

The [Summary of Recommendations](#) includes recommendations organized into the following categories: 1) Patient Cohorting and Isolation Precautions, 2) Hand Hygiene, 3) Patient Transfer and Ward Closure, 4) Indirect Patient Care Staff - Food Handlers in Healthcare, 5) Diagnostics, 6) Personal Protective Equipment, 7) Environmental Cleaning, 8) Staff Leave and Policy, 9) Visitors, 10) Education, 11) Active Case-finding, and 12) Communication and Notification.

Areas for further research identified during the evidence review are outlined in the Recommendations for Further Research. This section includes gaps that were identified during the literature review where specific recommendations could not be supported because of the absence of available information that matched the inclusion criteria for GRADE. These recommendations provide guidance for new research or methodological approaches that should be prioritized for future studies

Readers who wish to examine the primary evidence underlying the recommendations are referred to the Evidence Review in the body of the guideline, and the Evidence and GRADE Tables in the Appendices. The Evidence Review includes narrative summaries of the data presented in the Evidence and GRADE Tables. The Evidence Tables include all study-level data used in the guideline, and the GRADE Tables assess the overall quality of evidence for each question. The Appendices also contain a defined search strategy that will be used for periodic reviews to ensure that the guideline is updated as new information becomes available.

II. Summary of Recommendations

Table 1. HICPAC Categorization Scheme for Recommendations	
Category IA	A strong recommendation supported by high to moderate quality evidence suggesting net clinical benefits or harms.
Category IB	A strong recommendation supported by low-quality evidence suggesting net clinical benefits or harms, or an accepted practice (e.g., aseptic technique) supported by low to very low-quality evidence.
Category IC	A strong recommendation required by state or federal regulation.
Category II	A weak recommendation supported by any quality evidence suggesting a tradeoff between clinical benefits and harms.
Recommendation for further research	An unresolved issue for which there is low to very low-quality evidence with uncertain tradeoffs between benefits and harms.

*Please refer to Methods Section (p.23) and Umscheid et al. Updating the Guideline Methodology of the Healthcare Infection Control Practices Advisory Committee (HICPAC) (<http://www.cdc.gov/hicpac/guidelineMethod/guidelineMethod.html>) for the process used to grade quality of evidence and implications of category designation

**Key questions are described within the Evidence Review Section (p.31)

PATIENT COHORTING AND ISOLATION PRECAUTIONS

1. Avoid exposure to vomitus or diarrhea. Place patients on Contact Precautions in a single occupancy room if they have symptoms consistent with norovirus gastroenteritis. **(Category IB)** (Key Question 1.A.1)
 - 1a. When patients with norovirus gastroenteritis cannot be accommodated in single occupancy rooms, efforts should be made to separate them from asymptomatic patients. Dependent upon facility characteristics, approaches for cohorting patients during outbreaks may include placing patients in multi-occupancy rooms, or designating patient care areas or contiguous sections within a facility for patient cohorts. **(Category IB)** (Key Question 3C.4.b)
2. During outbreaks, place patients with norovirus gastroenteritis on Contact Precautions for a minimum of 48 hours after the resolution of symptoms to prevent further exposure of susceptible patients **(Category IB)** (Key Question 3.C.4.a)
 - 2a. Consider longer periods of isolation or cohorting precautions for complex medical patients (e.g., those with cardiovascular, autoimmune, immunosuppressive, or renal disorders) as they can experience protracted episodes of diarrhea and prolonged viral shedding. Patients with these or other comorbidities have the potential to relapse, and facilities may choose longer periods of isolation based on clinical judgment. **(Category II)** (Key Question 1.A.2.a)
 - 2b. Consider extending the duration of isolation or cohorting precautions for outbreaks among infants and young children (e.g., under 2 years), even after resolution of symptoms, as there is a potential for prolonged viral shedding and environmental contamination. Among infants, there is evidence to consider extending contact precautions for up to 5 days after the resolution of symptoms. **(Category II)** (Key Question 3.A.1)
3. Further research is needed to understand the correlation between prolonged shedding of norovirus and the risk of infection to susceptible patients **(No recommendation/unresolved issue)** (Key Question 3.A.2)
4. Consider minimizing patient movements within a ward or unit during norovirus gastroenteritis outbreaks. **(Category II)** (Key Question 3.C.4.c)
 - 4a. Consider restricting symptomatic and recovering patients from leaving the patient-care area unless it is for essential care or treatment to reduce the likelihood of environmental contamination and transmission of norovirus in unaffected clinical areas. **(Category II)** (Key Question 3.C.4.c.1)
5. Consider suspending group activities (e.g., dining events) for the duration of a norovirus outbreak. **(Category II)** (Key Question 3.C.4.d)
6. Staff who have recovered from recent suspected norovirus infection associated with an outbreak may be best suited to care for symptomatic patients until the outbreak resolves. **(Category II)**(Key Question 3.C.5.b)

HAND HYGIENE

7. Actively promote adherence to hand hygiene among healthcare personnel, patients, and visitors in patient care areas affected by outbreaks of norovirus gastroenteritis. **(Category IB)** (Key Question 3.C.1.a)
8. During outbreaks, use soap and water for hand hygiene after providing care or having contact with patients suspected or confirmed with norovirus gastroenteritis. **(Category IB)** (Key Question 3.C.1.b)
 - 8a. For all other hand hygiene indications (e.g., before having contact with norovirus patients) refer to the 2002 HICPAC Guideline for Hand Hygiene in Health-Care Settings (<http://www.cdc.gov/mmwr/PDF/rr/rr51116.pdf>), which includes the indications for use of FDA-compliant alcohol-based hand sanitizer. **(Category IB)** (Key Question 3.C.1.b.1)
 - 8a.1 Consider ethanol-based hand sanitizers (60-95%) as the preferred active agent compared to other alcohol or non-alcohol based hand sanitizer products during outbreaks of norovirus gastroenteritis. **(Category II)** (Key Question 3.C.1.b.2)
 - 8b. Further research is required to directly evaluate the efficacy of alcohol-based hand sanitizers against human strains of norovirus, or against a surrogate virus with properties convergent with human strains of norovirus. **(No recommendation/unresolved issue)** (Key Question 3.C.1.b.3)
9. More research is required to evaluate the virucidal capabilities of alcohol-based as well as non-alcohol based hand sanitizers against norovirus. **(No recommendation/unresolved issue)** (Key Question 3.C.12.e.4)

PATIENT TRANSFER AND WARD CLOSURE

10. Consider the closure of wards to new admissions or transfers as a measure to attenuate the magnitude of an outbreak of norovirus gastroenteritis. The threshold for ward closure varies and depends on risk assessments by infection prevention personnel and facility leadership. **(Category II)** (Key Question 3.C.6)
11. Consider limiting transfers to those for which the receiving facility is able to maintain Contact Precautions; otherwise, it may be prudent to postpone transfers until patients no longer require Contact Precautions. During outbreaks, medically suitable individuals recovering from norovirus gastroenteritis can be discharged to their place of residence. **(Category II)** (Key Question 3.C.11)
12. Implement systems to designate patients with symptomatic norovirus and to notify receiving healthcare facilities or personnel prior to transfer of such patients within or between facilities. **(Category IC)**

INDIRECT PATIENT CARE STAFF – FOOD HANDLERS IN HEALTHCARE

13. To prevent food-related outbreaks of norovirus gastroenteritis in healthcare settings, food handlers must perform hand hygiene prior to contact with or the preparation of food items and beverages

(<http://www.fda.gov/Food/FoodSafety/RetailFoodProtection/FoodCode/default.htm>). **(Category IC)** (Key Question 1.C.3.a)

14. Personnel who work with, prepare or distribute food must be excluded from duty if they develop symptoms of acute gastroenteritis. Personnel should not return to these activities until a minimum of 48 hours after the resolution of symptoms or longer as required by local health regulations (<http://www.fda.gov/Food/FoodSafety/RetailFoodProtection/FoodCode/default.htm>). **(Category IC)** (Key Question 1.C.3.b)
15. Remove all shared or communal food items for patients or staff from clinical areas for the duration of the outbreak. **(Category IB)** (Key Question 3.B.2)

DIAGNOSTICS

16. Consider the development and adoption of facility policies to enable rapid clinical and virological confirmation of suspected cases of symptomatic norovirus infection while implementing prompt control measures to reduce the magnitude of a potential norovirus outbreak. **(Category II)** (Key Question 1.C.1)
17. In the absence of clinical laboratory diagnostics or in the case of delay in obtaining laboratory results, use Kaplan's clinical and epidemiologic criteria to identify a norovirus gastroenteritis outbreak (see Table 4 for Kaplan's criteria). **(Category IA)** (Key Question 2.A.1)
18. Further research is needed to compare the Kaplan criteria with other early detection criteria for outbreaks of norovirus gastroenteritis in healthcare settings, and to assess whether additional clinical or epidemiologic criteria can be applied to detect norovirus clusters or outbreaks in healthcare settings. **(No recommendation/unresolved issue)** (Key Question 2.A.1)
19. Consider submitting stool specimens as early as possible during a suspected norovirus gastroenteritis outbreak and ideally from individuals during the acute phase of illness (within 2-3 days of onset). It is suggested that healthcare facilities consult with state or local public health authorities regarding the types of and number of specimens to obtain for testing. **(Category II)** (Key Question 2.B)
20. Use effective laboratory diagnostic protocols for testing of suspected cases of viral gastroenteritis (e.g., refer to the Centers for Disease Control and Prevention (CDC)'s most current recommendations for norovirus diagnostic testing at <http://www.cdc.gov/mmwr/pdf/rr/rr6003.pdf>). **(Category IB)** (Key Question 2.C)
21. Routine collecting and processing of environmental swabs during a norovirus outbreak is not required. When supported by epidemiologic evidence, environmental sampling can be considered useful to confirm specific sources of contamination during investigations. **(Category II)**
22. Specimens obtained from vomitus can be submitted for laboratory identification of norovirus when fecal specimens are unavailable. Testing of vomitus as compared to fecal specimens can be less sensitive due to lower detectable viral concentrations. **(Category II)**

PERSONAL PROTECTIVE EQUIPMENT

23. If norovirus infection is suspected, adherence to PPE use according to Contact and Standard Precautions is recommended for individuals entering the patient care area (i.e., gowns and gloves upon entry) to reduce the likelihood of exposure to infectious vomitus or fecal material. **(Category IB)** (Key Question 1.C.4)
24. Use a surgical or procedure mask and eye protection or a full face shield if there is an anticipated risk of splashes to the face during the care of patients, particularly among those who are vomiting. **(Category IB)** (Key Question 3.C.2.a)
25. More research is needed to evaluate the utility of implementing Universal Gloving (e.g., routine use of gloves for all patient care) during norovirus outbreaks. **(No recommendation/unresolved issue)**

ENVIRONMENTAL CLEANING

26. Perform routine cleaning and disinfection of frequently touched environmental surfaces and equipment in isolation and cohorted areas, as well as high-traffic clinical areas. Frequently touched surfaces include, but are not limited to, commodes, toilets, faucets, hand/bedrailing, telephones, door handles, computer equipment, and kitchen preparation surfaces. **(Category IB)** (Key Question 3.B.1)
27. Clean and disinfect shared equipment between patients using EPA-registered products with label claims for use in healthcare. Follow the manufacturer's recommendations for application and contact times. The EPA lists products with activity against norovirus on their website (<http://www.epa.gov/oppad001/chemregindex.htm>). **(Category IC)** (Key Question 3.C.12.a)
28. Increase the frequency of cleaning and disinfection of patient care areas and frequently touched surfaces during outbreaks of norovirus gastroenteritis (e.g., increase ward/unit level cleaning to twice daily to maintain cleanliness, with frequently touched surfaces cleaned and disinfected three times daily using EPA-approved products for healthcare settings). **(Category IB)** (Key Question 3.C.12.b.1)
29. Clean and disinfect surfaces starting from the areas with a lower likelihood of norovirus contamination (e.g., tray tables, counter tops) to areas with highly contaminated surfaces (e.g., toilets, bathroom fixtures). Change mop heads when a new bucket of cleaning solution is prepared, or after cleaning large spills of emesis or fecal material. **(Category IB)** (Key Question 3.C.12.b.2)
30. Consider discarding all disposable patient-care items and laundering unused linens from patient rooms after patients on isolation for norovirus gastroenteritis are discharged or transferred. Facilities can minimize waste by limiting the number of disposable items brought into rooms/areas on Contact Precautions. **(Category II)** (Key Question 3.C.12.c.1)
31. No additional provisions for using disposable patient service items such as utensils or dishware are suggested for patients with symptoms of norovirus infection. Silverware and dishware may undergo normal processing and cleaning using standard procedures. **(Category II)** (Key Question 3.C.12.c.2)
32. Use Standard Precautions for handling soiled patient-service items or linens, including the use of appropriate PPE. **(Category IB)** (Key Question 3.C.12.c.3)

33. Consider avoiding the use of upholstered furniture and rugs or carpets in patient care areas, as these objects are difficult to clean and disinfect completely. If this option is not possible, immediately clean soilage, such as emesis or fecal material, from upholstery, using a manufacturer-approved cleaning agent or detergent. Opt for seating in patient-care areas that can withstand routine cleaning and disinfection. **(Category II)** (Key Question 3.C.12.d.1)
34. Consider steam cleaning of upholstered furniture in patient rooms upon discharge. Consult with manufacturer's recommendations for cleaning and disinfection of these items. Consider discarding items that cannot be appropriately cleaned/disinfected. **(Category II)**(Key Question 3.C.12.d.2)
35. During outbreaks, change privacy curtains when they are visibly soiled and upon patient discharge or transfer. **(Category IB)** (Key Question 3.C.12.d.3)
36. Handle soiled linens carefully, without agitating them, to avoid dispersal of virus. Use Standard Precautions, including the use of appropriate PPE (e.g., gloves and gowns), to minimize the likelihood of cross-contamination. **(Category IB)** (Key Question 3.C.12.d.4)
37. Double bagging, incineration, or modifications for laundering are not indicated for handling or processing soiled linen. **(Category II)** (Key Question 3.C.12.d.5)
38. Clean surfaces and patient equipment prior to the application of a disinfectant. Follow the manufacturer's recommendations for optimal disinfectant dilution, application, and surface contact time with an EPA-approved product with claims against norovirus. **(Category IC)** (Key Question 3.C.12.e.1)
39. More research is required to clarify the effectiveness of cleaning and disinfecting agents against norovirus, either through the use of surrogate viruses or the development of human norovirus culture system. **(No recommendation/unresolved issue)** (Key Question 3.C.12.e.2)
40. More research is required to clarify the effectiveness and reliability of fogging, UV irradiation, and ozone mists to reduce norovirus environmental contamination. **(No recommendation/unresolved issue)** (Key Question 3.C.12.e.3)
41. Further research is required to evaluate the utility of medications that might attenuate the duration and severity of norovirus illness. **(No recommendation/unresolved issue)** (Key Question 3.D)

STAFF LEAVE AND POLICY

42. Develop and adhere to sick leave policies for healthcare personnel who have symptoms consistent with norovirus infection. **(Category IB)** (Key Question 3.C.3)
 - 42a. Exclude ill personnel from work for a minimum of 48 hours after the resolution of symptoms. Once personnel return to work, the importance of performing frequent hand hygiene should be reinforced, especially before and after each patient contact. **(Category IB)** (Key Question 3.C.3.a)
43. Establish protocols for staff cohorting in the event of an outbreak of norovirus gastroenteritis. Ensure staff care for one patient cohort on their ward and do not move between patient cohorts (e.g., patient cohorts may include symptomatic, asymptomatic exposed, or asymptomatic unexposed patient groups). **(Category IB)**(Key Question 3.C.5.a)

44. Exclude non-essential staff, students, and volunteers from working in areas experiencing outbreaks of norovirus gastroenteritis. **(Category IB)** (Key Question 3.C.5.c)

VISITORS

45. Establish visitor policies for acute gastroenteritis (e.g., norovirus) outbreaks. **(Category IB)** (Key Question 3.C.7.a)
46. Restrict non-essential visitors from affected areas of the facility during outbreaks of norovirus gastroenteritis. **(Category IB)** (Key Question 3.C.7.b)
- 46a. For those affected areas where it is necessary to have continued visitor privileges during outbreaks, screen and exclude visitors with symptoms consistent with norovirus infection and ensure that they comply with hand hygiene and Contact Precautions. **(Category IB)** (Key Question 3.C.7.b.1)

EDUCATION

47. Provide education to staff, patients, and visitors, including recognition of norovirus symptoms, preventing infection, and modes of transmission upon the recognition and throughout the duration of a norovirus gastroenteritis outbreak. **(Category IB)** (Key Question 3.C.8.a)
48. Consider providing educational sessions and making resources available on the prevention and management of norovirus before outbreaks occur, as part of annual trainings, and when sporadic cases are detected. **(Category II)** (Key Question 3.C.8.b)

ACTIVE CASE-FINDING

49. Begin active case-finding when a cluster of acute gastroenteritis cases is detected in the healthcare facility. Use a specified case definition, and implement line lists to track both exposed and symptomatic patients and staff. Collect relevant epidemiological, clinical, and demographic data as well as information on patient location and outcomes. **(Category IB)** (Key Question 3.C.9.a)

COMMUNICATION AND NOTIFICATION

50. Develop written policies that specify the chains of communication needed to manage and report outbreaks of norovirus gastroenteritis. Key stakeholders such as clinical staff, environmental services, laboratory administration, healthcare facility administration and public affairs, as well as state or local public health authorities, should be included in the framework. **(Category IB)** (Key Question 3.C.10)
- 50a. Provide timely communication to personnel and visitors when an outbreak of norovirus gastroenteritis is suspected and outline what policies and provisions need to be followed to prevent further transmission **(Category IB)** (Key Question 3.C.10.a)
51. As with all outbreaks, notify appropriate local and state health departments, as required by state and local public health regulations, if an outbreak of norovirus gastroenteritis is suspected. **(Category IC)** (Key Question 3.C.9.b)

III. Implementation and Audit

Prioritization of Recommendations

Category I recommendations in this guideline are all considered strong recommendations and should be implemented. If it is not feasible to implement all of these recommendations concurrently, e.g., due to differences in facility characteristics such as nursing homes and other non-hospital settings, priority should be given to the recommendations below. A limited number of Category II recommendations are included, and while these currently are limited by the strength of the available evidence, they are considered key activities in preventing further transmission of norovirus in healthcare settings.

PATIENT COHORTING AND ISOLATION PRECAUTIONS

1. Avoid exposure to vomitus or diarrhea. Place patients on Contact Precautions in a single occupancy room if they present with symptoms consistent with norovirus gastroenteritis. **(Category IB)** (Key Question 1.A.1)

HAND HYGIENE

8. During outbreaks, use soap and water for hand hygiene after providing care or having contact with patients suspected or confirmed with norovirus gastroenteritis. **(Category IB)** (Key Question 3.C.1.b)

PATIENT TRANSFER AND WARD CLOSURE

11. Consider limiting transfers to those for which the receiving facility is able to maintain Contact Precautions; otherwise, it may be prudent to postpone transfers until patients no longer require Contact Precautions. During outbreaks, medically suitable individuals recovering from norovirus gastroenteritis can be discharged to their place of residence. **(Category II)** (Key Question 3.C.11)

DIAGNOSTICS

17. In the absence of clinical laboratory diagnostics or in the case of delay in obtaining laboratory results, use Kaplan's clinical and epidemiologic criteria to identify a norovirus gastroenteritis outbreak. **(Category IA)** (Key Question 2.A.1)

ENVIRONMENTAL CLEANING

28. Increase the frequency of cleaning and disinfection of patient care areas and frequently touched surfaces during outbreaks of norovirus gastroenteritis (e.g., consider increasing ward/unit level cleaning to twice daily to maintain cleanliness, with frequently touched surfaces cleaned and disinfected three times daily using EPA-approved products for healthcare settings). **(Category IB)** (Key Question 3.C.12.b.1)

STAFF LEAVE AND POLICY

42. Develop and adhere to sick leave policies for healthcare personnel who have symptoms consistent with norovirus infection. **(Category IB)** (Key Question 3.C.3)

42a. Exclude ill personnel from work for a minimum of 48 hours after the resolution of symptoms. Once personnel return to work, the importance of performing frequent hand hygiene should be reinforced, especially before and after each patient contact. **(Category IB)** (Key Question 3.C.3.a)

43. Establish protocols for staff cohorting in the event of an outbreak of norovirus gastroenteritis. Ensure staff care for one patient cohort on their ward and do not move between patient cohorts (e.g., patient cohorts may include symptomatic, asymptomatic exposed, or asymptomatic unexposed patient groups). **(Category IB)**(Key Question 3.C.5.a)

COMMUNICATION AND NOTIFICATION

51. As with all outbreaks, notify appropriate local and state health departments, as required by state and local public health regulations, if an outbreak of norovirus gastroenteritis is suspected. **(Category IC)** (Key Question 3.C.9.b)

Performance Measures for Health Departments

Use of performance measures may assist individual healthcare facilities, as well as local and state health departments to recognize increasing and peak activities of norovirus infection, and may allow for prevention and awareness efforts to be implemented rapidly or as disease incidence escalates. Evaluate fluctuations in the incidence of norovirus in healthcare settings using the National Outbreak Reporting System (NORS) (<http://www.cdc.gov/outbreaknet/nors/>). This system monitors the reporting of waterborne, foodborne, enteric person-to-person, and animal contact-associated disease outbreaks to CDC by state and territorial public health agencies. This surveillance program was previously used only for reporting foodborne disease outbreaks, but it has now expanded to include all enteric outbreaks, regardless of mode of transmission. Additionally, CDC is currently implementing a national surveillance system (CaliciNet) for genetic sequences of noroviruses; this system may also be used to measure changes in the epidemiology of healthcare-associated norovirus infections.

IV. Recommendations for Further Research

The literature review for this guideline revealed that many of the studies addressing strategies to prevent norovirus gastroenteritis outbreaks in healthcare facilities were not of sufficient quality to allow firm conclusions regarding the benefit of certain interventions. Future studies of norovirus gastroenteritis prevention in healthcare settings should include:

1. Analyses of the impact of specific or bundled infection control interventions,
2. Use of controls or comparison groups in both clinical and laboratory trials,
3. Comparisons of surrogate and human norovirus strains, focusing on the differences in their survival and persistence after cleaning and disinfection, and compare the natural history of disease in animal models to that in human norovirus infections,
4. Assessment of healthcare-focused risk factors (e.g the impact of isolation vs. cohorting practices, duration of isolation, hand hygiene policies during outbreaks of norovirus, etc.)
5. Statistically powerful studies able to detect small but significant effects of norovirus infection control strategies or interventions, and
6. Quantitative assessments of novel, and practical methods for effective cleaning and disinfection during norovirus outbreaks.

The following are specific areas in need of further research in order to make more precise prevention recommendations (see also recommendations under the category of No recommendation/unresolved issue in the Evidence Review):

Measurement and Case Detection

1. Assess the benefit of using the Kaplan criteria as an early detection tool for outbreaks of norovirus gastroenteritis in healthcare settings and examine whether the Kaplan criteria are differentially predictive of select strains of norovirus.

Host Contagiousness and Transmission

1. Determine correlations between prolonged shedding of norovirus after symptoms have subsided and the likelihood of secondary transmission of norovirus infection.
2. Assess the utility of medications that may attenuate the duration and severity of norovirus illness.
3. Determine the role of asymptomatic shedding (among recovered persons and carriers) in secondary transmission.
4. Evaluate the duration of protective immunity and other protective host factors, including histo-blood group antigens (HBGA) and secretor status.
5. Assess the contribution of water or food sources to outbreaks of norovirus gastroenteritis in healthcare settings.

Environmental Issues

1. Quantify the effectiveness of cleaning and disinfecting agents against norovirus or appropriate surrogates.
2. Evaluate effectiveness and reliability of novel environmental disinfection strategies such as fogging, UV irradiation, vapor-phase hydrogen peroxides, and ozone mists to reduce norovirus contamination.
3. Develop methods to evaluate norovirus persistence in the environment, with a focus on persistent infectivity.
4. Identify a satisfactory animal model for surrogate testing of norovirus properties and pathogenesis. Translate laboratory findings into practical infection prevention strategies.

Hygiene and Infection Control

1. Evaluate the effectiveness of FDA-approved hand sanitizers against norovirus or appropriate surrogates, including viral persistence after treatment with non-alcohol based products.
2. Assess the benefits and impact of implementing Universal Gloving practices during outbreaks of norovirus gastroenteritis

V. Background

Norovirus is the most common etiological agent of acute gastroenteritis and is often responsible for outbreaks in a wide spectrum of community and healthcare settings. These single-stranded RNA viruses belong to the family *Caliciviridae*, which also includes the genera Sapovirus, Lagovirus, and Vesivirus.¹ Illness is typically self-limiting, with acute symptoms of fever, nausea, vomiting, cramping, malaise, and diarrhea persisting for 2 to 5 days.^{2,3} Noteworthy sequelae of norovirus infection include hypovolemia and electrolyte imbalance, as well as more severe medical presentations such as hypokalemia and renal insufficiency. As most healthy children and adults experience relatively mild symptoms, sporadic cases and outbreaks may be undetected or underreported. However, it is estimated that norovirus may be the causative agent in over 23 million gastroenteritis cases every year in the United States, representing approximately 60% of all acute gastroenteritis cases.⁴ Based on pooled analysis, it is estimated that norovirus may lead to over 91,000 emergency room visits and 23,000 hospitalizations for severe diarrhea among children under the age of five each year in the United States.^{5,6}

Noroviruses are classified into five genogroups, with most human infections resulting from genogroups GI and GII.⁶ Over 80% of confirmed human norovirus infections are associated with genotype GII.4.^{7,8} Since 2002, multiple new variants of the GII.4 genotype have emerged and quickly become the predominant cause of human norovirus disease.⁹ As recently as late 2006, two new GII.4 variants were detected across the United States and resulted in a 254% increase in acute gastroenteritis outbreaks in 2006 compared to 2005.¹⁰ The increase in incidence was likely associated with potential increases in pathogenicity and transmissibility of, and depressed population immunity to these new strains.¹⁰ CDC conducts surveillance for foodborne outbreaks, including norovirus or norovirus-like outbreaks, through voluntary state and local health reports using the Foodborne Disease Outbreak Surveillance System (FBDSS). CDC summary data for 2001-2005 indicate that caliciviruses (CaCV), primarily norovirus, were responsible for 29% of all reported foodborne outbreaks, while in 2006, 40% of foodborne outbreaks were attributed to norovirus.¹¹ In 2009, the National Outbreak Reporting System (NORS) was launched by the CDC after the Council of State

and Territorial Epidemiologists (CSTE) passed a resolution to commit states to reporting all acute gastroenteritis outbreaks, including those that involve person-to-person or waterborne transmission.

Norovirus infections are seen in all age groups, although severe outcomes and longer durations of illness are most likely to be reported among the elderly.² Among hospitalized persons who may be immunocompromised or have significant medical comorbidities, norovirus infection can directly result in a prolonged hospital stay, additional medical complications, and, rarely, death.¹⁰ Immunity after infection is strain-specific and appears to be limited in duration to a period of several weeks, despite the fact that seroprevalence of antibody to this virus reaches 80-90% as populations transition from childhood to adulthood.² There is currently no vaccine available for norovirus and, generally, no medical treatment is offered for norovirus infection apart from oral or intravenous repletion of volume.²

Food or water can be easily contaminated by norovirus, and numerous point-source outbreaks are attributed to improper handling of food by infected food-handlers, or through contaminated water sources where food is grown or cultivated (e.g., shellfish and produce) (<http://www.cdc.gov/mmwr/pdf/rr/rr6003.pdf>) The ease of its transmission, with a very low infectious dose of <10 -100 virions, primarily by the fecal-oral route, along with a short incubation period (24-48 hours)^{12,13}, environmental persistence, and lack of durable immunity following infection, enables norovirus to spread rapidly through confined populations.⁶

Institutional settings such as hospitals and long-term care facilities commonly report outbreaks of norovirus gastroenteritis, which may make up over 50% of reported outbreaks.¹¹ However, cases and outbreaks are also reported in a wide breadth of community settings such as cruise ships, schools, day-care centers, and food services, such as hotels and restaurants. In healthcare settings, norovirus may be introduced into a facility through ill patients, visitors, or staff. Typically, transmission occurs through exposure to direct or indirect fecal contamination found on fomites, by ingestion of fecally-contaminated food or water, or by exposure to aerosols of norovirus from vomiting persons.^{2,6} Healthcare facilities managing outbreaks of norovirus gastroenteritis may experience significant costs relating to isolation precautions and PPE, ward closures, supplemental environmental cleaning, staff cohorting or replacement, and sick time.

The pathogenesis of human norovirus infection

The P2 subdomain of the viral capsid is the likely binding site of norovirus, and is the most variable region on the norovirus genome.¹⁴ The P2 ligand is the natural binding site with human HBGA, which may be the point of initial viral attachment.¹⁴ HBGA is found on the surfaces of red blood cells and is also expressed in saliva, in the gut, and in respiratory epithelia. The strength of the virus binding may be dependent on the human host HBGA receptor sites, as well as on the infecting strain of norovirus. Infection appears to involve the lamina propria of the proximal portion of the small intestine,¹⁵ yet the cascade of changes to the local environment is unknown.

Clinical diagnosis of norovirus gastroenteritis is common, and, under outbreak conditions, the Kaplan Criteria are often used to determine whether gastroenteritis clusters or outbreaks of unknown etiology are likely to be attributable to norovirus.¹⁶ These criteria are:

1. Submitted fecal specimens negative for bacterial and if tested, parasitic pathogens,
2. Greater than 50% of cases reporting vomiting as a symptom of illness,
3. Mean or median duration of illness ranging between 12 and 60 hours, and
4. Mean or median incubation period ranging between 24 and 48 hours.

The current standard for norovirus diagnostics is reverse transcriptase polymerase chain reaction (RT-PCR), but clinical laboratories may use commercial enzyme immunoassays (EIA), or electron microscopy (EM).⁶ ELISA and transmission electron microscopy (TEM) demonstrate high sensitivity but lower specificities against the RT-PCR gold standard. The use of enzyme-linked immunosorbent assays (ELISA) and EM together can improve the overall test characteristics—particularly test specificity.¹⁷ Improvements in PCR have included the development of multiple nucleotide probes to detect a spectrum of genotypes as

well as methods to improve detection of norovirus from dilute samples or low viral loads and those containing PCR-inhibitors.¹⁸ While the currently available diagnostic methods are capable, with differing degrees of sensitivity and specificity, of detecting the physical presence of human norovirus from a sample, its detection does not directly translate into information about residual infectivity.

A significant challenge to controlling the environmental spread of norovirus in healthcare and other settings is the paucity of data available on the ability of human strains of norovirus to persist and remain infective in environments after cleaning and disinfection.¹⁹ Identifying the physical and chemical properties of norovirus is limited by the fact that human strains are presently uncultivable *in vitro*. The majority of research evaluating the efficacy of both environmental and hand disinfectants against human norovirus over the past two decades has primarily utilized feline calicivirus (FCV) as a surrogate. It is still unclear whether FCV is an appropriate surrogate for human norovirus, with some research suggesting that human norovirus may exhibit more resistance to disinfectants than does FCV.²⁰ Newer research has identified and utilized a murine norovirus (MNV) surrogate, which exhibits physical properties and pathophysiology more similar to those of human norovirus.²⁰ Currently, the Environmental Protection Agency (EPA) offers a list of approved disinfectants demonstrating efficacy against FCV, and the Federal Drug Administration (FDA) is responsible for evaluating hand disinfectants with label-claims against FCV as a surrogate for human norovirus (among other epidemiologically significant pathogens). It is unknown whether there are variations of physical and chemical tolerances to disinfectants and other virucidal agents among the various human norovirus genotypes. Other research pathways are evaluating the efficacy of fumigants, such as vapor phase hydrogen peroxides, as well as fogging methods as virucidal mechanisms to eliminate norovirus from environmental surfaces.

VI. Scope and Purpose

This guideline provides recommendations for the prevention and control of norovirus gastroenteritis outbreaks in healthcare settings. All patient populations and healthcare settings have been included in the review of the evidence. The guideline also includes specific recommendations for implementation, performance measurement, and surveillance strategies. Recommendations for further research are also included to address the knowledge gaps relating to norovirus gastroenteritis outbreak prevention and management that were identified during the literature review.

To evaluate the evidence on preventing and managing norovirus gastroenteritis outbreaks, three key questions were examined and addressed:

1. What host, viral, or environmental characteristics increase or decrease the risk of norovirus infection in healthcare settings?
2. What are the best methods to identify an outbreak of norovirus gastroenteritis in a healthcare setting?
3. What interventions best prevent or contain outbreaks of norovirus gastroenteritis in the healthcare setting?

This document is intended for use by infection prevention staff, healthcare epidemiologists, healthcare administrators, nurses, other healthcare providers, and persons responsible for developing, implementing, and evaluating infection prevention and control programs for healthcare settings across the continuum of care. The guideline can also be used as a resource for professional societies or organizations that wish to develop guidance on prevention or management of outbreaks of norovirus gastroenteritis for specialized settings or populations.

VII. Methods

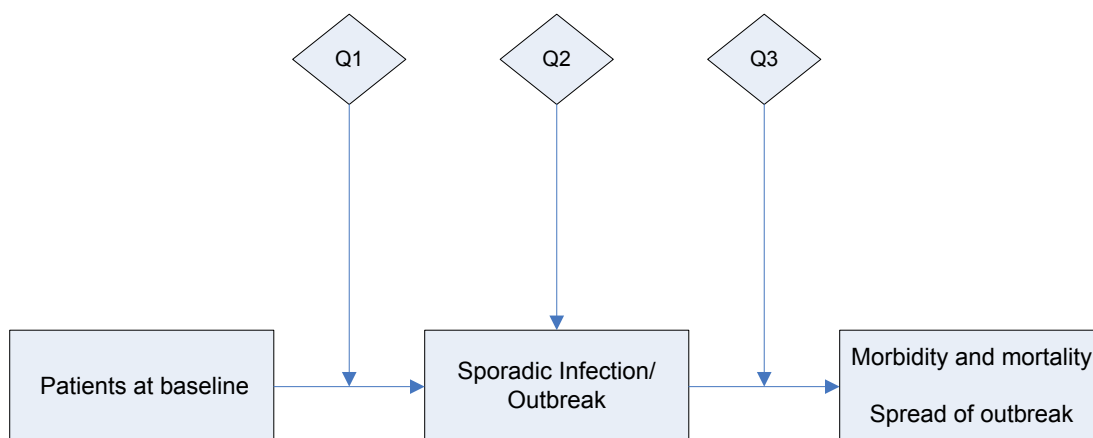
This guideline was based on a targeted systematic review of the best available evidence on the prevention and control of norovirus gastroenteritis outbreaks in healthcare settings. The Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach was used²¹⁻²⁴ to provide explicit links

between the available evidence and the resulting recommendations. Methods and/or details that were unique to this guideline are included below.

Development of Key Questions

First, an electronic search of the National Guideline Clearinghouse, MEDLINE, EMBASE, the Cochrane Health Technology Assessment Database, the NIH Consensus Development Program, and the National Institute for Health and Clinical Excellence, the Scottish Intercollegiate Guidelines Network and the United States Preventive Services Task Force databases was conducted for existing national and international guidelines relevant to norovirus. The strategy used for the guideline search and the search results can be found in *Appendix 1A*. A preliminary list of key questions was developed from a review of the relevant guidelines identified in the search.²⁵⁻⁴⁹ Key questions were put in final form after vetting them with a panel of content experts and HICPAC members. An analytic framework depicting the relationship among the key questions is included in *Figure 2*.

Figure 2. Norovirus Analytic Framework



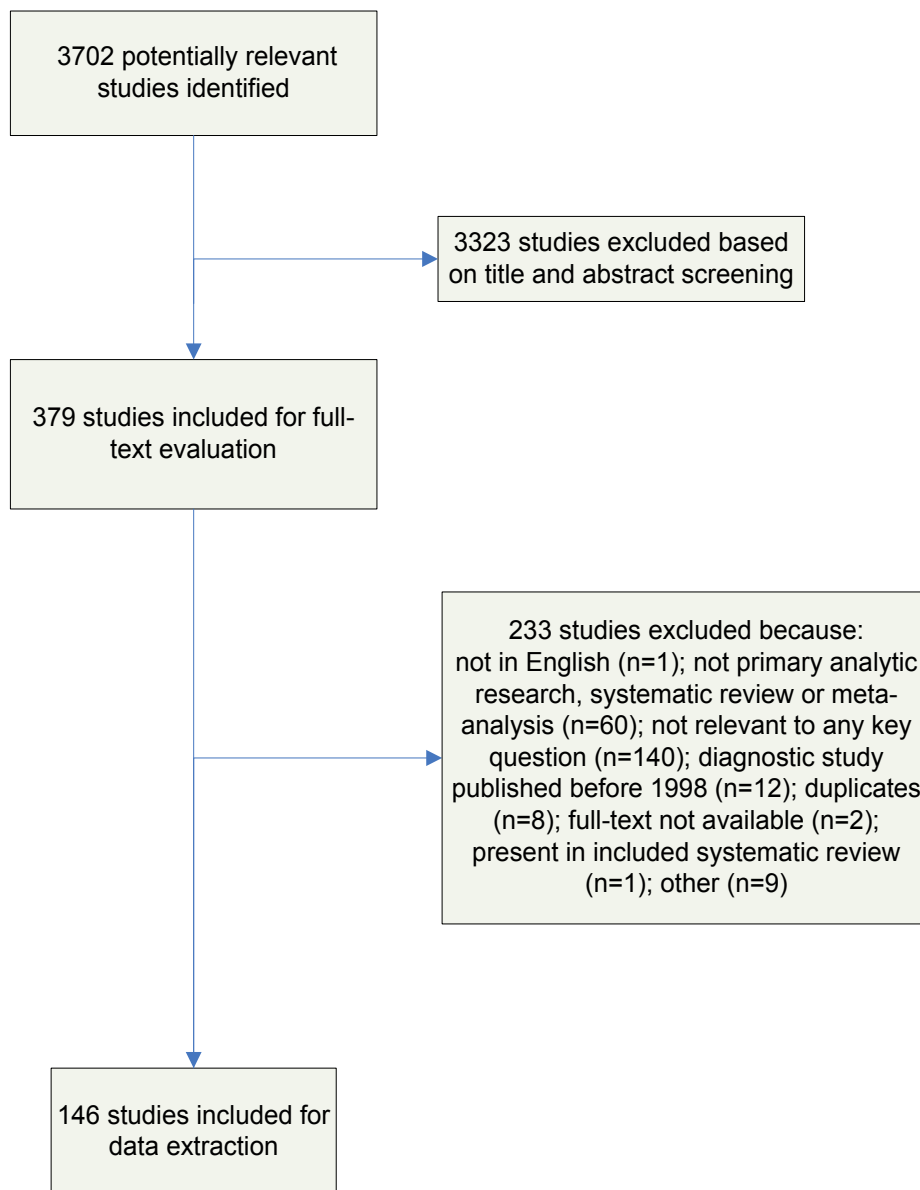
Literature Search

Following the development of the key questions, search terms were developed for identifying literature most relevant to those questions. For the purposes of quality assurance, these terms were compared to those used in relevant seminal studies and guidelines. These search terms were then incorporated into search strategies for the relevant electronic databases. Searches were performed in MEDLINE, EMBASE, CINAHL, the Cochrane Library, Global Health and ISI Web of Science (all databases were searched to the end of February 2008), and the resulting references were imported into a reference manager, where duplicates were resolved. The detailed search strategy used for identifying primary literature and the results of the search can be found in *Appendix 1B*.

Study Selection

Titles and abstracts from references were screened by a single reviewer (T.M. or K.B.S.). Full text articles were retrieved if they were 1) relevant to one or more key questions, 2) primary research, systematic reviews or meta-analyses, and 3) written in English. To be included, studies had to measure ≥ 1 clinically relevant outcome. For Key Questions 1 and 3, this included symptoms of norovirus infection, or stool antigen, virus, or EM results. For Key Question 2, this included any study published after 1997 that reported test characteristics (e.g., sensitivity, specificity, predictive values, likelihood ratios). Outbreak descriptions were included if: 1) norovirus was confirmed as the cause by EM, PCR, or antigen tests AND 2) the outbreak occurred in a healthcare setting and included a list of interventions or practices used to prevent or contain the outbreak OR 3) the outbreak occurred in any setting, but the report included statistical analyses. Full-text articles were screened by two independent reviewers (T.M., and I.L., or K.B.S.) and disagreements were resolved by discussion. The results of this process are depicted in *Figure 3*.

Figure 3. Results of the Study Selection Process



Data Extraction and Synthesis

For those studies meeting inclusion criteria, data on the study author, year, design, objective, population, setting, sample size, power, follow-up, and definitions and results of clinically relevant outcomes were extracted into standardized data extraction forms (*Appendix 3*). From these, three evidence tables were developed, each of which represented one of the key questions (*Appendix 2*). Studies were extracted into the most relevant evidence table. Then, studies were organized by the common themes that emerged within each evidence table. Data were extracted by a single author (R.K.A or I.L.) and cross-checked by another author (R.K.A or I.L.). Disagreements were resolved by the remaining authors. Data and analyses were extracted as originally presented in the included studies. Meta-analyses were performed only where

their use was deemed critical to a recommendation and only in circumstances in which multiple studies with sufficiently homogenous populations, interventions, and outcomes could be analyzed. Systematic reviews were included in this review. To avoid duplication of data, primary studies were excluded if they were also included in a systematic review captured through the broader search strategy. The only exception to this was if the primary study also addressed a relevant question that was outside the scope of the included systematic review. Before exclusion, data from primary studies that were originally captured were abstracted into the evidence tables and reviewed. Systematic reviews that analyzed primary studies that were fully captured in a more recent systematic review were excluded. The only exception to this was if the older systematic review also addressed a relevant question that was outside the scope of the newer systematic review. To ensure that all relevant studies were captured in the search, the bibliography was vetted by a panel of content experts. For the purposes of the review, statistical significance was defined as $p \leq 0.05$.

For all other methods (i.e., Grading of Evidence, Formulation of Recommendations, and Finalizing of the Guideline) please refer to the [Guideline Methods supplement](#).

Updating the Guideline

Future revisions to this guideline will be dictated by new research and technological advancements for preventing and managing norovirus gastroenteritis outbreaks.

VIII. Evidence Review

Question 1: What host, viral or environmental characteristics increase or decrease the risk of norovirus infection in healthcare settings?

To answer this question, the quality of evidence was evaluated among risk factors identified in 57 studies. In areas for which the outcome of symptomatic norovirus infection was available, this was considered the critical outcome in decision-making. The evidence for this question consisted of one systematic review,⁵⁶ 51 observational,^{57-62,62-64,64-77,77-107} and 4 descriptive studies,¹⁰⁸⁻¹¹¹ as well as one basic science study.¹¹² The paucity of randomized controlled trials (RCT) and the large number of observational studies greatly influenced the quality of evidence supporting the conclusions in the evidence review. Based on the available evidence, the risk factors were categorized as host, viral or environmental characteristics. Host characteristics were further categorized into demographics, clinical characteristics, and laboratory characteristics. Environmental characteristics were further categorized into institution, pets, diet, and exposure. The findings of the evidence review and the grades for all clinically relevant outcomes are shown in Evidence and Grade Table 1.

Q1.A Person characteristics

Q1.A.1 Demographic characteristics

Low-quality evidence was available to support age as a risk factor for norovirus infection,^{57-60,62-64} and very low-quality evidence to support black race as a protective factor.⁶⁴ Three studies indicated that persons over the age of 65 may be at greater risk than younger patients for prolonged duration and recovery from diarrhea in healthcare settings.⁵⁷⁻⁵⁹ Studies including children under the age of five showed an increased risk of household transmission as well as asymptomatic infection compared with older children and adults.^{60,62}

A single but large-scale observational study among military personnel found blacks to be at lower risk of infection than whites.⁶⁴ Very low-quality evidence failed to demonstrate meaningful differences in the risk of infection corresponding to strata on the basis of educational background (in the community setting).⁶¹ Based upon very low-quality evidence, outbreaks originating from patients were more likely to affect a large proportion of patients than were outbreaks originating from staff.⁵⁶ Exposure to vomitus and patients with diarrhea increased the likelihood that long-term care facility staff would develop norovirus infection.⁶⁶

The search did not identify studies that established a clear association between sex and symptomatic norovirus infection or complications of norovirus infection.^{57,59, 79, 98} Low-quality evidence from one prospective controlled trial did not identify sex as a significant predictor of symptomatic norovirus in univariate analyses.⁵⁷ There is low-quality evidence suggesting that sex is not a risk factor for protracted illness or complications of norovirus infection including acute renal failure and hypokalemia.⁵⁷

Q1.A.2 Clinical characteristics

Review of the available studies revealed very low-quality evidence identifying clinical characteristics as risk factors for norovirus infection.^{57,60,65,68} One small study found hospitalized children with human immunodeficiency virus (HIV) and chronic diarrhea were more likely to have symptomatic infection with small round structured virus (SRSV) than those without HIV and affected with chronic diarrhea.^{65,68} Adult patients with symptomatic norovirus receiving immunosuppressive therapy or admitted with underlying trauma were at risk for a greater than 10% rise in their serum creatinine.⁵⁷ Norovirus-infected patients with cardiovascular disease or having had a renal transplant were at greater risk for a decrease in their potassium levels by greater than 20%.⁵⁷ Observational, univariate study data also supported an increased duration of diarrhea (longer than two days) among hospitalized patients of advanced age and those with

malignancies.⁵⁷ This search did not reveal data on the risk of norovirus acquisition among those co-infected with other acute gastrointestinal infections, such as *C. difficile*.

Q1.A.3 Laboratory characteristics

Q1.A.3.a Antibody levels

There was very low-quality evidence to support limited protective effects of serum antibody levels against subsequent norovirus infection.⁷⁴⁻⁷⁶ In two challenge studies, adult and pediatric subjects with prior exposure to norovirus showed higher antibody titers than found in previously unexposed subjects after initial infection and after challenge.^{74,76} The detection of preexisting serum antibody does not appear to correlate with protection against subsequent norovirus challenge, nor did increasing detectable pre-existing antibody titres correlate with attenuations in the clinical severity of disease.^{74,75} In one study, symptoms such as vomiting, nausea, headaches, and arthralgia were correlated with increasing antibody titres.⁷⁴ In a serial challenge study, 50% of participants (n=6) developed infection, and upon subsequent challenge 27-42 months later, only those same participants developed symptoms. A third challenge 4-8 weeks after the second series resulted in symptoms in just a single volunteer.⁷⁶ Pre-existing antibody may offer protection to susceptible persons only for a limited window of time, on the order of a few weeks. The search strategy did not reveal data on the persistence of immunity to norovirus nor elevations in antibody titers that were consistently suggestive of immunity.

Q1.A.3.b Secretor genotype

Review of the outlined studies demonstrated high-quality evidence to support the protective effects of human host non-secretor genotypes against norovirus infection.^{70-72,113} Two observational studies and one intervention study examined volunteers with and without the expression of the secretor (FUT2) genotype after norovirus challenge.⁷⁰⁻⁷² Statistically significant differences were reported with secretor-negative persons demonstrating a greater likelihood of protection against, or innate resistance to symptomatic and asymptomatic norovirus infection than seen in persons with secretor-positive genotypes. This search did not reveal data on the dose-response effects of norovirus in persons with homozygous and heterozygous secretor genotypes. Because the FUT2-mediated secretor positive phenotype appears to confer susceptibility to subsequent norovirus infection following challenge, there is an association between this phenotype and measurable circulating antibody (suggesting prior infection) in the population. One study estimated that 80% of the population is secretor-positive (or susceptible to norovirus) and 20% is secretor-negative (resistant to norovirus challenge independent of inoculum dose). Among susceptible persons, approximately 35% are protected from infection. This protection is potentially linked to a memory-mediated rapid mucosal IgA response to norovirus exposure that is not seen in the other 45% of susceptibles, who demonstrate delayed mucosal IgA and serum IgG responses.⁷² Although elevated antibody levels following infection appear to confer some protective immunity to subsequent challenge, paradoxically, measurable antibody titers in the population may be a marker of *increased* susceptibility to norovirus because of the association between such antibodies and FUT2-positive status.

Q1.A.3.c ABO phenotype

There was low-quality evidence suggesting any association of ABO blood type with the risk of norovirus infection.^{69,72,73,77,78,114,115} An RCT suggested that persons with histo-blood group type O was associated with an increased risk of symptomatic or asymptomatic norovirus infection among secretor-positive patients.⁷² Binding of norovirus to the mucosal epithelium may be facilitated by ligands associated with type-O blood. The other blood types—A, B, and AB—were not associated with norovirus infection after controlling for secretor status. Three studies showed no protective effect of any of the blood types against norovirus.^{69,77,78} The search strategy did not reveal prospective cohort data to correlate the role of ABO blood types with risk of norovirus infection.

Q1.B Viral characteristics

There was very low-quality evidence to suggest an association of virus characteristics with norovirus infection.^{57,108-110} Very low-quality descriptive evidence suggested that increases in overall norovirus activity may result from the emergence of new variants among circulating norovirus strains, and strains may differ in pathogenicity, particularly among GII.3 and GII.4 variants.¹⁰⁸⁻¹¹⁰ In recent years, GII.4 strains are increasingly reported in the context of healthcare-associated outbreaks, but further epidemiologic and laboratory studies are required to expand on this body of information. This search did not identify studies examining genotypic characteristics of viruses associated with healthcare-acquired norovirus infection.

Q1.C Environmental characteristics

Q1.C.1 Institutional characteristics

Very low-quality evidence was available to support the association of institutional characteristics with symptomatic norovirus infection.^{82,99} Among two observational studies, the number of beds within a ward, nurse understaffing, admission to an acute care hospital (compared to smaller community-based facilities), and having experienced a prior outbreak of norovirus gastroenteritis within the past 30 days were all possible risk factors for new infections.^{82,99} These increased institutional risks were identified from univariate analyses in pediatric and adult hospital populations. There were statistically significant, increased risks of infection among those admitted to geriatric, mental health, orthopedic, and general medicine wards. The review process did not reveal data on the comparative risks of infection among those admitted to private and shared patient rooms.

Q1.C.2 Pets

Review of the outlined studies demonstrated very low-quality evidence to support exposure to pets (e.g., cats and dogs) as a risk factor for norovirus infection.⁶¹ One case-control study examined pet exposure among households in the community and concluded that the effect of cats was negligible.⁶¹ The single study did not demonstrate any evidence of transmission between pets and humans of norovirus infection. This search strategy did not reveal studies that evaluated the impact of therapy pets in healthcare settings during outbreaks of norovirus gastroenteritis or data examining domestic animals as reservoirs for human infection.

Q1.C.3 Diet

There was low-quality evidence to suggest that extrinsically contaminated food items are commonly implicated as vehicles of norovirus exposure in healthcare settings.^{61,77,80,84,86,87,89-97,100-102,104-107,111} Nineteen observational studies itemized statistically significant food sources implicated in community outbreaks.^{80,81,84,86,87,89-97,100,101,104-106} Common to most of these food sources was a symptomatic or asymptomatic food-handler. Sauces, sandwiches, fruits and vegetables, salads, and other moisture-containing foods were most often cited as extrinsically contaminated sources of outbreaks of norovirus gastroenteritis. Importantly, these data reflected the breadth of foods that can become contaminated. Tap water and ice were also associated with norovirus contamination during an outbreak with an ill food-handler. This literature review did not identify studies that examined the introduction of intrinsically contaminated produce or meats as a nidus for norovirus infection and dissemination within healthcare facilities.

Q1.C.4 Proximity to infected persons

This review demonstrated high-quality evidence to suggest that proximity to infected persons with norovirus is associated with increased risk of symptomatic infection.^{61,62,64,79,83,88,98,103,111} Eight observational studies found statistically significant factors such as proximate exposure to an infected source within households or in crowded quarters increased infection risk, as did exposures to any or frequent vomiting episodes.^{61,62,64,79,83,88,98,103} These data suggest person-to-person transmission is dependent on close or direct contact as well as short-range aerosol exposures. One observational study established a linear relationship

between a point source exposure and attack rate based on proximity to an infected and vomiting source.⁸⁸ This search process did not identify studies that quantified the spatial radius necessary for transmission to successfully occur.

Q1 Recommendations

1.A.1 Avoid exposure to vomitus or diarrhea. Place patients on Contact Precautions in a single occupancy room if they have symptoms consistent with norovirus gastroenteritis. **(Category IB)** (Key Question 1A)

1.A.2.a Consider longer periods of isolation or cohorting precautions for complex medical patients (e.g., those with cardiovascular, autoimmune, immunosuppressive, or renal disorders) as they can experience protracted episodes of diarrhea and prolonged viral shedding. Patients with these or other comorbidities have the potential to relapse and facilities may choose longer periods of isolation based on clinical judgment. **(Category II)** (Key Question 1A)

1.C.1 Consider the development and adoption of facility policies to enable rapid clinical and virological confirmation of suspected cases of symptomatic norovirus infection while implementing prompt control measures to reduce the magnitude of a potential norovirus outbreak. **(Category II)** (Key Question 1C)

1.C.3.a To prevent food-related outbreaks of norovirus gastroenteritis in healthcare settings, food handlers must perform hand hygiene prior to contact with or the preparation of food items and beverages (<http://www.fda.gov/Food/FoodSafety/RetailFoodProtection/FoodCode/default.htm>). **(Category IC)** (Key Question 1C)

1.C.3.b Personnel who work with, prepare or distribute food must be excluded from duty if they develop symptoms of acute gastroenteritis. Personnel should not return to these activities until a minimum of 48 hours after the resolution of symptoms or longer as required by local health regulations (<http://www.fda.gov/Food/FoodSafety/RetailFoodProtection/FoodCode/default.htm>). **(Category IC)** (Key Question 1C)

1.C.4 If norovirus infection is suspected, adherence to PPE use according to Contact and Standard Precautions is recommended for individuals entering the patient care area (i.e., gowns and gloves upon entry) to reduce the likelihood of exposure to infectious vomitus or fecal material. **(Category IB)** (Key Question 1C)

Question 2: What are the best methods to identify an outbreak of norovirus gastroenteritis in a healthcare setting?

To address this question, studies that provided test characteristics for the diagnosis of norovirus or outbreaks of norovirus gastroenteritis were critically reviewed. The available data examined the use of clinical criteria for the diagnosis of an outbreak of norovirus, methods of specimen collection for the diagnosis of a norovirus outbreak, and characteristics of tests used to diagnose norovirus. The evidence consisted of 33 diagnostic studies.^{17,18,116-146} The findings from the evidence review and the grades of evidence for clinically relevant outcomes are shown in Evidence and Grade Table 2.

Q2.A Clinical Criteria

There was moderate quality evidence from a single diagnostic study supporting the use of the Kaplan criteria to detect outbreaks of norovirus gastroenteritis.^{16,116} Of 362 confirmed gastroenteritis outbreaks with complete clinical or laboratory data, the sensitivity of the Kaplan Criteria to detect an outbreak of norovirus gastroenteritis without an identified bacterial pathogen was 68.2%, with a specificity of 98.6%. The positive predictive value (PPV) was 97.1% and the negative predictive value was 81.8%. Individual criteria, such as vomiting among >50% of a patient cohort, brief duration of illness (12-60 hours), or mean incubation time of 24-48 hours, demonstrated high sensitivities (85.8-89.2%), but specificities were low (60.7-69.6%). The use of additional criteria, such as the ratios of fever-to-vomiting and diarrhea-to-vomiting, provided sensitivities of 90.1% and 96.6%, and specificities of 46.6% and 44.5%, respectively. Applied to the 1141 outbreaks of unconfirmed etiology, suspected norovirus or bacterial sources with complete data, the Kaplan criteria estimated that 28% of all 1998-2000 CDC-reported *foodborne* outbreaks might be attributable to norovirus. The search strategy did not identify studies that have assessed the utility of the Kaplan criteria in healthcare-associated outbreaks of norovirus gastroenteritis.

Q2.B Specimen Collection

There was low-quality evidence from three diagnostic studies outlining the minimum number of stool samples from symptomatic patients required to confirm an outbreak of norovirus gastroenteritis.^{117,119,120,122,123} In modeling analyses using a hypothetical test demonstrating 100% sensitivity and 100% specificity, obtaining a positive EIA result from two or more submitted samples demonstrated a sensitivity of 52.2-57%, with a peak in sensitivity when at least one from a total of six submitted samples was positive for norovirus (71.4-92%). Specificity was 100% when at least one positive EIA was obtained from a minimum of two submitted stool samples.

Using a reverse transcriptase polymerase chain reaction (RT-PCR) method, if at least one positive test was identified among 2 to 4 submitted stool specimens from symptomatic persons, the test sensitivity was greater than 84%. When 5-11 stool samples were submitted and at least 2 were confirmed as positive, the sensitivity of PCR was greater than 92%. When at least one stool specimen was submitted for identification, PCR confirmed norovirus as the causative agent in a larger proportion of outbreaks than those using EM or ELISA methods, and is currently the Gold Standard. This evaluation was unable to determine how diagnostic test characteristics are affected by the timing of specimen collection relative to the disease process.

Q2.C Diagnostic Methods

28 diagnostic studies^{17,18,118-120,122,124-139,141-145,147} and 1 descriptive study¹²¹ that evaluated the test characteristics of EIA such as ELISA, EM, reverse transcriptase PCR, and nucleic acid sequence-based amplification (NASBA) in the detection of norovirus in human fecal specimens were summarized. Test characteristics for the most common or commercially-available norovirus diagnostics are summarized in the following Table.

Q2 Recommendations

2.A.1 In the absence of clinical laboratory diagnostics or in the case of delay in obtaining laboratory results, use Kaplan's clinical and epidemiologic criteria to identify a norovirus gastroenteritis outbreak (see Table 4 for Kaplan's criteria). **(Category IA)** (Key Question 2A)

2.A.2 Further research is needed to compare the Kaplan criteria with other early detection criteria for outbreaks of norovirus gastroenteritis in healthcare settings, and to assess whether additional clinical or epidemiologic criteria can be applied to detect norovirus clusters or outbreaks in healthcare settings. **(No recommendation/unresolved issue)** (Key Question 2A)

2.B Consider submitting stool specimens as early as possible during a suspected norovirus gastroenteritis outbreak and ideally from individuals during the acute phase of illness (within 2-3 days of onset). It is suggested that healthcare facilities consult with state or local public health regarding the types of and number of specimens to obtain for testing. **(Category II)** (Key Question 2B)

2.C Use effective laboratory diagnostic protocols for testing of suspected cases of viral gastroenteritis (e.g., refer to the Centers for Disease Control and Prevention (CDC)'s most current recommendations for norovirus diagnostic testing at <http://www.cdc.gov/mmwr/pdf/rr/rr6003.pdf>. **(Category IB)** (Key Question 2C)

Table 3. Test Characteristics for Norovirus in Fecal Specimens

Diagnostic method	Reference standard	Quantity and type of evidence	Findings*			
			Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Kaplan criteria	PCR	1 DIAG ¹¹⁶	68	99	97	82
EIA/ELISA	PCR	10 DIAG ^{17,118-120,123-128,139}	31 – 90	65 – 100	52 – 100	56-97
EM	PCR	2 DIAG ^{17,119}	24 – 58	98-99	88-94	71-91
NASBA	PCR	1 DIAG ¹⁴⁴	100	50	-	-

* Range from studies that reported test characteristics
Negative predictive Value, NPV; Positive predictive value, PPV

Table 4. Kaplan Criteria¹⁶

- 1) Vomiting in more than half of symptomatic cases
- 2) Mean (or median) incubation period of 24 to 48 hours
- 3) Mean (or median) duration of illness of 12 to 60 hours
- 4) No bacterial pathogen isolated in stool culture

Question 3: What interventions best prevent or contain outbreaks of norovirus gastroenteritis in the healthcare setting?

To address this question, 69 studies^{58,63,66,79,83-85,87,89,92,102,103,112,148-203} were critically reviewed for evidence of interventions that might prevent or attenuate an outbreak of norovirus. The available data dealt with viral shedding, recovery of norovirus, and components of an outbreak prevention or containment program, including the use of medications. The evidence consisted of 1 randomized controlled trial,²⁰² 1 systematic review,¹⁵³ 20 basic science studies,^{112,162,163,185-201} 43 descriptive studies,^{58,63,79,83-85,87,89,92,102,103,149-152,154-161,165-184} and 4 observational studies.^{66,148,164,203} The findings from the evidence review and the grades of evidence for clinically relevant outcomes are shown in Evidence and Grade Table 3.

Q3.A Viral Shedding

This review did not identify studies demonstrating direct associations between viral shedding and infectivity. However, there was low-quality evidence to support an association between age and duration of viral shedding.^{149,150} One observational study suggested that children under the age of six months may be at an increased risk of prolonged viral shedding (greater than two weeks), even after the resolution of symptoms.¹⁴⁸ Other findings suggest that infants can shed higher titers of virus than levels reported in other

age groups.¹⁴⁹ High-quality evidence was available to demonstrate the presence of viral shedding in asymptomatic subjects, and low-quality evidence demonstrating that shedding can persist for up to 22 days following infection and 5 days after the resolution of symptoms.¹⁵⁰⁻¹⁵² The search strategy employed did not identify studies that correlated other clinical factors to duration of viral shedding.

Q3.B Recovery of Norovirus

Q3.B.1 Fomites

There was low-quality evidence positively associating fomite contamination with norovirus infection.^{153-159,161,163,194} Similarly, there was low-quality evidence demonstrating transfer of norovirus from fomites to hands.¹⁹⁴ One basic science study demonstrated that norovirus on surfaces can be readily transferred to other fomites (telephones, taps, door handles) via fingertips in 30-50% of opportunities even when virus has been left to dry for 15 minutes.¹⁹⁴ There was moderate quality evidence examining the norovirus contamination of the environment.^{153-159,161,163} A single systematic review evaluated 5 outbreaks with environmental sampling data.¹⁵³ Three of those outbreaks confirmed environmental contamination with norovirus. Of the over 200 swabs examined from the 5 outbreaks in this review, 36% identified norovirus contamination on various fomites such as curtains, carpets, cushions, commodes and toilets, furnishings and equipment within 3-4 feet of the patient, handrails, faucets, telephones, and door handles. However, in two outbreaks from which 47 environmental samples were collected, norovirus was not detected. Additional studies detected norovirus on kitchen surfaces, elevator buttons, and other patient equipment.^{154-157, 194}

There was low-quality evidence regarding the duration of norovirus persistence.^{154,155,157-159,161} Norovirus can persist in a dried state at room temperature for up to 21-28 days and, in a single observational study, was undetectable in areas of previously known contamination after 5 months had elapsed.¹⁵⁹ Laboratory studies comparing FCV and MNV-1 also demonstrated persistence of virus in both dried and in fecal suspensions for a minimum of seven days on stainless steel preparations at 4°C and at room temperature.²⁰ Within a systematic review, it was observed that norovirus may remain viable in carpets up to 12 days, despite regular vacuuming.¹⁵³ Similarly, a cultivable surrogate for human strains of norovirus (FCV) was detected on computer keyboards and mice, as well as telephone components up to 72 hrs from its initial inoculation.¹⁵⁶ This search strategy did not find studies in which the recovery of norovirus from fomites, food, and water sources was directly associated with transmission of infection in healthcare settings; however transmission from these sources has been well documented in other settings.

Q3.B.2 Foods and Food Preparation Surfaces

There was low-quality evidence suggesting that foods and food-preparation surfaces are significant sources of norovirus transmission in healthcare settings.^{112,162,163} There was moderate quality evidence among three basic science studies to suggest that norovirus can be recovered from foods such as meats and produce as well as from utensils and non-porous surfaces (e.g., stainless steel, laminate, ceramics) upon which foods are prepared.^{112,162,163} Two of these studies, comprised of low-quality evidence, suggested that the transfer of diluted aliquots of norovirus from stainless steel surfaces to wet and dry food, and through contaminated gloves was detectable using PCR methods. Norovirus transfer was statistically more efficient when it was inoculated onto moist surfaces compared to dry ones.^{162,163}

There was low-quality evidence to suggest that norovirus persists for longer periods in meats compared to other foods and non-porous surfaces, both at 4°C and at room temperature.¹¹² There was moderate quality evidence demonstrating that over a period of 7 days after application, both human norovirus genogroup I and a surrogate (FCV) could be detected among all surfaces tested.^{112,162} Within the first hour, the log₁₀ of FCV titers declined by 2-3, with an additional drop of 2-4 after 48 hours elapsed.¹⁶² Food and food-preparation areas can serve as a common source of contamination with norovirus in the absence of cleaning and disinfection.

Q3.B.3 Water

This search strategy did not identify studies that measured the contribution of norovirus-contaminated water to outbreaks in the healthcare setting. However, there was moderate quality evidence to suggest that norovirus could be recovered from water.^{155,158,160} Among three outbreaks that examined water as a source, one identified norovirus in 3 of 7 water samples.¹⁶⁰ In outbreaks in the community, which were outside the scope of this review, contaminated surface water sources, well water, and recreational water venues have been associated with outbreaks of norovirus gastroenteritis.²⁰⁴

Q3.C Components of an Outbreak Prevention/Containment Program

As with most infection-prevention and control activities, multiple strategies are instituted simultaneously during outbreaks in healthcare settings. Thus, it is difficult to single out particular interventions that may be more influential than others, as it is normally a combination of prudent interventions that reduce disease transmission. Numerous studies cite the early recognition of cases and the rapid implementation of infection control measures as key to controlling disease transmission. The following interventions represent a summary of key components in light of published primary literature and addressed in seminal guidelines on outbreaks of norovirus gastroenteritis.

Q3.C.1 Hand Hygiene

Q3.C.1.a Handwashing with soap and water

Very low-quality evidence was available to confirm that handwashing with soap and water prevents symptomatic norovirus infections.^{63,66,79,85,89,102,103,165,166,168-171,173-177,183} Several descriptive studies emphasized hand hygiene as a primary prevention behavior and promoted it simultaneously with other practical interventions. Several outbreaks centered in healthcare augmented or reinforced hand hygiene behavior as an early intervention and considered it an effective measure aimed at outbreak control.^{103,165,168,170,174,176,177,183} The protocols for hand hygiene that were reviewed included switching to the exclusive use of handwashing with soap and water, and a blend of handwashing with the adjunct use of alcohol-based hand sanitizers. Additional guidance is available in the 2002 HICPAC Guideline for Hand Hygiene in Health-Care Settings (<http://www.cdc.gov/mmwr/PDF/rr/rr5116.pdf>).

Q3.C.1.b Alcohol-based hand sanitizers

Very low-quality evidence was available to suggest that hand hygiene using alcohol-based hand sanitizers may reduce the likelihood of symptomatic norovirus infection.^{66,87,169,171,205} Several studies used FDA-compliant alcohol-based hand antiseptics during periods of norovirus activity as an adjunct measure of hand hygiene.^{66,87,168,169,171,205,206} Two studies used a commercially available 95% ethanol-based hand sanitizer along with handwashing with soap and water; but without a control group and with hand hygiene comprising one of several interventions, the relative contribution of hand hygiene to attenuating transmission was difficult to evaluate.^{169,171} In the laboratory, even with 95% ethanol products, the maximum mean reduction in log₁₀ titer reduction was 2.17.¹⁹³ Evidence to evaluate the efficacy of alcohol-based hand disinfectants consisted of basic science studies using FCV as a surrogate for norovirus. Moderate quality evidence supported ethanol as a superior active ingredient in alcohol-based hand disinfectants compared to 1-propanol, particularly when simulated organic loads (e.g. fecal material) were used in conjunction with exposure to norovirus.^{189,191,193,196} The use of hand sanitizers with mixtures of ethanol and propanol have shown effectiveness against FCV compared to products with single active ingredients (70% ethanol or propanol) under controlled conditions.¹⁸⁹ There were no studies available to evaluate the effect of non-alcohol based hand sanitizers on norovirus persistence on skin surfaces.

Q3.C.1.c Role of artificial nails

Very low-quality evidence suggested that the magnitude in reduction of a norovirus surrogate (FCV) using a spectrum of soaps and hand disinfectants was significantly greater among volunteers with natural nails compared to those with artificial nails.¹⁹⁷ A subanalysis showed that longer fingernails were associated with consistently greater hand contamination. Further evidence summarizing the impact of artificial and long fingernails in healthcare settings can be found in the HICPAC Guidelines for Hand Hygiene in Healthcare Settings (<http://www.cdc.gov/Handhygiene/>).

Q3.C.2 Personal Protective Equipment

Very low-quality evidence among 1 observational⁶⁶ and 13 descriptive studies^{167-173,176-179,181,183} support the use of personal protective equipment (PPE) as a prevention measure against symptomatic norovirus infection. A single retrospective study failed to support the use of gowns as a significantly protective measure against norovirus infection during the outbreak among staff but did not consider the role of wearing gowns in avoiding patient-to-patient transmission.⁶⁶ Mask or glove use was not evaluated in the self-administered questionnaire used in the study. Several observational and descriptive studies emphasized the use of gloves and isolation gowns for routine care of symptomatic patients, with the use of masks recommended when staff anticipated exposure to emesis or circumstances where virus may be aerosolized.^{167-173,176-179,181,183} The use of PPE was advocated for both staff and visitors in two outbreak studies.^{169,179}

Q3.C.3 Leave Policies for Staff

There was very low-quality evidence among several studies to support the implementation of staff exclusion policies to prevent symptomatic norovirus infections in healthcare settings.^{84,85,92,165,167-169,172,174,176,177,179-181,183,184} Fifteen descriptive studies emphasized granting staff sick time from the time of symptom onset to a minimum of 24 hours after symptom resolution.^{84,85,92,167-169,172,176,177,179,180,183,184} The majority of studies opted for 48 hours after symptom resolution before staff could return to the workplace.^{84,92,167,169,172,176,177,179,180,183,184} One study instituted a policy to exclude symptomatic staff from work until they had remained symptom-free for 72 hours.¹⁶⁸ While selected studies have identified the ability of persons to shed virus for protracted periods post-infection, it is not well understood whether virus detection translates to norovirus infectivity. The literature search was unable to determine whether return to work policies were effective in reducing secondary transmission of norovirus in healthcare facilities.

Q3.C.4 Isolation/Cohorting of Symptomatic Patients

There was very low-quality evidence among several descriptive studies to support patient cohorting or placing patients on Contact Precautions as an intervention to prevent symptomatic norovirus infections in healthcare settings.^{87,166-171,173,176,177,179-182,184} No evidence was available to encourage the use of Contact Precautions for sporadic cases, and the standard of care in these circumstances is to manage such cases with Standard Precautions (<http://www.cdc.gov/ncidod/dhqp/pdf/guidelines/Isolation2007.pdf>). Fifteen descriptive studies used isolation precautions or cohorting practices as a primary means of outbreak management.^{87,166-171,173,176,177,179-182,184} Patients were cared for in single occupancy (e.g., private) rooms, physically grouped into cohorts of symptomatic, exposed but asymptomatic, or unexposed within a ward, or alternatively, with entire wards placed under Contact Precautions. Exposure status typically was based on a person's symptoms and/or physical and temporal proximity to norovirus activity. A few studies cited restricting patient movements within the ward, suspending group activities, and special considerations for therapy or other medical appointments during outbreak periods as adjunct measures to control the spread of norovirus.^{63,169,182,183}

Q3.C.5 Staff Cohorting

Very low-quality evidence supported the implementation of staff cohorting and the exclusion of non-essential staff and volunteers to prevent symptomatic norovirus infections.^{87,103,165,168-170,172,173,177,179,180,182,183} All studies addressing this topic were descriptive. Staff was designated to care for one cohort of patients

(symptomatic, exposed but asymptomatic, or unexposed). Exposed staff was discouraged from working in unaffected clinical areas and from returning to care for unexposed patients before, at a minimum, allowing 48 hours from their last putative exposure to elapse.¹⁷⁷ The search strategy did not identify healthcare personnel other than nursing, medical, environmental services, and paramedical staff who were assigned to staff cohorting. There were no identified studies that evaluated the infectious risk of assigning recovered staff as caregivers for asymptomatic patients.

Q3.C.6 Ward Closure

Low-quality evidence was available to support ward closure as an intervention to prevent symptomatic norovirus infections.^{85,164-166,168,173,176-179,183,184} Ward closure focused on temporarily suspending transfers in or out of the ward, and discouraged or disallowed staff from working in clinical areas outside of the closed ward. One prospective controlled study evaluating 227 ward-level outbreaks between 2002 and 2003 demonstrated that outbreaks were significantly shorter (7.9 vs. 15.4 days, $p < 0.01$) when wards were closed to new admissions.¹⁶⁴ The mean duration of ward closure was 9.65 days, with a loss of 3.57 bed-days for each day the ward was closed. The duration of ward closure in the descriptive studies examined was dependent on facility resources and magnitude of the outbreaks. Allowing at least 48 hours from the resolution of the last case, followed by thorough environmental cleaning and disinfection was common before re-opening a ward. Other community-based studies have used closures as an opportunity to perform thorough environmental cleaning and disinfection before re-opening. Two studies moved all patients with symptoms of norovirus infection to a closed infectious disease ward and then performed thorough terminal cleaning of the vacated area.^{170,172} In most instances, studies defended that it was preferable to minimize patient movements and transfers in an effort to contain environmental contamination.

Q3.C.7 Visitor Policies

There was very low-quality evidence demonstrating the impact of restriction and/or screening of visitors for symptoms consistent with norovirus infection.^{168,170,173,182,183} In two studies, visitors were screened for symptoms of gastroenteritis using a standard questionnaire or evaluated by nursing staff prior to ward entry as part of multi-faceted outbreak control measures.^{168,170} Other studies restricted visitors to immediate family, suspended all visitor privileges, or curtailed visitors from accessing multiple clinical areas.^{182,183} The reviewed literature failed to identify research that considered the impact of different levels of visitor restrictions on outbreak containment.

Q3.C.8 Education

There was very low-quality evidence on the impact of staff and/or patient education on symptomatic norovirus infections.^{166,168,169,172,173,182} Six studies simply described education promoted during outbreaks.^{166,168,169,172,173,182} Content for education included recognizing symptoms of norovirus, understanding basic principles of disease transmission, understanding the components of transmission-based precautions, patient discharges and transfer policies, as well as cleaning and disinfection procedures. While many options are available, the studies that were reviewed used posters to emphasize hand hygiene and conducted one-on-one teaching with patients and visitors, as well as holding departmental seminars for staff. The literature reviewed failed to identify research that examined the impact of educational measures on the magnitude and duration of outbreaks of norovirus gastroenteritis, or what modes of education were most effective in promoting adherence to outbreak measures.

Q3.C.9 Surveillance

There was very low-quality evidence to suggest that surveillance for norovirus activity was an important measure in preventing symptomatic infection.^{58,84,166,170} Four descriptive studies identified surveillance as a component of outbreak measurement and containment. Establishing a working case definition and performing active surveillance through contact tracing, admission screening, and patient chart review were suggested as actionable items during outbreaks. There was no available literature to determine whether

active case-finding and tracking of new norovirus cases were directly associated with shorter outbreaks or more efficient outbreak containment.

Q3.C.10 Policy Development and Communication

Very low-quality evidence was available to support the benefits of having established written policies and a pre-arranged communication framework in facilitating the prevention and management of symptomatic norovirus infections.^{63,84,172,182-184} Six descriptive studies outlined the need for mechanisms to disseminate outbreak information and updates to staff, laboratory liaisons, healthcare facility administration, and public health departments.^{63,84,172,182-184} The search of the literature did not yield any studies to demonstrate that facilities with written norovirus policies already in place had fewer or shorter outbreaks of norovirus gastroenteritis.

Q3.C.11 Patient Transfers and Discharges

There was very low-quality evidence examining the benefit of delayed discharge or transfer for patients with symptomatic norovirus infection.^{172,179,183,184} Transfer of patients after symptom resolution was supported in one study but discouraged unless medically necessary in three others. Discharge home was supported once a minimum of 48 hours had elapsed since the patient's symptoms had resolved. For transfers to long-term care or assisted living, patients were held for five days after symptom resolution before transfer occurred. The literature search was unable to identify studies that compared the impact of conservative patient discharge policies for recovered, asymptomatic patients.

Q3.C.12 Environmental Disinfection

Q3.C.12.a Targeted surface disinfection

Very low-quality evidence was available to support cleaning and disinfection of frequently touched surfaces to prevent symptomatic norovirus infection.^{79,153,168,183} One systematic review¹⁵³ and three descriptive studies^{79,168,183} highlighted the need to routinely clean and disinfect frequently touched surfaces (e.g., patient and staff bathrooms and clean and dirty utility rooms, tables, chairs, commodes, computer keyboards and mice, and items in close proximity to symptomatic patients). One systematic review¹⁵³ and two descriptive studies^{102,177,183,184} supported-steam cleaning carpets once an outbreak was declared over. Within the review, a single case report suggested that contaminated carpets may contain viable virus for a minimum of twelve days even after routine dry vacuuming.¹⁵³ Routine cleaning and disinfection of non-porous flooring were supported by several studies, with particular attention to prompt cleaning of visible soiling from emesis or fecal material.^{153,168} There were no studies directly addressing the impact of surface disinfection of frequently touched areas on outbreak prevention or containment.

Q3.C.12.b Process of environmental disinfection

There was very low-quality evidence supportive of enhanced cleaning during an outbreak of norovirus gastroenteritis.^{168,170,177,179} Several studies cited increasing the frequency of cleaning and disinfection during outbreaks of norovirus gastroenteritis.^{168,170,177,179} Ward-level cleaning was performed once to twice per day, with frequently touched surfaces and bathrooms cleaned and disinfected more frequently (e.g., hourly, once per shift, or three times daily). Studies also described enhancements to the process of environmental cleaning. Environmental services staff wore PPE while cleaning patient-care areas during outbreaks of norovirus gastroenteritis.^{176,177,179,205} Personnel first cleaned the rooms of unaffected patients and then moved to the symptomatic patient areas¹⁵⁹. Adjunct measures to minimize environmental contamination from two descriptive studies included labeling patient commodes and expanding the cleaning radius for enhanced cleaning within the immediate patient area to include other proximal fixtures and equipment.^{170,177} In another study, mop heads were changed at an interval of once every three rooms.¹⁶⁸ This literature search was not able to identify whether there was an association with enhanced cleaning regimens during outbreaks of norovirus gastroenteritis and the attenuation in outbreak magnitude or duration.

Q3.C.12.c Patient-service items

There was very low-quality evidence to support the cleaning of patient equipment or service items to reduce symptomatic norovirus infections.^{168,172,177} Three descriptive studies suggested that patient equipment/service items be cleaned and disinfected after use, with disposable patient care items discarded from patient rooms upon discharge.^{168,172,177} A single descriptive study used disposable dishware and cutlery for symptomatic patients.¹⁷² There were no identified studies that directly examined the impact of disinfection of patient equipment on outbreaks of norovirus gastroenteritis.

Q3.C.12.d Fabrics

Very low-quality evidence was available to examine the impact of fabric disinfection on norovirus infections.^{153,168,177,183} One systematic review¹⁵³ and three descriptive studies^{168,177,183} suggested changing patient privacy curtains if they are visibly soiled or upon patient discharge. One descriptive study suggested that soiled, upholstered patient equipment should be steam cleaned^{135, 159}. If this was not possible, those items were discarded. Two descriptive studies emphasized careful handling of soiled linens to minimize re-aerosolization of virus.^{177,183} Wheeling hampers to the bedside or using hot soluble hamper bags (e.g., disposable) were suggested mechanisms to reduce self-contamination. This literature search did not identify studies that examined the direct impact of disinfection of fabrics on outbreaks of norovirus gastroenteritis or whether self-contamination with norovirus was associated with new infection.

Q.3.C.12.e Cleaning and disinfection agents

The overall quality of evidence on cleaning and disinfection agents was very low.^{63,83,87,89,153,167,168,170,174,176-179,182,184} The outcomes examined were symptomatic norovirus infection, inactivation of human norovirus, and inactivation of FCV. Evidence for efficacy against norovirus was usually based on studies using FCV as a surrogate. However, FCV and norovirus exhibit different physiochemical properties and it is unclear whether inactivation of FCV reflects efficacy against human strains of norovirus. One systematic review¹⁵³ and 14 descriptive studies^{63,83,87,89,167,168,170,174,176-179,182,184} outlined strategies for containing environmental bioburden. The majority of outbreaks were managed with sodium hypochlorite in various concentrations as the primary disinfectant. The concentrations for environmental cleaning among these studies ranged from 0.1% to 6.15% sodium hypochlorite.

There was found moderate quality evidence to examine the impact of disinfection agents on human norovirus inactivation.^{187,194,201} Three basic science studies evaluated the virucidal effects of select disinfectants against norovirus.^{187,194,201} A decline of 3 in the log₁₀ of human norovirus exposed to disinfectants in the presence of fecal material, a fetal bovine serum protein load, or both was achieved with 5% organic acid after 60 minutes of contact time, 6000 ppm free chlorine with 15 minutes of contact time, or a 1 or 2% peroxide solution for 60 minutes.¹⁸⁷ This study also demonstrated that the range of disinfectants more readily inactivated FCV than human norovirus samples, suggesting that FCV may not have equivalent physical properties to those of human norovirus. One basic science study demonstrated a procedure to eliminate norovirus (genogroup II) from a melamine substrate using a two step process - a cleaning step to remove gross fecal material, followed by a 5000-ppm hypochlorite product with a one minute contact time.¹⁹⁴ Cleaning with a detergent, or using a disinfectant alone failed to eliminate the virus.

Moderate quality evidence was available on the impact of disinfection agents on the human norovirus surrogate, FCV.^{185,187,188,190-192,198-200} Nine basic science studies evaluated the activity of several disinfectants against FCV.^{185,187,188,190-192,198-200} Only a single study showed equivalent efficacy between a quaternary ammonium compound and 1000 ppm hypochlorite on non-porous surfaces.¹⁸⁸ In contrast, selected quaternary ammonium based-products, ethanol, and a 1% anionic detergent were all unable to inactivate FCV beyond a reduction of 1.25 in the log₁₀ of virus, compared to 1000 ppm and 5000 ppm hypochlorite, 0.8% iodine, and 0.5% glutaraldehyde products.²⁰⁰ 4% organic acid, 1% peroxide, and >2% aldehyde products showed inactivation of FCV but only with impractical contact times exceeding 1 hour.¹⁸⁷

Studies of disinfecting non-porous surfaces and hands evaluated the efficacy of varying dilutions of ethanol and isopropanol and determined that 70-90% ethanol was more efficacious at inactivating FCV compared to isopropanol, but unable to achieve a reduction of 3 in the log₁₀ of the viral titer (99.9%), even after 10 minutes of contact.¹⁹¹ Other studies have shown that combinations of phenolic and quaternary ammonium compounds and peroxyacetic acid were only effective against FCV if they exceeded the manufacturers' recommended concentrations by a factor of 2 to 4.¹⁹⁹ The included basic science studies agents demonstrating complete inactivation of FCV were those containing hypochlorite, glutaraldehyde, hydrogen peroxide, iodine, or >5% sodium bicarbonate active ingredients. Not all of these products are feasible for use in healthcare settings.

In applications to various fabrics (100% cotton, 100% polyester, and cotton blends), FCV was inactivated completely by 2.6% glutaraldehyde, and showed >90% reductions of FCV titers when phenolics, 2.5% or 10% sodium bicarbonate, or 70% isopropanol were evaluated.¹⁹⁰ In carpets consisting of olefin, polyester, nylon, or blends, 2.6% glutaraldehyde demonstrated >99.7% inactivation of FCV, with other disinfectants showing moderate to modest reductions in FCV titers.¹⁹⁰ The experimental use of monochloramine as an alternative disinfectant to free chlorine in water treatment systems only demonstrated modest reductions in viral titer after 3 hours of contact time. The literature search did not evaluate publications using newer methods for environmental disinfection, such as ozone mist from a humidifying device, fumigation, UV irradiation, and fogging.

This search strategy was unable to find well-designed studies that compared virucidal efficacy of products on human norovirus, FCV, or other surrogate models among commonly used hospital disinfectants agents to establish practical standards, conditions, concentrations, and contact times. Ongoing laboratory studies are now exploring murine models as a surrogate that may exhibit greater similarity to human norovirus than FCV. Forthcoming research using this animal model may provide clearer direction regarding which disinfectants reduce norovirus environmental contamination from healthcare environments, while balancing occupational safety issues with the practicality of efficient and ready-to-use products.

Q3.D Medications

There was very low-quality evidence suggesting that select medications may reduce the risk of illness or attenuate symptoms of norovirus.^{202,203} Among elderly psychiatric patients, those on antipsychotic drugs plus trihexyphenidyl or benztropine were less likely to become symptomatic, as were those taking psyllium hydrophilic mucilloid.²⁰³ The pharmacodynamics to explain this outcome are unknown, and it is likely that these medications may either be a surrogate marker for another biologically plausible protective factor, or may impact norovirus through central or local effects on gastrointestinal motility. Those who received nitazoxanide, an anti-protozoal drug, were more likely to exhibit longer periods of norovirus illness than those patients who received placebo.²⁰² The search strategy used in this review did not identify research that considered the effect of anti-peristaltics on the duration or outcomes of norovirus infection.

Q3 Recommendations

3.A.1 Consider extending the duration of isolation or cohorting precautions for outbreaks among infants and young children (e.g., under 2 years), even after resolution of symptoms, as there is a potential for prolonged viral shedding and environmental contamination. Among infants, there is evidence to consider extending contact precautions for up to 5 days after the resolution of symptoms. **(Category II)** (Key Question 3A)

3.A.2 Further research is needed to understand the correlation between prolonged shedding of norovirus and the risk of infection to susceptible patients **(No recommendation/unresolved issue)** (Key Question 3A)

3.B.1 Perform routine cleaning and disinfection of frequently touched environmental surfaces and equipment in isolation and cohorted areas, as well as high-traffic clinical areas. Frequently touched surfaces include, but are not limited to, commodes, toilets, faucets, hand/bedrailing, telephones, door handles, computer equipment, and kitchen preparation surfaces. **(Category IB)** (Key Question 3B)

3.B.2 Remove all shared or communal food items for patients or staff from clinical areas for the duration of the outbreak. **(Category IB)** (Key Question 3B)

3.C.1.a. Actively promote adherence to hand hygiene among healthcare personnel, patients, and visitors in patient care areas affected by outbreaks of norovirus gastroenteritis. **(Category IB)** (Key Question 3C)

3.C.1.b. During outbreaks, use soap and water for hand hygiene after providing care or having contact with patients suspected or confirmed with norovirus gastroenteritis. **(Category IB)** (Key Question 3C)

3.C.1.b.1. For all other hand hygiene indications (e.g., when hands are not visibly soiled and have not been in contact with diarrheal patients, contaminated surfaces, or other body fluids) refer to the 2002 HICPAC Guideline for Hand Hygiene in Health-Care Settings (<http://www.cdc.gov/mmwr/PDF/rr/rr51116.pdf>), which includes the indications for use of FDA-compliant alcohol based hand sanitizer. **(Category IB)** (Key Question 3C)

3.C.1.b.2. Consider ethanol-based hand sanitizers (60-95%) as the preferred active agent compared to other alcohol or non-alcohol based hand sanitizer products during outbreaks of norovirus gastroenteritis. **(Category II)** (Key Question 3C)

3.C.1.b.3. Further research is required to directly evaluate the efficacy of alcohol-based hand sanitizers against human strains of norovirus, or against a surrogate virus with properties convergent with human strains of norovirus. **(No recommendation/unresolved issue)** (Key Question 3C)

3.C.2.a Use a surgical or procedure mask and eye protection or a full face shield if there is an anticipated risk of splashes to the face during the care of patients, particularly among those who are vomiting. **(Category IB)** (Key Question 3C)

3.C.3 Develop and adhere to sick leave policies for healthcare personnel who have symptoms consistent with norovirus infection. **(Category IB)** (Key Question 3C)

3.C.3.a Exclude ill personnel from work for a minimum of 48 hours after the resolution of symptoms. Once personnel return to work, the importance of performing frequent hand hygiene should be reinforced, especially before and after each patient contact. **(Category IB)** (Key Question 3C)

3.C.4.a During outbreaks, place patients with norovirus gastroenteritis on Contact Precautions for a minimum of 48 hours after the resolution of symptoms to prevent further transmission. **(Category IB)** (Key Question 3C)

3.C.4.b When patients with norovirus gastroenteritis cannot be accommodated in single occupancy rooms, efforts should be made to separate them from asymptomatic patients. Dependent upon facility characteristics, approaches for cohorting patients during outbreaks may include placing patients in multi-occupancy rooms, or designating patient care areas or contiguous sections within a facility for patient cohorts. **(Category IB)** (Key Question 3C)

3.C.4.c Consider minimizing patient movements within a ward or unit during norovirus gastroenteritis outbreaks. **(Category II)** (Key Question 3C)

3.C.4.c.1 Consider restricting symptomatic and recovering patients from leaving the patient-care area

unless it is for essential care or treatment to reduce the likelihood of environmental contamination and transmission of norovirus in unaffected clinical areas. **(Category II)** (Key Question 3C)

3.C.4.d Consider suspending group activities (e.g., dining events) for the duration of a norovirus outbreak. **(Category II)** (Key Question 3C)

3.C.5.a Establish protocols for staff cohorting in the event of an outbreak of norovirus gastroenteritis. Ensure staff care for one patient cohort on their ward and do not move between patient cohorts (e.g., patient cohorts may include symptomatic, asymptomatic exposed, or asymptomatic unexposed patient groups). **(Category IB)** (Key Question 3C)

3.C.5.b Staff who have recovered from recent suspected norovirus infection associated with this outbreak may be best suited to care for symptomatic patients until the outbreak resolves. **(Category II)** (Key Question 3C)

3.C.5.c Exclude non-essential staff, students, and volunteers from working in areas experiencing outbreaks of norovirus gastroenteritis. **(Category IB)** (Key Question 3C)

3.C.6 Consider the closure of wards to new admissions or transfers as a measure to attenuate the magnitude of an outbreak of norovirus gastroenteritis. The threshold for ward closure varies and depends on risk assessments by infection prevention personnel and facility leadership. **(Category II)** (Key Question 3C)

3.C.7.a Establish visitor policies for acute gastroenteritis (e.g., norovirus) outbreaks. **(Category IB)** (Key Question 3C)

3.C.7.b Restrict non-essential visitors from affected areas of the facility during outbreaks of norovirus gastroenteritis. **(Category IB)** (Key Question 3C)

3.C.7.b.1 For those affected areas where it is necessary to have continued visitor privileges during outbreaks, screen and exclude visitors with symptoms consistent with norovirus infection and ensure that they comply with hand hygiene and Contact Precautions. **(Category IB)** (Key Question 3C)

3.C.8.a Provide education to staff, patients, and visitors, including recognition of norovirus symptoms, preventing infection, and modes of transmission upon the recognition and throughout the duration of a norovirus gastroenteritis outbreak. **(Category IB)** (Key Question 3C)

3.C.8.b Consider providing educational sessions and making resources available on the prevention and management of norovirus before outbreaks occur, as part of annual trainings, and when sporadic cases are detected. **(Category II)** (Key Question 3C)

3.C.9.a Begin active case-finding when a cluster of acute gastroenteritis cases is detected in the healthcare facility. Use a specified case definition, and implement line lists to track both exposed and symptomatic patients and staff. Collect relevant epidemiological, clinical, and demographic data as well as information on patient location and outcomes. **(Category IB)** (Key Question 3C)

3.C.9.b As with all outbreaks, notify appropriate local and state health departments, as required by state and local public health regulations, if an outbreak of norovirus gastroenteritis is suspected. **(Category IC)** (Key Question 3C)

3.C.10 Develop written policies that specify the chains of communication needed to manage and report outbreaks of norovirus gastroenteritis. Key stakeholders such as clinical staff, environmental services, laboratory administration, healthcare facility administration and public affairs, as well as state or local public

health authorities, should be included in the framework. **(Category IB)** (Key Question 3C)

3.C.10.a Provide timely communication to personnel and visitors when an outbreak of norovirus gastroenteritis is identified and outline what policies and provisions need to be followed to prevent further transmission **(Category IB)** (Key Question 3C)

3.C.11 Consider limiting transfers to those for which the receiving facility is able to maintain Contact Precautions; otherwise, it may be prudent to postpone transfers until patients no longer require Contact Precautions. During outbreaks, medically suitable individuals recovering from norovirus gastroenteritis can be discharged to their place of residence. **(Category II)** (Key Question 3C)

3.C.12.a Clean and disinfect shared equipment between patients using EPA-registered products with label claims for use in healthcare. Follow the manufacturer's recommendations for application and contact times. The EPA lists products with activity against norovirus on their website (<http://www.epa.gov/oppad001/chemregindex.htm>). **(Category IC)** (Key Question 3C)

3.C.12.b.1 Increase the frequency of cleaning and disinfection of patient care areas and frequently touched surfaces during outbreaks of norovirus gastroenteritis (e.g., consider increasing ward/unit level cleaning to twice daily to maintain cleanliness, with frequently touched surfaces cleaned and disinfected three times daily using EPA-approved products for healthcare settings). **(Category IB)** (Key Question 3C)

3.C.12.b.2 Clean and disinfect surfaces starting from the areas with a lower likelihood of norovirus contamination (e.g., tray tables, counter tops) to areas with highly contaminated surfaces (e.g., toilets, bathroom fixtures). Change mop heads when a new bucket of cleaning solution is prepared, or after cleaning large spills of emesis or fecal material. **(Category IB)** (Key Question 3C)

3.C.12.c.1 Consider discarding all disposable patient-care items and laundering unused linens from patient rooms after patients on isolation for norovirus gastroenteritis are discharged or transferred. Facilities can minimize waste by limiting the number of disposable items brought into rooms/areas on Contact Precautions. **(Category II)** (Key Question 3C)

3.C.12.c.2 No additional provisions for using disposable patient service items such as utensils or dishware are suggested for patients with symptoms of norovirus infection. Silverware and dishware may undergo normal processing and cleaning using standard procedures. **(Category II)** (Key Question 3C)

3.C.12.c.3 Use Standard Precautions for handling soiled patient-service items or linens, including the use of appropriate PPE. **(Category IB)** (Key Question 3C)

3.C.12.d.1 Consider avoiding the use of upholstered furniture and rugs or carpets in patient care areas, as these objects are difficult to clean and disinfect completely. If this option is not possible, immediately clean soilage, such as emesis or fecal material, from upholstery, using a manufacturer-approved cleaning agent or detergent. Opt for seating in patient-care areas that can withstand routine cleaning and disinfection. **(Category II)** (Key Question 3C)

3.C.12.d.2 Consider steam cleaning of upholstered furniture in patient rooms upon discharge. Consult with manufacturer's recommendations for cleaning and disinfection of these items. Consider discarding items that cannot be appropriately cleaned/disinfected. **(Category II)**(Key Question 3C)

3.C.12.d.3 During outbreaks, change privacy curtains when they are visibly soiled and upon patient discharge or transfer. **(Category IB)** (Key Question 3C)

3.C.12.d.4 Handle soiled linens carefully, without agitating them, to avoid dispersal of virus. Use Standard

Precautions, including the use of appropriate PPE (e.g., gloves and gowns), to minimize the likelihood of cross-contamination. **(Category IB)** (Key Question 3C)

3.C.12.d.5 Double bagging, incineration, or modifications for laundering are not indicated for handling or processing soiled linen. **(Category II)** (Key Question 3C)

3.C.12.e.1 Clean surfaces and patient equipment prior to the application of a disinfectant. Follow the manufacturer's recommendations for optimal disinfectant dilution, application, and surface contact time with an EPA-approved product with claims against norovirus. **(Category IC)** (Key Question 3C)

3.C.12.e.2 More research is required to clarify the effectiveness of cleaning and disinfecting agents against norovirus, either through the use of surrogate viruses or the development of human norovirus culture system. **(No recommendation/unresolved issue)** (Key Question 3C)

3.C.12.e.3 More research is required to clarify the effectiveness and reliability of fogging, UV irradiation, and ozone mists to reduce norovirus environmental contamination. **(No recommendation/unresolved issue)** (Key Question 3C)

3.C.12.e.4 More research is required to evaluate the virucidal capabilities of alcohol-based as well as non-alcohol based hand sanitizers against norovirus. **(No recommendation/unresolved issue)** (Key Question 3C)

3.D Further research is required to evaluate the utility of medications that may attenuate the duration and severity of norovirus illness. **(No recommendation/unresolved issue)** (Key Question 3D)

References

1. Green KY, Ando T, Balayan, et al. Taxonomy of the caliciviruses. *J Infect Dis.* 2000;181 (Supplement 2):S322-30.
2. Atmar RL, Estes MK. The epidemiologic and clinical importance of norovirus infection. *Gastroenterol Clin North Am.* 2006;35(2):275-290.
3. Kaplan JE, Schonberger LB, Varano G, Jackman N, Bied J, Gary GW. An outbreak of acute nonbacterial gastroenteritis in a nursing home. Demonstration of person-to-person transmission by temporal clustering of cases. *Am J Epidemiol.* 1982;116(6):940-948.
4. Mead PS, Slutsker L, Dietz V, et al. Food-related illness and death in the United States. *Emerg Infect Dis.* 1999;5(5):607-625.
5. Widdowson MA, Meltzer MI, Zhang X, Bresee JS, Parashar UD, Glass RI. Cost-effectiveness and potential impact of rotavirus vaccination in the United States. *Pediatrics.* 2007;119(4):684-697.
6. Patel MM, Widdowson MA, Glass RI, Akazawa K, Vinje J, Parashar UD. Systematic literature review of role of noroviruses in sporadic gastroenteritis. *Emerg Infect Dis.* 2008;14(8):1224-1231.
7. Fankhauser RL, Monroe SS, Noel JS, et al. Epidemiologic and molecular trends of "Norwalk-like viruses" associated with outbreaks of gastroenteritis in the United States. *J Infect Dis.* 2002;186(1):1-7.
8. Widdowson MA, Cramer EH, Hadley L, et al. Outbreaks of acute gastroenteritis on cruise ships and on land: identification of a predominant circulating strain of norovirus--United States, 2002. *J Infect Dis.* 2004;190(1):27-36.
9. Siebenga JJ, Vennema H, Zheng DP, et al. Norovirus illness is a global problem: emergence and spread of norovirus GII.4 variants, 2001-2007. *J Infect Dis.* 2009;200(5):802-812.
10. Centers for Disease Control and Prevention (CDC). Norovirus activity--United States, 2006-2007. *MMWR.* 2007;56(33):842-846.
11. Centers for Disease Control and Prevention (CDC). Surveillance for foodborne disease outbreaks - United States, 2006. *MMWR.* 2009;58(22):609-615.

12. Caul EO. Small round structured viruses: airborne transmission and hospital control. *Lancet*. 1994;343(8908):1240-1242.
13. Hutson AM, Atmar RL, Estes MK. Norovirus disease: changing epidemiology and host susceptibility factors. *Trends Microbiol*. 2004;12(6):279-287.
14. Donaldson EF, Lindesmith LC, Lobue AD, Baric RS. Norovirus pathogenesis: mechanisms of persistence and immune evasion in human populations. *Immunol Rev*. 2008;225:190-211.
15. Widerlite L, Trier JS, Blacklow NR, Schreiber DS. Structure of the gastric mucosa in acute infectious bacterial gastroenteritis. *Gastroenterology*. 1975;68(3):425-430.
16. Kaplan JE, Feldman R, Campbell DS, Lookabaugh C, Gary GW. The frequency of a Norwalk-like pattern of illness in outbreaks of acute gastroenteritis. *Am J Public Health*. 1982;72(12):1329-1332.
17. Rabenau HF, Sturmer M, Buxbaum S, Walczok A, Preiser W, Doerr HW. Laboratory diagnosis of norovirus: which method is the best?. *Intervirology*. 2003;46(4):232-238.
18. Trujillo A, McCaustland K, Zheng D, et al. Use of TaqMan real-time reverse transcription-PCR for rapid detection, quantification, and typing of norovirus. *Journal of Clinical Microbiology*. American Society for Microbiology (ASM), Washington, USA. 2006;44(4):1405-1412. <http://dx.doi.org/10.1128/JCM.44.4.1405-1412.2006>.
19. Said MA, Perl TM, Sears CL. Healthcare epidemiology: gastrointestinal flu: norovirus in health care and long-term care facilities. *Clin Infect Dis*. 2008;47(9):1202-1208.
20. Cannon JL, Papafragkou E, Park GW, Osborne J, Jaykus LA, Vinje J. Surrogates for the study of norovirus stability and inactivation in the environment: a comparison of murine norovirus and feline calicivirus. *J Food Prot*. 2006;69(11):2761-2765.
21. Guyatt GH, Oxman AD, Vist GE, et al. GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. *BMJ*. 2008;336(7650):924-926.
22. Guyatt GH, Oxman AD, Kunz R, et al. What is "quality of evidence" and why is it important to clinicians? *BMJ*. 2008;336(7651):995-998.
23. Guyatt GH, Oxman AD, Kunz R, et al. Going from evidence to recommendations. *BMJ*. 2008;336(7652):1049-1051.
24. Schunemann HJ, Oxman AD, Brozek J, et al. Grading quality of evidence and strength of recommendations for diagnostic tests and strategies. *BMJ*. 2008;336(7653):1106-1110.
25. Vessel Sanitation Program Construction Guideline, July 2005. Available at: <http://www.cdc.gov/nceh/vsp/ConstructionGuidelines/ConstructionGuidelines2005.pdf>. Accessed September 24, 2007.
26. Vessel Sanitation Program Operations Manual, August 2005. Available at: <http://www.cdc.gov/nceh/vsp/operationsmanual/OPSMannual2005.pdf>. Accessed September 24, 2007.
27. Guidance for the Management of Norovirus Infection in Cruise Ships. July 2007. Available at: http://www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1206520183347. Accessed September 24, 2007.
28. Viral Gastroenteritis: Leeds Teaching Hospitals Trust Infection Control Policies. 2008. Available at: http://www.leedsteachinghospitals.com/sites/infection_control/documents/VGRevisedJan08.pdf. Accessed September 24, 2007.
29. National Guidelines on the Management of Outbreaks of Norovirus Infection in Healthcare Settings. 2003. Available at: <http://www.hpsc.ie/hpsc/A-Z/Gastroenteric/ViralGastroenteritis/Publications/File,1194,en.pdf>. Accessed September 24, 2007.
30. Netherlands: Norovirus Guidelines for Cruiseships and Hotels. 2007. Accessed September 24, 2007.
31. Gastroenteritis in an Institution Response Protocol for New South Wales Public Health Units, NSW Health 2005. (updated 2010). Available at: <http://www.health.nsw.gov.au/factsheets/guideline/gastro.html>. Accessed September 24, 2007.
32. Guidelines for the Management of Norovirus Outbreaks in Hospitals and Elderly Care Institutions, July 2007, Auckland Regional Public Health Service, New Zealand. (updated 2008). Available at: http://www.arphs.govt.nz/notifiable/downloads/Norovirus_Guidelines_2008.pdf. Accessed September 24, 2007.
33. Viral Gastroenteritis Outbreaks: Information for Supervisors in the Child Care and Hospitality Industries. Queensland Health, Australia. (updated 2010). Available at: <http://www.health.qld.gov.au/ph/documents/cdb/26888.pdf>. Accessed September 24, 2007.

34. Aide-Memoire for Managing Norovirus Outbreaks in Healthcare Settings Scottish Centre for Infection and Environmental Health, National Services Scotland. 2004. Available at: <http://www.hps.scot.nhs.uk/giz/publicationsdetail.aspx?id=22582>. Accessed September 24, 2007.
35. The Identification and Management of Outbreaks of Norovirus Infection in Tourists and Leisure Industry Settings. 2005. Available at: http://www.sefton.gov.uk/pdf/epd_norovirusguide.pdf. Accessed September 24, 2007.
36. Guidelines for the Management of Infectious Gastroenteritis in Aged Care Facilities in South Australia. 2005. Available at: <http://www.publications.health.sa.gov.au/cgi/viewcontent.cgi?article=1014&context=cdc>. Accessed September 24, 2007.
37. General Guidelines for the Management of Viral Gastroenteritis, Community Infection Prevention and Control Policy and Procedure, Gloucestershire Primary Care Trust, 2008. Available at: <http://www.glospct.nhs.uk/pdf/policies/infectioncontrol/gpct%20viral%20gastroenteritis.pdf>. Accessed September 24, 2007.
38. Recommendations for the Prevention and Control of Viral Gastroenteritis Outbreaks in California Long-Term Care Facilities. 2006 Available at: <http://www.cdph.ca.gov/pubsforms/Guidelines/Documents/PCofViralGastroenteritisOutbreaks.pdf>. Accessed September 24, 2007.
39. Control of Viral Gastroenteritis Outbreaks in Illinois Long-Term Care Facilities. Illinois Department of Public Health. 2006. Available at: <http://www.co.mchenry.il.us/departments/health/pdfDocs/PHS/CD/NoroLongFacGuide.pdf>. Accessed September 24, 2007.
40. Norovirus Prevention Guidance for Institutions/Facilities. Virginia Department of Health, January 2007. Available at: http://www.flpic.com/Norovirus_Prev_Guidance_Institutions_2007.pdf. Accessed September 24, 2007.
41. Viral Gastroenteritis Outbreak Guidelines for Child Care Facilities. Washoe County District Health Department, Reno, Nevada. http://www.co.washoe.nv.us/repository/files//4/Childcare_guidelines_for_norovirus.pdf. Accessed September 24, 2007
42. Viral Gastroenteritis Outbreak Guidelines for Community Living Facilities. Washoe County District Health Department, Reno, Nevada. 2005. Available at: <http://www.co.washoe.nv.us/repository/files/4/Guidelines%20for%20Norovirus%20in%20ECFs.pdf>. Accessed September 24, 2007.
43. Guidelines for the Epidemiological Investigation of Gastroenteritis Outbreaks in Long Term Care Facilities, Maryland Department of Health and Mental Hygiene. Revised 2001. Available at: <http://ideha.dhmm.maryland.gov/pdf/guidelines/gastroenteritis.aspx>. Accessed September 24, 2007.
44. Recommendations for the Control of Viral Gastroenteritis Outbreaks in Long-Term Care Facilities and Viral Gastroenteritis Outbreaks in Nursing Homes or Long-Term Care Facilities Guidelines for Environmental Decontamination. Georgia Department of Community Health. Available at: http://health.state.ga.us/pdfs/epi/notifiable/outbreaks/Norovirus%20cleaning%20guidelines_LTC.pdf. Accessed September 24, 2007.
45. Recommendations for the Prevention and Control of Viral Gastroenteritis Outbreaks in Wisconsin Long-Term Care Facilities. Wisconsin Division of Public Health. (updated 2009). Available at: http://www.dhs.wisconsin.gov/rl_dsl/Providers/norovirusRecoLTCF09.pdf. Accessed September 24, 2007.
46. Norovirus (Viral Gastroenteritis) Control Measures for Skilled Nursing Facilities. Los Angeles County Public Health. 2006. Available at: http://www.publichealth.lacounty.gov/acd/docs/Norovirus/NorovirusControlMeasures_12_1_06.pdf. Accessed September 24, 2007.
47. Norovirus (Viral Gastroenteritis): Information Packet for Nursing Facilities. Washington State Department of Health, May 2005. Available at: <http://www.co.thurston.wa.us/health/personalhealth/communicabledisease/DrYuUpdates/PDF/2011noropacket.pdf>. Accessed September 24, 2007.
48. Viral Gastroenteritis Guidance. Royal Devon and Exeter. NHS, August 2007. (revised July 2009). Available at: http://www.rdehospital.nhs.uk/docs/patients/services/infection_control/Viral%20gastroenteritis-Aug2009.pdf. Accessed September 24, 2007.
49. Norovirus Infections. Public Health Importance and Outbreak Management. Public Health-Seattle and King County. Updated March 2010. Available at:

<http://www.kingcounty.gov/healthservices/health/communicable/diseases/NorovirusControl.aspx>. Accessed September 24, 2007.

50. von Elm E, Altman DG, Egger M, et al. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *Ann Intern Med*. 2007;147(8):573-577.
51. Shea BJ, Grimshaw JM, Wells GA, et al. Development of AMSTAR: a measurement tool to assess the methodological quality of systematic reviews. *BMC Med Res Methodol*. 2007;7:10.
52. Sanderson S, Tatt ID, Higgins JP. Tools for assessing quality and susceptibility to bias in observational studies in epidemiology: a systematic review and annotated bibliography. *Int J Epidemiol*. 2007;36(3):666-676.
53. Moher D, Schulz KF, Altman D, CONSORT Group (Consolidated Standards of Reporting Trials). The CONSORT statement: revised recommendations for improving the quality of reports of parallel-group randomized trials. *JAMA*. 2001;285(15):1987-1991.
54. Jadad AR, Moore RA, Carroll D, et al. Assessing the quality of reports of randomized clinical trials: is blinding necessary? *Control Clin Trials*. 1996;17(1):1-12.
55. Yale Center for Medical Informatics. 2009. Available at: <http://ycmi.med.yale.edu/>. Accessed April 27, 2011.
56. Mattner F, Mattner L, Borck HU, Gastmeier P. Evaluation of the impact of the source (patient versus staff) on nosocomial norovirus outbreak severity. *Infect Control Hosp Epidemiol*. 2005;26(3):268-272.
57. Mattner F, Sohr D, Heim A, Gastmeier P, Vennema H, Koopmans M. Risk groups for clinical complications of norovirus infections: an outbreak investigation. *Clin Microbiol Infect*. 2006;12(1):69-74.
58. Lopman BA, Reacher MH, Vipond IB, Sarangi J, Brown DW. Clinical manifestation of norovirus gastroenteritis in health care settings. *Clin Infect Dis*. 2004;39(3):318-324.
59. Lee N, Chan MCW, Wong B, et al. Fecal viral concentration and diarrhea in norovirus gastroenteritis. *Emerg Infect Dis*. 2007;13(9):1399-1401. <http://www.cdc.gov/eid/content/13/9/pdfs/1399.pdf>.
60. Rodriguez-Guillen L, Vizzi E, Alcalá AC, Pujol FH, Liprandi F, Ludert JE. Calicivirus infection in human immunodeficiency virus seropositive children and adults. *J Clin Virol*. 2005;33(2):104-109.
61. de Wit MA, Koopmans MP, van Duynhoven YT. Risk factors for norovirus, Sapporo-like virus, and group A rotavirus gastroenteritis. *Emerg Infect Dis*. 2003;9(12):1563-1570.
62. Gotz H, Ekdahl K, Lindback J, de Jong B, Hedlund KO, Giesecke J. Clinical spectrum and transmission characteristics of infection with Norwalk-like virus: findings from a large community outbreak in Sweden. *Clin Infect Dis*. 2001;33(5):622-628.
63. Oppermann H, Mueller B, Takkinen J, Klauditz W, Schreier E, Ammon A. An outbreak of viral gastroenteritis in a mother-and-child health clinic. *Int J Hyg Environ Health*. 2001;203(4):369-373.
64. Sharp TW, Hyams KC, Watts D, et al. Epidemiology of Norwalk virus during an outbreak of acute gastroenteritis aboard a US aircraft carrier. *J Med Virol*. 1995;45(1):61-67.
65. Thea DM, Glass R, Grohmann GS, et al. Prevalence of enteric viruses among hospital patients with AIDS in Kinshasa, Zaire. *Trans R Soc Trop Med Hyg*. 1993;87(3):263-266.
66. Marx A, Shay DK, Noel JS, et al. An outbreak of acute gastroenteritis in a geriatric long-term-care facility: combined application of epidemiological and molecular diagnostic methods. *Infect Control Hosp Epidemiol*. 1999;20(5):306-311.
67. Caceres VM, Kim DK, Bresee JS, et al. A viral gastroenteritis outbreak associated with person-to-person spread among hospital staff. *Infect Control Hosp Epidemiol*. 1998;19(3):162-167.
68. Cegielski JP, Msengi AE, Miller SE. Enteric viruses associated with HIV infection in Tanzanian children with chronic diarrhea. *Pediatr AIDS HIV Infect*. 1994;5(5):296-299.
69. Halperin T, Vennema H, Koopmans M, et al. No association between histo-blood group antigens and susceptibility to clinical infections with genogroup II norovirus. *J Infect Dis*. 2008;197(1):63-65.
70. Hutson AM, Airaud F, LePendu J, Estes MK, Atmar RL. Norwalk virus infection associates with secretor status genotyped from sera. *J Med Virol*. 2005;77(1):116-120.
71. Thorven M, Grahn A, Hedlund KO, et al. A homozygous nonsense mutation (428G-->A) in the human secretor (FUT2) gene provides resistance to symptomatic norovirus (GGII) infections. *J Virol*. 2005;79(24):15351-15355.
72. Lindesmith L, Moe C, Marionneau S, et al. Human susceptibility and resistance to Norwalk virus infection. *Nat Med*. 2003;9(5):548-553.
73. Hutson AM, Atmar RL, Graham DY, Estes MK. Norwalk virus infection and disease is associated with ABO histo-blood group type. *J Infect Dis*. 2002;185(9):1335-1337.

74. Graham DY, Jiang X, Tanaka T, Opekun AR, Madore HP, Estes MK. Norwalk virus infection of volunteers: new insights based on improved assays. *J Infect Dis.* 1994;170(1):34-43.
75. Nakata S, Chiba S, Terashima H, Yokoyama T, Nakao T. Humoral immunity in infants with gastroenteritis caused by human calicivirus. *J Infect Dis.* 1985;152(2):274-279.
76. Parrino TA, Schreiber DS, Trier JS, Kapikian AZ, Blacklow NR. Clinical immunity in acute gastroenteritis caused by Norwalk agent. *N Engl J Med.* 1977;297(2):86-89.
77. Fretz R, Svoboda P, Schorr D, Tanner M, Baumgartner A. Risk factors for infections with Norovirus gastrointestinal illness in Switzerland. *Eur J Clin Microbiol Infect Dis.* 2005;24(4):256-261.
78. Meyer E, Ebner W, Scholz R, Dettkenkofer M, Daschner FD. Nosocomial outbreak of norovirus gastroenteritis and investigation of ABO histo-blood group type in infected staff and patients. *J Hosp Infect.* 2004;56(1):64-66.
79. Centers for Disease Control and Prevention (CDC). Norovirus outbreak in an elementary school--District of Columbia, February 2007. *MMWR.* 2008;56(51-52):1340-1343.
80. Centers for Disease Control and Prevention (CDC). Multistate outbreak on norovirus gastroenteritis among attendees at a family reunion -- Grant County, West Virginia, October 2006. *MMWR.* 2007;56(27):673-678.
81. Costas L, Vilella A, Llupia A, Bosch J, Jimenez de Anta MT, Trilla A. Outbreak of norovirus gastroenteritis among staff at a hospital in Barcelona, Spain, September 2007. *Euro Surveill.* 2007;12(11):E071122.5.
82. Lopman BA, Andrews N, Sarangi J, Vipond IB, Brown DW, Reacher MH. Institutional risk factors for outbreaks of nosocomial gastroenteritis: survival analysis of a cohort of hospital units in South-west England, 2002-2003. *J Hosp Infect.* 2005;60(2):135-143.
83. Evans MR, Meldrum R, Lane W, et al. An outbreak of viral gastroenteritis following environmental contamination at a concert hall. *Epidemiol Infect.* 2002;129(2):355-360.
84. Lachlan M, Licence K, Oates K, Vaughan S, Hill R. Practical lessons from the management of an outbreak of small round structured virus (Norwalk-like virus) gastroenteritis. *Commun Dis Public Health.* 2002;5(1):43-47.
85. Love SS, Jiang X, Barrett E, Farkas T, Kelly S. A large hotel outbreak of Norwalk-like virus gastroenteritis among three groups of guests and hotel employees in Virginia. *Epidemiol Infect.* 2002;129(1):127-132.
86. Anderson AD, Garrett VD, Sobel J, et al. Multistate outbreak of Norwalk-like virus gastroenteritis associated with a common caterer. *Am J Epidemiol.* 2001;154(11):1013-1019.
87. Cunney RJ, Costigan P, McNamara EB, et al. Investigation of an outbreak of gastroenteritis caused by Norwalk-like virus, using solid phase immune electron microscopy. *J Hosp Infect.* 2000;44(2):113-118.
88. Marks PJ, Vipond IB, Carlisle D, Deakin D, Fey RE, Caul EO. Evidence for airborne transmission of Norwalk-like virus (NLV) in a hotel restaurant. *Epidemiol Infect.* 2000;124(3):481-487.
89. Lo SV, Connolly AM, Palmer SR, Wright D, Thomas PD, Joynson D. The role of the pre-symptomatic food handler in a common source outbreak of food-borne SRSV gastroenteritis in a group of hospitals. *Epidemiol Infect.* 1994;113(3):513-521.
90. Patterson T, Hutchings P, Palmer S. Outbreak of SRSV gastroenteritis at an international conference traced to food handled by a post-symptomatic caterer. *Epidemiol Infect.* 1993;111(1):157-162.
91. Alexander WJ, Holmes JR, Shaw JF, Riley WE, Roper WL. Norwalk virus outbreak at a college campus. *South Med J.* 1986;79(1):33-36.
92. Wit MA, Widdowson V, H., Bruin Ed, Fernandes T, Koopmans M. Large outbreak of norovirus: the baker who should have known better. *Journal of Infection.* Elsevier, Amsterdam, Netherlands. 2007;55(2):188-193.
93. Centers for Disease Control and Prevention (CDC). Norovirus outbreak associated with ill food-service workers-- Michigan, January-February 2006. *MMWR.* 2007;56(46):1212-1216.
94. Rizzo C, Di Bartolo I, Santantonio M, et al. Epidemiological and virological investigation of a Norovirus outbreak in a resort in Puglia, Italy. *BMC Infect Dis.* 2007;7:135.
95. Schmid D, Stuger HP, Lederer I, et al. A foodborne norovirus outbreak due to manually prepared salad, Austria 2006. *Infection.* 2007;35(4):232-239.
96. Payne J, Hall M, Lutzke M, Armstrong C, King J. Multisite outbreak of norovirus associated with a franchise restaurant - Kent County, Michigan, May 2005. *MMWRt.* 2006;55(14):395-397.
97. Grotto I, Huerta M, Balicer RD, et al. An outbreak of norovirus gastroenteritis on an Israeli military base. *Infection.* 2004;32(6):339-343.
98. Marks PJ, Vipond IB, Regan FM, Wedgwood K, Fey RE, Caul EO. A school outbreak of Norwalk-like virus: evidence for airborne transmission. *Epidemiol Infect.* 2003;131(1):727-736.

99. Stegenga J, Bell E, Matlow A. The role of nurse understaffing in nosocomial viral gastrointestinal infections on a general pediatrics ward. *Infect Control Hosp Epidemiol.* 2002;23(3):133-136.
100. Becker KM, Moe CL, Southwick KL, MacCormack JN. Transmission of Norwalk virus during football game. *N Engl J Med.* 2000;343(17):1223-1227.
101. Parashar UD, Dow L, Fankhauser RL, et al. An outbreak of viral gastroenteritis associated with consumption of sandwiches: implications for the control of transmission by food handlers. *Epidemiol Infect.* 1998;121(3):615-621.
102. McEvoy M, Blake W, Brown D, Green J, Cartwright R. An outbreak of viral gastroenteritis on a cruise ship. *Commun Dis Rep CDR Rev.* 1996;6(13):R188-92.
103. Chadwick PR, McCann R. Transmission of a small round structured virus by vomiting during a hospital outbreak of gastroenteritis. *J Hosp Infect.* 1994;26(4):251-259.
104. Reid JA, Caul EO, White DG, Palmer SR. Role of infected food handler in hotel outbreak of Norwalk-like viral gastroenteritis: implications for control. *Lancet.* 1988;2(8606):321-323.
105. Iversen AM, Gill M, Bartlett CL, Cubitt WD, McSwiggan DA. Two outbreaks of foodborne gastroenteritis caused by a small round structured virus: evidence of prolonged infectivity in a food handler. *Lancet.* 1987;2(8558):556-558.
106. White KE, Osterholm MT, Mariotti JA, et al. A foodborne outbreak of Norwalk virus gastroenteritis. Evidence for post-recovery transmission. *Am J Epidemiol.* 1986;124(1):120-126.
107. Kaplan JE, Gary GW, Baron RC, et al. Epidemiology of Norwalk gastroenteritis and the role of Norwalk virus in outbreaks of acute nonbacterial gastroenteritis. *Ann Intern Med.* 1982;96(6 Pt 1):756-761.
108. Tu ETV, Bull RA, Greening GE, et al. Epidemics of gastroenteritis during 2006 were associated with the spread of norovirus GII.4 variants 2006a and 2006b. *Clinical Infectious Diseases.* 2008;46(3):413-420.
109. Adamson WE, Gunson RN, Maclean A, Carman WF. Emergence of a new norovirus variant in Scotland in 2006. *J Clin Microbiol.* 2007;45(12):4058-4060.
110. Gallimore CI, Cubitt D, du Plessis N, Gray JJ. Asymptomatic and symptomatic excretion of noroviruses during a hospital outbreak of gastroenteritis. *J Clin Microbiol.* 2004;42(5):2271-2274.
111. Blanton LH, Adams SM, Beard RS, et al. Molecular and epidemiologic trends of caliciviruses associated with outbreaks of acute gastroenteritis in the United States, 2000-2004. *J Infect Dis.* 2006;193(3):413-421.
112. Mattison K, Karthikeyan K, Abebe M, et al. Survival of calicivirus in foods and on surfaces: experiments with feline calicivirus as a surrogate for norovirus. *J Food Prot.* 2007;70(2):500-503.
113. Lindesmith L, Moe C, Lependu J, Frelinger JA, Treanor J, Baric RS. Cellular and humoral immunity following Snow Mountain virus challenge. *J Virol.* 2005;79(5):2900-2909.
114. Fretz R, Herrmann L, Christen A, et al. Frequency of Norovirus in stool samples from patients with gastrointestinal symptoms in Switzerland. *Eur J Clin Microbiol Infect Dis.* 2005;24(3):214-216.
115. Fretz R, Svoboda P, Luthi TM, Tanner M, Baumgartner A. Outbreaks of gastroenteritis due to infections with Norovirus in Switzerland, 2001-2003. *Epidemiol Infect.* 2005;133(3):429-437.
116. Turcios RM, Widdowson MA, Sulka AC, Mead PS, Glass RI. Reevaluation of epidemiological criteria for identifying outbreaks of acute gastroenteritis due to norovirus: United States, 1998-2000. *Clin Infect Dis.* 2006;42(7):964-969.
117. Duizer E, Pielaat A, Vennema H, Kroneman A, Koopmans M. Probabilities in norovirus outbreak diagnosis. *J Clin Virol.* 2007;40(1):38-42.
118. Gray JJ, Kohli E, Ruggeri FM, et al. European multicenter evaluation of commercial enzyme immunoassays for detecting norovirus antigen in fecal samples. *Clin Vaccine Immunol.* 2007;14(10):1349-1355.
119. Richards AF, Lopman B, Gunn A, et al. Evaluation of a commercial ELISA for detecting Norwalk-like virus antigen in faeces. *J Clin Virol.* 2003;26(1):109-115.
120. Khamrin P, Nguyen TA, Phan TG, et al. Evaluation of immunochromatography and commercial enzyme-linked immunosorbent assay for rapid detection of norovirus antigen in stool samples. *J Virol Methods.* 2008;147(2):360-363.
121. Wiechers C, Bissinger AL, Hamprecht K, Kimmig P, Jahn G, Poets CF. Apparently non-specific results found using a norovirus antigen immunoassay for fecal specimens from neonates. *Journal of Perinatology.* 2008;28(1):79-81.
122. Castriciano S, Luinstra K, Petrich A, et al. Comparison of the RIDASCREEN norovirus enzyme immunoassay to IDEIA NLV GI/GII by testing stools also assayed by RT-PCR and electron microscopy. *J Virol Methods.* 2007;141(2):216-219.

123. Wilhelmi de Cal I, Revilla A, del Alamo JM, Roman E, Moreno S, Sanchez-Fauquier A. Evaluation of two commercial enzyme immunoassays for the detection of norovirus in faecal samples from hospitalised children with sporadic acute gastroenteritis. *Clin Microbiol Infect.* 2007;13(3):341-343.
124. de Bruin E, Duizer E, Vennema H, Koopmans MP. Diagnosis of Norovirus outbreaks by commercial ELISA or RT-PCR. *J Virol Methods.* 2006;137(2):259-264.
125. Okitsu-Negishi S, Okame M, Shimizu Y, et al. Detection of norovirus antigens from recombinant virus-like particles and stool samples by a commercial norovirus enzyme-linked immunosorbent assay kit. *J Clin Microbiol.* 2006;44(10):3784-3786.
126. Burton-MacLeod JA, Kane EM, Beard RS, Hadley LA, Glass RI, Ando T. Evaluation and comparison of two commercial enzyme-linked immunosorbent assay kits for detection of antigenically diverse human noroviruses in stool samples. *J Clin Microbiol.* 2004;42(6):2587-2595.
127. Christen A, Fretz R, Tanner M, Svoboda P. Evaluation of a commercial ELISA kit for the detection of Norovirus antigens in human stool specimens. *Mitteilungen aus Lebensmitteluntersuchung und Hygiene. Bundesamt für Gesundheit.* 2003;94(6):594-602.
128. Gunson RN, Miller J, Carman WF. Comparison of real-time PCR and EIA for the detection of outbreaks of acute gastroenteritis caused by norovirus. *Commun Dis Public Health.* 2003;6(4):297-299.
129. Nordgren J, Bucardo F, Dienus O, Svensson L, Lindgren PE. Novel light-upon-extension real-time PCR assays for detection and quantification of genogroup I and II noroviruses in clinical specimens. *J Clin Microbiol.* 2008;46(1):164-170.
130. De Medici D, Suffredini E, Crudeli S, Ruggeri FM. Effectiveness of an RT-booster-PCR method for detection of noroviruses in stools collected after an outbreak of gastroenteritis. *J Virol Methods.* 2007;144(1-2):161-164.
131. Hymas W, Atkinson A, Stevenson J, Hillyard D. Use of modified oligonucleotides to compensate for sequence polymorphisms in the real-time detection of norovirus. *J Virol Methods.* 2007;142(1-2):10-14.
132. Logan C, O'Leary JJ, O'Sullivan N. Real-time reverse transcription PCR detection of norovirus, sapovirus and astrovirus as causative agents of acute viral gastroenteritis. *J Virol Methods.* 2007;146(1-2):36-44.
133. Menton JF, Kearney K, Morgan JG. Development of a real-time RT-PCR and Reverse Line probe Hybridisation assay for the routine detection and genotyping of Noroviruses in Ireland. *Virol J.* 2007;4:86.
134. Wolf S, Williamson WM, Hewitt J, et al. Sensitive multiplex real-time reverse transcription-PCR assay for the detection of human and animal noroviruses in clinical and environmental samples. *Appl Environ Microbiol.* 2007;73(17):5464-5470.
135. Yoda T, Suzuki Y, Yamazaki K, et al. Evaluation and application of reverse transcription loop-mediated isothermal amplification for detection of noroviruses. *J Med Virol.* 2007;79(3):326-334.
136. Antonishyn NA, Crozier NA, McDonald RR, Levett PN, Horsman GB. Rapid detection of Norovirus based on an automated extraction protocol and a real-time multiplexed single-step RT-PCR. *J Clin Virol.* 2006;37(3):156-161.
137. Hohne M, Schreier E. Detection and Characterization of Norovirus Outbreaks in Germany: Application of a One-Tube RT-PCR Using a Fluorogenic Real-Time Detection System. *J Med Virol.* 2004;72(2):312-319.
138. Rohayem J, Berger S, Juretzek T, et al. A simple and rapid single-step multiplex RT-PCR to detect Norovirus, Astrovirus and Adenovirus in clinical stool samples. *J Virol Methods.* 2004;118(1):49-59.
139. Schmid M, Oehme R, Schalasta G, Brockmann S, Kimmig P, Enders G. Fast detection of Noroviruses using a real-time PCR assay and automated sample preparation. *BMC Infect Dis.* 2004;4:15.
140. Vinje J, Vennema H, Maunula L, et al. International collaborative study to compare reverse transcriptase PCR assays for detection and genotyping of noroviruses. *J Clin Microbiol.* 2003;41(4):1423-1433.
141. Tatsumi M, Nakata S, Sakai Y, Honma S, Numata-Kinoshita K, Chiba S. Detection and differentiation of Norwalk virus by reverse transcription-polymerase chain reaction and enzyme-linked immunosorbent assay. *J Med Virol.* 2002;68(2):285-290.
142. O'Neill HJ, McCaughey C, Wyatt DE, Mitchell F, Coyle PV. Gastroenteritis outbreaks associated with Norwalk-like viruses and their investigation by nested RT-PCR. *BMC Microbiol.* 2001;1:14.
143. Jean J, D'Souza D, Jaykus LA. Transcriptional enhancement of RT-PCR for rapid and sensitive detection of Noroviruses. *FEMS Microbiol Lett.* 2003;226(2):339-345.
144. Greene SR, Moe CL, Jaykus LA, Cronin M, Grosso L, Aarle P. Evaluation of the NucliSens Basic Kit assay for detection of Norwalk virus RNA in stool specimens. *J Virol Methods.* 2003;108(1):123-131.

145. Tian P, Mandrell R. Detection of norovirus capsid proteins in faecal and food samples by a real time immuno-PCR method. *Journal of Applied Microbiology*. 2006;100(3):564-574.
146. Beuret C. A simple method for isolation of enteric viruses (noroviruses and enteroviruses) in water. *J Virol Methods*. 2003;107(1):1-8.
147. Vinje J, Hamidjaja RA, Sobsey MD. Development and application of a capsid VP1 (region D) based reverse transcription PCR assay for genotyping of genogroup I and II noroviruses. *J Virol Methods*. 2004;116(2):109-117.
148. Murata T, Katsushima N, Mizuta K, Muraki Y, Hongo S, Matsuzaki Y. Prolonged norovirus shedding in infants <or=6 months of age with gastroenteritis. *Pediatr Infect Dis J*. 2007;26(1):46-49.
149. Rockx B, De Wit M, Vennema H, et al. Natural history of human calicivirus infection: a prospective cohort study. *Clin Infect Dis*. 2002;35(3):246-253.
150. Marshall JA, Salamone S, Yuen L, Catton MG, Wright JP. High level excretion of Norwalk-like virus following resolution of clinical illness. *Pathology*. 2001;33(1):50-52.
151. Hedlund K-, Bennet R, Eriksson M, Ehrnst A. Norwalk-like virus as a cause of diarrhea in a pediatric hospital. *Clinical Microbiology and Infection*. 1998;4(8):417-421.
152. Chiba S, Sakuma Y, Kogasaka R, et al. Fecal shedding of virus in relation to the days of illness in infantile gastroenteritis due to calicivirus. *J Infect Dis*. 1980;142(2):247-249.
153. Dalling J. A review of environmental contamination during outbreaks of Norwalk-like virus. *Br J Infect Control*. 2004;5(2):9-13.
154. Wu HM, Fornek M, Schwab KJ, et al. A norovirus outbreak at a long-term-care facility: the role of environmental surface contamination. *Infect Control Hosp Epidemiol*. 2005;26(10):802-810.
155. Jones EL, Kramer A, Gaither M, Gerba CP. Role of fomite contamination during an outbreak of norovirus on houseboats. *Int J Environ Health Res*. 2007;17(2):123-131.
156. Clay S, Maherchandani S, Malik YS, Goyal SM. Survival on uncommon fomites of feline calicivirus, a surrogate of noroviruses. *Am J Infect Control*. 2006;34(1):41-43.
157. Gallimore CI, Taylor C, Gennery AR, et al. Environmental monitoring for gastroenteric viruses in a pediatric primary immunodeficiency unit. *J Clin Microbiol*. 2006;44(2):395-399.
158. Kuusi M, Nuorti JP, Maunula L, et al. A prolonged outbreak of Norwalk-like calicivirus (NLV) gastroenteritis in a rehabilitation centre due to environmental contamination. *Epidemiol Infect*. 2002;129(1):133-138.
159. Cheesbrough JS, Green J, Gallimore CI, Wright PA, Brown DW. Widespread environmental contamination with Norwalk-like viruses (NLV) detected in a prolonged hotel outbreak of gastroenteritis. *Epidemiol Infect*. 2000;125(1):93-98.
160. Schvoerer E, Bonnet F, Dubois V, et al. A hospital outbreak of gastroenteritis possibly related to the contamination of tap water by a small round structured virus. *J Hosp Infect*. 1999;43(2):149-154.
161. Green J, Wright PA, Gallimore CI, Mitchell O, Morgan-Capner P, Brown DW. The role of environmental contamination with small round structured viruses in a hospital outbreak investigated by reverse-transcriptase polymerase chain reaction assay. *J Hosp Infect*. 1998;39(1):39-45.
162. D'Souza DH, Sair A, Williams K, et al. Persistence of caliciviruses on environmental surfaces and their transfer to food. *Int J Food Microbiol*. 2006;108(1):84-91.
163. Paulson DS. The transmission of surrogate Norwalk virus - from inanimate surfaces to gloved hands: is it a threat? *Food Protection Trends*. International Association for Food Protection. 2005;25(6):450-454.
164. Lopman BA, Reacher MH, Vipond IB, et al. Epidemiology and cost of nosocomial gastroenteritis, Avon, England, 2002-2003. *Emerg Infect Dis*. 2004;10(10):1827-1834.
165. Billgren M, Christenson B, Hedlund KO, Vinje J. Epidemiology of Norwalk-like human caliciviruses in hospital outbreaks of acute gastroenteritis in the Stockholm area in 1996. *J Infect*. 2002;44(1):26-32.
166. Hansen S, Stamm-Balderjahn S, Zuschneid I, et al. Closure of medical departments during nosocomial outbreaks: data from a systematic analysis of the literature. *J Hosp Infect*. 2007;65(4):348-353.
167. Zingg W, Colombo C, Jucker T, Bossart W, Rued C. Impact of an outbreak of norovirus infection on hospital resources. *Infect Control Hosp Epidemiol*. 2005;26(3):263-267.
168. Johnston CP, Qiu H, Ticehurst JR, et al. Outbreak management and implications of a nosocomial norovirus outbreak. *Clin Infect Dis*. 2007;45(5):534-540.
169. Leuenberger S, Widdowson MA, Feilchenfeldt J, Egger R, Streuli RA. Norovirus outbreak in a district general hospital--new strain identified. *Swiss Med Wkly*. 2007;137(3-4):57-81.

170. Cheng FW, Leung TF, Lai RW, Chan PK, Hon EK, Ng PC. Rapid control of norovirus gastroenteritis outbreak in an acute paediatric ward. *Acta Paediatr.* 2006;95(5):581-586.
171. Simon A, Schildgen O, Maria Eis-Hubinger A, et al. Norovirus outbreak in a pediatric oncology unit. *Scand J Gastroenterol.* 2006;41(6):693-699.
172. Conway R, Bunt S, Mathias E, Said H. The Norovirus experience: an exercise in outbreak management at a tertiary referral hospital. *Aust Infect Control.* 2005;10(3):95, 97-102.
173. Cooper E, Blamey S. A norovirus gastroenteritis epidemic in a long-term-care facility. *Infection Control and Hospital Epidemiology.* 2005;26(3):256-258.
174. Navarro G, Sala RM, Segura F, et al. An outbreak of norovirus infection in a long-term-care unit in Spain. *Infect Control Hosp Epidemiol.* 2005;26(3):259-262.
175. Schmid D, Lederer I, Pichler AM, Berghold C, Schreier E, Allerberger F. An outbreak of Norovirus infection affecting an Austrian nursing home and a hospital. *Wien Klin Wochenschr.* 2005;117(23-24):802-808.
176. Weber DJ, Sickbert-Bennett EE, Vinje J, et al. Lessons learned from a norovirus outbreak in a locked pediatric inpatient psychiatric unit. *Infect Control Hosp Epidemiol.* 2005;26(10):841-843.
177. Lynn S, Toop J, Hanger C, Millar N. Norovirus outbreaks in a hospital setting: the role of infection control. *N Z Med J.* 2004;117(1189):U771.
178. Khanna N, Goldenberger D, Graber P, Battegay M, Widmer AF. Gastroenteritis outbreak with norovirus in a Swiss university hospital with a newly identified virus strain. *J Hosp Infect.* 2003;55(2):131-136.
179. McCall J, Smithson R. Rapid response and strict control measures can contain a hospital outbreak of Norwalk-like virus. *Commun Dis Public Health.* 2002;5(3):243-246.
180. Milazzo A, Tribe IG, Ratcliff R, Doherty C, Higgins G, Givney R. A large, prolonged outbreak of human calicivirus infection linked to an aged-care facility. *Commun Dis Intell.* 2002;26(2):261-264.
181. Miller M, Carter L, Scott K, Millard G, Lynch B, Guest C. Norwalk-like virus outbreak in Canberra: implications for infection control in aged care facilities. *Commun Dis Intell.* 2002;26(4):555-561.
182. Hoyle J. Managing the challenge of an acute gastroenteritis outbreak caused by a Norwalk-like virus in a 239 bed long-term care facility. *Aust Infect Control.* 2001;6(4):128-133.
183. Russo PL, Spelman DW, Harrington GA, et al. Concise communications. Hospital outbreak of Norwalk-like virus. *Infect Control Hosp Epidemiol.* 1997;18(8):576-579.
184. Stevenson P, McCann R, Duthie R, Glew E, Ganguli L. A hospital outbreak due to Norwalk virus. *J Hosp Infect.* 1994;26(4):261-272.
185. Hudson JB, Sharma M, Petric M. Inactivation of Norovirus by ozone gas in conditions relevant to healthcare. *J Hosp Infect.* 2007;66(1):40-45.
186. Park GW, Boston DM, Kase JA, Sampson MN, Sobsey MD. Evaluation of liquid- and fog-based application of Sterilox hypochlorous acid solution for surface inactivation of human norovirus. *Appl Environ Microbiol.* 2007;73(14):4463-4468.
187. Poschetto LF, Ike A, Papp T, Mohn U, Bohm R, Marschang RE. Comparison of the sensitivities of noroviruses and feline calicivirus to chemical disinfection under field-like conditions. *Appl Environ Microbiol.* 2007;73(17):5494-5500.
188. Jimenez L, Chiang M. Virucidal activity of a quaternary ammonium compound disinfectant against feline calicivirus: a surrogate for norovirus. *Am J Infect Control.* 2006;34(5):269-273.
189. Kramer A, Galabov AS, Sattar SA, et al. Virucidal activity of a new hand disinfectant with reduced ethanol content: comparison with other alcohol-based formulations. *J Hosp Infect.* 2006;62(1):98-106.
190. Malik YS, Allwood PB, Hedberg CW, Goyal SM. Disinfection of fabrics and carpets artificially contaminated with calicivirus: relevance in institutional and healthcare centres. *J Hosp Infect.* 2006;63(2):205-210.
191. Malik YS, Maherchandani S, Goyal SM. Comparative efficacy of ethanol and isopropanol against feline calicivirus, a norovirus surrogate. *Am J Infect Control.* 2006;34(1):31-35.
192. Malik YS, Goyal SM. Virucidal efficacy of sodium bicarbonate on a food contact surface against feline calicivirus, a norovirus surrogate. *International Journal of Food Microbiology.* 2006;109(1/2):160-163.
193. Kampf G, Grotheer D, Steinmann J. Efficacy of three ethanol-based hand rubs against feline calicivirus, a surrogate virus for norovirus. *J Hosp Infect.* 2005;60(2):144-149.
194. Barker J, Vipond IB, Bloomfield SF. Effects of cleaning and disinfection in reducing the spread of Norovirus contamination via environmental surfaces. *J Hosp Infect.* 2004;58(1):42-49.

195. Duizer E, Bijkerk P, Rockx B, De Groot A, Twisk F, Koopmans M. Inactivation of caliciviruses. *Appl Environ Microbiol.* 2004;70(8):4538-4543.
196. Gehrke C, Steinmann J, Goroncy-Bermes P. Inactivation of feline calicivirus, a surrogate of norovirus (formerly Norwalk-like viruses), by different types of alcohol in vitro and in vivo. *J Hosp Infect.* 2004;56(1):49-55.
197. Lin CM, Wu FM, Kim HK, Doyle MP, Michael BS, Williams LK. A comparison of hand washing techniques to remove *Escherichia coli* and caliciviruses under natural or artificial fingernails. *J Food Prot.* 2003;66(12):2296-2301.
198. Nuanualsuwan S, Mariam T, Himathongkham S, Cliver DO. Ultraviolet inactivation of feline calicivirus, human enteric viruses and coliphages. *Photochemistry and Photobiology.* 2002;76(4):406-410.
199. Gulati BR, Allwood PB, Hedberg CW, Goyal SM. Efficacy of commonly used disinfectants for the inactivation of calicivirus on strawberry, lettuce, and a food-contact surface. *J Food Prot.* 2001;64(9):1430-1434.
200. Doultree JC, Druce JD, Birch CJ, Bowden DS, Marshall JA. Inactivation of feline calicivirus, a Norwalk virus surrogate. *J Hosp Infect.* 1999;41(1):51-57.
201. Shin GA, Sobsey MD. Reduction of norwalk virus, poliovirus 1 and coliphage MS2 by monochloramine disinfection of water. *Water Science and Technology.* 1998;38(12):151-154.
202. Rossignol JF, El-Gohary YM. Nitazoxanide in the treatment of viral gastroenteritis: a randomized double-blind placebo-controlled clinical trial. *Aliment Pharmacol Ther.* 2006;24(10):1423-1430.
203. Gustafson TL, Kobylik B, Hutcheson RH, Schaffner W. Protective effect of anticholinergic drugs and psyllium in a nosocomial outbreak of Norwalk gastroenteritis. *J Hosp Infect.* 1983;4(4):367-374.
204. Yoder J. Roberts V. Craun GF. Hill V. Hicks LA. Alexander NT. Radke V. Calderon RL. Hlavsa MC. Beach MJ. Roy SL. Centers for Disease Control and Prevention (CDC). Surveillance for waterborne disease and outbreaks associated with drinking water and water not intended for drinking--United States, 2005-2006. *Morb Mortal Wkly Rep Surveill Summ.* 2008;57(9):39-62.
205. Cooper E, Blamey S. A norovirus gastroenteritis epidemic in a long-term-care facility. *Infect Control Hosp Epidemiol.* 2005;26(3):256-258.
206. Russo PL, Spelman DW, Harrington GA, et al. Hospital outbreak of Norwalk-like virus. *Infect Control Hosp Epidemiol.* 1997;18(8):576-579.
207. Centers for Disease Control and Prevention (CDC). Multisite outbreak of norovirus associated with a franchise restaurant--Kent County, Michigan, May 2005. *MMWR.* 2006;55(14):395-397.



MMWRTM

Morbidity and Mortality Weekly Report

Recommendations and Reports

October 25, 2002 / Vol. 51 / No. RR-16

Guideline for Hand Hygiene in Health-Care Settings

**Recommendations of the Healthcare Infection Control Practices
Advisory Committee and the HICPAC/SHEA/APIC/IDSA
Hand Hygiene Task Force**

INSIDE: Continuing Education Examination

CENTERS FOR DISEASE CONTROL AND PREVENTION

SAFER • HEALTHIER • PEOPLETM

The *MMWR* series of publications is published by the Epidemiology Program Office, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30333.

SUGGESTED CITATION

Centers for Disease Control and Prevention. Guideline for Hand Hygiene in Health-Care Settings: Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force. *MMWR* 2002;51(No. RR-16):[inclusive page numbers].

Centers for Disease Control and Prevention

Julie L. Gerberding, M.D., M.P.H.

Director

David W. Fleming, M.D.

Deputy Director for Science and Public Health

Dixie E. Snider, Jr., M.D., M.P.H.

Associate Director for Science

Epidemiology Program Office

Stephen B. Thacker, M.D., M.Sc.

Director

Office of Scientific and Health Communications

John W. Ward, M.D.

Director

Editor, MMWR Series

Suzanne M. Hewitt, M.P.A.

Managing Editor

Rachel J. Wilson

Douglas W. Weatherwax

Project Editors

Malbea A. Heilman

Beverly J. Holland

Visual Information Specialists

Quang M. Doan

Erica R. Shaver

Information Technology Specialists

CONTENTS

Part I. Review of the Scientific Data Regarding

Hand Hygiene	1
Historical Perspective	1
Normal Bacterial Skin Flora	2
Physiology of Normal Skin	2
Definition of Terms	3
Evidence of Transmission of Pathogens on Hands	4
Models of Hand Transmission	5
Relation of Hand Hygiene and Acquisition of Health-Care–Associated Pathogens	5
Methods Used To Evaluate the Efficacy of Hand-Hygiene Products	6
Review of Preparations Used for Hand Hygiene	8
Activity of Antiseptic Agents Against Spore-Forming Bacteria	16
Reduced Susceptibility of Bacteria to Antiseptics	17
Surgical Hand Antisepsis	17
Relative Efficacy of Plain Soap, Antiseptic Soap/Detergent, and Alcohols	18
Irritant Contact Dermatitis Resulting from Hand-Hygiene Measures	18
Proposed Methods for Reducing Adverse Effects of Agents	19
Factors To Consider When Selecting Hand-Hygiene Products	20
Hand-Hygiene Practices Among HCWs	21
Lessons Learned from Behavioral Theories	25
Methods Used To Promote Improved Hand Hygiene	26
Efficacy of Promotion and Impact of Improved Hand Hygiene	27
Other Policies Related to Hand Hygiene	29
Hand-Hygiene Research Agenda	30
Web-Based Hand-Hygiene Resources	30
Part II. Recommendations	31
Categories	31
Recommendations	32
Part III. Performance Indicators	34
References	34
Appendix	45
Continuing Education Activity	CE-1

Guideline for Hand Hygiene in Health-Care Settings

Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force

Prepared by
John M. Boyce, M.D.¹
Didier Pittet, M.D.²
¹Hospital of Saint Raphael
New Haven, Connecticut
²University of Geneva
Geneva, Switzerland

Summary

The Guideline for Hand Hygiene in Health-Care Settings provides health-care workers (HCWs) with a review of data regarding handwashing and hand antisepsis in health-care settings. In addition, it provides specific recommendations to promote improved hand-hygiene practices and reduce transmission of pathogenic microorganisms to patients and personnel in health-care settings. This report reviews studies published since the 1985 CDC guideline (Garner JS, Favero MS. CDC guideline for handwashing and hospital environmental control, 1985. Infect Control 1986;7:231–43) and the 1995 APIC guideline (Larson EL, APIC Guidelines Committee. APIC guideline for handwashing and hand antisepsis in health care settings. Am J Infect Control 1995;23:251–69) were issued and provides an in-depth review of hand-hygiene practices of HCWs, levels of adherence of personnel to recommended handwashing practices, and factors adversely affecting adherence. New studies of the in vivo efficacy of alcohol-based hand rubs and the low incidence of dermatitis associated with their use are reviewed. Recent studies demonstrating the value of multidisciplinary hand-hygiene promotion programs and the potential role of alcohol-based hand rubs in improving hand-hygiene practices are summarized. Recommendations concerning related issues (e.g., the use of surgical hand antiseptics, hand lotions or creams, and wearing of artificial fingernails) are also included.

Part I. Review of the Scientific Data Regarding Hand Hygiene

Historical Perspective

For generations, handwashing with soap and water has been considered a measure of personal hygiene (1). The concept of cleansing hands with an antiseptic agent probably emerged in the early 19th century. As early as 1822, a French pharmacist demonstrated that solutions containing chlorides of lime or soda could eradicate the foul odors associated with human corpses and that such solutions could be used as disinfectants and antiseptics (2). In a paper published in 1825, this pharmacist stated that physicians and other persons attending patients with contagious diseases would benefit from moistening their hands with a liquid chloride solution (2).

In 1846, Ignaz Semmelweis observed that women whose babies were delivered by students and physicians in the First Clinic at the General Hospital of Vienna consistently had a

higher mortality rate than those whose babies were delivered by midwives in the Second Clinic (3). He noted that physicians who went directly from the autopsy suite to the obstetrics ward had a disagreeable odor on their hands despite washing their hands with soap and water upon entering the obstetrics clinic. He postulated that the puerperal fever that affected so many parturient women was caused by “cadaverous particles” transmitted from the autopsy suite to the obstetrics ward via the hands of students and physicians. Perhaps because of the known deodorizing effect of chlorine compounds, as of May 1847, he insisted that students and physicians clean their hands with a chlorine solution between each patient in the clinic. The maternal mortality rate in the First Clinic subsequently dropped dramatically and remained low for years. This intervention by Semmelweis represents the first evidence indicating that cleansing heavily contaminated hands with an antiseptic agent between patient contacts may reduce health-care–associated transmission of contagious diseases more effectively than handwashing with plain soap and water.

In 1843, Oliver Wendell Holmes concluded independently that puerperal fever was spread by the hands of health personnel (1). Although he described measures that could be taken to limit its spread, his recommendations had little impact on

The material in this report originated in the National Center for Infectious Diseases, James M. Hughes, M.D., Director; and the Division of Healthcare Quality Promotion, Steve Solomon, M.D., Acting Director.

obstetric practices at the time. However, as a result of the seminal studies by Semmelweis and Holmes, handwashing gradually became accepted as one of the most important measures for preventing transmission of pathogens in health-care facilities.

In 1961, the U. S. Public Health Service produced a training film that demonstrated handwashing techniques recommended for use by health-care workers (HCWs) (4). At the time, recommendations directed that personnel wash their hands with soap and water for 1–2 minutes before and after patient contact. Rinsing hands with an antiseptic agent was believed to be less effective than handwashing and was recommended only in emergencies or in areas where sinks were unavailable.

In 1975 and 1985, formal written guidelines on handwashing practices in hospitals were published by CDC (5,6). These guidelines recommended handwashing with non-antimicrobial soap between the majority of patient contacts and washing with antimicrobial soap before and after performing invasive procedures or caring for patients at high risk. Use of waterless antiseptic agents (e.g., alcohol-based solutions) was recommended only in situations where sinks were not available.

In 1988 and 1995, guidelines for handwashing and hand antisepsis were published by the Association for Professionals in Infection Control (APIC) (7,8). Recommended indications for handwashing were similar to those listed in the CDC guidelines. The 1995 APIC guideline included more detailed discussion of alcohol-based hand rubs and supported their use in more clinical settings than had been recommended in earlier guidelines. In 1995 and 1996, the Healthcare Infection Control Practices Advisory Committee (HICPAC) recommended that either antimicrobial soap or a waterless antiseptic agent be used for cleaning hands upon leaving the rooms of patients with multidrug-resistant pathogens (e.g., vancomycin-resistant enterococci [VRE] and methicillin-resistant *Staphylococcus aureus* [MRSA]) (9,10). These guidelines also provided recommendations for handwashing and hand antisepsis in other clinical settings, including routine patient care. Although the APIC and HICPAC guidelines have been adopted by the majority of hospitals, adherence of HCWs to recommended handwashing practices has remained low (11,12).

Recent developments in the field have stimulated a review of the scientific data regarding hand hygiene and the development of new guidelines designed to improve hand-hygiene practices in health-care facilities. This literature review and accompanying recommendations have been prepared by a Hand Hygiene Task Force, comprising representatives from HICPAC, the Society for Healthcare Epidemiology of America (SHEA), APIC, and the Infectious Diseases Society of America (IDSA).

Normal Bacterial Skin Flora

To understand the objectives of different approaches to hand cleansing, a knowledge of normal bacterial skin flora is essential. Normal human skin is colonized with bacteria; different areas of the body have varied total aerobic bacterial counts (e.g., 1×10^6 colony forming units (CFUs)/cm² on the scalp, 5×10^5 CFUs/cm² in the axilla, 4×10^4 CFUs/cm² on the abdomen, and 1×10^4 CFUs/cm² on the forearm) (13). Total bacterial counts on the hands of medical personnel have ranged from 3.9×10^4 to 4.6×10^6 (14–17). In 1938, bacteria recovered from the hands were divided into two categories: transient and resident (14). Transient flora, which colonize the superficial layers of the skin, are more amenable to removal by routine handwashing. They are often acquired by HCWs during direct contact with patients or contact with contaminated environmental surfaces within close proximity of the patient. Transient flora are the organisms most frequently associated with health-care-associated infections. Resident flora, which are attached to deeper layers of the skin, are more resistant to removal. In addition, resident flora (e.g., coagulase-negative staphylococci and diphtheroids) are less likely to be associated with such infections. The hands of HCWs may become persistently colonized with pathogenic flora (e.g., *S. aureus*), gram-negative bacilli, or yeast. Investigators have documented that, although the number of transient and resident flora varies considerably from person to person, it is often relatively constant for any specific person (14,18).

Physiology of Normal Skin

The primary function of the skin is to reduce water loss, provide protection against abrasive action and microorganisms, and act as a permeability barrier to the environment. The basic structure of skin includes, from outer- to innermost layer, the superficial region (i.e., the stratum corneum or horny layer, which is 10- to 20- μ m thick), the viable epidermis (50- to 100- μ m thick), the dermis (1- to 2-mm thick), and the hypodermis (1- to 2-mm thick). The barrier to percutaneous absorption lies within the stratum corneum, the thinnest and smallest compartment of the skin. The stratum corneum contains the corneocytes (or horny cells), which are flat, polyhedral-shaped nonnucleated cells, remnants of the terminally differentiated keratinocytes located in the viable epidermis. Corneocytes are composed primarily of insoluble bundled keratins surrounded by a cell envelope stabilized by cross-linked proteins and covalently bound lipid. Interconnecting the corneocytes of the stratum corneum are polar structures (e.g., corneodesmosomes), which contribute to stratum corneum cohesion.

The intercellular region of the stratum corneum is composed of lipid primarily generated from the exocytosis of lamellar bodies during the terminal differentiation of the keratinocytes. The intercellular lipid is required for a competent skin barrier and forms the only continuous domain. Directly under the stratum corneum is a stratified epidermis, which is composed primarily of 10–20 layers of keratinizing epithelial cells that are responsible for the synthesis of the stratum corneum. This layer also contains melanocytes involved in skin pigmentation; Langerhans cells, which are important for antigen presentation and immune responses; and Merkel cells, whose precise role in sensory reception has yet to be fully delineated. As keratinocytes undergo terminal differentiation, they begin to flatten out and assume the dimensions characteristic of the corneocytes (i.e., their diameter changes from 10–12 μm to 20–30 μm , and their volume increases by 10- to 20-fold). The viable epidermis does not contain a vascular network, and the keratinocytes obtain their nutrients from below by passive diffusion through the interstitial fluid.

The skin is a dynamic structure. Barrier function does not simply arise from the dying, degeneration, and compaction of the underlying epidermis. Rather, the processes of cornification and desquamation are intimately linked; synthesis of the stratum corneum occurs at the same rate as loss. Substantial evidence now confirms that the formation of the skin barrier is under homeostatic control, which is illustrated by the epidermal response to barrier perturbation by skin stripping or solvent extraction. Circumstantial evidence indicates that the rate of keratinocyte proliferation directly influences the integrity of the skin barrier. A general increase in the rate of proliferation results in a decrease in the time available for 1) uptake of nutrients (e.g., essential fatty acids), 2) protein and lipid synthesis, and 3) processing of the precursor molecules required for skin-barrier function. Whether chronic but quantitatively smaller increases in rate of epidermal proliferation also lead to changes in skin-barrier function remains unclear. Thus, the extent to which the decreased barrier function caused by irritants is caused by an increased epidermal proliferation also is unknown.

The current understanding of the formation of the stratum corneum has come from studies of the epidermal responses to perturbation of the skin barrier. Experimental manipulations that disrupt the skin barrier include 1) extraction of skin lipids with apolar solvents, 2) physical stripping of the stratum corneum using adhesive tape, and 3) chemically induced irritation. All of these experimental manipulations lead to a decreased skin barrier as determined by transepidermal water loss (TEWL). The most studied experimental system is the treatment of mouse skin with acetone. This experiment

results in a marked and immediate increase in TEWL, and therefore a decrease in skin-barrier function. Acetone treatment selectively removes glycerolipids and sterols from the skin, which indicates that these lipids are necessary, though perhaps not sufficient in themselves, for barrier function. Detergents act like acetone on the intercellular lipid domain. The return to normal barrier function is biphasic: 50%–60% of barrier recovery typically occurs within 6 hours, but complete normalization of barrier function requires 5–6 days.

Definition of Terms

Alcohol-based hand rub. An alcohol-containing preparation designed for application to the hands for reducing the number of viable microorganisms on the hands. In the United States, such preparations usually contain 60%–95% ethanol or isopropanol.

Antimicrobial soap. Soap (i.e., detergent) containing an antiseptic agent.

Antiseptic agent. Antimicrobial substances that are applied to the skin to reduce the number of microbial flora. Examples include alcohols, chlorhexidine, chlorine, hexachlorophene, iodine, chloroxylenol (PCMX), quaternary ammonium compounds, and triclosan.

Antiseptic handwash. Washing hands with water and soap or other detergents containing an antiseptic agent.

Antiseptic hand rub. Applying an antiseptic hand-rub product to all surfaces of the hands to reduce the number of microorganisms present.

Cumulative effect. A progressive decrease in the numbers of microorganisms recovered after repeated applications of a test material.

Decontaminate hands. To Reduce bacterial counts on hands by performing antiseptic hand rub or antiseptic handwash.

Detergent. Detergents (i.e., surfactants) are compounds that possess a cleaning action. They are composed of both hydrophilic and lipophilic parts and can be divided into four groups: anionic, cationic, amphoteric, and nonionic detergents. Although products used for handwashing or antiseptic handwash in health-care settings represent various types of detergents, the term “soap” is used to refer to such detergents in this guideline.

Hand antiseptics. Refers to either antiseptic handwash or antiseptic hand rub.

Hand hygiene. A general term that applies to either handwashing, antiseptic handwash, antiseptic hand rub, or surgical hand antiseptics.

Handwashing. Washing hands with plain (i.e., non-antimicrobial) soap and water.

Persistent activity. Persistent activity is defined as the prolonged or extended antimicrobial activity that prevents or inhibits the proliferation or survival of microorganisms after application of the product. This activity may be demonstrated by sampling a site several minutes or hours after application and demonstrating bacterial antimicrobial effectiveness when compared with a baseline level. This property also has been referred to as “residual activity.” Both substantive and nonsubstantive active ingredients can show a persistent effect if they substantially lower the number of bacteria during the wash period.

Plain soap. Plain soap refers to detergents that do not contain antimicrobial agents or contain low concentrations of antimicrobial agents that are effective solely as preservatives.

Substantivity. Substantivity is an attribute of certain active ingredients that adhere to the stratum corneum (i.e., remain on the skin after rinsing or drying) to provide an inhibitory effect on the growth of bacteria remaining on the skin.

Surgical hand antisepsis. Antiseptic handwash or antiseptic hand rub performed preoperatively by surgical personnel to eliminate transient and reduce resident hand flora. Antiseptic detergent preparations often have persistent antimicrobial activity.

Visibly soiled hands. Hands showing visible dirt or visibly contaminated with proteinaceous material, blood, or other body fluids (e.g., fecal material or urine).

Waterless antiseptic agent. An antiseptic agent that does not require use of exogenous water. After applying such an agent, the hands are rubbed together until the agent has dried.

Food and Drug Administration (FDA) product categories. The 1994 FDA Tentative Final Monograph for Health-Care Antiseptic Drug Products divided products into three categories and defined them as follows (19):

- **Patient preoperative skin preparation.** A fast-acting, broad-spectrum, and persistent antiseptic-containing preparation that substantially reduces the number of microorganisms on intact skin.
- **Antiseptic handwash or HCW handwash.** An antiseptic-containing preparation designed for frequent use; it reduces the number of microorganisms on intact skin to an initial baseline level after adequate washing, rinsing, and drying; it is broad-spectrum, fast-acting, and if possible, persistent.
- **Surgical hand scrub.** An antiseptic-containing preparation that substantially reduces the number of microorganisms on intact skin; it is broad-spectrum, fast-acting, and persistent.

Evidence of Transmission of Pathogens on Hands

Transmission of health-care-associated pathogens from one patient to another via the hands of HCWs requires the following sequence of events:

- Organisms present on the patient’s skin, or that have been shed onto inanimate objects in close proximity to the patient, must be transferred to the hands of HCWs.
- These organisms must then be capable of surviving for at least several minutes on the hands of personnel.
- Next, handwashing or hand antisepsis by the worker must be inadequate or omitted entirely, or the agent used for hand hygiene must be inappropriate.
- Finally, the contaminated hands of the caregiver must come in direct contact with another patient, or with an inanimate object that will come into direct contact with the patient.

Health-care-associated pathogens can be recovered not only from infected or draining wounds, but also from frequently colonized areas of normal, intact patient skin (20–31). The perineal or inguinal areas are usually most heavily colonized, but the axillae, trunk, and upper extremities (including the hands) also are frequently colonized (23,25,26,28,30–32). The number of organisms (e.g., *S. aureus*, *Proteus mirabilis*, *Klebsiella* spp., and *Acinetobacter* spp.) present on intact areas of the skin of certain patients can vary from 100 to 10⁶/cm² (25,29,31,33). Persons with diabetes, patients undergoing dialysis for chronic renal failure, and those with chronic dermatitis are likely to have areas of intact skin that are colonized with *S. aureus* (34–41). Because approximately 10⁶ skin squames containing viable microorganisms are shed daily from normal skin (42), patient gowns, bed linen, bedside furniture, and other objects in the patient’s immediate environment can easily become contaminated with patient flora (30,43–46). Such contamination is particularly likely to be caused by staphylococci or enterococci, which are resistant to desiccation.

Data are limited regarding the types of patient-care activities that result in transmission of patient flora to the hands of personnel (26,45–51). In the past, attempts have been made to stratify patient-care activities into those most likely to cause hand contamination (52), but such stratification schemes were never validated by quantifying the level of bacterial contamination that occurred. Nurses can contaminate their hands with 100–1,000 CFUs of *Klebsiella* spp. during “clean” activities (e.g., lifting a patient; taking a patient’s pulse, blood pressure, or oral temperature; or touching a patient’s hand, shoulder, or groin) (48). Similarly, in another study, hands were cultured of nurses who touched the groins of patients heavily colonized with *P. mirabilis* (25); 10–600 CFUs/mL of this

organism were recovered from glove juice samples from the nurses' hands. Recently, other researchers studied contamination of HCWs' hands during activities that involved direct patient-contact wound care, intravascular catheter care, respiratory-tract care, and the handling of patient secretions (51). Agar fingertip impression plates were used to culture bacteria; the number of bacteria recovered from fingertips ranged from 0 to 300 CFUs. Data from this study indicated that direct patient contact and respiratory-tract care were most likely to contaminate the fingers of caregivers. Gram-negative bacilli accounted for 15% of isolates and *S. aureus* for 11%. Duration of patient-care activity was strongly associated with the intensity of bacterial contamination of HCWs' hands.

HCWs can contaminate their hands with gram-negative bacilli, *S. aureus*, enterococci, or *Clostridium difficile* by performing "clean procedures" or touching intact areas of the skin of hospitalized patients (26,45,46,53). Furthermore, personnel caring for infants with respiratory syncytial virus (RSV) infections have acquired RSV by performing certain activities (e.g., feeding infants, changing diapers, and playing with infants) (49). Personnel who had contact only with surfaces contaminated with the infants' secretions also acquired RSV by contaminating their hands with RSV and inoculating their oral or conjunctival mucosa. Other studies also have documented that HCWs may contaminate their hands (or gloves) merely by touching inanimate objects in patient rooms (46,53–56). None of the studies concerning hand contamination of hospital personnel were designed to determine if the contamination resulted in transmission of pathogens to susceptible patients.

Other studies have documented contamination of HCWs' hands with potential health-care-associated pathogens, but did not relate their findings to the specific type of preceding patient contact (15,17,57–62). For example, before glove use was common among HCWs, 15% of nurses working in an isolation unit carried a median of 1×10^4 CFUs of *S. aureus* on their hands (61). Of nurses working in a general hospital, 29% had *S. aureus* on their hands (median count: 3,800 CFUs), whereas 78% of those working in a hospital for dermatology patients had the organism on their hands (median count: 14.3×10^6 CFUs). Similarly, 17%–30% of nurses carried gram-negative bacilli on their hands (median counts: 3,400–38,000 CFUs). One study found that *S. aureus* could be recovered from the hands of 21% of intensive-care-unit personnel and that 21% of physician and 5% of nurse carriers had $>1,000$ CFUs of the organism on their hands (59). Another study found lower levels of colonization on the hands of personnel working in a neurosurgery unit, with an average of 3 CFUs of *S. aureus* and 11 CFUs of gram-negative bacilli (16). Serial

cultures revealed that 100% of HCWs carried gram-negative bacilli at least once, and 64% carried *S. aureus* at least once.

Models of Hand Transmission

Several investigators have studied transmission of infectious agents by using different experimental models. In one study, nurses were asked to touch the groins of patients heavily colonized with gram-negative bacilli for 15 seconds — as though they were taking a femoral pulse (25). Nurses then cleaned their hands by washing with plain soap and water or by using an alcohol hand rinse. After cleaning their hands, they touched a piece of urinary catheter material with their fingers, and the catheter segment was cultured. The study revealed that touching intact areas of moist skin of the patient transferred enough organisms to the nurses' hands to result in subsequent transmission to catheter material, despite handwashing with plain soap and water.

The transmission of organisms from artificially contaminated "donor" fabrics to clean "recipient" fabrics via hand contact also has been studied. Results indicated that the number of organisms transmitted was greater if the donor fabric or the hands were wet upon contact (63). Overall, only 0.06% of the organisms obtained from the contaminated donor fabric were transferred to recipient fabric via hand contact. *Staphylococcus saprophyticus*, *Pseudomonas aeruginosa*, and *Serratia* spp. were also transferred in greater numbers than was *Escherichia coli* from contaminated fabric to clean fabric after hand contact (64). Organisms are transferred to various types of surfaces in much larger numbers (i.e., $>10^4$) from wet hands than from hands that are thoroughly dried (65).

Relation of Hand Hygiene and Acquisition of Health-Care-Associated Pathogens

Hand antisepsis reduces the incidence of health-care-associated infections (66,67). An intervention trial using historical controls demonstrated in 1847 that the mortality rate among mothers who delivered in the First Obstetrics Clinic at the General Hospital of Vienna was substantially lower when hospital staff cleaned their hands with an antiseptic agent than when they washed their hands with plain soap and water (3).

In the 1960s, a prospective, controlled trial sponsored by the National Institutes of Health and the Office of the Surgeon General demonstrated that infants cared for by nurses who did not wash their hands after handling an index infant colonized with *S. aureus* acquired the organism more often and more rapidly than did infants cared for by nurses who used hexachlorophene to clean their hands between infant

contacts (68). This trial provided evidence that, when compared with no handwashing, washing hands with an antiseptic agent between patient contacts reduces transmission of health-care-associated pathogens.

Trials have studied the effects of handwashing with plain soap and water versus some form of hand antiseptics on health-care-associated infection rates (69,70). Health-care-associated infection rates were lower when antiseptic handwashing was performed by personnel (69). In another study, antiseptic handwashing was associated with lower health-care-associated infection rates in certain intensive-care units, but not in others (70).

Health-care-associated infection rates were lower after antiseptic handwashing using a chlorhexidine-containing detergent compared with handwashing with plain soap or use of an alcohol-based hand rinse (71). However, because only a minimal amount of the alcohol rinse was used during periods when the combination regimen also was in use and because adherence to policies was higher when chlorhexidine was available, determining which factor (i.e., the hand-hygiene regimen or differences in adherence) accounted for the lower infection rates was difficult. Investigators have determined also that health-care-associated acquisition of MRSA was reduced when the antimicrobial soap used for hygienic handwashing was changed (72,73).

Increased handwashing frequency among hospital staff has been associated with decreased transmission of *Klebsiella* spp. among patients (48); these studies, however, did not quantify the level of handwashing among personnel. In a recent study, the acquisition of various health-care-associated pathogens was reduced when hand antiseptics was performed more frequently by hospital personnel (74); both this study and another (75) documented that the prevalence of health-care-associated infections decreased as adherence to recommended hand-hygiene measures improved.

Outbreak investigations have indicated an association between infections and understaffing or overcrowding; the association was consistently linked with poor adherence to hand hygiene. During an outbreak investigation of risk factors for central venous catheter-associated bloodstream infections (76), after adjustment for confounding factors, the patient-to-nurse ratio remained an independent risk factor for bloodstream infection, indicating that nursing staff reduction below a critical threshold may have contributed to this outbreak by jeopardizing adequate catheter care. The understaffing of nurses can facilitate the spread of MRSA in intensive-care settings (77) through relaxed attention to basic control measures (e.g., hand hygiene). In an outbreak of *Enterobacter cloacae* in a neonatal intensive-care unit (78), the daily number of

hospitalized children was above the maximum capacity of the unit, resulting in an available space per child below current recommendations. In parallel, the number of staff members on duty was substantially less than the number necessitated by the workload, which also resulted in relaxed attention to basic infection-control measures. Adherence to hand-hygiene practices before device contact was only 25% during the workload peak, but increased to 70% after the end of the understaffing and overcrowding period. Surveillance documented that being hospitalized during this period was associated with a fourfold increased risk of acquiring a health-care-associated infection. This study not only demonstrates the association between workload and infections, but it also highlights the intermediate cause of antimicrobial spread: poor adherence to hand-hygiene policies.

Methods Used To Evaluate the Efficacy of Hand-Hygiene Products

Current Methods

Investigators use different methods to study the in vivo efficacy of handwashing, antiseptic handwash, and surgical hand antiseptics protocols. Differences among the various studies include 1) whether hands are purposely contaminated with bacteria before use of test agents, 2) the method used to contaminate fingers or hands, 3) the volume of hand-hygiene product applied to the hands, 4) the time the product is in contact with the skin, 5) the method used to recover bacteria from the skin after the test solution has been used, and 6) the method of expressing the efficacy of the product (i.e., either percent reduction in bacteria recovered from the skin or log reduction of bacteria released from the skin). Despite these differences, the majority of studies can be placed into one of two major categories: studies focusing on products to remove transient flora and studies involving products that are used to remove resident flora from the hands. The majority of studies of products for removing transient flora from the hands of HCWs involve artificial contamination of the volunteer's skin with a defined inoculum of a test organism before the volunteer uses a plain soap, an antimicrobial soap, or a waterless antiseptic agent. In contrast, products tested for the preoperative cleansing of surgeons' hands (which must comply with surgical hand-antiseptics protocols) are tested for their ability to remove resident flora from without artificially contaminating the volunteers' hands.

In the United States, antiseptic handwash products intended for use by HCWs are regulated by FDA's Division of Over-the-Counter Drug Products (OTC). Requirements for in vitro and in vivo testing of HCW handwash products and surgical

hand scrubs are outlined in the FDA Tentative Final Monograph for Healthcare Antiseptic Drug Products (TFM) (19). Products intended for use as HCW handwashes are evaluated by using a standardized method (19). Tests are performed in accordance with use directions for the test material. Before baseline bacterial sampling and before each wash with the test material, 5 mL of a standardized suspension of *Serratia marcescens* are applied to the hands and then rubbed over the surfaces of the hands. A specified volume of the test material is dispensed into the hands and is spread over the hands and lower one third of the forearms. A small amount of tap water is added to the hands, and hands are completely lathered for a specified time, covering all surfaces of the hands and the lower third of the forearms. Volunteers then rinse hands and forearms under 40°C tap water for 30 seconds. Ten washes with the test formulation are required. After the first, third, seventh, and tenth washes, rubber gloves or polyethylene bags used for sampling are placed on the right and left hands, and 75 mL of sampling solution is added to each glove; gloves are secured above the wrist. All surfaces of the hand are massaged for 1 minute, and samples are obtained aseptically for quantitative culture. No neutralizer of the antimicrobial is routinely added to the sampling solution, but if dilution of the antimicrobial in the sampling fluid does not result in demonstrable neutralization, a neutralizer specific for the test formulation is added to the sampling solution. For waterless formulations, a similar procedure is used. TFM criteria for efficacy are as follows: a 2- \log_{10} reduction of the indicator organism on each hand within 5 minutes after the first use, and a 3- \log_{10} reduction of the indicator organism on each hand within 5 minutes after the tenth use (19).

Products intended for use as surgical hand scrubs have been evaluated also by using a standardized method (19). Volunteers clean under fingernails with a nail stick and clip their fingernails. All jewelry is removed from hands and arms. Hands and two thirds of forearms are rinsed with tap water (38°C–42°C) for 30 seconds, and then they are washed with a non-antimicrobial soap for 30 seconds and are rinsed for 30 seconds under tap water. Baseline microbial hand counts can then be determined. Next, a surgical scrub is performed with the test formulation using directions provided by the manufacturer. If no instructions are provided with the formulation, two 5-minute scrubs of hands and forearms followed by rinsing are performed. Reduction from baseline microbial hand counts is determined in a series of 11 scrubs conducted during 5 days. Hands are sampled at 1 minute, 3 hours, and 6 hours after the first scrubs on day 1, day 2, and day 5. After washing, volunteers wear rubber gloves; 75 mL of sampling solution are then added to one glove, and all surfaces of the hands are massaged

for 1 minute. Samples are then taken aseptically and cultured quantitatively. The other glove remains on the other hand for 6 hours and is sampled in the same manner. TFM requires that formulations reduce the number of bacteria 1 \log_{10} on each hand within 1 minute of product application and that the bacterial cell count on each hand does not subsequently exceed baseline within 6 hours on day 1; the formulation must produce a 2- \log_{10} reduction in microbial flora on each hand within 1 minute of product application by the end of the second day of enumeration and a 3- \log_{10} reduction of microbial flora on each hand within 1 minute of product use by the end of the fifth day when compared with the established baseline (19).

The method most widely used in Europe to evaluate the efficacy of hand-hygiene agents is European Standard 1500–1997 (EN 1500—Chemical disinfectants and antiseptics. Hygienic hand-rub test method and requirements) (79). This method requires 12–15 test volunteers and an 18- to 24-hour growth of broth culture of *E. coli* K12. Hands are washed with a soft soap, dried, and then immersed halfway to the metacarpals in the broth culture for 5 seconds. Hands are removed from the broth culture, excess fluid is drained off, and hands are dried in the air for 3 minutes. Bacterial recovery for the initial value is obtained by kneading the fingertips of each hand separately for 60 seconds in 10 mL of tryptic soy broth (TSB) without neutralizers. The hands are removed from the broth and disinfected with 3 mL of the hand-rub agent for 30 seconds in a set design. The same operation is repeated with total disinfection time not exceeding 60 seconds. Both hands are rinsed in running water for 5 seconds and water is drained off. Fingertips of each hand are kneaded separately in 10 mL of TSB with added neutralizers. These broths are used to obtain the final value. \log_{10} dilutions of recovery medium are prepared and plated out. Within 3 hours, the same volunteers are tested with the reference disinfectant (60% 2-propanol [isopropanol]) and the test product. Colony counts are performed after 24 and 48 hours of incubation at 36°C. The average colony count of both left and right hand is used for evaluation. The log-reduction factor is calculated and compared with the initial and final values. The reduction factor of the test product should be superior or the same as the reference alcohol-based rub for acceptance. If a difference exists, then the results are analyzed statistically using the Wilcoxon test. Products that have log reductions substantially less than that observed with the reference alcohol-based hand rub (i.e., approximately 4 \log_{10} reduction) are classified as not meeting the standard.

Because of different standards for efficacy, criteria cited in FDA TFM and the European EN 1500 document for establishing alcohol-based hand rubs vary (1, 19, 79). Alcohol-based

hand rubs that meet TFM criteria for efficacy may not necessarily meet the EN 1500 criteria for efficacy (80). In addition, scientific studies have not established the extent to which counts of bacteria or other microorganisms on the hands need to be reduced to minimize transmission of pathogens in health-care facilities (1,8); whether bacterial counts on the hands must be reduced by 1 log₁₀ (90% reduction), 2 log₁₀ (99%), 3 log₁₀ (99.9%), or 4 log₁₀ (99.99%) is unknown. Several other methods also have been used to measure the efficacy of antiseptic agents against various viral pathogens (81–83).

Shortcomings of Traditional Methodologies

Accepted methods of evaluating hand-hygiene products intended for use by HCWs require that test volunteers wash their hands with a plain or antimicrobial soap for 30 seconds or 1 minute, despite the observation in the majority of studies that the average duration of handwashing by hospital personnel is <15 seconds (52,84–89). A limited number of investigators have used 15-second handwashing or hygienic hand-wash protocols (90–94). Therefore, almost no data exist regarding the efficacy of plain or antimicrobial soaps under conditions in which they are actually used by HCWs. Similarly, certain accepted methods for evaluating waterless antiseptic agents for use as antiseptic hand rubs require that 3 mL of alcohol be rubbed into the hands for 30 seconds, followed by a repeat application for the same duration. This type of protocol also does not reflect actual usage patterns among HCWs. Furthermore, volunteers used in evaluations of products are usually surrogates for HCWs, and their hand flora may not reflect flora found on the hands of personnel working in health-care settings. Further studies should be conducted among practicing HCWs using standardized protocols to obtain more realistic views of microbial colonization and risk of bacterial transfer and cross-transmission (51).

Review of Preparations Used for Hand Hygiene

Plain (Non-Antimicrobial) Soap

Soaps are detergent-based products that contain esterified fatty acids and sodium or potassium hydroxide. They are available in various forms including bar soap, tissue, leaflet, and liquid preparations. Their cleaning activity can be attributed to their detergent properties, which result in removal of dirt, soil, and various organic substances from the hands. Plain soaps have minimal, if any, antimicrobial activity. However, handwashing with plain soap can remove loosely adherent transient flora. For example, handwashing with plain soap and water for 15 seconds reduces bacterial counts on the skin by 0.6–1.1 log₁₀, whereas washing for 30 seconds reduces counts

by 1.8–2.8 log₁₀ (1). However, in several studies, handwashing with plain soap failed to remove pathogens from the hands of hospital personnel (25,45). Handwashing with plain soap can result in paradoxical increases in bacterial counts on the skin (92,95–97). Non-antimicrobial soaps may be associated with considerable skin irritation and dryness (92,96,98), although adding emollients to soap preparations may reduce their propensity to cause irritation. Occasionally, plain soaps have become contaminated, which may lead to colonization of hands of personnel with gram-negative bacilli (99).

Alcohols

The majority of alcohol-based hand antiseptics contain either isopropanol, ethanol, n-propanol, or a combination of two of these products. Although n-propanol has been used in alcohol-based hand rubs in parts of Europe for many years, it is not listed in TFM as an approved active agent for HCW handwashes or surgical hand-scrub preparations in the United States. The majority of studies of alcohols have evaluated individual alcohols in varying concentrations. Other studies have focused on combinations of two alcohols or alcohol solutions containing limited amounts of hexachlorophene, quaternary ammonium compounds, povidone-iodine, triclosan, or chlorhexidine gluconate (61,93,100–119).

The antimicrobial activity of alcohols can be attributed to their ability to denature proteins (120). Alcohol solutions containing 60%–95% alcohol are most effective, and higher concentrations are less potent (120–122) because proteins are not denatured easily in the absence of water (120). The alcohol content of solutions may be expressed as percent by weight (w/w), which is not affected by temperature or other variables, or as percent by volume (vol/vol), which can be affected by temperature, specific gravity, and reaction concentration (123). For example, 70% alcohol by weight is equivalent to 76.8% by volume if prepared at 15°C, or 80.5% if prepared at 25°C (123). Alcohol concentrations in antiseptic hand rubs are often expressed as percent by volume (19).

Alcohols have excellent in vitro germicidal activity against gram-positive and gram-negative vegetative bacteria, including multidrug-resistant pathogens (e.g., MRSA and VRE), *Mycobacterium tuberculosis*, and various fungi (120–122,124–129). Certain enveloped (lipophilic) viruses (e.g., herpes simplex virus, human immunodeficiency virus [HIV], influenza virus, respiratory syncytial virus, and vaccinia virus) are susceptible to alcohols when tested in vitro (120,130,131) (Table 1). Hepatitis B virus is an enveloped virus that is somewhat less susceptible but is killed by 60%–70% alcohol; hepatitis C virus also is likely killed by this percentage of alcohol (132). In a porcine tissue carrier model used to study antiseptic activity, 70% ethanol and 70% isopropanol were found to

TABLE 1. Virucidal activity of antiseptic agents against enveloped viruses

Ref. no.	Test method	Viruses	Agent	Results
(379)	Suspension	HIV	19% EA	LR = 2.0 in 5 minutes
(380)	Suspension	HIV	50% EA 35% IPA	LR > 3.5 LR > 3.7
(381)	Suspension	HIV	70% EA	LR = 7.0 in 1 minute
(382)	Suspension	HIV	70% EA	LR = 3.2B 5.5 in 30 seconds
(383)	Suspension	HIV	70% IPA/0.5% CHG 4% CHG	LR = 6.0 in 15 seconds LR = 6.0 in 15 seconds
(384)	Suspension	HIV	Chloroxylenol Benzalkonium chloride	Inactivated in 1 minute Inactivated in 1 minute
(385)	Suspension	HIV	Povidone-iodine Chlorhexidine	Inactivated Inactivated
(386)	Suspension	HIV	Detergent/0.5% PCMX	Inactivated in 30 seconds
(387)	Suspension/dried plasma chimpanzee challenge	HBV	70% IPA	LR = 6.0 in 10 minutes
(388)	Suspension/plasma chimpanzee challenge	HBV	80% EA	LR = 7.0 in 2 minutes
(389)	Suspension	HSV	95% EA 75% EA 95% IPA 70% EA + 0.5% CHG	LR > 5.0 in 1 minute LR > 5.0 LR > 5.0 LR > 5.0
(130)	Suspension	RSV	35% IPA 4% CHG	LR > 4.3 in 1 minute LR > 3.3
(141)	Suspension	Influenza Vaccinia	95% EA 95% EA	Undetectable in 30 seconds Undetectable in 30 seconds
(141)	Hand test	Influenza Vaccinia	95% EA 95% EA	LR > 2.5 LR > 2.5

Note: HIV = human immunodeficiency virus, EA = ethanol, LR = Log₁₀ reduction, IPA = isopropanol, CHG = chlorhexidine gluconate, HBV = hepatitis B virus, RSV = respiratory syncytial virus, HSV = herpes simplex virus, HAV = hepatitis A virus, and PCMX = chloroxylenol.

reduce titers of an enveloped bacteriophage more effectively than an antimicrobial soap containing 4% chlorhexidine gluconate (133). Despite its effectiveness against these organisms, alcohols have very poor activity against bacterial spores, protozoan oocysts, and certain nonenveloped (nonlipophilic) viruses.

Numerous studies have documented the *in vivo* antimicrobial activity of alcohols. Alcohols effectively reduce bacterial counts on the hands (14, 121, 125, 134). Typically, log reductions of the release of test bacteria from artificially contaminated hands average 3.5 log₁₀ after a 30-second application and 4.0–5.0 log₁₀ after a 1-minute application (1). In 1994, the FDA TFM classified ethanol 60%–95% as a Category I agent (i.e., generally safe and effective for use in antiseptic handwash or HCW hand-wash products) (19). Although TFM placed isopropanol 70%–91.3% in category IIIIE (i.e., insufficient data to classify as effective), 60% isopropanol has subse-

quently been adopted in Europe as the reference standard against which alcohol-based hand-rub products are compared (79). Alcohols are rapidly germicidal when applied to the skin, but they have no appreciable persistent (i.e., residual) activity. However, regrowth of bacteria on the skin occurs slowly after use of alcohol-based hand antiseptics, presumably because of the sublethal effect alcohols have on some of the skin bacteria (135, 136). Addition of chlorhexidine, quaternary ammonium compounds, octenidine, or triclosan to alcohol-based solutions can result in persistent activity (1).

Alcohols, when used in concentrations present in alcohol-based hand rubs, also have *in vivo* activity against several nonenveloped viruses (Table 2). For example, 70% isopropanol and 70% ethanol are more effective than medicated soap or nonmedicated soap in reducing rotavirus titers on fingerpads (137, 138). A more recent study using the same test methods evaluated a commercially available product containing 60%

TABLE 2. Virucidal activity of antiseptic agents against nonenveloped viruses

Ref. no.	Test method	Viruses	Antiseptic	Result
(390)	Suspension	Rotavirus	4% CHG 10% Povidone-Iodine 70% IPA/0.1% HCP	LR < 3.0 in 1 minute LR > 3.0 LR > 3.0
(141)	Hand test	Adenovirus Poliovirus Coxsackie	95% EA 95% EA 95% EA	LR > 1.4 LR = 0.2–1.0 LR = 1.1–1.3
	Finger test	Adenovirus Poliovirus Coxsackie	95% EA 95% EA 95% EA	LR > 2.3 LR = 0.7–2.5 LR = 2.9
(389)	Suspension	ECHO virus	95% EA 75% EA 95% IPA 70% IPA + 0.5% CHG	LR > 3.0 in 1 minute LR ≤ 1.0 LR = 0 LR = 0
(140)	Finger pad	HAV	70% EA 62% EA foam plain soap 4% CHG 0.3% Triclosan	87.4% reduction 89.3% reduction 78.0% reduction 89.6% reduction 92.0% reduction
(105)	Finger tips	Bovine Rotavirus	n-propanol + IPA 70% IPA 70% EA 2% triclosan water (control) 7.5% povidone-iodine plain soap 4% CHG	LR = 3.8 in 30 seconds LR = 3.1 LR = 2.9 LR = 2.1 LR = 1.3 LR = 1.3 LR = 1.2 LR = 0.5
(137)	Finger pad	Human Rotavirus	70% IPA plain soap	98.9% decrease in 10 seconds 77.1%
(138)	Finger pad	Human Rotavirus	70% IPA 2% CHG plain soap	99.6% decrease in 10 seconds 80.3% 72.5%
(81)	Finger pad	Rotavirus Rhinovirus Adenovirus	60% EA gel 60% EA gel 60% EA gel	LR > 3.0 in 10 seconds LR > 3.0 LR > 3.0
(139)	Finger pad	Poliovirus	70% EA 70% IPA	LR = 1.6 in 10 seconds LR = 0.8
(200)	Finger tips	Poliovirus	Plain soap 80% EA	LR = 2.1 LR = 0.4

Note: HIV = human immunodeficiency virus, EA = ethanol, LR = Log₁₀ reduction, IPA = isopropanol, CHG = chlorhexidine gluconate, HBV = hepatitis B virus, RSV = respiratory syncytial virus, HSV = herpes simplex virus, and HAV = hepatitis A virus.

ethanol and found that the product reduced the infectivity titers of three nonenveloped viruses (i.e., rotavirus, adenovirus, and rhinovirus) by >3 logs (81). Other nonenveloped viruses such as hepatitis A and enteroviruses (e.g., poliovirus) may require 70%–80% alcohol to be reliably inactivated (82,139). However, both 70% ethanol and a 62% ethanol foam product with emollients reduced hepatitis A virus titers on whole hands or fingertips more than nonmedicated soap; both were equally as effective as antimicrobial soap containing 4% chlorhexidine gluconate in reducing reduced viral counts on hands (140). In the same study, both 70% ethanol and the 62% ethanol foam product demonstrated greater virucidal activity against poliovirus than either non-antimicrobial

soap or a 4% chlorhexidine gluconate-containing soap (140). However, depending on the alcohol concentration, the amount of time that hands are exposed to the alcohol, and viral variant, alcohol may not be effective against hepatitis A and other nonlipophilic viruses. The inactivation of nonenveloped viruses is influenced by temperature, disinfectant-virus volume ratio, and protein load (141). Ethanol has greater activity against viruses than isopropanol. Further in vitro and in vivo studies of both alcohol-based formulations and antimicrobial soaps are warranted to establish the minimal level of virucidal activity that is required to interrupt direct contact transmission of viruses in health-care settings.

Alcohols are not appropriate for use when hands are visibly dirty or contaminated with proteinaceous materials. However, when relatively small amounts of proteinaceous material (e.g., blood) are present, ethanol and isopropanol may reduce viable bacterial counts on hands more than plain soap or antimicrobial soap (142).

Alcohol can prevent the transfer of health-care-associated pathogens (25,63,64). In one study, gram-negative bacilli were transferred from a colonized patient's skin to a piece of catheter material via the hands of nurses in only 17% of experiments after antiseptic hand rub with an alcohol-based hand rinse (25). In contrast, transfer of the organisms occurred in 92% of experiments after handwashing with plain soap and water. This experimental model indicates that when the hands of HCWs are heavily contaminated, an antiseptic hand rub using an alcohol-based rinse can prevent pathogen transmission more effectively than can handwashing with plain soap and water.

Alcohol-based products are more effective for standard handwashing or hand antisepsis by HCWs than soap or antimicrobial soaps (Table 3) (25,53,61,93,106–112,119,143–152). In all but two of the trials that compared alcohol-based solutions with antimicrobial soaps or detergents, alcohol reduced bacterial counts on hands more than washing hands with soaps or detergents containing hexachlorophene, povidone-iodine, 4% chlorhexidine, or triclosan. In studies exam-

ining antimicrobial-resistant organisms, alcohol-based products reduced the number of multidrug-resistant pathogens recovered from the hands of HCWs more effectively than did handwashing with soap and water (153–155).

Alcohols are effective for preoperative cleaning of the hands of surgical personnel (1,101,104,113–119,135,143,147,156–159) (Tables 4 and 5). In multiple studies, bacterial counts on the hands were determined immediately after using the product and again 1–3 hours later; the delayed testing was performed to determine if regrowth of bacteria on the hands is inhibited during operative procedures. Alcohol-based solutions were more effective than washing hands with plain soap in all studies, and they reduced bacterial counts on the hands more than antimicrobial soaps or detergents in the majority of experiments (101,104,113–119,135,143,147,157–159). In addition, the majority of alcohol-based preparations were more effective than povidone-iodine or chlorhexidine.

The efficacy of alcohol-based hand-hygiene products is affected by several factors, including the type of alcohol used, concentration of alcohol, contact time, volume of alcohol used, and whether the hands are wet when the alcohol is applied. Applying small volumes (i.e., 0.2–0.5 mL) of alcohol to the hands is not more effective than washing hands with plain soap and water (63,64). One study documented that 1 mL of alcohol was substantially less effective than 3 mL (91). The ideal volume of product to apply to the hands is not known

TABLE 3. Studies comparing the relative efficacy (based on log₁₀ reductions achieved) of plain soap or antimicrobial soaps versus alcohol-based antiseptics in reducing counts of viable bacteria on hands

Ref. no.	Year	Skin contamination	Assay method	Time (sec)	Relative efficacy
(143)	1965	Existing hand flora	Finger-tip agar culture	60	Plain soap < HCP < 50% EA foam
(119)	1975	Existing hand flora	Hand-rub broth culture	—	Plain soap < 95% EA
(106)	1978	Artificial contamination	Finger-tip broth culture	30	Plain soap < 4% CHG < P-I < 70% EA = alc. CHG
(144)	1978	Artificial contamination	Finger-tip broth culture	30	Plain soap < 4% CHG < 70% EA
(107)	1979	Existing hand flora	Hand-rub broth culture	120	Plain soap < 0.5% aq. CHG < 70% EA < 4% CHG < alc.CHG
(145)	1980	Artificial contamination	Finger-tip broth culture	60–120	4% CHG < P-I < 60% IPA
(53)	1980	Artificial contamination	Finger-tip broth culture	15	Plain soap < 3% HCP < P-I < 4% CHG < 70% EA
(108)	1982	Artificial contamination	Glove juice test	15	P-I < alc. CHG
(109)	1983	Artificial contamination	Finger-tip broth culture	120	0.3–2% triclosan = 60% IPA = alc. CHG < alc. triclosan
(146)	1984	Artificial contamination	Finger-tip agar culture	60	Phenolic < 4% CHG < P-I < EA < IPA < n-P
(147)	1985	Existing hand flora	Finger-tip agar culture	60	Plain soap < 70% EA < 95% EA
(110)	1986	Artificial contamination	Finger-tip broth culture	60	Phenolic = P-I < alc. CHG < n-P
(93)	1986	Existing hand flora	Sterile-broth bag technique	15	Plain soap < IPA < 4% CHG = IPA-E = alc. CHG
(61)	1988	Artificial contamination	Finger-tip broth culture	30	Plain soap < triclosan < P-I < IPA < alc. CHG < n-P
(25)	1991	Patient contact	Glove-juice test	15	Plain soap < IPA-E
(148)	1991	Existing hand flora	Agar-plate/image analysis	30	Plain soap < 1% triclosan < P-I < 4% CHG < IPA
(111)	1992	Artificial contamination	Finger-tip agar culture	60	Plain soap < IPA < EA < alc. CHG
(149)	1992	Artificial contamination	Finger-tip broth culture	60	Plain soap < 60% n-P
(112)	1994	Existing hand flora	Agar-plate/image analysis	30	Plain soap < alc. CHG
(150)	1999	Existing hand flora	Agar-plate culture	N.S.	Plain soap < commercial alcohol mixture
(151)	1999	Artificial contamination	Glove-juice test	20	Plain soap < 0.6% PCMX < 65% EA
(152)	1999	Artificial contamination	Finger-tip broth culture	30	4% CHG < plain soap < P-I < 70% EA

Note: Existing hand flora = without artificially contaminating hands with bacteria, alc. CHG = alcoholic chlorhexidine gluconate, aq. CHG = aqueous chlorhexidine gluconate, 4% CHG = chlorhexidine gluconate detergent, EA = ethanol, HCP = hexachlorophene soap/detergent, IPA = isopropanol, IPA-E = isopropanol + emollients, n-P = n-propanol, PCMX = chloroxyleneol detergent, P-I = povidone-iodine detergent, and N.S. = not stated.

TABLE 4. Studies comparing the relative efficacy of plain soap or antimicrobial soap versus alcohol-containing products in reducing counts of bacteria recovered from hands immediately after use of products for pre-operative cleansing of hands

Ref. no.	Year	Assay method	Relative efficacy
(143)	1965	Finger-tip agar culture	HCP < 50% EA foam + QAC
(157)	1969	Finger-tip agar culture	HCP < P-I < 50% EA foam + QAC
(101)	1973	Finger-tip agar culture	HCP soap < EA foam + 0.23% HCP
(135)	1974	Broth culture	Plain soap < 0.5% CHG < 4% CHG < alc. CHG
(119)	1975	Hand-broth test	Plain soap < 0.5% CHG < 4% CHG < alc. CHG
(118)	1976	Glove-juice test	0.5% CHG < 4% CHG < alc. CHG
(114)	1977	Glove-juice test	P-I < CHG < alc. CHG
(117)	1978	Finger-tip agar culture	P-I = 46% EA + 0.23% HCP
(113)	1979	Broth culture of hands	Plain soap < P-I < alc. CHG < alc. P-I
(116)	1979	Glove-juice test	70% IPA = alc. CHG
(147)	1985	Finger-tip agar culture	Plain soap < 70% - 90% EA
(115)	1990	Glove-juice test, modified	Plain soap < triclosan < CHG < P-I < alc. CHG
(104)	1991	Glove-juice test	Plain soap < 2% triclosan < P-I < 70% IPA
(158)	1998	Finger-tip broth culture	70% IPA < 90% IPA = 60% n-P
(159)	1998	Glove-juice test	P-I < CHG < 70% EA

Note: QAC = quaternary ammonium compound, alc. CHG = alcoholic chlorhexidine gluconate, CHG = chlorhexidine gluconate detergent, EA = ethanol, HCP = hexachlorophene detergent, IPA = isopropanol, and P-I = povidone-iodine detergent.

TABLE 5. Efficacy of surgical hand-rub solutions in reducing the release of resident skin flora from clean hands

Study	Rub	Concentration* (%)	Time (min)	Mean log reduction				
				Immediate	Sustained (3 hr)			
1	n-Propanol	60	5	2.9 [†]	1.6 [†]			
2			5	2.7 [†]	NA			
3			5	2.5 [†]	1.8 [†]			
4			5	2.3 [†]	1.6 [†]			
5			3	2.9 [§]	NA			
4			3	2.0 [†]	1.0 [†]			
4			1	1.1 [†]	0.5 [†]			
6			Isopropanol	90	3	2.4 [§]	1.4 [§]	
6					3	2.3 [§]	1.2 [§]	
7					5	2.4 [†]	2.1 [†]	
4	5	2.1 [†]			1.0 [†]			
6	3	2.0 [§]			0.7 [§]			
5	3	1.7 ^c			NA			
4	3	1.5 [†]			0.8 [†]			
8	2	1.2			0.8			
4	1	0.7 [†]	0.2					
9	1	0.8	NA					
10	Isopropanol + chlorhexidine gluc. (w/v)	60	5	1.7	1.0			
7			70 + 0.5	5	2.5 [†]	2.7 [†]		
8				2	1.0	1.5		
11				Ethanol	95	2.1	NA	
5					85	2.4 [§]	NA	
12					80	1.5	NA	
8					70	1.0	0.6	
13				Ethanol + chlorhexidine gluc. (w/v)	95 + 0.5	2	1.7	NA
14					77 + 0.5	5	2.0	1.5 [¶]
8					70 + 0.5	2	0.7	1.4
8	Chlorhexidine gluc. (aq. Sol., w/v)	0.5		2	0.4	1.2		
15	Povidone-iodine (aq. Sol., w/v)	1.0	5	1.9 [†]	0.8 [†]			
16	Peracetic acid (w/v)	0.5	5	1.9	NA			

Note: NA = not available.

Source: Rotter M. Hand washing and hand disinfection [Chapter 87]. In: Mayhall CG, ed. Hospital epidemiology and infection control. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999. Table 5 is copyrighted by Lippincott Williams & Wilkins; it is reprinted here with their permission and permission from Manfred Rotler, M.D., Professor of Hygiene and Microbiology, Klinisches Institute für Hygiene der Universität Wien, Germany.

* Volume/volume unless otherwise stated.

[†] Tested according to Deutsche Gesellschaft für Hygiene, and Mikrobiologic (DGHM)-German Society of Hygiene and Microbiology method.

[§] Tested according to European Standard prEN.

[¶] After 4 hours.

and may vary for different formulations. However, if hands feel dry after rubbing hands together for 10–15 seconds, an insufficient volume of product likely was applied. Because alcohol-impregnated towelettes contain a limited amount of alcohol, their effectiveness is comparable to that of soap and water (63,160,161).

Alcohol-based hand rubs intended for use in hospitals are available as low viscosity rinses, gels, and foams. Limited data are available regarding the relative efficacy of various formulations. One field trial demonstrated that an ethanol gel was slightly more effective than a comparable ethanol solution at reducing bacterial counts on the hands of HCWs (162). However, a more recent study indicated that rinses reduced bacterial counts on the hands more than the gels tested (80). Further studies are warranted to determine the relative efficacy of alcohol-based rinses and gels in reducing transmission of health-care-associated pathogens.

Frequent use of alcohol-based formulations for hand antisepsis can cause drying of the skin unless emollients, humectants, or other skin-conditioning agents are added to the formulations. The drying effect of alcohol can be reduced or eliminated by adding 1%–3% glycerol or other skin-conditioning agents (90,93,100,101,106,135,143,163,164). Moreover, in several recent prospective trials, alcohol-based rinses or gels containing emollients caused substantially less skin irritation and dryness than the soaps or antimicrobial detergents tested (96,98,165,166). These studies, which were conducted in clinical settings, used various subjective and objective methods for assessing skin irritation and dryness. Further studies are warranted to establish whether products with different formulations yield similar results.

Even well-tolerated alcohol hand rubs containing emollients may cause a transient stinging sensation at the site of any broken skin (e.g., cuts and abrasions). Alcohol-based hand-rub preparations with strong fragrances may be poorly tolerated by HCWs with respiratory allergies. Allergic contact dermatitis or contact urticaria syndrome caused by hypersensitivity to alcohol or to various additives present in certain alcohol hand rubs occurs only rarely (167,168).

Alcohols are flammable. Flash points of alcohol-based hand rubs range from 21°C to 24°C, depending on the type and concentration of alcohol present (169). As a result, alcohol-based hand rubs should be stored away from high temperatures or flames in accordance with National Fire Protection Agency recommendations. In Europe, where alcohol-based hand rubs have been used extensively for years, the incidence of fires associated with such products has been low (169). One recent U.S. report described a flash fire that occurred as a result of an unusual series of events, which included an HCW applying an alcohol gel to her hands, immediately removing a

polyester isolation gown, and then touching a metal door before the alcohol had evaporated (170). Removing the polyester gown created a substantial amount of static electricity that generated an audible static spark when the HCW touched the metal door, igniting the unevaporated alcohol on her hands (170). This incident emphasizes the need to rub hands together after application of alcohol-based products until all the alcohol has evaporated.

Because alcohols are volatile, containers should be designed to minimize evaporation. Contamination of alcohol-based solutions has seldom been reported. One report documented a cluster of pseudoinfections caused by contamination of ethyl alcohol by *Bacillus cereus* spores (171).

Chlorhexidine

Chlorhexidine gluconate, a cationic bisbiguanide, was developed in England in the early 1950s and was introduced into the United States in the 1970s (8,172). Chlorhexidine base is only minimally soluble in water, but the digluconate form is water-soluble. The antimicrobial activity of chlorhexidine is likely attributable to attachment to, and subsequent disruption of, cytoplasmic membranes, resulting in precipitation of cellular contents (1,8). Chlorhexidine's immediate antimicrobial activity occurs more slowly than that of alcohols. Chlorhexidine has good activity against gram-positive bacteria, somewhat less activity against gram-negative bacteria and fungi, and only minimal activity against tubercle bacilli (1,8,172). Chlorhexidine is not sporicidal (1,172). It has in vitro activity against enveloped viruses (e.g., herpes simplex virus, HIV, cytomegalovirus, influenza, and RSV) but substantially less activity against nonenveloped viruses (e.g., rotavirus, adenovirus, and enteroviruses) (130,131,173). The antimicrobial activity of chlorhexidine is only minimally affected by the presence of organic material, including blood. Because chlorhexidine is a cationic molecule, its activity can be reduced by natural soaps, various inorganic anions, nonionic surfactants, and hand creams containing anionic emulsifying agents (8,172,174). Chlorhexidine gluconate has been incorporated into a number of hand-hygiene preparations. Aqueous or detergent formulations containing 0.5% or 0.75% chlorhexidine are more effective than plain soap, but they are less effective than antiseptic detergent preparations containing 4% chlorhexidine gluconate (135,175). Preparations with 2% chlorhexidine gluconate are slightly less effective than those containing 4% chlorhexidine (176).

Chlorhexidine has substantial residual activity (106,114–116,118,135,146,175). Addition of low concentrations (0.5%–1.0%) of chlorhexidine to alcohol-based preparations results in greater residual activity than alcohol alone (116,135). When used as recommended, chlorhexidine has a good safety

record (172). Minimal, if any, absorption of the compound occurs through the skin. Care must be taken to avoid contact with the eyes when using preparations with $\geq 1\%$ chlorhexidine, because the agent can cause conjunctivitis and severe corneal damage. Ototoxicity precludes its use in surgery involving the inner or middle ear. Direct contact with brain tissue and the meninges should be avoided. The frequency of skin irritation is concentration-dependent, with products containing 4% most likely to cause dermatitis when used frequently for antiseptic handwashing (177); allergic reactions to chlorhexidine gluconate are uncommon (118,172). Occasional outbreaks of nosocomial infections have been traced to contaminated solutions of chlorhexidine (178–181).

Chloroxylenol

Chloroxylenol, also known as parachlorometaxylenol (PCMX), is a halogen-substituted phenolic compound that has been used as a preservative in cosmetics and other products and as an active agent in antimicrobial soaps. It was developed in Europe in the late 1920s and has been used in the United States since the 1950s (182).

The antimicrobial activity of PCMX likely is attributable to inactivation of bacterial enzymes and alteration of cell walls (1). It has good in vitro activity against gram-positive organisms and fair activity against gram-negative bacteria, mycobacteria, and certain viruses (1,7,182). PCMX is less active against *P. aeruginosa*, but addition of ethylenediaminetetraacetic acid (EDTA) increases its activity against *Pseudomonas* spp. and other pathogens.

A limited number of articles focusing on the efficacy of PCMX-containing preparations intended for use by HCWs have been published in the last 25 years, and the results of studies have sometimes been contradictory. For example, in studies in which antiseptics were applied to abdominal skin, PCMX had the weakest immediate and residual activity of any of the agents studied (183). However, when 30-second handwashes were performed using 0.6% PCMX, 2% chlorhexidine gluconate, or 0.3% triclosan, the immediate effect of PCMX was similar to that of the other agents. When used 18 times per day for 5 consecutive days, PCMX had less cumulative activity than did chlorhexidine gluconate (184). When PCMX was used as a surgical scrub, one report indicated that 3% PCMX had immediate and residual activity comparable to 4% chlorhexidine gluconate (185), whereas two other studies demonstrated that the immediate and residual activity of PCMX was inferior to both chlorhexidine gluconate and povidone-iodine (176,186). The disparity between published studies may be associated with the various concentrations of PCMX included in the preparations evaluated and with other aspects of the formulations tested, including the

presence or absence of EDTA (7,182). PCMX is not as rapidly active as chlorhexidine gluconate or iodophors, and its residual activity is less pronounced than that observed with chlorhexidine gluconate (7,182). In 1994, FDA TFM tentatively classified PCMX as a Category III SE active agent (i.e., insufficient data are available to classify this agent as safe and effective) (19). Further evaluation of this agent by the FDA is ongoing.

The antimicrobial activity of PCMX is minimally affected by the presence of organic matter, but it is neutralized by non-ionic surfactants. PCMX, which is absorbed through the skin (7,182), is usually well-tolerated, and allergic reactions associated with its use are uncommon. PCMX is available in concentrations of 0.3%–3.75%. In-use contamination of a PCMX-containing preparation has been reported (187).

Hexachlorophene

Hexachlorophene is a bisphenol composed of two phenolic groups and three chlorine moieties. In the 1950s and early 1960s, emulsions containing 3% hexachlorophene were widely used for hygienic handwashing, as surgical scrubs, and for routine bathing of infants in hospital nurseries. The antimicrobial activity of hexachlorophene results from its ability to inactivate essential enzyme systems in microorganisms. Hexachlorophene is bacteriostatic, with good activity against *S. aureus* and relatively weak activity against gram-negative bacteria, fungi, and mycobacteria (7).

Studies of hexachlorophene as a hygienic handwash and surgical scrub demonstrated only modest efficacy after a single handwash (53,143,188). Hexachlorophene has residual activity for several hours after use and gradually reduces bacterial counts on hands after multiple uses (i.e., it has a cumulative effect) (1,101,188,189). With repeated use of 3% hexachlorophene preparations, the drug is absorbed through the skin. Infants bathed with hexachlorophene and personnel regularly using a 3% hexachlorophene preparation for handwashing have blood levels of 0.1–0.6 ppm hexachlorophene (190). In the early 1970s, certain infants bathed with hexachlorophene developed neurotoxicity (vacuolar degeneration) (191). As a result, in 1972, the FDA warned that hexachlorophene should no longer be used routinely for bathing infants. However, after routine use of hexachlorophene for bathing infants in nurseries was discontinued, investigators noted that the incidence of health-care-associated *S. aureus* infections in hospital nurseries increased substantially (192,193). In several instances, the frequency of infections decreased when hexachlorophene bathing of infants was reinstated. However, current guidelines still recommend against the routine bathing of neonates with hexachlorophene because of its potential neurotoxic effects (194). The agent is classified by FDA TFM as not

generally recognized as safe and effective for use as an antiseptic handwash (19). Hexachlorophene should not be used to bathe patients with burns or extensive areas of susceptible, sensitive skin. Soaps containing 3% hexachlorophene are available by prescription only (7).

Iodine and Iodophors

Iodine has been recognized as an effective antiseptic since the 1800s. However, because iodine often causes irritation and discoloring of skin, iodophors have largely replaced iodine as the active ingredient in antiseptics.

Iodine molecules rapidly penetrate the cell wall of microorganisms and inactivate cells by forming complexes with amino acids and unsaturated fatty acids, resulting in impaired protein synthesis and alteration of cell membranes (195). Iodophors are composed of elemental iodine, iodide or triiodide, and a polymer carrier (i.e., the complexing agent) of high molecular weight. The amount of molecular iodine present (so-called “free” iodine) determines the level of antimicrobial activity of iodophors. “Available” iodine refers to the total amount of iodine that can be titrated with sodium thiosulfate (196). Typical 10% povidone-iodine formulations contain 1% available iodine and yield free iodine concentrations of 1 ppm (196). Combining iodine with various polymers increases the solubility of iodine, promotes sustained release of iodine, and reduces skin irritation. The most common polymers incorporated into iodophors are polyvinyl pyrrolidone (i.e., povidone) and ethoxylated nonionic detergents (i.e., poloxamers) (195,196). The antimicrobial activity of iodophors also can be affected by pH, temperature, exposure time, concentration of total available iodine, and the amount and type of organic and inorganic compounds present (e.g., alcohols and detergents).

Iodine and iodophors have bactericidal activity against gram-positive, gram-negative, and certain spore-forming bacteria (e.g., clostridia and *Bacillus* spp.) and are active against mycobacteria, viruses, and fungi (8,195,197–200). However, in concentrations used in antiseptics, iodophors are not usually sporicidal (201). In vivo studies have demonstrated that iodophors reduce the number of viable organisms that are recovered from the hands of personnel (113,145,148,152,155). Povidone-iodine 5%–10% has been tentatively classified by FDA TFM as a Category I agent (i.e., a safe and effective agent for use as an antiseptic handwash and an HCW handwash) (19). The extent to which iodophors exhibit persistent antimicrobial activity after they have been washed off the skin is unclear. In one study, persistent activity was noted for 6 hours (176); however, several other studies demonstrated persistent activity for only 30–60 minutes after washing hands with an iodophor (61,117,202). In studies in which bacterial counts

were obtained after gloves were worn for 1–4 hours after washing, iodophors have demonstrated poor persistent activity (1,104,115,189,203–208). The in vivo antimicrobial activity of iodophors is substantially reduced in the presence of organic substances (e.g., blood or sputum) (8).

The majority of iodophor preparations used for hand hygiene contain 7.5%–10% povidone-iodine. Formulations with lower concentrations also have good antimicrobial activity because dilution can increase free iodine concentrations (209). However, as the amount of free iodine increases, the degree of skin irritation also may increase (209). Iodophors cause less skin irritation and fewer allergic reactions than iodine, but more irritant contact dermatitis than other antiseptics commonly used for hand hygiene (92). Occasionally, iodophor antiseptics have become contaminated with gram-negative bacilli as a result of poor manufacturing processes and have caused outbreaks or pseudo-outbreaks of infection (196).

Quaternary Ammonium Compounds

Quaternary ammonium compounds are composed of a nitrogen atom linked directly to four alkyl groups, which may vary in their structure and complexity (210). Of this large group of compounds, alkyl benzalkonium chlorides are the most widely used as antiseptics. Other compounds that have been used as antiseptics include benzethonium chloride, cetrимide, and cetylpyridium chloride (1). The antimicrobial activity of these compounds was first studied in the early 1900s, and a quaternary ammonium compound for preoperative cleaning of surgeons' hands was used as early as 1935 (210). The antimicrobial activity of this group of compounds likely is attributable to adsorption to the cytoplasmic membrane, with subsequent leakage of low molecular weight cytoplasmic constituents (210).

Quaternary ammonium compounds are primarily bacteriostatic and fungistatic, although they are microbicidal against certain organisms at high concentrations (1); they are more active against gram-positive bacteria than against gram-negative bacilli. Quaternary ammonium compounds have relatively weak activity against mycobacteria and fungi and have greater activity against lipophilic viruses. Their antimicrobial activity is adversely affected by the presence of organic material, and they are not compatible with anionic detergents (1,210). In 1994, FDA TFM tentatively classified benzalkonium chloride and benzethonium chloride as Category IIISE active agents (i.e., insufficient data exists to classify them as safe and effective for use as an antiseptic handwash) (19). Further evaluation of these agents by FDA is in progress.

Quaternary ammonium compounds are usually well tolerated. However, because of weak activity against

gram-negative bacteria, benzalkonium chloride is prone to contamination by these organisms. Several outbreaks of infection or pseudoinfection have been traced to quaternary ammonium compounds contaminated with gram-negative bacilli (211–213). For this reason, in the United States, these compounds have been seldom used for hand antisepsis during the last 15–20 years. However, newer handwashing products containing benzalkonium chloride or benzethonium chloride have recently been introduced for use by HCWs. A recent study of surgical intensive-care unit personnel found that cleaning hands with antimicrobial wipes containing a quaternary ammonium compound was about as effective as using plain soap and water for handwashing; both were less effective than decontaminating hands with an alcohol-based hand rub (214). One laboratory-based study reported that an alcohol-free hand-rub product containing a quaternary ammonium compound was efficacious in reducing microbial counts on the hands of volunteers (215). Further studies of such products are needed to determine if newer formulations are effective in health-care settings.

Triclosan

Triclosan (chemical name: 2,4,4'-trichloro-2'-hydroxydiphenyl ether) is a nonionic, colorless substance that was developed in the 1960s. It has been incorporated into soaps for use by HCWs and the public and into other consumer products. Concentrations of 0.2%–2% have antimicrobial activity. Triclosan enters bacterial cells and affects the cytoplasmic membrane and synthesis of RNA, fatty acids, and proteins (216). Recent studies indicate this agent's antibacterial activity is attributable to binding to the active site of enoyl-acyl carrier protein reductase (217,218).

Triclosan has a broad range of antimicrobial activity, but it is often bacteriostatic (1). Minimum inhibitory concentrations (MICs) range from 0.1 to 10 µg/mL, whereas minimum bactericidal concentrations are 25–500 µg/mL. Triclosan's activity against gram-positive organisms (including MRSA) is greater than against gram-negative bacilli, particularly *P. aeruginosa* (1,216). The agent possesses reasonable activity against mycobacterial and *Candida* spp., but it has limited activity against filamentous fungi. Triclosan (0.1%) reduces bacterial counts on hands by 2.8 log₁₀ after a 1-minute hygienic handwash (1). In several studies, log reductions have been lower after triclosan is used than when chlorhexidine, iodophors, or alcohol-based products are applied (1,61,149,184,219). In 1994, FDA TFM tentatively classified triclosan ≤1.0% as a Category IIISE active agent (i.e., insufficient data exist to classify this agent as safe and effective for use as an antiseptic handwash) (19). Further evaluation of this agent by the FDA is underway. Like chlorhexidine, triclosan has persistent activity on the skin. Its activity in

hand-care products is affected by pH, the presence of surfactants, emollients, or humectants and by the ionic nature of the particular formulation (1,216). Triclosan's activity is not substantially affected by organic matter, but it can be inhibited by sequestration of the agent in micelle structures formed by surfactants present in certain formulations. The majority of formulations containing <2% triclosan are well-tolerated and seldom cause allergic reactions. Certain reports indicate that providing hospital personnel with a triclosan-containing preparation for hand antisepsis has led to decreased MRSA infections (72,73). Triclosan's lack of potent activity against gram-negative bacilli has resulted in occasional reports of contamination (220).

Other Agents

Approximately 150 years after puerperal-fever-related maternal mortality rates were demonstrated by Semmelweis to be reduced by use of a hypochlorite hand rinse, the efficacy of rubbing hands for 30 seconds with an aqueous hypochlorite solution was studied once again (221). The solution was demonstrated to be no more effective than distilled water. The regimen used by Semmelweis, which called for rubbing hands with a 4% [w/w] hypochlorite solution until the hands were slippery (approximately 5 minutes), has been revisited by other researchers (222). This more current study indicated that the regimen was 30 times more effective than a 1-minute rub using 60% isopropanol. However, because hypochlorite solutions are often irritating to the skin when used repeatedly and have a strong odor, they are seldom used for hand hygiene.

Certain other agents are being evaluated by FDA for use in health-care-related antiseptics (19). However, the efficacy of these agents has not been evaluated adequately for use in handwashing preparations intended for use by HCWs. Further evaluation of these agents is warranted. Products that use different concentrations of traditional antiseptics (e.g., low concentrations of iodophor) or contain novel compounds with antiseptic properties are likely to be introduced for use by HCWs. For example, preliminary studies have demonstrated that adding silver-containing polymers to an ethanol carrier (i.e., Surfacine®) results in a preparation that has persistent antimicrobial activity on animal and human skin (223). New compounds with good in vitro activity must be tested in vivo to determine their abilities to reduce transient and resident skin flora on the hands of HCWs.

Activity of Antiseptic Agents Against Spore-Forming Bacteria

The widespread prevalence of health-care-associated diarrhea caused by *Clostridium difficile* and the recent occurrence

in the United States of human *Bacillus anthracis* infections associated with contaminated items sent through the postal system has raised concern regarding the activity of antiseptic agents against spore-forming bacteria. None of the agents (including alcohols, chlorhexidine, hexachlorophene, iodophors, PCMX, and triclosan) used in antiseptic handwash or antiseptic hand-rub preparations are reliably sporicidal against *Clostridium* spp. or *Bacillus* spp. (120,172,224,225). Washing hands with non-antimicrobial or antimicrobial soap and water may help to physically remove spores from the surface of contaminated hands. HCWs should be encouraged to wear gloves when caring for patients with *C. difficile*-associated diarrhea (226). After gloves are removed, hands should be washed with a non-antimicrobial or an antimicrobial soap and water or disinfected with an alcohol-based hand rub. During outbreaks of *C. difficile*-related infections, washing hands with a non-antimicrobial or antimicrobial soap and water after removing gloves is prudent. HCWs with suspected or documented exposure to *B. anthracis*-contaminated items also should be encouraged to wash their hands with a non-antimicrobial or antimicrobial soap and water.

Reduced Susceptibility of Bacteria to Antiseptics

Reduced susceptibility of bacteria to antiseptic agents can either be an intrinsic characteristic of a species or can be an acquired trait (227). Several reports have described strains of bacteria that appear to have acquired reduced susceptibility (when defined by MICs established in vitro) to certain antiseptics (e.g., chlorhexidine, quaternary ammonium compounds, and triclosan) (227–230). However, because the antiseptic concentrations that are actually used by HCWs are often substantially higher than the MICs of strains with reduced antiseptic susceptibility, the clinical relevance of the in vitro findings is questionable. For example, certain strains of MRSA have chlorhexidine and quaternary ammonium compound MICs that are several-fold higher than methicillin-susceptible strains, and certain strains of *S. aureus* have elevated MICs to triclosan (227,228). However, such strains were readily inhibited by the concentrations of these antiseptics that are actually used by practicing HCWs (227,228). The description of a triclosan-resistant bacterial enzyme has raised the question of whether resistance to this agent may develop more readily than to other antiseptic agents (218). In addition, exposing *Pseudomonas* strains containing the MexAB-OprM efflux system to triclosan may select for mutants that are resistant to multiple antibiotics, including fluoroquinolones (230). Further studies are needed to determine whether reduced susceptibility to antiseptic agents is of epidemiologic

significance and whether resistance to antiseptics has any influence on the prevalence of antibiotic-resistant strains (227).

Surgical Hand Antisepsis

Since the late 1800s, when Lister promoted the application of carbolic acid to the hands of surgeons before procedures, preoperative cleansing of hands and forearms with an antiseptic agent has been an accepted practice (231). Although no randomized, controlled trials have been conducted to indicate that surgical-site infection rates are substantially lower when preoperative scrubbing is performed with an antiseptic agent rather than a non-antimicrobial soap, certain other factors provide a strong rationale for this practice. Bacteria on the hands of surgeons can cause wound infections if introduced into the operative field during surgery (232); rapid multiplication of bacteria occurs under surgical gloves if hands are washed with a non-antimicrobial soap. However, bacterial growth is slowed after preoperative scrubbing with an antiseptic agent (14,233). Reducing resident skin flora on the hands of the surgical team for the duration of a procedure reduces the risk of bacteria being released into the surgical field if gloves become punctured or torn during surgery (1,156,169). Finally, at least one outbreak of surgical-site infections occurred when surgeons who normally used an antiseptic surgical scrub preparation began using a non-antimicrobial product (234).

Antiseptic preparations intended for use as surgical hand scrubs are evaluated for their ability to reduce the number of bacteria released from hands at different times, including 1) immediately after scrubbing, 2) after wearing surgical gloves for 6 hours (i.e., persistent activity), and 3) after multiple applications over 5 days (i.e., cumulative activity). Immediate and persistent activity are considered the most important in determining the efficacy of the product. U.S. guidelines recommend that agents used for surgical hand scrubs should substantially reduce microorganisms on intact skin, contain a nonirritating antimicrobial preparation, have broad-spectrum activity, and be fast-acting and persistent (19,235).

Studies have demonstrated that formulations containing 60%–95% alcohol alone or 50%–95% when combined with limited amounts of a quaternary ammonium compound, hexachlorophene, or chlorhexidine gluconate, lower bacterial counts on the skin immediately postscrub more effectively than do other agents (Table 4). The next most active agents (in order of decreasing activity) are chlorhexidine gluconate, iodophors, triclosan, and plain soap (104,119,186,188,203,204,206,208,236). Because studies of PCMX as a surgical scrub have yielded contradictory results, further studies are needed to establish how the efficacy of this compound compares with the other agents (176,185,186).

Although alcohols are not considered to have persistent antimicrobial activity, bacteria appear to reproduce slowly on the hands after a surgical scrub with alcohol, and bacterial counts on hands after wearing gloves for 1–3 hours seldom exceed baseline (i.e., prescrub) values (1). However, a recent study demonstrated that a formulation containing 61% ethanol alone did not achieve adequate persistent activity at 6 hours postscrub (237). Alcohol-based preparations containing 0.5% or 1% chlorhexidine gluconate have persistent activity that, in certain studies, has equaled or exceeded that of chlorhexidine gluconate-containing detergents (1,118,135,237).*

Persistent antimicrobial activity of detergent-based surgical scrub formulations is greatest for those containing 2% or 4% chlorhexidine gluconate, followed by hexachlorophene, triclosan, and iodophors (1,102,113–115,159,189,203,204,206–208,236). Because hexachlorophene is absorbed into the blood after repeated use, it is seldom used as a surgical scrub.

Surgical staff have been traditionally required to scrub their hands for 10 minutes preoperatively, which frequently leads to skin damage. Several studies have demonstrated that scrubbing for 5 minutes reduces bacterial counts as effectively as a 10-minute scrub (117,238,239). In other studies, scrubbing for 2 or 3 minutes reduced bacterial counts to acceptable levels (156,205,207,240,241).

Studies have indicated that a two-stage surgical scrub using an antiseptic detergent, followed by application of an alcohol-containing preparation, is effective. For example, an initial 1- or 2-minute scrub with 4% chlorhexidine gluconate or povidone-iodine followed by application of an alcohol-based product has been as effective as a 5-minute scrub with an antiseptic detergent (114,242).

Surgical hand-antiseptic protocols have required personnel to scrub with a brush. But this practice can damage the skin of personnel and result in increased shedding of bacteria from the hands (95,243). Scrubbing with a disposable sponge or combination sponge-brush has reduced bacterial counts on the hands as effectively as scrubbing with a brush (244–246). However, several studies indicate that neither a brush nor a

sponge is necessary to reduce bacterial counts on the hands of surgical personnel to acceptable levels, especially when alcohol-based products are used (102,117,159,165,233,237,247,248). Several of these studies performed cultures immediately or at 45–60 minutes postscrub (102,117,233,247,248), whereas in other studies, cultures were obtained 3 and 6 hours postscrub (159,237). For example, a recent laboratory-based study using volunteers demonstrated that brushless application of a preparation containing 1% chlorhexidine gluconate plus 61% ethanol yielded lower bacterial counts on the hands of participants than using a sponge/brush to apply a 4% chlorhexidine-containing detergent preparation (237).

Relative Efficacy of Plain Soap, Antiseptic Soap/Detergent, and Alcohols

Comparing studies related to the in vivo efficacy of plain soap, antimicrobial soaps, and alcohol-based hand rubs is problematic, because certain studies express efficacy as the percentage reduction in bacterial counts achieved, whereas others give log₁₀ reductions in counts achieved. However, summarizing the relative efficacy of agents tested in each study can provide an overview of the in vivo activity of various formulations intended for handwashing, hygienic handwash, antiseptic hand rub, or surgical hand antiseptics (Tables 2–4).

Irritant Contact Dermatitis Resulting from Hand-Hygiene Measures

Frequency and Pathophysiology of Irritant Contact Dermatitis

In certain surveys, approximately 25% of nurses report symptoms or signs of dermatitis involving their hands, and as many as 85% give a history of having skin problems (249). Frequent and repeated use of hand-hygiene products, particularly soaps and other detergents, is a primary cause of chronic irritant contact dermatitis among HCWs (250). The potential of detergents to cause skin irritation can vary considerably and can be ameliorated by the addition of emollients and humectants. Irritation associated with antimicrobial soaps may be caused by the antimicrobial agent or by other ingredients of the formulation. Affected persons often complain of a feeling of dryness or burning; skin that feels “rough;” and erythema, scaling, or fissures. Detergents damage the skin by causing denaturation of stratum corneum proteins, changes in intercellular lipids (either depletion or reorganization of lipid moieties), decreased corneocyte cohesion, and decreased stratum corneum water-binding capacity (250,251). Damage

* In a recent randomized clinical trial, surgical site infection rates were monitored among patients who were operated on by surgical personnel who cleaned their hands preoperatively either by performing a traditional 5-minute surgical hand scrub using 4% povidone-iodine or 4% antiseptic antimicrobial soap, or by washing their hands for 1 minute with a non-antimicrobial soap followed by a 5-minute hand-rubbing technique using an alcohol-based hand rinse containing 0.2% mectronium etilsulfate. The incidence of surgical site infections was virtually identical in the two groups of patients. (Source: Parienti JJ, Thibon P, Heller R, et al. for Members of the Antiseptic Chirurgicale des Mains Study Group. Hand-rubbing with an aqueous alcoholic solution vs traditional surgical hand-scrubbing and 30-day surgical site infection rates: a randomized equivalence study. JAMA 2002;288:722–7).

to the skin also changes skin flora, resulting in more frequent colonization by staphylococci and gram-negative bacilli (17,90). Although alcohols are among the safest antiseptics available, they can cause dryness and irritation of the skin (1,252). Ethanol is usually less irritating than n-propanol or isopropanol (252).

Irritant contact dermatitis is more commonly reported with iodophors (92). Other antiseptic agents that can cause irritant contact dermatitis (in order of decreasing frequency) include chlorhexidine, PCMX, triclosan, and alcohol-based products. Skin that is damaged by repeated exposure to detergents may be more susceptible to irritation by alcohol-based preparations (253). The irritancy potential of commercially prepared hand-hygiene products, which is often determined by measuring transepidermal water loss, may be available from the manufacturer. Other factors that can contribute to dermatitis associated with frequent handwashing include using hot water for handwashing, low relative humidity (most common in winter months), failure to use supplementary hand lotion or cream, and the quality of paper towels (254,255). Shear forces associated with wearing or removing gloves and allergy to latex proteins may also contribute to dermatitis of the hands of HCWs.

Allergic Contact Dermatitis Associated with Hand-Hygiene Products

Allergic reactions to products applied to the skin (i.e., contact allergies) may present as delayed type reactions (i.e., allergic contact dermatitis) or less commonly as immediate reactions (i.e., contact urticaria). The most common causes of contact allergies are fragrances and preservatives; emulsifiers are less common causes (256–259). Liquid soaps, hand lotions or creams, and “udder ointments” may contain ingredients that cause contact allergies among HCWs (257,258).

Allergic reactions to antiseptic agents, including quaternary ammonium compounds, iodine or iodophors, chlorhexidine, triclosan, PCMX, and alcohols have been reported (118,167,172,256,260–265). Allergic contact dermatitis associated with alcohol-based hand rubs is uncommon. Surveillance at a large hospital in Switzerland, where a commercial alcohol hand rub has been used for >10 years, failed to identify a single case of documented allergy to the product (169). In late 2001, a Freedom of Information Request for data in the FDA’s Adverse Event Reporting System regarding adverse reactions to popular alcohol hand rubs in the United States yielded only one reported case of an erythematous rash reaction attributed to such a product (John M. Boyce, M.D., Hospital of St. Raphael, New Haven, Connecticut, personal communication, 2001). However, with increasing use of such products by HCWs, true allergic reactions to such products likely will be encountered.

Allergic reactions to alcohol-based products may represent true allergy to alcohol, allergy to an impurity or aldehyde metabolite, or allergy to another constituent of the product (167). Allergic contact dermatitis or immediate contact urticarial reactions may be caused by ethanol or isopropanol (167). Allergic reactions can be caused by compounds that may be present as inactive ingredients in alcohol-based hand rubs, including fragrances, benzyl alcohol, stearyl or isostearyl alcohol, phenoxyethanol, myristyl alcohol, propylene glycol, parabens, and benzalkonium chloride (167,256,266–270).

Proposed Methods for Reducing Adverse Effects of Agents

Potential strategies for minimizing hand-hygiene-related irritant contact dermatitis among HCWs include reducing the frequency of exposure to irritating agents (particularly anionic detergents), replacing products with high irritation potential with preparations that cause less damage to the skin, educating personnel regarding the risks of irritant contact dermatitis, and providing caregivers with moisturizing skin-care products or barrier creams (96,98,251,271–273). Reducing the frequency of exposure of HCWs to hand-hygiene products would prove difficult and is not desirable because of the low levels of adherence to hand-hygiene policies in the majority of institutions. Although hospitals have provided personnel with non-antimicrobial soaps in hopes of minimizing dermatitis, frequent use of such products may cause greater skin damage, dryness, and irritation than antiseptic preparations (92,96,98). One strategy for reducing the exposure of personnel to irritating soaps and detergents is to promote the use of alcohol-based hand rubs containing various emollients. Several recent prospective, randomized trials have demonstrated that alcohol-based hand rubs containing emollients were better tolerated by HCWs than washing hands with non-antimicrobial soaps or antimicrobial soaps (96,98,166). Routinely washing hands with soap and water immediately after using an alcohol hand rub may lead to dermatitis. Therefore, personnel should be reminded that it is neither necessary nor recommended to routinely wash hands after each application of an alcohol hand rub.

Hand lotions and creams often contain humectants and various fats and oils that can increase skin hydration and replace altered or depleted skin lipids that contribute to the barrier function of normal skin (251,271). Several controlled trials have demonstrated that regular use (e.g., twice a day) of such products can help prevent and treat irritant contact dermatitis caused by hand-hygiene products (272,273). In one study, frequent and scheduled use of an oil-containing lotion improved skin condition, and thus led to a 50% increase in

handwashing frequency among HCWs (273). Reports from these studies emphasize the need to educate personnel regarding the value of regular, frequent use of hand-care products.

Recently, barrier creams have been marketed for the prevention of hand-hygiene-related irritant contact dermatitis. Such products are absorbed to the superficial layers of the epidermis and are designed to form a protective layer that is not removed by standard handwashing. Two recent randomized, controlled trials that evaluated the skin condition of caregivers demonstrated that barrier creams did not yield better results than did the control lotion or vehicle used (272,273). As a result, whether barrier creams are effective in preventing irritant contact dermatitis among HCWs remains unknown.

In addition to evaluating the efficacy and acceptability of hand-care products, product-selection committees should inquire about the potential deleterious effects that oil-containing products may have on the integrity of rubber gloves and on the efficacy of antiseptic agents used in the facility (8,236).

Factors To Consider When Selecting Hand-Hygiene Products

When evaluating hand-hygiene products for potential use in health-care facilities, administrators or product-selection committees must consider factors that can affect the overall efficacy of such products, including the relative efficacy of antiseptic agents against various pathogens (Appendix) and acceptance of hand-hygiene products by personnel (274,275). Soap products that are not well-accepted by HCWs can be a deterrent to frequent handwashing (276). Characteristics of a product (either soap or alcohol-based hand rub) that can affect acceptance by personnel include its smell, consistency (i.e., “feel”), and color (92,277,278). For soaps, ease of lathering also may affect user preference.

Because HCWs may wash their hands from a limited number of times per shift to as many as 30 times per shift, the tendency of products to cause skin irritation and dryness is a substantial factor that influences acceptance, and ultimate usage (61,98,274,275,277,279). For example, concern regarding the drying effects of alcohol was a primary cause of poor acceptance of alcohol-based hand-hygiene products in hospitals in the United States (5,143). However, several studies have demonstrated that alcohol-based hand rubs containing emollients are acceptable to HCWs (90,93,98,100,101,106,143,163,164,166). With alcohol-based products, the time required for drying may also affect user acceptance.

Studies indicate that the frequency of handwashing or antiseptic handwashing by personnel is affected by the accessibility of hand-hygiene facilities (280–283). In certain health-care

facilities, only one sink is available in rooms housing several patients, or sinks are located far away from the door of the room, which may discourage handwashing by personnel leaving the room. In intensive-care units, access to sinks may be blocked by bedside equipment (e.g., ventilators or intravenous infusion pumps). In contrast to sinks used for handwashing or antiseptic handwash, dispensers for alcohol-based hand rubs do not require plumbing and can be made available adjacent to each patient’s bed and at many other locations in patient-care areas. Pocket carriage of alcohol-based hand-rub solutions, combined with availability of bedside dispensers, has been associated with substantial improvement in adherence to hand-hygiene protocols (74,284). To avoid any confusion between soap and alcohol hand rubs, alcohol hand-rub dispensers should not be placed adjacent to sinks. HCWs should be informed that washing hands with soap and water after each use of an alcohol hand rub is not necessary and is not recommended, because it may lead to dermatitis. However, because personnel feel a “build-up” of emollients on their hands after repeated use of alcohol hand gels, washing hands with soap and water after 5–10 applications of a gel has been recommended by certain manufacturers.

Automated handwashing machines have not been demonstrated to improve the quality or frequency of handwashing (88,285). Although technologically advanced automated handwashing devices and monitoring systems have been developed recently, only a minimal number of studies have been published that demonstrate that use of such devices results in enduring improvements in hand-hygiene adherence among HCWs. Further evaluation of automated handwashing facilities and monitoring systems is warranted.

Dispenser systems provided by manufacturers or vendors also must be considered when evaluating hand-hygiene products. Dispensers may discourage use by HCWs when they 1) become blocked or partially blocked and do not deliver the product when accessed by personnel, and 2) do not deliver the product appropriately onto the hands. In one hospital where a viscous alcohol-based hand rinse was available, only 65% of functioning dispensers delivered product onto the caregivers’ hands with one press of the dispenser lever, and 9% of dispensers were totally occluded (286). In addition, the volume delivered was often suboptimal, and the product was sometimes squirted onto the wall instead of the caregiver’s hand.

Only limited information is available regarding the cost of hand-hygiene products used in health-care facilities (165,287). These costs were evaluated in patient-care areas at a 450-bed community teaching hospital (287); the hospital spent \$22,000 (\$0.72 per patient-day) on 2% chlorhexidine-containing preparations, plain soap, and an alcohol hand rinse. (287) When

hand-hygiene supplies for clinics and nonpatient care areas were included, the total annual budget for soaps and hand antiseptic agents was \$30,000 (approximately \$1 per patient-day). Annual hand-hygiene product budgets at other institutions vary considerably because of differences in usage patterns and varying product prices. One researcher (287) determined that if non-antimicrobial liquid soap were assigned an arbitrary relative cost of 1.0, the cost per liter would be 1.7 times as much for 2% chlorhexidine gluconate detergent, 1.6–2.0 times higher for alcohol-based hand-rub products, and 4.5 times higher for an alcohol-based foam product. A recent cost comparison of surgical scrubbing with an antimicrobial soap versus brushless scrubbing with an alcohol-based hand rub revealed that costs and time required for preoperative scrubbing were less with the alcohol-based product (165). In a trial conducted in two critical-care units, the cost of using an alcohol hand rub was half as much as using an antimicrobial soap for handwashing (\$0.025 versus \$0.05 per application, respectively) (166).

To put expenditures for hand-hygiene products into perspective, health-care facilities should consider comparing their budget for hand-hygiene products to estimated excess hospital costs resulting from health-care-associated infections. The excess hospital costs associated with only four or five health-care-associated infections of average severity may equal the entire annual budget for hand-hygiene products used in inpatient-care areas. Just one severe surgical site infection, lower respiratory tract infection, or bloodstream infection may cost the hospital more than the entire annual budget for antiseptic agents used for hand hygiene (287). Two studies provided certain quantitative estimates of the benefit of hand-hygiene-promotion programs (72,74). One study demonstrated a cost saving of approximately \$17,000 resulting from reduced use of vancomycin after the observed decrease in MRSA incidence in a 7-month period (72). In another study that examined both direct costs associated with the hand-hygiene promotion program (increased use of hand-rub solution and poster production) and indirect costs associated with health-care-personnel time (74), costs of the program were an estimated \$57,000 or less per year (an average of \$1.42 per patient admitted). Supplementary costs associated with the increased use of alcohol-based hand-rub solution averaged \$6.07 per 100 patient-days. Based on conservative estimates of \$2,100 saved per infection averted and on the assumption that only 25% of the observed reduction in the infection rate was associated with improved hand-hygiene practice, the program was substantially cost-effective. Thus, hospital administrators must consider that by purchasing more effective or more acceptable hand-hygiene products to improve hand-hygiene practices, they

will avoid the occurrence of nosocomial infections; preventing only a limited number of additional health-care-associated infections per year will lead to savings that will exceed any incremental costs of improved hand-hygiene products.

Hand-Hygiene Practices Among HCWs

In observational studies conducted in hospitals, HCWs washed their hands an average of five times per shift to as many as 30 times per shift (Table 6) (17,61,90,98,274,288); certain nurses washed their hands ≤ 100 times per shift (90). Hospitalwide surveillance of hand hygiene reveals that the average number of handwashing opportunities varies markedly between hospital wards. For example, nurses in pediatric wards had an average of eight opportunities for hand hygiene per hour of patient care compared with an average of 20 for nurses in intensive-care units (11). The duration of handwashing or hygienic handwash episodes by HCWs has averaged 6.6–24.0 seconds in observational studies (Table 7) (17,52,59,84–87,89,249,279). In addition to washing their

TABLE 6. Handwashing frequency among health-care workers

Ref. no.	Year	Avg. no./time period	Range	Avg. no./hr
(61)	1988	5/8 hour	N.S.	
(89)	1984	5–10/shift	N.S.	
(96)	2000	10/shift	N.S.	
(273)	2000	12–18/day	2–60	
(98)	2000	13–15/8 hours	5–27	1.6–1.8/hr
(90)	1977	20–42/8 hours	10–100	
(391)	2000	21/12 hours	N.S.	
(272)	2000	22/day	0–70	
(88)	1991			1.7–2.1/hr
(17)	1998			2.1/hr
(279)	1978			3/hr
(303)	1994			3.3/hr

Note: N.S. = Not Stated.

TABLE 7. Average duration of handwashing by health-care workers

Ref. no.	Year	Mean/median time
(392)	1997	4.7–5.3 seconds
(303)	1994	6.6 seconds
(52)	1974	8–9.3 seconds
(85)	1984	8.6 seconds
(86)	1994	<9 seconds
(87)	1994	9.5 seconds
(88)	1991	<10 seconds
(294)	1990	10 seconds
(89)	1984	11.6 seconds
(300)	1992	12.5 seconds
(59)	1988	15.6–24.4 seconds
(17)	1998	20.6 seconds
(279)	1978	21 seconds
(293)	1989	24 seconds

hands for limited time periods, personnel often fail to cover all surfaces of their hands and fingers (288).

Adherence of HCWs to Recommended Hand-Hygiene Practices

Observational Studies of Hand-Hygiene Adherence. Adherence of HCWs to recommended hand-hygiene procedures has been poor, with mean baseline rates of 5%–81% (overall average: 40%) (Table 8) (71,74,86,87,276,280,281,283,285,289–313). The methods used for defining adherence (or non-adherence) and those used for conducting observations vary considerably among studies, and reports do not provide

detailed information concerning the methods and criteria used. The majority of studies were conducted with hand-hygiene adherence as the major outcome measure, whereas a limited number measured adherence as part of a broader investigation. Several investigators reported improved adherence after implementing various interventions, but the majority of studies had short follow-up periods and did not confirm whether behavioral improvements were long-lasting. Other studies established that sustained improvements in handwashing behavior occurred during a long-term program to improve adherence to hand-hygiene policies (74,75).

TABLE 8. Hand-hygiene adherence by health-care workers (1981–2000)

Ref. no.	Year	Setting	Before/after	Adherence baseline	Adherence after intervention	Intervention
(280)	1981	ICU	A	16%	30%	More convenient sink locations
(289)	1981	ICU	A	41%	—	
		ICU	A	28%	—	
(290)	1983	All wards	A	45%	—	
(281)	1986	SICU	A	51%	—	
		MICU	A	76%	—	
(276)	1986	ICU	A	63%	92%	Performance feedback
(291)	1987	PICU	A	31%	30%	Wearing overgown
(292)	1989	MICU	B/A	14%/28%*	73%/81%	Feedback, policy reviews, memo, and posters
		MICU	B/A	26%/23%	38%/60%	
(293)	1989	NICU	A/B	75%/50%	—	
(294)	1990	ICU	A	32%	45%	Alcohol rub introduced
(295)	1990	ICU	A	81%	92%	Inservices first, then group feedback
(296)	1990	ICU	B/A	22%	30%	
(297)	1991	SICU	A	51%	—	
(298)	1991	Pedi OPDs	B	49%	49%	Signs, feedback, and verbal reminders to physicians
(299)	1991	Nursery and NICU	B/A†	28%	63%	Feedback, dissemination of literature, and results of environmental cultures
(300)	1992	NICU/others	A	29%	—	
(71)	1992	ICU	N.S.	40%	—	
(301)	1993	ICUs	A	40%	—	
(87)	1994	Emergency Room	A	32%	—	
(86)	1994	All wards	A	32%	—	
(285)	1994	SICU	A	22%	38%	Automated handwashing machines available
(302)	1994	NICU	A	62%	60%	No gowning required
(303)	1994	ICU Wards	AA	30%/29%	—	
(304)	1995	ICU Oncol Ward	A	56%	—	
(305)	1995	ICU	N.S.	5%	63%	Lectures, feedback, and demonstrations
(306)	1996	PICU	B/A	12%/11%	68%/65%	Overt observation, followed by feedback
(307)	1996	MICU	A	41%	58%	Routine wearing of gowns and gloves
(308)	1996	Emergency Dept	A	54%	64%	Signs/distributed review paper
(309)	1998	All wards	A	30%	—	
(310)	1998	Pediatric wards	B/A	52%/49%	74%/69%	Feedback, movies, posters, and brochures
(311)	1999	MICU	B/A	12%/55%	—	
(74)	2000	All wards	B/A	48%	67%	Posters, feedback, administrative support, and alcohol rub
(312)	2000	MICU	A	42%	61%	Alcohol hand rub made available
(283)	2000	MICU	B/A	10%/22%	23%/48%	Education, feedback, and alcohol gel made available
		CTICU	B/A	4%/13%	7%/14%	
(313)	2000	Medical wards	A	60%	52%	Education, reminders, and alcohol gel made available

Note: ICU = intensive care unit, SICU = surgical ICU, MICU = medical ICU, PICU = pediatric ICU, NICU = neonatal ICU, Emerg = emergency, Oncol = oncology, CTICU = cardiothoracic ICU, and N.S. = not stated.

* Percentage compliance before/after patient contact.

† After contact with inanimate objects.

Factors Affecting Adherence. Factors that may influence hand hygiene include those identified in epidemiologic studies and factors reported by HCWs as being reasons for lack of adherence to hand-hygiene recommendations. Risk factors for poor adherence to hand hygiene have been determined objectively in several observational studies or interventions to improve adherence (11,12,274,292,295,314–317). Among these, being a physician or a nursing assistant, rather than a nurse, was consistently associated with reduced adherence (Box 1).

In the largest hospitalwide survey of hand-hygiene practices among HCWs (11), predictors of poor adherence to recommended hand-hygiene measures were identified. Predictor variables included professional category, hospital ward, time of day/week, and type and intensity of patient care, defined as the number of opportunities for hand hygiene per hour of patient care. In 2,834 observed opportunities for hand hygiene, average adherence was 48%. In multivariate analysis, nonadherence was lowest among nurses and during weekends

BOX 1. Factors influencing adherence to hand-hygiene practices*

Observed risk factors for poor adherence to recommended hand-hygiene practices

- Physician status (rather than a nurse)
- Nursing assistant status (rather than a nurse)
- Male sex
- Working in an intensive-care unit
- Working during the week (versus the weekend)
- Wearing gowns/gloves
- Automated sink
- Activities with high risk of cross-transmission
- High number of opportunities for hand hygiene per hour of patient care

Self-reported factors for poor adherence with hand hygiene

- Handwashing agents cause irritation and dryness
- Sinks are inconveniently located/shortage of sinks
- Lack of soap and paper towels
- Often too busy/insufficient time
- Understaffing/overcrowding
- Patient needs take priority
- Hand hygiene interferes with health-care worker relationships with patients
- Low risk of acquiring infection from patients
- Wearing of gloves/beliefs that glove use obviates the need for hand hygiene
- Lack of knowledge of guidelines/protocols
- Not thinking about it/forgetfulness
- No role model from colleagues or superiors
- Skepticism regarding the value of hand hygiene
- Disagreement with the recommendations
- Lack of scientific information of definitive impact of improved hand hygiene on health-care-associated infection rates

Additional perceived barriers to appropriate hand hygiene

- Lack of active participation in hand-hygiene promotion at individual or institutional level
- Lack of role model for hand hygiene
- Lack of institutional priority for hand hygiene
- Lack of administrative sanction of noncompliers/rewarding compliers
- Lack of institutional safety climate

* Source: Adapted from Pittet D. Improving compliance with hand hygiene in hospitals. *Infect Control Hosp Epidemiol* 2000;21:381–6.

(Odds Ratio [OR]: 0.6; 95% confidence interval [CI] = 0.4–0.8). Nonadherence was higher in intensive-care units compared with internal medicine wards (OR: 2.0; 95% CI = 1.3–3.1), during procedures that carried a high risk of bacterial contamination (OR: 1.8; 95% CI = 1.4–2.4), and when intensity of patient care was high (21–40 handwashing opportunities — OR: 1.3; 95% CI = 1.0–1.7; 41–60 opportunities — OR: 2.1; 95% CI = 1.5–2.9; >60 opportunities — OR: 2.1; 95% CI = 1.3–3.5). The higher the demand for hand hygiene, the lower the adherence; on average, adherence decreased by 5% (\pm 2%) for each increase of 10 opportunities per hour when the intensity of patient care exceeded 10 opportunities per hour. Similarly, the lowest adherence rate (36%) was found in intensive-care units, where indications for hand hygiene were typically more frequent (on average, 20 opportunities per patient-hour). The highest adherence rate (59%) was observed in pediatrics wards, where the average intensity of patient care was lower than in other hospital areas (an average of eight opportunities per patient-hour). The results of this study indicate that full adherence to previous guidelines may be unrealistic, and that facilitated access to hand hygiene could help improve adherence (11,12,318).

Perceived barriers to adherence with hand-hygiene practice recommendations include skin irritation caused by hand-hygiene agents, inaccessible hand-hygiene supplies, interference with HCW-patient relationships, priority of care (i.e., the patients' needs are given priority over hand hygiene), wearing of gloves, forgetfulness, lack of knowledge of the guidelines, insufficient time for hand hygiene, high workload and understaffing, and the lack of scientific information indicating a definitive impact of improved hand hygiene on health-care-associated infection rates (11,274,292,295,315–317). Certain perceived barriers to adherence with hand-hygiene guidelines have been assessed or quantified in observational studies (12,274,292,295,314–317) (Box 1).

Skin irritation by hand-hygiene agents constitutes a substantial barrier to appropriate adherence (319). Because soaps and detergents can damage skin when applied on a regular basis, HCWs must be better informed regarding the possible adverse effects associated with hand-hygiene agents. Lack of knowledge and education regarding this subject is a barrier to motivation. In several studies, alcohol-based hand rubs containing emollients (either isopropanol, ethanol, or n-propanol in 60%–90% vol/vol) were less irritating to the skin than the soaps or detergents tested. In addition, the alcohol-based products containing emollients that were tested were at least as tolerable and efficacious as the detergents tested. Also, studies demonstrate that several hand lotions have reduced skin scaling and cracking, which may reduce microbial shedding from the hands (67,272,273).

Easy access to hand-hygiene supplies, whether sink, soap, medicated detergent, or alcohol-based hand-rub solution, is essential for optimal adherence to hand-hygiene recommendations. The time required for nurses to leave a patient's bedside, go to a sink, and wash and dry their hands before attending the next patient is a deterrent to frequent handwashing or hand antisepsis (11,318). Engineering controls could facilitate adherence, but careful monitoring of hand-hygiene behavior should be conducted to exclude the possible negative effect of newly introduced handwashing devices (88).

The impact of wearing gloves on adherence to hand-hygiene policies has not been definitively established, because published studies have yielded contradictory results (87,290,301,320). Hand hygiene is required regardless of whether gloves are used or changed. Failure to remove gloves after patient contact or between "dirty" and "clean" body-site care on the same patient must be regarded as nonadherence to hand-hygiene recommendations (11). In a study in which experimental conditions approximated those occurring in clinical practice (321), washing and reusing gloves between patient contacts resulted in observed bacterial counts of 0–4.7 log on the hands after glove removal. Therefore, this practice should be discouraged; handwashing or disinfection should be performed after glove removal.

Lack of 1) knowledge of guidelines for hand hygiene, 2) recognition of hand-hygiene opportunities during patient care, and 3) awareness of the risk of cross-transmission of pathogens are barriers to good hand-hygiene practices. Furthermore, certain HCWs believe they have washed their hands when necessary, even when observations indicate they have not (89,92,295,296,322).

Perceived barriers to hand-hygiene behavior are linked not only to the institution, but also to HCWs' colleagues. Therefore, both institutional and small-group dynamics need to be considered when implementing a system change to secure an improvement in HCWs' hand-hygiene practice.

Possible Targets for Hand-Hygiene Promotion

Targets for the promotion of hand hygiene are derived from studies assessing risk factors for nonadherence, reported reasons for the lack of adherence to recommendations, and additional factors perceived as being important to facilitate appropriate HCW behavior. Although certain factors cannot be modified (Box 1), others can be changed.

One factor that must be addressed is the time required for HCWs to clean their hands. The time required for traditional handwashing may render full adherence to previous guidelines unrealistic (11,12,318) and more rapid access to hand-hygiene materials could help improve adherence. One study conducted in an intensive-care unit demonstrated that it took

nurses an average of 62 seconds to leave a patient's bedside, walk to a sink, wash their hands, and return to patient care (318). In contrast, an estimated one fourth as much time is required when using alcohol-based hand rub placed at each patient's bedside. Providing easy access to hand-hygiene materials is mandatory for appropriate hand-hygiene behavior and is achievable in the majority of health-care facilities (323). In particular, in high-demand situations (e.g., the majority of critical-care units), under hectic working conditions, and at times of overcrowding or understaffing, HCWs may be more likely to use an alcohol-based hand rub than to wash their hands (323). Further, using alcohol-based hand rubs may be a better option than traditional handwashing with plain soap and water or antiseptic handwash, because they not only require less time (166,318) but act faster (1) and irritate hands less often (1,67,96,98,166). They also were used in the only program that reported a sustained improvement in hand-hygiene adherence associated with decreased infection rates (74). However, making an alcohol-based hand rub available to personnel without providing ongoing educational and motivational activities may not result in long-lasting improvement in hand-hygiene practices (313). Because increased use of hand-hygiene agents might be associated with skin dryness, the availability of free skin-care lotion is recommended.

Education is a cornerstone for improvement with hand-hygiene practices. Topics that must be addressed by educational programs include the lack of 1) scientific information for the definitive impact of improved hand hygiene on health-care-associated infection and resistant organism transmission rates; 2) awareness of guidelines for hand hygiene and insufficient knowledge concerning indications for hand hygiene during daily patient care; 3) knowledge concerning the low average adherence rate to hand hygiene by the majority of HCWs; and 4) knowledge concerning the appropriateness, efficacy, and understanding of the use of hand-hygiene and skin-care-protection agents.

HCWs necessarily evolve within a group that functions within an institution. Possible targets for improvement in hand-hygiene behavior not only include factors linked to individual HCWs, but also those related to the group(s) and the institution as a whole (317,323). Examples of possible targets for hand-hygiene promotion at the group level include education and performance feedback on hand-hygiene adherence; efforts to prevent high workload, downsizing, and understaffing; and encouragement and provision of role models from key members in the work unit. At the institutional level, targets for improvement include 1) written guidelines, hand-hygiene agents, skin-care promotions and agents, or hand-hygiene facilities; 2) culture or tradition of adherence; and 3)

administrative leadership, sanction, support, and rewards. Several studies, conducted in various types of institutions, reported modest and even low levels of adherence to recommended hand-hygiene practices, indicating that such adherence varied by hospital ward and by type of HCW. These results indicate educational sessions may need to be designed specifically for certain types of personnel (11,289,290,294,317,323).

Lessons Learned from Behavioral Theories

In 1998, the prevailing behavioral theories and their applications with regard to the health professions were reviewed by researchers in an attempt to better understand how to target more successful interventions (317). The researchers proposed a hypothetical framework to enhance hand-hygiene practices and stressed the importance of considering the complexity of individual and institutional factors when designing behavioral interventions.

Although behavioral theories and secondary interventions have primarily targeted individual workers, this practice might be insufficient to produce sustained change (317,324,325). Interventions aimed at improving hand-hygiene practices must account for different levels of behavior interaction (12,317,326). Thus, the interdependence of individual factors, environmental constraints, and the institutional climate must be taken into account in the strategic planning and development of hand-hygiene campaigns. Interventions to promote hand hygiene in hospitals should consider variables at all these levels. Various factors involved in hand-hygiene behavior include intention, attitude towards the behavior, perceived social norm, perceived behavioral control, perceived risk for infection, hand-hygiene practices, perceived role model, perceived knowledge, and motivation (317). The factors necessary for change include 1) dissatisfaction with the current situation, 2) perception of alternatives, and 3) recognition, both at the individual and institutional level, of the ability and potential to change. Although the latter implies education and motivation, the former two necessitate a system change.

Among the reported reasons for poor adherence with hand-hygiene recommendations (Box 1), certain ones are clearly associated with the institution or system (e.g., lack of institutional priority for hand hygiene, administrative sanctions, and a safety climate). Although all of these reasons would require a system change in the majority of institutions, the third requires management commitment, visible safety programs, an acceptable level of work stress, a tolerant and supportive attitude toward reported problems, and belief in the efficacy

of preventive strategies (12,317,325,327). Most importantly, an improvement in infection-control practices requires 1) questioning basic beliefs, 2) continuous assessment of the group (or individual) stage of behavioral change, 3) intervention(s) with an appropriate process of change, and 4) supporting individual and group creativity (317). Because of the complexity of the process of change, single interventions often fail. Thus, a multimodal, multidisciplinary strategy is likely necessary (74,75,317,323,326).

Methods Used To Promote Improved Hand Hygiene

Hand-hygiene promotion has been challenging for >150 years. In-service education, information leaflets, workshops and lectures, automated dispensers, and performance feedback on hand-hygiene adherence rates have been associated with transient improvement (291,294–296,306,314).

Several strategies for promotion of hand hygiene in hospitals have been published (Table 9). These strategies require education, motivation, or system change. Certain strategies are based on epidemiologic evidence, others on the authors' and other investigators' experience and review of current knowledge. Some strategies may be unnecessary in certain circumstances, but may be helpful in others. In particular, changing the hand-hygiene agent could be beneficial in institutions or hospital wards with a high workload and a high demand for hand hygiene when alcohol-based hand rubs are not available (11,73,78,328). However, a change in the recommended hand-hygiene agent could be deleterious if introduced during winter, at a time of higher hand-skin irritability, and if not accompanied by the provision of skin-care products (e.g., pro-

TECTIVE creams and lotions). Additional specific elements should be considered for inclusion in educational and motivational programs (Box 2).

Several strategies that could potentially be associated with successful promotion of hand hygiene require a system change (Box 1). Hand-hygiene adherence and promotion involve factors at both the individual and system level. Enhancing individual and institutional attitudes regarding the feasibility of making changes (self-efficacy), obtaining active participation of personnel at both levels, and promoting an institutional safety climate represent challenges that exceed the current perception of the role of infection-control professionals.

Whether increased education, individual reinforcement technique, appropriate rewarding, administrative sanction, enhanced self-participation, active involvement of a larger number of organizational leaders, enhanced perception of health threat, self-efficacy, and perceived social pressure (12,317,329,330), or combinations of these factors can improve HCWs' adherence with hand hygiene needs further investigation. Ultimately, adherence to recommended hand-hygiene practices should become part of a culture of patient safety where a set of interdependent quality elements interact to achieve a shared objective (331).

On the basis of both these hypothetical considerations and successful, actual experiences in certain institutions, strategies to improve adherence to hand-hygiene practices should be both multimodal and multidisciplinary. However, strategies must be further researched before they are implemented.

TABLE 9. Strategies for successful promotion of hand hygiene in hospitals

Strategy	Tool for change*	Selected references†
Education	E (M, S)	(74,295,306,326,393)
Routine observation and feedback	S (E, M)	(74,294,306,326,393)
Engineering control		
Make hand hygiene possible, easy, and convenient	S	(74,281,326,393)
Make alcohol-based hand rub available	S	(74)
(at least in high-demand situations)	S	(74,283,312)
Patient education	S (M)	(283,394)
Reminders in the workplace	S	(74,395)
Administrative sanction/rewarding	S	(12,317)
Change in hand-hygiene agent	S (E)	(11,67,71,283,312)
Promote/facilitate skin care for health-care-workers' hands	S (E)	(67,74,274,275)
Obtain active participation at individual and institutional level	E, M, S	(74,75,317)
Improve institutional safety climate	S (M)	(74,75,317)
Enhance individual and institutional self-efficacy	S (E, M)	(74,75,317)
Avoid overcrowding, understaffing, and excessive workload	S	(11,74,78,297,396)
Combine several of above strategies	E, M, S	(74,75,295,306,317,326)

* The dynamic of behavioral change is complex and involves a combination of education (E), motivation (M), and system change (S).

† Only selected references have been listed; readers should refer to more extensive reviews for exhaustive reference lists (1,8,317,323,397).

BOX 2. Elements of health-care worker educational and motivational programs**Rationale for hand hygiene**

- Potential risks of transmission of microorganisms to patients
- Potential risks of health-care worker colonization or infection caused by organisms acquired from the patient
- Morbidity, mortality, and costs associated with health-care–associated infections

Indications for hand hygiene

- Contact with a patient's intact skin (e.g., taking a pulse or blood pressure, performing physical examinations, lifting the patient in bed) (25,26,45,48,51,53)
- Contact with environmental surfaces in the immediate vicinity of patients (46,51,53,54)
- After glove removal (50,58,71)

Techniques for hand hygiene

- Amount of hand-hygiene solution
- Duration of hand-hygiene procedure
- Selection of hand-hygiene agents
 - Alcohol-based hand rubs are the most efficacious agents for reducing the number of bacteria on the hands of personnel. Antiseptic soaps and detergents are the next most effective, and non-antimicrobial soaps are the least effective (1,398).
 - Soap and water are recommended for visibly soiled hands.
 - Alcohol-based hand rubs are recommended for routine decontamination of hands for all clinical indications (except when hands are visibly soiled) and as one of the options for surgical hand hygiene.

Methods to maintain hand skin health

- Lotions and creams can prevent or minimize skin dryness and irritation caused by irritant contact dermatitis
- Acceptable lotions or creams to use
- Recommended schedule for applying lotions or creams

Expectations of patient care managers/administrators

- Written statements regarding the value of, and support for, adherence to recommended hand-hygiene practices
- Role models demonstrating adherence to recommended hand hygiene practices (399)

Indications for, and limitations of, glove use

- Hand contamination may occur as a result of small, undetected holes in examination gloves (321,361)
- Contamination may occur during glove removal (50)
- Wearing gloves does not replace the need for hand hygiene (58)
- Failure to remove gloves after caring for a patient may lead to transmission of microorganisms from one patient to another (373).

Efficacy of Promotion and Impact of Improved Hand Hygiene

The lack of scientific information of the definitive impact of improved hand hygiene on health-care–associated infection rates is a possible barrier to appropriate adherence with hand-hygiene recommendations (Box 1). However, evidence supports the belief that improved hand hygiene can reduce health-care–associated infection rates. Failure to perform appropriate hand hygiene is considered the leading cause of

health-care–associated infections and spread of multiresistant organisms and has been recognized as a substantial contributor to outbreaks.

Of nine hospital-based studies of the impact of hand hygiene on the risk of health-care–associated infections (Table 10) (48,69–75,296), the majority demonstrated a temporal relationship between improved hand-hygiene practices and reduced infection rates.

In one of these studies, endemic MRSA in a neonatal intensive-care unit was eliminated 7 months after introduction of a new

TABLE 10. Association between improved adherence with hand-hygiene practice and health-care–associated infection rates

Year	Ref. no.	Hospital setting	Results	Duration of follow-up
1977	(48)	Adult ICU	Reduction in health-care–associated infections caused by endemic <i>Klebsiella</i> spp.	2 years
1982	(69)	Adult ICU	Reduction in health-care-associated infection rates	N.S.
1984	(70)	Adult ICU	Reduction in health-care–associated infection rates	N.S.
1990	(296)	Adult ICU	No effect (average hand hygiene adherence improvement did not reach statistical significance)	11 months
1992	(71)	Adult ICU	Substantial difference between rates of health-care–associated infection between two different hand-hygiene agents	8 months
1994	(72)	NICU	Elimination of MRSA, when combined with multiple other infection-control measures. Reduction of vancomycin use	9 months
1995	(73)	Newborn nursery	Elimination of MRSA, when combined with multiple other infection-control measures	3.5 years
2000	(75)	MICU/NICU	85% relative reduction of VRE rate in the intervention hospital; 44% relative reduction in control hospital; no change in MRSA	8 months
2000	(74)	Hospitalwide	Substantial reduction in the annual overall prevalence of health-care–associated infections and MRSA cross-transmission rates. Active surveillance cultures and contact precautions were implemented during same period	5 years

Note: ICU = intensive care unit, NICU = neonatal ICU, MRSA = methicillin-resistant *Staphylococcus aureus*, MICU = medical ICU, and N.S. = not stated.

hand antiseptic (1% triclosan); all other infection-control measures remained in place, including the practice of conducting weekly active surveillance by obtaining cultures (72). Another study reported an MRSA outbreak involving 22 infants in a neonatal unit (73). Despite intensive efforts, the outbreak could not be controlled until a new antiseptic was added (i.e., 0.3% triclosan); all previously used control measures remained in place, including gloves and gowns, cohorting, and obtaining cultures for active surveillance.

The effectiveness of a longstanding, hospitalwide program to promote hand hygiene at the University of Geneva hospitals was recently reported (74). Overall adherence to hand-hygiene guidelines during routine patient care was monitored during hospitalwide observational surveys. These surveys were conducted biannually during December 1994–December 1997, before and during implementation of a hand-hygiene campaign that specifically emphasized the practice of bedside, alcohol-based hand disinfection. Individual-sized bottles of hand-rub solution were distributed to all wards, and custom-made holders were mounted on all beds to facilitate access to hand disinfection. HCWs were also encouraged to carry bottles in their pockets, and in 1996, a newly designed flat (instead of round) bottle was made available to further facilitate pocket carriage. The promotional strategy was multimodal and involved a multidisciplinary team of HCWs, the use of wall posters, the promotion of antiseptic hand rubs located at bed-sides throughout the institution, and regular performance feedback to all HCWs (see <http://www.hopisafe.ch> for further

details on methodology). Health-care–associated infection rates, attack rates of MRSA cross-transmission, and consumption of hand-rub disinfectant were measured. Adherence to recommended hand-hygiene practices improved progressively from 48% in 1994 to 66% in 1997 ($p < 0.001$). Whereas recourse to handwashing with soap and water remained stable, frequency of hand disinfection markedly increased during the study period ($p < 0.001$), and the consumption of alcohol-based hand-rub solution increased from 3.5 to 15.4 liters per 1,000 patient-days during 1993–1998 ($p < 0.001$). The increased frequency of hand disinfection was unchanged after adjustment for known risk factors of poor adherence. During the same period, both overall health-care–associated infection and MRSA transmission rates decreased (both $p < 0.05$). The observed reduction in MRSA transmission may have been affected by both improved hand-hygiene adherence and the simultaneous implementation of active surveillance cultures for detecting and isolating patients colonized with MRSA (332). The experience from the University of Geneva hospitals constitutes the first report of a hand-hygiene campaign with a sustained improvement over several years. An additional multimodal program also yielded sustained improvements in hand-hygiene practices over an extended period (75); the majority of studies have been limited to a 6- to 9-month observation period.

Although these studies were not designed to assess the independent contribution of hand hygiene on the prevention of health-care–associated infections, the results indicate that

improved hand-hygiene practices reduce the risk of transmission of pathogenic microorganisms. The beneficial effects of hand-hygiene promotion on the risk of cross-transmission also have been reported in surveys conducted in schools and day care centers (333–338), as well as in a community setting (339–341).

Other Policies Related to Hand Hygiene

Fingernails and Artificial Nails

Studies have documented that subungual areas of the hand harbor high concentrations of bacteria, most frequently coagulase-negative staphylococci, gram-negative rods (including *Pseudomonas* spp.), Corynebacteria, and yeasts (14,342,343). Freshly applied nail polish does not increase the number of bacteria recovered from periungual skin, but chipped nail polish may support the growth of larger numbers of organisms on fingernails (344,345). Even after careful handwashing or the use of surgical scrubs, personnel often harbor substantial numbers of potential pathogens in the subungual spaces (346–348).

Whether artificial nails contribute to transmission of health-care-associated infections is unknown. However, HCWs who wear artificial nails are more likely to harbor gram-negative pathogens on their fingertips than are those who have natural nails, both before and after handwashing (347–349). Whether the length of natural or artificial nails is a substantial risk factor is unknown, because the majority of bacterial growth occurs along the proximal 1 mm of the nail adjacent to subungual skin (345,347,348). Recently, an outbreak of *P. aeruginosa* in a neonatal intensive care unit was attributed to two nurses (one with long natural nails and one with long artificial nails) who carried the implicated strains of *Pseudomonas* spp. on their hands (350). Patients were substantially more likely than controls to have been cared for by the two nurses during the exposure period, indicating that colonization of long or artificial nails with *Pseudomonas* spp. may have contributed to causing the outbreak. Personnel wearing artificial nails also have been epidemiologically implicated in several other outbreaks of infection caused by gram-negative bacilli and yeast (351–353). Although these studies provide evidence that wearing artificial nails poses an infection hazard, additional studies are warranted.

Gloving Policies

CDC has recommended that HCWs wear gloves to 1) reduce the risk of personnel acquiring infections from patients, 2) prevent health-care worker flora from being transmitted to patients, and 3) reduce transient contamination of the hands

of personnel by flora that can be transmitted from one patient to another (354). Before the emergence of the acquired immunodeficiency syndrome (AIDS) epidemic, gloves were worn primarily by personnel caring for patients colonized or infected with certain pathogens or by personnel exposed to patients with a high risk of hepatitis B. Since 1987, a dramatic increase in glove use has occurred in an effort to prevent transmission of HIV and other bloodborne pathogens from patients to HCWs (355). The Occupational Safety and Health Administration (OSHA) mandates that gloves be worn during all patient-care activities that may involve exposure to blood or body fluids that may be contaminated with blood (356).

The effectiveness of gloves in preventing contamination of HCWs' hands has been confirmed in several clinical studies (45,51,58). One study found that HCWs who wore gloves during patient contact contaminated their hands with an average of only 3 CFUs per minute of patient care, compared with 16 CFUs per minute for those not wearing gloves (51). Two other studies, involving personnel caring for patients with *C. difficile* or VRE, revealed that wearing gloves prevented hand contamination among the majority of personnel having direct contact with patients (45,58). Wearing gloves also prevented personnel from acquiring VRE on their hands when touching contaminated environmental surfaces (58). Preventing heavy contamination of the hands is considered important, because handwashing or hand antisepsis may not remove all potential pathogens when hands are heavily contaminated (25,111).

Several studies provide evidence that wearing gloves can help reduce transmission of pathogens in health-care settings. In a prospective controlled trial that required personnel to routinely wear vinyl gloves when handling any body substances, the incidence of *C. difficile* diarrhea among patients decreased from 7.7 cases/1,000 patient discharges before the intervention to 1.5 cases/1,000 discharges during the intervention (226). The prevalence of asymptomatic *C. difficile* carriage also decreased substantially on "glove" wards, but not on control wards. In intensive-care units where VRE or MRSA have been epidemic, requiring all HCWs to wear gloves to care for all patients in the unit (i.e., universal glove use) likely has helped control outbreaks (357,358).

The influence of glove use on the hand-hygiene habits of personnel is not clear. Several studies found that personnel who wore gloves were less likely to wash their hands upon leaving a patient's room (290,320). In contrast, two other studies found that personnel who wore gloves were substantially more likely to wash their hands after patient care (87,301).

The following caveats regarding use of gloves by HCWs must be considered. Personnel should be informed that gloves

do not provide complete protection against hand contamination. Bacterial flora colonizing patients may be recovered from the hands of $\leq 30\%$ of HCWs who wear gloves during patient contact (50,58). Further, wearing gloves does not provide complete protection against acquisition of infections caused by hepatitis B virus and herpes simplex virus (359,360). In such instances, pathogens presumably gain access to the caregiver's hands via small defects in gloves or by contamination of the hands during glove removal (50,321,359,361).

Gloves used by HCWs are usually made of natural rubber latex and synthetic nonlatex materials (e.g., vinyl, nitrile, and neoprene [polymers and copolymers of chloroprene]). Because of the increasing prevalence of latex sensitivity among HCWs and patients, FDA has approved several powdered and powder-free latex gloves with reduced protein contents, as well as synthetic gloves that can be made available by health-care institutions for use by latex-sensitive employees. In published studies, the barrier integrity of gloves varies on the basis of type and quality of glove material, intensity of use, length of time used, manufacturer, whether gloves were tested before or after use, and method used to detect glove leaks (359,361–366). In published studies, vinyl gloves have had defects more frequently than latex gloves, the difference in defect frequency being greatest after use (359,361,364,367). However, intact vinyl gloves provide protection comparable to that of latex gloves (359). Limited studies indicate that nitrile gloves have leakage rates that approximate those of latex gloves (368–371). Having more than one type of glove available is desirable, because it allows personnel to select the type that best suits their patient-care activities. Although recent studies indicate that improvements have been made in the quality of gloves (366), hands should be decontaminated or washed after removing gloves (8,50,58,321,361). Gloves should not be washed or reused (321,361). Use of petroleum-based hand lotions or creams may adversely affect the integrity of latex gloves (372). After use of powdered gloves, certain alcohol hand rubs may interact with residual powder on the hands of personnel, resulting in a gritty feeling on the hands. In facilities where powdered gloves are commonly used, various alcohol-based hand rubs should be tested after removal of powdered gloves to avoid selecting a product that causes this undesirable reaction. Personnel should be reminded that failure to remove gloves between patients may contribute to transmission of organisms (358,373).

Jewelry

Several studies have demonstrated that skin underneath rings is more heavily colonized than comparable areas of skin on fingers without rings (374–376). One study found that 40% of nurses harbored gram-negative bacilli (e.g., *E. cloacae*, *Klebsiella*, and *Acinetobacter*) on skin under rings and that certain nurses carried the same organism under their rings for several months (375). In a more recent study involving >60 intensive care unit nurses, multivariable analysis revealed that rings were the only substantial risk factor for carriage of gram-negative bacilli and *S. aureus* and that the concentration of organisms recovered correlated with the number of rings worn (377). Whether the wearing of rings results in greater transmission of pathogens is unknown. Two studies determined that mean bacterial colony counts on hands after handwashing were similar among persons wearing rings and those not wearing rings (376,378). Further studies are needed to establish if wearing rings results in greater transmission of pathogens in health-care settings.

Hand-Hygiene Research Agenda

Although the number of published studies concerning hand hygiene has increased considerably in recent years, many questions regarding hand-hygiene products and strategies for improving adherence of personnel to recommended policies remain unanswered. Several concerns must still be addressed by researchers in industry and by clinical investigators (Box 3).

Web-Based Hand-Hygiene Resources

Additional information regarding improving hand hygiene is available at <http://www.hopisafe.ch>

University of Geneva Hospitals, Geneva, Switzerland

<http://www.cdc.gov/ncidod/hip>

CDC, Atlanta, Georgia

<http://www.jr2.ox.ac.uk/bandolier/band88/b88-8.html>

Bandolier journal, United Kingdom

<http://www.med.upenn.edu>

University of Pennsylvania, Philadelphia, Pennsylvania

BOX 3. Hand-hygiene research agenda**Education and promotion**

- Provide health-care workers (HCWs) with better education regarding the types of patient care activities that can result in hand contamination and cross-transmission of microorganisms.
- Develop and implement promotion hand-hygiene programs in pregraduate courses.
- Study the impact of population-based education on hand-hygiene behavior.
- Design and conduct studies to determine if frequent glove use should be encouraged or discouraged.
- Determine evidence-based indications for hand cleansing (considering that it might be unrealistic to expect HCWs to clean their hands after every contact with the patient).
- Assess the key determinants of hand-hygiene behavior and promotion among the different populations of HCWs.
- Develop methods to obtain management support.
- Implement and evaluate the impact of the different components of multimodal programs to promote hand hygiene.

Hand-hygiene agents and hand care

- Determine the most suitable formulations for hand-hygiene products.
- Determine if preparations with persistent antimicrobial activity reduce infection rates more effectively than do preparations whose activity is limited to an immediate effect.
- Study the systematic replacement of conventional handwashing by the use of hand disinfection.
- Develop devices to facilitate the use and optimal application of hand-hygiene agents.
- Develop hand-hygiene agents with low irritancy potential.
- Study the possible advantages and eventual interaction of hand-care lotions, creams, and other barriers to help minimize the potential irritation associated with hand-hygiene agents.

Laboratory-based and epidemiologic research and development

- Develop experimental models for the study of cross-contamination from patient to patient and from environment to patient.
- Develop new protocols for evaluating the in vivo efficacy of agents, considering in particular short application times and volumes that reflect actual use in health-care facilities.
- Monitor hand-hygiene adherence by using new devices or adequate surrogate markers, allowing frequent individual feedback on performance.
- Determine the percentage increase in hand-hygiene adherence required to achieve a predictable risk reduction in infection rates.
- Generate more definitive evidence for the impact on infection rates of improved adherence to recommended hand-hygiene practices.
- Provide cost-effectiveness evaluation of successful and unsuccessful promotion campaigns.

Part II. Recommendations**Categories**

These recommendations are designed to improve hand-hygiene practices of HCWs and to reduce transmission of pathogenic microorganisms to patients and personnel in health-care settings. This guideline and its recommendations are not intended for use in food processing or food-service establishments, and are not meant to replace guidance provided by FDA's Model Food Code.

As in previous CDC/HICPAC guidelines, each recommendation is categorized on the basis of existing scientific data, theoretical rationale, applicability, and economic impact. The CDC/HICPAC system for categorizing recommendations is as follows:

Category IA. Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiologic studies.

Category IB. Strongly recommended for implementation and supported by certain experimental, clinical, or epidemiologic studies and a strong theoretical rationale.

Category IC. Required for implementation, as mandated by federal or state regulation or standard.

Category II. Suggested for implementation and supported by suggestive clinical or epidemiologic studies or a theoretical rationale.

No recommendation. Unresolved issue. Practices for which insufficient evidence or no consensus regarding efficacy exist.

Recommendations

1. Indications for handwashing and hand antisepsis
 - A. When hands are visibly dirty or contaminated with proteinaceous material or are visibly soiled with blood or other body fluids, wash hands with either a non-antimicrobial soap and water or an antimicrobial soap and water (IA) (66).
 - B. If hands are not visibly soiled, use an alcohol-based hand rub for routinely decontaminating hands in all other clinical situations described in items 1C–J (IA) (74,93,166,169,283,294,312,398). Alternatively, wash hands with an antimicrobial soap and water in all clinical situations described in items 1C–J (IB) (69-71,74).
 - C. Decontaminate hands before having direct contact with patients (IB) (68,400).
 - D. Decontaminate hands before donning sterile gloves when inserting a central intravascular catheter (IB) (401,402).
 - E. Decontaminate hands before inserting indwelling urinary catheters, peripheral vascular catheters, or other invasive devices that do not require a surgical procedure (IB) (25,403).
 - F. Decontaminate hands after contact with a patient's intact skin (e.g., when taking a pulse or blood pressure, and lifting a patient) (IB) (25,45,48,68).
 - G. Decontaminate hands after contact with body fluids or excretions, mucous membranes, nonintact skin, and wound dressings if hands are not visibly soiled (IA) (400).
 - H. Decontaminate hands if moving from a contaminated-body site to a clean-body site during patient care (II) (25,53).
 - I. Decontaminate hands after contact with inanimate objects (including medical equipment) in the immediate vicinity of the patient (II) (46,53,54).
 - J. Decontaminate hands after removing gloves (IB) (50,58,321).
 - K. Before eating and after using a restroom, wash hands with a non-antimicrobial soap and water or with an antimicrobial soap and water (IB) (404-409).
 - L. Antimicrobial-impregnated wipes (i.e., towelettes) may be considered as an alternative to washing hands with non-antimicrobial soap and water. Because they are not as effective as alcohol-based hand rubs or washing hands with an antimicrobial soap and water for reducing bacterial counts on the hands of HCWs, they are not a substitute for using an alcohol-based hand rub or antimicrobial soap (IB) (160,161).
 - M. Wash hands with non-antimicrobial soap and water or with antimicrobial soap and water if exposure to *Bacillus anthracis* is suspected or proven. The physical action of washing and rinsing hands under such circumstances is recommended because alcohols, chlorhexidine, iodophors, and other antiseptic agents have poor activity against spores (II) (120,172,224,225).
 - N. No recommendation can be made regarding the routine use of nonalcohol-based hand rubs for hand hygiene in health-care settings. Unresolved issue.
2. Hand-hygiene technique
 - A. When decontaminating hands with an alcohol-based hand rub, apply product to palm of one hand and rub hands together, covering all surfaces of hands and fingers, until hands are dry (IB) (288,410). Follow the manufacturer's recommendations regarding the volume of product to use.
 - B. When washing hands with soap and water, wet hands first with water, apply an amount of product recommended by the manufacturer to hands, and rub hands together vigorously for at least 15 seconds, covering all surfaces of the hands and fingers. Rinse hands with water and dry thoroughly with a disposable towel. Use towel to turn off the faucet (IB) (90-92,94,411). Avoid using hot water, because repeated exposure to hot water may increase the risk of dermatitis (IB) (254,255).
 - C. Liquid, bar, leaflet or powdered forms of plain soap are acceptable when washing hands with a non-antimicrobial soap and water. When bar soap is used, soap racks that facilitate drainage and small bars of soap should be used (II) (412-415).
 - D. Multiple-use cloth towels of the hanging or roll type are not recommended for use in health-care settings (II) (137,300).
3. Surgical hand antisepsis
 - A. Remove rings, watches, and bracelets before beginning the surgical hand scrub (II) (375,378,416).
 - B. Remove debris from underneath fingernails using a nail cleaner under running water (II) (14,417).

- C. Surgical hand antisepsis using either an antimicrobial soap or an alcohol-based hand rub with persistent activity is recommended before donning sterile gloves when performing surgical procedures (IB) (115,159,232,234,237,418).
 - D. When performing surgical hand antisepsis using an antimicrobial soap, scrub hands and forearms for the length of time recommended by the manufacturer, usually 2–6 minutes. Long scrub times (e.g., 10 minutes) are not necessary (IB) (117,156,205,207,238-241).
 - E. When using an alcohol-based surgical hand-scrub product with persistent activity, follow the manufacturer's instructions. Before applying the alcohol solution, prewash hands and forearms with a non-antimicrobial soap and dry hands and forearms completely. After application of the alcohol-based product as recommended, allow hands and forearms to dry thoroughly before donning sterile gloves (IB) (159,237).
4. Selection of hand-hygiene agents
- A. Provide personnel with efficacious hand-hygiene products that have low irritancy potential, particularly when these products are used multiple times per shift (IB) (90,92,98,166,249). This recommendation applies to products used for hand antisepsis before and after patient care in clinical areas and to products used for surgical hand antisepsis by surgical personnel.
 - B. To maximize acceptance of hand-hygiene products by HCWs, solicit input from these employees regarding the feel, fragrance, and skin tolerance of any products under consideration. The cost of hand-hygiene products should not be the primary factor influencing product selection (IB) (92,93,166,274,276-278).
 - C. When selecting non-antimicrobial soaps, antimicrobial soaps, or alcohol-based hand rubs, solicit information from manufacturers regarding any known interactions between products used to clean hands, skin care products, and the types of gloves used in the institution (II) (174,372).
 - D. Before making purchasing decisions, evaluate the dispenser systems of various product manufacturers or distributors to ensure that dispensers function adequately and deliver an appropriate volume of product (II) (286).
 - E. Do not add soap to a partially empty soap dispenser. This practice of “topping off” dispensers can lead to bacterial contamination of soap (IA) (187,419).
5. Skin care
- A. Provide HCWs with hand lotions or creams to minimize the occurrence of irritant contact dermatitis associated with hand antisepsis or handwashing (IA) (272,273).
 - B. Solicit information from manufacturers regarding any effects that hand lotions, creams, or alcohol-based hand antiseptics may have on the persistent effects of antimicrobial soaps being used in the institution (IB) (174,420,421).
6. Other Aspects of Hand Hygiene
- A. Do not wear artificial fingernails or extenders when having direct contact with patients at high risk (e.g., those in intensive-care units or operating rooms) (IA) (350–353).
 - B. Keep natural nails tips less than 1/4-inch long (II) (350).
 - C. Wear gloves when contact with blood or other potentially infectious materials, mucous membranes, and nonintact skin could occur (IC) (356).
 - D. Remove gloves after caring for a patient. Do not wear the same pair of gloves for the care of more than one patient, and do not wash gloves between uses with different patients (IB) (50,58,321,373).
 - E. Change gloves during patient care if moving from a contaminated body site to a clean body site (II) (50,51,58).
 - F. No recommendation can be made regarding wearing rings in health-care settings. Unresolved issue.
7. Health-care worker educational and motivational programs
- A. As part of an overall program to improve hand-hygiene practices of HCWs, educate personnel regarding the types of patient-care activities that can result in hand contamination and the advantages and disadvantages of various methods used to clean their hands (II) (74,292,295,299).
 - B. Monitor HCWs' adherence with recommended hand-hygiene practices and provide personnel with information regarding their performance (IA) (74,276,292,295,299,306,310).
 - C. Encourage patients and their families to remind HCWs to decontaminate their hands (II) (394,422).
8. Administrative measures
- A. Make improved hand-hygiene adherence an institutional priority and provide appropriate

- administrative support and financial resources (IB) (74,75).
- B. Implement a multidisciplinary program designed to improve adherence of health personnel to recommended hand-hygiene practices (IB) (74,75).
 - C. As part of a multidisciplinary program to improve hand-hygiene adherence, provide HCWs with a readily accessible alcohol-based hand-rub product (IA) (74,166,283,294,312).
 - D. To improve hand-hygiene adherence among personnel who work in areas in which high workloads and high intensity of patient care are anticipated, make an alcohol-based hand rub available at the entrance to the patient's room or at the bedside, in other convenient locations, and in individual pocket-sized containers to be carried by HCWs (IA) (11,74,166,283,284,312,318,423).
 - E. Store supplies of alcohol-based hand rubs in cabinets or areas approved for flammable materials (IC).

Part III. Performance Indicators

1. The following performance indicators are recommended for measuring improvements in HCWs' hand-hygiene adherence:
 - A. Periodically monitor and record adherence as the number of hand-hygiene episodes performed by personnel/number of hand-hygiene opportunities, by ward or by service. Provide feedback to personnel regarding their performance.
 - B. Monitor the volume of alcohol-based hand rub (or detergent used for handwashing or hand antisepsis) used per 1,000 patient-days.
 - C. Monitor adherence to policies dealing with wearing of artificial nails.
 - D. When outbreaks of infection occur, assess the adequacy of health-care worker hand hygiene.

References

1. Rotter M. Hand washing and hand disinfection [Chapter 87]. In: Mayhall CG, ed. Hospital epidemiology and infection control. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999.
2. Labarraque AG. Instructions and observations regarding the use of the chlorides of soda and lime. Porter J, ed. [French] New Haven, CT: Baldwin and Treadway, 1829.
3. Semmelweis I. Etiology, concept, and prophylaxis of childbed fever. Carter KC, ed. 1st ed. Madison, WI: The University of Wisconsin Press, 1983.
4. Coppage CM. Hand washing in patient care [Motion picture]. Washington, DC: US Public Health Service, 1961.
5. Steere AC, Mallison GF. Handwashing practices for the prevention of nosocomial infections. *Ann Intern Med* 1975;83:683–90.
6. Garner JS, Favero MS. CDC guideline for handwashing and hospital environmental control, 1985. *Infect Control* 1986;7:231–43.
7. Larson E. Guideline for use of topical antimicrobial agents. *Am J Infect Control* 1988;16:253–66.
8. Larson EL, APIC Guidelines Committee. APIC guideline for handwashing and hand antisepsis in health care settings. *Am J Infect Control* 1995;23:251–69.
9. Hospital Infection Control Practices Advisory Committee (HICPAC). Recommendations for preventing the spread of vancomycin resistance. *Infect Control Hosp Epidemiol* 1995;16:105–13.
10. Garner JS, Hospital Infection Control Practices Advisory Committee. Guideline for isolation precautions in hospitals. *Infect Control Hosp Epidemiol* 1996;17:53–80.
11. Pittet D, Mourouga P, Perneger TV, Members of the Infection Control Program. Compliance with handwashing in a teaching hospital. *Ann Intern Med* 1999;130:126–30.
12. Boyce JM. It is time for action: improving hand hygiene in hospitals. *Ann Intern Med* 1999;130:153–5.
13. Selwyn S. Microbiology and ecology of human skin. *Practitioner* 1980;224:1059–62.
14. Price PB. Bacteriology of normal skin: a new quantitative test applied to a study of the bacterial flora and the disinfectant action of mechanical cleansing. *J Infect Dis* 1938;63:301–18.
15. Larson E. Effects of handwashing agent, handwashing frequency, and clinical area on hand flora. *Am J Infect Control* 1984;11:76–82.
16. Maki D. Control of colonization and transmission of pathogenic bacteria in the hospital. *Ann Intern Med* 1978;89(Pt 2):777–80.
17. Larson EL, Norton Hughes CA, Pyrak JD, Sparks SM, Cagatay EU, Bartkus JM. Changes in bacterial flora associated with skin damage on hands of health care personnel. *Am J Infect Control* 1998;26:513–21.
18. Sprunt K, Redman W, Leidy G. Antibacterial effectiveness of routine hand washing. *Pediatrics* 1973;52:264–71.
19. Food and Drug Administration. Tentative final monograph for healthcare antiseptic drug products; proposed rule. *Federal Register* 1994;59:31441–52.
20. Lowbury EJJ. Gram-negative bacilli on the skin. *Br J Dermatol* 1969;81(suppl 1):55–61.
21. Noble WC. Distribution of the Micrococccaceae. *Br J Dermatol* 1969;81(suppl 1):27–31.
22. McBride ME, Duncan WC, Bodey GP, McBride CM. Microbial skin flora of selected cancer patients and hospital personnel. *J Clin Microbiol* 1976;3:14–20.
23. Casewell MW. Role of hands in nosocomial gram-negative infection. In: Maibach HI, Aly R, eds. Skin microbiology: relevance to clinical infection. New York, NY: Springer-Verlag, 1981.
24. Larson EL, McGinley KJ, Foglia AR, Talbot GH, Leyden JJ. Composition and antimicrobial resistance of skin flora in hospitalized and healthy adults. *J Clin Microbiol* 1986;23:604–8.
25. Ehrenkranz NJ, Alfonso BC. Failure of bland soap handwash to prevent hand transfer of patient bacteria to urethral catheters. *Infect Control Hosp Epidemiol* 1991;12:654–62.
26. Sanderson PJ, Weissler S. Recovery of coliforms from the hands of nurses and patients: activities leading to contamination. *J Hosp Infect* 1992;21:85–93.
27. Coello R, Jiménez J, García M, et al. Prospective study of infection, colonization and carriage of methicillin-resistant *Staphylococcus aureus* in an outbreak affecting 990 patients. *Eur J Clin Microbiol Infect Dis* 1994;13:74–81.

28. Sanford MD, Widmer AF, Bale MJ, Jones RN, Wenzel RP. Efficient detection and long-term persistence of the carriage of methicillin-resistant *Staphylococcus aureus*. Clin Infect Dis 1994;19:1123–8.
29. Bertone SA, Fisher MC, Mortensen JE. Quantitative skin cultures at potential catheter sites in neonates. Infect Control Hosp Epidemiol 1994;15:315–8.
30. Bonten MJM, Hayden MK, Nathan C, VanVoorhis J, et al. Epidemiology of colonisation of patients and environment with vancomycin-resistant enterococci. Lancet 1996;348:1615–9.
31. Larson EL, Cronquist AB, Whittier S, Lai L, Lyle CT, Della Latta P. Differences in skin flora between inpatients and chronically ill patients. Heart Lung 2000;29:298–305.
32. Polakoff S, Richards IDG, Parker MT, Lidwell OM. Nasal and skin carriage of *Staphylococcus aureus* by patients undergoing surgical operation. J Hyg (Lond) 1967;65:559–66.
33. Leyden JJ, McGinley KJ, Nordstrom KM, Webster GF. Skin microflora. J Invest Dermatol 1987;88:65s–72s.
34. Tuazon CU, Perez A, Kishaba T, Sheagren JN. *Staphylococcus aureus* among insulin-injecting diabetic patients. JAMA 1975;231:1272.
35. Kaplowitz LG, Comstock JA, Landwehr DM, Dalton HP, Mayhall CG. Prospective study of microbial colonization of the nose and skin and infection of the vascular access site in hemodialysis patients. J Clin Microbiol 1988;26:1257–62.
36. Aly R, Maibach HI, Shinefield HR. Microbial flora of atopic dermatitis. Arch Dermatol 1977;113:780–2.
37. Kirmani N, Tuazon CU, Murray HW, Parrish AE, Sheagren JN. *Staphylococcus aureus* carriage rate of patients receiving long-term hemodialysis. Arch Intern Med 1978;138:1657–9.
38. Goldblum SE, Ulrich JA, Goldman RS, Reed WP. Nasal and cutaneous flora among hemodialysis patients and personnel: quantitative and qualitative characterization and patterns of staphylococcal carriage. Am J Kidney Dis 1982;11:281–6.
39. Boelaert JR, Van Landuyt HW, Gordts BZ, De Baere YA, Messer SA, Herwaldt LA. Nasal and cutaneous carriage of *Staphylococcus aureus* in hemodialysis patients: the effect of nasal mupirocin. Infect Control Hosp Epidemiol 1996;17:809–11.
40. Zimakoff J, Pedersen FB, Bergen L, et al. *Staphylococcus aureus* carriage and infections among patients in four haemo- and peritoneal-dialysis centres in Denmark. J Hosp Infect 1996;33:289–300.
41. Bibel DJ, Greenbert JH, Cook JL. *Staphylococcus aureus* and the microbial ecology of atopic dermatitis. Can J Microbiol 1997;23:1062–8.
42. Noble WC. Dispersal of skin microorganisms. Br J Dermatol 1975;93:477–85.
43. Walter CW, Kundsins RB, Shilkret MA, Day MM. Spread of staphylococci to the environment. Antibiotics Annual 1959:952–7.
44. Boyce JM, Opal SM, Chow JW, et al. Outbreak of multidrug-resistant *Enterococcus faecium* with transferable *vanB* class vancomycin resistance. J Clin Microbiol 1994;32:1148–53.
45. McFarland LV, Mulligan ME, Kwok RYY, Stamm WE. Nosocomial acquisition of *Clostridium difficile* infection. N Engl J Med 1989;320:204–10.
46. Samore MH, Venkataraman L, DeGirolami PC, Levin E, Arbeit RD, Karchmer AW. Clinical and molecular epidemiology of sporadic and clustered cases of nosocomial *Clostridium difficile* diarrhea. Am J Med 1996;100:32–40.
47. Lidwell OM, Towers AG, Ballard J, Gladstone B. Transfer of microorganisms between nurses and patients in a clean air environment. J Appl Bacteriol 1974;37:649–56.
48. Casewell M, Phillips I. Hands as route of transmission for *Klebsiella* species. Br Med J 1977;2:1315–7.
49. Hall CB, Douglas RG. Modes of transmission of respiratory syncytial virus. J Pediatr 1981;99:100–2.
50. Olsen RJ, Lynch P, Coyle MB, Cummings J, Bokete T, Stamm WE. Examination gloves as barriers to hand contamination in clinical practice. JAMA 1993;270:350–3.
51. Pittet D, Dharan S, Touveneau S, Sauvan V, Perneger TV. Bacterial contamination of the hands of hospital staff during routine patient care. Arch Intern Med 1999;159:821–6.
52. Fox MK, Langner SB, Wells RW. How good are hand washing practices? Am J Nursing 1974;74:1676–8.
53. Ojajärvi J. Effectiveness of hand washing and disinfection methods in removing transient bacteria after patient nursing. J Hyg (Lond) 1980;85:193–203.
54. Boyce JM, Potter-Bynoe G, Chenevert C, King T. Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. Infect Control Hosp Epidemiol 1997;18:622–7.
55. Hayden MK, Blom, DW, Lyle, EA, et al. The risk of hand and glove contamination by healthcare workers (HCWs) after contact with a VRE (+) patient (pt) or the pts environment (env) [Abstract K-1334]. Presented at the 41st Interscience Conference on Antimicrobial Agents and Chemotherapy Chicago: American Society for Microbiology, 2001.
56. Scott E, Bloomfield SF. The survival and transfer of microbial contamination via cloths, hands and utensils. J Appl Bacteriol 1990;68:271–8.
57. Bauer TM, Ofner E, Just HM, Just H, Daschner FD. An epidemiological study assessing the relative importance of airborne and direct contact transmission of microorganisms in a medical intensive care unit. J Hosp Infect 1990;15:301–9.
58. Tenorio AR, Badri SM, Sahgal NB, et al. Effectiveness of gloves in the prevention of hand carriage of vancomycin-resistant *Enterococcus* species by health care workers after patient care. Clin Infect Dis 2001;32:826–9.
59. Daschner FD. How cost-effective is the present use of antiseptics? J Hosp Infect 1988;11 (suppl A):227–35.
60. Knittle MA, Eitzman DV, Baer H. Role of hand contamination of personnel in the epidemiology of gram-negative nosocomial infections. J Pediatr 1975;86:433–7.
61. Ayliffe GAJ, Babb JR, Davies JG, Lilly HA. Hand disinfection: a comparison of various agents in laboratory and ward studies. J Hosp Infect 1988;11:226–43.
62. Strausbaugh LJ, Sewell DL, Ward TT, Pfaller MA, Heitzman T, Tjoelker R. High frequency of yeast carriage on hands of hospital personnel. J Clin Microbiol 1994;32:2299–300.
63. Marples RR, Towers AG. A laboratory model for the investigation of contact transfer of micro-organisms. J Hyg (Lond) 1979;82:237–48.
64. Mackintosh CA, Hoffman PN. An extended model for transfer of micro-organisms via the hands: differences between organisms and the effect of alcohol disinfection. J Hyg (Lond) 1984;92:345–55.
65. Patrick DR, Findon G, Miller TE. Residual moisture determines the level of touch-contact-associated bacterial transfer following hand washing. Epidemiol Infect 1997;119:319–25.
66. Larson E. A causal link between handwashing and risk of infection? Examination of the evidence. Infect Control Hosp Epidemiol 1988;9:28–36.
67. Larson E. Skin hygiene and infection prevention: more of the same or different approaches? Clin Infect Dis 1999;29:1287–94.

68. Mortimer EA Jr, Lipsitz PJ, Wolinsky E, Gonzaga AJ, Rammelkamp CH Jr. Transmission of staphylococci between newborns. *Am J Dis Child* 1962;104:289–95.
69. Maki DG. The use of antiseptics for handwashing by medical personnel. *J Chemother* 1989;1(suppl 1):3–11.
70. Massanari RM, Hierholzer WJ Jr. A crossover comparison of antiseptic soaps on nosocomial infection rates in intensive care units. *Am J Infect Control* 1984;12:247–8.
71. Doebbeling BN, Stanley GL, Sheetz CT, et al. Comparative efficacy of alternative hand-washing agents in reducing nosocomial infections in intensive care units. *N Engl J Med* 1992;327:88–93.
72. Webster J, Faoagali JL, Cartwright D. Elimination of methicillin-resistant *Staphylococcus aureus* from a neonatal intensive care unit after hand washing with triclosan. *J Paediatr Child Health* 1994;30:59–64.
73. Zafar AB, Butler RC, Reese DJ, Gaydos LA, Mennonona PA. Use of 0.3% triclosan (Bacti-Stat*) to eradicate an outbreak of methicillin-resistant *Staphylococcus aureus* in a neonatal nursery. *Am J Infect Control* 1995;23:200–8.
74. Pittet D, Hugonnet S, Harbarth S, Mourouga P, Sauvan V, Touveneau S. Effectiveness of a hospital-wide programme to improve compliance with hand hygiene. *Lancet* 2000;356:1307–12.
75. Larson EL, Early E, Cloonan P, Sugrue S, Parides M. An organizational climate intervention associated with increased handwashing and decreased nosocomial infections. *Behav Med* 2000;26:14–22.
76. Fridkin SK, Pear SM, Williamson TH, Galgiani JN, Jarvis WR. The role of understaffing in central venous catheter-associated bloodstream infections. *Infect Control Hosp Epidemiol* 1996;17:150–8.
77. Vicca AF. Nursing staff workload as a determinant of methicillin-resistant *Staphylococcus aureus* spread in an adult intensive therapy unit. *J Hosp Infect* 1999;43:109–13.
78. Harbarth S, Sudre P, Dharan S, Cadenas M, Pittet D. Outbreak of *Enterobacter cloacae* related to understaffing, overcrowding, and poor hygiene practices. *Infect Control Hosp Epidemiol* 1999;20:598–603.
79. European Committee for Standardization. Chemical disinfectants and antiseptics—hygienic handrub—test method and requirements (phase 2/step 2) [European standard EN 1500]. Brussels, Belgium: Central Secretariat; 1997.
80. Kramer A, Rudolph P, Kampf G, Pittet D. Limited efficacy of alcohol-based hand gels. *Lancet* 2002;359:1489–90.
81. Sattar SA, Abebe M, Bueti AJ, Jampani H, Newman J, Hua S. Activity of an alcohol-based hand gel against human adeno-, rhino-, and rotaviruses using the fingerpad method. *Infect Control Hosp Epidemiol* 2000;21:516–9.
82. Wolff MH, Schmitt J, Rahaus M, König A. Hepatitis A virus: a test method for virucidal activity. *J Hosp Infect* 2001;48(suppl A):S18–S22.
83. Steinmann J. Some principles of virucidal testing. *J Hosp Infect* 2001;48(suppl A):S15–S17.
84. Gould D, Ream E. Assessing nurses' hand decontamination performance. *Nursing Times* 1993;89:47–50.
85. Quraishi ZA, McGuckin M, Blais FX. Duration of handwashing in intensive care units: a descriptive study. *Am J Infect Control* 1984;11:83–7.
86. Lund S, Jackson J, Leggett J, Hales L, Dworkin R, Gilbert D. Reality of glove use and handwashing in a community hospital. *Am J Infect Control* 1994;22:352–7.
87. Meengs MR, Giles BK, Chisholm CD, Cordell WH, Nelson DR. Hand washing frequency in an emergency department. *Ann Emerg Med* 1994;23:1307–12.
88. Larson E, McGeer A, Quraishi ZA, et al. Effect of an automated sink on handwashing practices and attitudes in high-risk units. *Infect Control Hosp Epidemiol* 1991;12:422–8.
89. Broughall JM, Marshman C, Jackson B, Bird P. An automatic monitoring system for measuring handwashing frequency. *J Hosp Infect* 1984;5:447–53.
90. Ojajärvi J, Mäkelä P, Rantasalo I. Failure of hand disinfection with frequent hand washing: a need for prolonged field studies. *J Hyg (Lond)* 1977;79:107–19.
91. Larson EL, Eke PI, Wilder MP, Laughon BE. Quantity of soap as a variable in handwashing. *Infect Control* 1987;8:371–5.
92. Larson E, Leyden JJ, McGinley KJ, Grove GL, Talbot GH. Physiologic and microbiologic changes in skin related to frequent handwashing. *Infect Control* 1986;7:59–63.
93. Larson EL, Eke PI, Laughon BE. Efficacy of alcohol-based hand rinses under frequent-use conditions. *Antimicrob Agents Chemother* 1986;30:542–4.
94. Larson EL, Laughon BE. Comparison of four antiseptic products containing chlorhexidine gluconate. *Antimicrob Agents Chemother* 1987;31:1572–4.
95. Meers PD, Yeo GA. Shedding of bacteria and skin squames after handwashing. *J Hyg (Lond)* 1978;81:99–105.
96. Winnefeld M, Richard MA, Drancourt M, Grobb JJ. Skin tolerance and effectiveness of two hand decontamination procedures in everyday hospital use. *Br J Dermatol* 2000;143:546–50.
97. Maki DG, Zilz MA, Alvarado CJ. Evaluation of the antibacterial efficacy of four agents for handwashing. In: Nelson JC, Grassi C, eds. Current chemotherapy and infectious disease proceedings of the 11th International Congress on Chemotherapy and the 19th ICACC. Washington, DC: American Society for Microbiology, 1979.
98. Boyce JM, Kelliher S, Vallande N. Skin irritation and dryness associated with two hand-hygiene regimens: soap-and-water handwashing versus hand antiseptics with an alcoholic hand gel. *Infect Control Hosp Epidemiol* 2000;21:442–8.
99. Sartor C, Jacomo V, Duvivier C, Tissot-Dupont H, Sambuc R, Drancourt M. Nosocomial *Serratia marcescens* infections associated with extrinsic contamination of a liquid nonmedicated soap. *Infect Control Hosp Epidemiol* 2000;21:196–9.
100. Walter CW. Editorial: disinfection of hands. *Am J Surg* 1965;109:691–3.
101. Gravens DL, Butcher HR Jr, Ballinger WF, Dewar NE. Septisol antiseptic foam for hands of operating room personnel: an effective antibacterial agent. *Surgery* 1973;73:360–7.
102. Eitzen HE, Ritter MA, French MLV, Gioe TJ. A microbiological in-use comparison of surgical hand-washing agents. *J Bone Joint Surg Am* 1979;61–A:403–6.
103. Minakuchi K, Yamamoto Y, Matsunaga K, et al. The antiseptic effect of a quick drying rubbing type povidone-iodine alcoholic disinfectant solution. *Postgrad Med J* 1993;69(suppl 3):S23–S26.
104. Babb JR, Davies JG, Ayliffe GAJ. A test procedure for evaluating surgical hand disinfection. *J Hosp Infect* 1991;18(suppl B):41–9.
105. Bellamy K, Alcock R, Babb JR, Davies JG, Ayliffe GA. A test for the assessment of 'hygienic' hand disinfection using rotavirus. *J Hosp Infect* 1993;24:201–10.
106. Ayliffe GAJ, Babb JR, Quraishi AH. A test for 'hygienic' hand disinfection. *J Clin Pathol* 1978;31:923–8.
107. Lilly HA, Lowbury EJJ, Wilkins MD. Detergents compared with each other and with antiseptics as skin 'degerming' agents. *J Hyg (Lond)* 1979;82:89–93.

108. Ulrich JA. Clinical study comparing hibistat (0.5% chlorhexidine gluconate in 70% isopropyl alcohol) and betadine surgical scrub (7.5% povidone-iodine) for efficacy against experimental contamination of human skin. *Curr Ther Res* 1982;31:27–30.
109. Bartzokas CA, Gibson MF, Graham R, Pinder DC. A comparison of triclosan and chlorhexidine preparations with 60 per cent isopropyl alcohol for hygienic hand disinfection. *J Hosp Infect* 1983;4:245–55.
110. Rotter ML, Koller W, Wewalka G, Werner HP, Ayliffe GAJ, Babb JR. Evaluation of procedures for hygienic hand-disinfection: controlled parallel experiments on the Vienna test model. *J Hyg (Lond)* 1986;96:27–37.
111. Kjrlen H, Andersen BM. Handwashing and disinfection of heavily contaminated hands—effective or ineffective? *J Hosp Infect* 1992;21:61–71.
112. Namura S, Nishijima S, Asada Y. An evaluation of the residual activity of antiseptic handrub lotions: an ‘in use’ setting study. *J Dermatol* 1994;21:481–5.
113. Jarvis JD, Wynne CD, Enwright L, Williams JD. Handwashing and antiseptic-containing soaps in hospital. *J Clin Path* 1979;32:732–7.
114. Pereira LJ, Lee GM, Wade KJ. An evaluation of five protocols for surgical handwashing in relation to skin condition and microbial counts. *J Hosp Infect* 1997;36:49–65.
115. Larson EL, Butz AM, Gullette DL, Laughon BA. Alcohol for surgical scrubbing? *Infect Control Hosp Epidemiol* 1990;11:139–43.
116. Aly R, Maibach HI. Comparative study on the antimicrobial effect of 0.5% chlorhexidine gluconate and 70% isopropyl alcohol on the normal flora of hands. *Appl Environ Microbiol* 1979;37:610–3.
117. Galle PC, Homesley HD, Rhyne AL. Reassessment of the surgical scrub. *Surg Gynecol Obstet* 1978;147:215–8.
118. Rosenberg A, Alatary SD, Peterson AF. Safety and efficacy of the antiseptic chlorhexidine gluconate. *Surg Gynecol Obstet* 1976;143:789–92.
119. Ayliffe GAJ, Babb JR, Bridges K, et al. Comparison of two methods for assessing the removal of total organisms and pathogens from the skin. *J Hyg (Lond)* 1975;75:259–74.
120. Larson EL, Morton HE. Alcohols [Chapter 11]. In: Block SS, ed. *Disinfection, sterilization and preservation*. 4th ed. Philadelphia, PA: Lea and Febiger, 1991:642–54.
121. Price PB. Ethyl alcohol as a germicide. *Arch Surg* 1939;38:528–42.
122. Harrington C, Walker H. The germicidal action of alcohol. *Boston Medical and Surgical Journal* 1903;148:548–52.
123. Price PB. New studies in surgical bacteriology and surgical technic. *JAMA* 1938;111:1993–6.
124. Coulthard CE, Sykes G. The germicidal effect of alcohol with special reference to its action on bacterial spores. *Pharmaceutical Journal* 1936;137:79–81.
125. Pohle WD, Stuart LS. The germicidal action of cleaning agents—a study of a modification of Price’s procedure. *J Infect Dis* 1940;67:275–81.
126. Gardner AD. Rapid disinfection of clean unwashed skin: further experiments. *Lancet* 1948;760–3.
127. Sakuragi T, Yanagisawa K, Dan K. Bactericidal activity of skin disinfectants on methicillin-resistant *Staphylococcus aureus*. *Anesth Analg* 1995;81:555–8.
128. Kampf G, Jarosch R, Rüden H. Limited effectiveness of chlorhexidine based hand disinfectants against methicillin-resistant *Staphylococcus aureus* (MRSA). *J Hosp Infect* 1998;38:297–303.
129. Kampf G, Höfer M, Wendt C. Efficacy of hand disinfectants against vancomycin-resistant enterococci in vitro. *J Hosp Infect* 1999;42:143–50.
130. Platt J, Bucknall RA. The disinfection of respiratory syncytial virus by isopropanol and a chlorhexidine-detergent handwash. *J Hosp Infect* 1985;6:89–94.
131. Krilov LR, Harkness SH. Inactivation of respiratory syncytial virus by detergents and disinfectants. *Pediatr Infect Dis* 1993;12:582–4.
132. Sattar SA, Tetro J, Springthorpe VS, Giulivi A. Preventing the spread of hepatitis B and C viruses: where are germicides relevant? *Am J Infect Control* 2001;29:187–97.
133. Woolwine JD, Gerberding JL. Effect of testing method on apparent activities of antiviral disinfectants and antiseptics. *Antimicrob Agents Chemother* 1995;39:921–3.
134. Pillsbury DM, Livingood CS, Nichols AC. Bacterial flora of the normal skin: report on the effect of various ointment and solutions, with comments on the clinical significance of this study. *Arch Dermatol* 1942;45:61–80.
135. Lowbury EJJ, Lilly HA, Ayliffe GAJ. Preoperative disinfection of surgeons’ hands: use of alcoholic solutions and effects of gloves on skin flora. *Br Med J* 1974;4:369–72.
136. Lilly HA, Lowbury EJJ, Wilkins MD, Zaggy A. Delayed antimicrobial effects of skin disinfection by alcohol. *J Hyg (Lond)* 1979;82:497–500.
137. Ansari SA, Springthorpe VS, Sattar SA, Tostowaryk W, Wells GA. Comparison of cloth, paper, and warm air drying in eliminating viruses and bacteria from washed hands. *Am J Infect Control* 1991;19:243–9.
138. Ansari SA, Sattar SA, Springthorpe VS, Wells GA, Tostowaryk W. In vivo protocol for testing efficacy of hand-washing agents against viruses and bacteria: experiments with rotavirus and *Escherichia coli*. *Appl Environ Microbiol* 1989;55:3113–8.
139. Steinmann J, Nehrkorn R, Meyer A, Becker K. Two in-vivo protocols for testing virucidal efficacy of handwashing and hand disinfection. *Zentralbl Hyg Umweltmed.* 1995;196:425–36.
140. Mbithi JN, Springthorpe VS, Sattar SA. Comparative in vivo efficiencies of hand-washing agents against hepatitis A virus (HM-175) and poliovirus type 1 (Sabin). *Appl Environ Microbiol* 1993;59:3463–9.
141. Schurmann W, Eggers HJ. Antiviral activity of an alcoholic hand disinfectant: comparison of the in vitro suspension test with in vivo experiments on hands, and on individual fingertips. *Antiviral Res* 1983;3:25–41.
142. Larson E, Bobo L. Effective hand degerming in the presence of blood. *J Emerg Med* 1992;10:7–11.
143. Dineen P, Hildick-Smith G. Antiseptic care of the hands [Chapter 21]. In: Maibach HI, Hildick-Smith G, eds. *Skin bacteria and their role in infection*. New York: McGraw-Hill, 1965.
144. Lilly HA, Lowbury EJJ. Transient skin flora: their removal by cleansing or disinfection in relation to their mode of deposition. *J Clin Path* 1978;31:919–22.
145. Rotter M, Koller W, Wewalka G. Povidone-iodine and chlorhexidine gluconate-containing detergents for disinfection of hands. *J Hosp Infect* 1980;1:149–58.
146. Rotter ML. Hygienic hand disinfection. *Infect Control* 1984;1:18–22.
147. Blech MF, Hartemann P, Paquin JL. Activity of non antiseptic soaps and ethanol for hand disinfection. *Zentralbl Bakteriolog Hyg [B]* 1985;181:496–512.

148. Leyden JJ, McGinley KJ, Kaminer MS, et al. Computerized image analysis of full-hand touch plates: a method for quantification of surface bacteria on hands and the effect of antimicrobial agents. *J Hosp Infect* 1991;18(suppl B):13–22.
149. Rotter ML, Koller W. Test models for hygienic handrub and hygienic handwash: the effects of two different contamination and sampling techniques. *J Hosp Infect* 1992;20:163–71.
150. Zaragoza M, Sallés M, Gomez J, Bayas JM, Trilla A. Handwashing with soap or alcoholic solutions? A randomized clinical trial of its effectiveness. *Am J Infect Control* 1999;27:258–61.
151. Paulson DS, Fendler EJ, Dolan MJ, Williams RA. A close look at alcohol gel as an antimicrobial sanitizing agent. *Am J Infect Control* 1999;27:332–8.
152. Cardoso CL, Pereira HH, Zequim JC, Guilhermetti M. Effectiveness of hand-cleansing agents for removing *Acinetobacter baumannii* strain from contaminated hands. *Am J Infect Control* 1999;27:327–31.
153. Casewell MW, Law MM, Desai N. A laboratory model for testing agents for hygienic hand disinfection: handwashing and chlorhexidine for the removal of klebsiella. *J Hosp Infect* 1988;12:163–75.
154. Wade JJ, Desai N, Casewell MW. Hygienic hand disinfection for the removal of epidemic vancomycin-resistant *Enterococcus faecium* and gentamicin-resistant *Enterobacter cloacae*. *J Hosp Infect* 1991;18:211–8.
155. Huang Y, Oie S, Kamiya A. Comparative effectiveness of hand-cleansing agents for removing methicillin-resistant *Staphylococcus aureus* from experimentally contaminated fingertips. *Am J Infect Control* 1994;22:224–7.
156. Lowbury EJJ, Lilly HA. Disinfection of the hands of surgeons and nurses. *Br Med J* 1960;1:5184.
157. Berman RE, Knight RA. Evaluation of hand antisepsis. *Arch Environ Health* 1969;18:781–3.
158. Rotter ML, Simpson RA, Koller W. Surgical hand disinfection with alcohols at various concentrations: parallel experiments using the new proposed European standards method. *Infect Control Hosp Epidemiol* 1998;19:778–81.
159. Hobson DW, Woller W, Anderson L, Guthery E. Development and evaluation of a new alcohol-based surgical hand scrub formulation with persistent antimicrobial characteristics and brushless application. *Am J Infect Control* 1998;26:507–12.
160. Jones MV, Rowe GB, Jackson B, Pritchard NJ. The use of alcoholic paper wipes for routine hand cleansing: results of trials in two hospitals. *J Hosp Infect* 1986;8:268–74.
161. Butz AM, Laughon BE, Gullette DL, Larson EL. Alcohol-impregnated wipes as an alternative in hand hygiene. *Am J Infect Control* 1990;18:70–6.
162. Ojajarvi J. Handwashing in Finland. *J Hosp Infect* 1991;18(suppl B):35–40.
163. Newman JL, Seitz JC. Intermittent use of an antimicrobial hand gel for reducing soap-induced irritation of health care personnel. *Am J Infect Control* 1990;18:194–200.
164. Rotter ML, Koller W, Neumann R. The influence of cosmetic additives on the acceptability of alcohol-based hand disinfectants. *J Hosp Infect* 1991;18 (suppl B):57–63.
165. Larson EL, Aiello AE, Heilman JM, et al. Comparison of different regimens for surgical hand preparation. *AORN J* 2001;73:412–20.
166. Larson EL, Aiello AE, Bastyr J, et al. Assessment of two hand hygiene regimens for intensive care unit personnel. *Crit Care Med* 2001;29:944–51.
167. Ophaswongse S, Maibach HI. Alcohol dermatitis: allergic contact dermatitis and contact urticaria syndrome: a review. *Contact Dermatitis* 1994;30:1–6.
168. Rilliet A, Hunziker N, Brun R. Alcohol contact urticaria syndrome (immediate-type hypersensitivity): case report. *Dermatologica* 1980;161:361–4.
169. Widmer AF. Replace hand washing with use of a waterless alcohol hand rub? *Clin Infect Dis* 2000;31:136–43.
170. Bryant KA, Pearce J, Stover B. Flash fire associated with the use of alcohol-based antiseptic agent [Letter]. *Am J Infect Control* 2002;30:256–7.
171. Hsueh PR, Teng LJ, Yang PC, Pan HL, Ho SW, Luh KT. Nosocomial pseudoepidemic caused by *Bacillus cereus* traced to contaminated ethyl alcohol from a liquor factory. *J Clin Microbiol* 1999;37:2280–4.
172. Denton GW. Chlorhexidine [Chapter 16]. In: Block SS, ed. *Disinfection, sterilization and reservation*. 4th ed. Philadelphia, PA: Lea and Febiger, 1991.
173. Narang HK, Codd AA. Action of commonly used disinfectants against enteroviruses. *J Hosp Infect* 1983;4:209–12.
174. Walsh B, Blakemore PH, Drabu YJ. The effect of handcream on the antibacterial activity of chlorhexidine gluconate. *J Hosp Infect* 1987;9:30–3.
175. Lowbury EJJ, Lilly HA. Use of 4% chlorhexidine detergent solution (Hibiscrub) and other methods of skin disinfection. *Br Med J* 1973;1:510–5.
176. Paulson DS. Comparative evaluation of five surgical hand scrub preparations. *AORN J* 1994;60:246–56.
177. Stringeni L, Lapomarda V, Lisi P. Occupational hand dermatitis in hospital environments. *Contact Dermatitis* 1995;33:172–6.
178. Marrie TJ, Costerton JW. Prolonged survival of *Serratia marcescens* in chlorhexidine. *Appl Environ Microbiol* 1981;42:1093–102.
179. McAllister TA, Lucas CE, Mocan H, et al. *Serratia marcescens* outbreak in a paediatric oncology unit traced to contaminated chlorhexidine. *Scott Med J* 1989;34:525–8.
180. Vigeant P, Loo VG, Bertrand C, et al. An outbreak of *Serratia marcescens* infections related to contaminated chlorhexidine. *Infect Control Hosp Epidemiol* 1998;19:791–4.
181. Vu-Thien H, Darbord JC, Moissenet D, et al. Investigation of an outbreak of wound infections due to *Alcaligenes xylosoxidans* transmitted by chlorhexidine in a burns unit. *Eur J Clin Microbiol* 1998;17:724–6.
182. Larson E, Talbot GH. An approach for selection of health care personnel handwashing agents. *Infect Control* 1986;7:419–24.
183. Davies J, Babb JR, Ayliffe GAJ, Wilkins MD. Disinfection of the skin of the abdomen. *Br J Surg* 1978;65:855–8.
184. Larson E, Mayur K, Laughon BA. Influence of two handwashing frequencies on reduction in colonizing flora with three handwashing products used by health care personnel. *Am J Infect Control* 1988;17:83–8.
185. Soulsby ME, Barnett JB, Maddox S. Brief report: the antiseptic efficacy of chlorxylenol-containing vs. chlorhexidine gluconate-containing surgical scrub preparations. *Infect Control* 1986;7:223–6.
186. Aly R, Maibach HI. Comparative antibacterial efficacy of a 2-minute surgical scrub with chlorhexidine gluconate, povidone-iodine, and chloroxylenol sponge-brushes. *Am J Infect Control* 1988;16:173–7.
187. Archibald LK, Corl A, Shah B, et al. *Serratia marcescens* outbreak associated with extrinsic contamination of 1% chlorxylenol soap. *Infect Control Hosp Epidemiol* 1997;18:704–9.
188. Lowbury EJJ, Lilly HA, Bull JP. Disinfection of hands: removal of resident bacteria. *Br Med J* 1963;1:1251–6.

189. Kundsinn RB, Walter CW. The surgical scrub—practical consideration. *Arch Surg* 1973;107:75–7.
190. Lockhart J. How toxic is hexachlorophene? *Pediatrics* 1972;50:229–35.
191. Shuman RM, Leech RW, Alvord EC Jr. Neurotoxicity of hexachlorophene in humans: II. a clinicopathological study of 46 premature infants. *Arch Neurol* 1975;32:320–5.
192. Dixon RE, Kaslow RA, Mallison GF, Bennett JV. Staphylococcal disease outbreaks in hospital nurseries in the United States—December 1971 through March 1972. *Pediatrics* 1973;51:413–6.
193. Kaslow RA, Dixon RE, Martin SM, et al. Staphylococcal disease related to hospital nursery bathing practices—a nationwide epidemiologic investigation. *Pediatrics* 1973;51:418–29.
194. American Academy of Pediatrics, American College of Obstetricians and Gynecologists. Guidelines for perinatal care. 4th ed. Elk Grove Village, IL: American Academy of Pediatrics; Washington, DC: American Academy of Obstetricians and Gynecologists, 1997.
195. Gottardi W. Iodine and iodine compounds [Chapter 8]. In: Block SS, ed. *Disinfection, sterilization and preservation*. 4th ed. Philadelphia, PA: Lea & Febiger; 1991.
196. Anderson RL. Iodophor antiseptics: intrinsic microbial contamination with resistant bacteria. *Infect Control Hosp Epidemiol* 1989;10:443–6.
197. Goldenheim PD. In vitro efficacy of povidone-iodine solution and cream against methicillin-resistant *Staphylococcus aureus*. *Postgrad Med J* 1993;69(suppl 3):S62–S65.
198. Traoré O, Fayard SF, Laveran H. An in-vitro evaluation of the activity of povidone-iodine against nosocomial bacterial strains. *J Hosp Infect* 1996;34:217–22.
199. McLure AR, Gordon J. In-vitro evaluation of povidone-iodine and chlorhexidine against methicillin-resistant *Staphylococcus aureus*. *J Hosp Infect* 1992;21:291–9.
200. Davies JG, Babb JR, Bradley CR, Ayliffe GAJ. Preliminary study of test methods to assess the virucidal activity of skin disinfectants using poliovirus and bacteriophages. *J Hosp Infect* 1993;25:125–31.
201. Rotter ML. Chapter 79/Hand washing and hand disinfection. In: Mayhall CG, ed. *Hospital Epidemiology and Infection Control*. Baltimore, MD: Williams & Wilkins, 1996.
202. Wade JJ, Casewell MW. The evaluation of residual antimicrobial activity on hands and its clinical relevance. *J Hosp Infect* 1991;18 (suppl B):23–8.
203. Aly R, Maibach HI. Comparative evaluation of chlorhexidine gluconate (Hibiclens[®]) and povidone-iodine (E-Z Scrub[®]) sponge/brushes for presurgical hand scrubbing. *Curr Ther Res* 1983;34:740–5.
204. Herruzo-Cabrera R, Vizcaino-Alcaide MJ, Fdez-AciZero MJ. Usefulness of an alcohol solution of N-duopropenide for the surgical antisepsis of the hands compared with handwashing with iodine-povidone and chlorhexidine: clinical essay. *J Surgical Research* 2000;94:6–12.
205. Hingst V, Juditzki I, Heeg P, Sonntag HG. Evaluation of the efficacy of surgical hand disinfection following a reduced application time of 3 instead of 5 min. *J Hosp Infect* 1992;20:79–86.
206. Faoagali J, Fong J, George N, Mahoney P, O'Rourke V. Comparison of the immediate, residual, and cumulative antibacterial effects of Novaderm R, Novascrub R, Betadine Surgical Scrub, Hibiclens, and liquid soap. *Am J Infect Control* 1995;23:337–43.
207. Pereira LJ, Lee GM, Wade KJ. The effect of surgical handwashing routines on the microbial counts of operating room nurses. *Am J Infect Control* 1990;18:354–64.
208. Peterson AF, Rosenberg A. Comparative evaluation of surgical scrub preparations. *Surg Gynecol Obstet* 1978;146:63–5.
209. Berkelman RL, Holland BW, Anderson RL. Increased bactericidal activity of dilute preparations of povidone-iodine solutions. *J Clin Microbiol* 1982;15:635–9.
210. Merianos JJ. Quaternary ammonium antimicrobial compounds [Chapter 13]. In: Block SS, ed. *Disinfection, Sterilization, and Preservation*. 4th ed. Philadelphia, PA: Lea and Febiger; 1991.
211. Dixon RE, Kaslow RA, Mackel DC, Fulkerson CC, Mallison GF. Aqueous quaternary ammonium antiseptics and disinfectants: use and misuse. *JAMA* 1976;236:2415–7.
212. Sautter RL, Mattman LH, Legaspi RC. *Serratia marcescens* meningitis associated with a contaminated benzalkonium chloride solution. *Infect Control* 1984;5:223–5.
213. Oie S, Kamiya A. Microbial contamination of antiseptics and disinfectants. *Am J Infect Control* 1996;24:389–95.
214. Hayes RA, Trick WE, Vernon MO, et al. Comparison of three hand hygiene (HH) methods in a surgical intensive care unit (SICU) [Abstract K-1337]. Presented at the 41st Interscience Conference on Antimicrobial Agents and Chemotherapy Chicago, IL: American Society for Microbiology, 2001.
215. Dyer DL, Gerenraich KB, Wadhams PS. Testing a new alcohol-free hand sanitizer to combat infection. *AORN J* 1998;68:239–51.
216. Jones RD, Jampani HB, Newman JL, Lee AS. Triclosan: a review of effectiveness and safety in health care settings. *Am J Infect Control* 2000;28:184–96.
217. Ward WH, Holdgate GA, Rowsell S, et al. Kinetic and structural characteristics of the inhibition of enoyl (acyl carrier protein) reductase by triclosan. *Biochemistry* 1999;38:12514–25.
218. Heath RJ, Li J, Roland GE. Inhibition of the *Staphylococcus aureus* NADPH-dependent enoyl-acyl carrier protein reductase by triclosan and hexachlorophene. *J Biol Chem* 2000;275:4654–9.
219. Faoagali JL, George N, Fong J, Davy J, Dowser M. Comparison of the antibacterial efficacy of 4% chlorhexidine gluconate and 1% triclosan handwash products in an acute clinical ward. *Am J Infect Control* 1999;27:320–6.
220. Barry MA, Craven DE, Goularte TA, Lichtenberg DA. *Serratia marcescens* contamination of antiseptic soap containing triclosan: implications for nosocomial infection. *Infect Control* 1984;5:427–30.
221. Lowbury EJL, Lilly HA, Bull JP. Disinfection of hands: removal of transient organisms. *Br Med J* 1964;2:230–3.
222. Rotter ML. Semmelweis' sesquicentennial: a little-noted anniversary of handwashing. *Current Opinion in Infectious Diseases* 1998;11:457–60.
223. Manivannan G, Brady MJ, Cahalan PT, et al. Immediate, persistent and residual antimicrobial efficiency of Surfaccine[™] hand sanitizer [Abstract]. *Infection Control Hosp Epidemiol* 2000;21:105.
224. Gershenfeld L. Povidone-iodine as a sporicide. *Am J Pharm* 1962;134:79–81.
225. Russell AD. Chemical sporicidal and sporostatic agents [Chapter 22]. In: Block SS, ed. *Disinfection, sterilization and preservation*. 4th ed. Philadelphia, PA: Lea and Febiger, 1991.
226. Johnson S, Gerding DN, Olson MM, et al. Prospective, controlled study of vinyl glove use to interrupt *Clostridium difficile* nosocomial transmission. *Am J Med* 1990;88:137–40.
227. Russell AD. Mechanisms of bacterial insusceptibility to biocides. *Am J Infect Control* 2001;29:259–61.
228. Cookson BD, Bolton MC, Platt JH. Chlorhexidine resistance in methicillin-resistant *Staphylococcus aureus* or just an elevated MIC? An in vitro and in vivo assessment. *Antimicrob Agents Chemother* 1991;35:1997–2002.

229. McMurry LM, Oethinger M, Levy SB. Overexpression of *marA*, *soxS*, or *acrAB* produces resistance to triclosan in laboratory and clinical strains of *Escherichia coli*. *FEMS Microbiol Lett* 1998;166:305–9.
230. Chuanchuen R, Beinlich K, Hoang TT, et al. Cross-resistance between triclosan and antibiotics in *Pseudomonas aeruginosa* is mediated by multidrug efflux pumps: exposure of a susceptible mutant strain to triclosan selects *nfxB* mutants overexpressing MexCD-OprJ. *Antimicrob Agents Chemother* 2001;45:428–32.
231. Gröschel DHM, Pruett TL. Surgical antisepsis [Chapter 36]. In: Block SS, ed. *Disinfection, sterilization and preservation*. 4th ed. Philadelphia, PA: Lea and Febiger, 1991.
232. Boyce JM, Potter-Bynoe G, Opal SM, Dziobek L, Medeiros AA. A common-source outbreak of *Staphylococcus epidermidis* infections among patients undergoing cardiac surgery. *J Infect Dis* 1990;161:493–9.
233. Dewar NE, Gravens DL. Effectiveness of seprisol antiseptic foam as a surgical scrub agent. *Appl Microbiol* 1973;26:544–9.
234. Grinbaum RS, de Mendonça JS, Cardo DM. An outbreak of handscrubbing-related surgical site infections in vascular surgical procedures. *Infect Control Hosp Epidemiol* 1995;16:198–202.
235. AORN Recommended Practices Committee. Recommended practices for surgical hand scrubs. In: Fogg D, Parker N, Shevlin D, eds. *Standards, Recommended Practices, and Guidelines*. Denver, CO: AORN, 2001.
236. Larson E, Anderson JK, Baxendale L, Bobo L. Effects of a protective foam on scrubbing and gloving. *Am J Infect Control* 1993;21:297–301.
237. Mulberry G, Snyder AT, Heilman J, Pyrek J, Stahl J. Evaluation of a waterless, scrubless chlorhexidine gluconate/ethanol surgical scrub for antimicrobial efficacy. *Am J Infect Control* 2001;29:377–82.
238. Dineen P. An evaluation of the duration of the surgical scrub. *Surg Gynecol Obstet* 1969;129:1181–4.
239. O'Farrell DA, Kenny G, O'Sullivan M, Nicholson P, Stephens M, Hone R. Evaluation of the optimal hand-scrub duration prior to total hip arthroplasty. *J Hosp Infect* 1994;26:93–8.
240. O'Shaughnessy M, O'Malley VP, Corbett G, Given HF. Optimum duration of surgical scrub-time [Short note]. *Br J Surg* 1991;78:685–6.
241. Wheelock SM, Lookinland S. Effect of surgical hand scrub time on subsequent bacterial growth. *AORN J* 1997;65:1087–98.
242. Deshmukh N, Kjellberg SI, Kramer JW. A comparison of 5-minute povidone-iodine scrub and 1-minute povidone-iodine scrub followed by alcohol foam. *Military Medicine* 1998;163:145–7.
243. Kikuchi-Numagami K, Saishu T, Fukaya M, Kanazawa E, Tagami H. Irritancy of scrubbing up for surgery with or without a brush. *Acta Derm Venereol* 1999;79:230–2.
244. Dineen P. The use of a polyurethane sponge in surgical scrubbing. *Surg Gynecol Obstet* 1966;123:595–8.
245. Bornside GH, Crowder VH Jr, Cohn I Jr. A bacteriological evaluation of surgical scrubbing with disposable iodophor-soap impregnated polyurethane scrub sponges. *Surgery* 1968;64:743–51.
246. McBride ME, Duncan WC, Knox JM. An evaluation of surgical scrub brushes. *Surg Gynecol Obstet* 1973;137:934–6.
247. Berman RE, Knight RA. Evaluation of hand antisepsis. *Arch Environ Health* 1969;18:781–3.
248. Loeb MB, Wilcox L, Smaill F, Walter S, Duff Z. A randomized trial of surgical scrubbing with a brush compared to antiseptic soap alone. *Am J Infect Control* 1997;25:11–5.
249. Larson E, Friedman C, Cohran J, Treston-Aurand J, Green S. Prevalence and correlates of skin damage on the hands of nurses. *Heart Lung* 1997;26:404–12.
250. Tupker RA. Detergents and cleansers [Chapter 7]. In: van der Valk PGM, Maibach HI, eds. *The Irritant Contact Dermatitis Syndrome*. New York, NY: CRC Press, 1996.
251. Wilhelm KP. Prevention of surfactant-induced irritant contact dermatitis. *Curr Probl Dermatol* 1996;25:78–85.
252. de Haan P, Meester HHM, Bruynzeel DP. Irritancy of alcohols [Chapter 6]. In: van der Valk PGM, Maibach HI, eds. *The Irritant Contact Dermatitis Syndrome*. New York, NY: CRC Press, 1996.
253. Lübke J, Ruffieux C, van Melle G, Perrenoud D. Irritancy of the skin disinfectant n-propanol. *Contact Dermatitis* 2001;45:226–31.
254. qhlenschlaeger J, Friberg J, Ramsing D, Agner T. Temperature dependency of skin susceptibility to water and detergents. *Acta Derm Venereol* 1996;76:274–6.
255. Emilson A, Lindberg M, Forslind B. The temperature effect of in vitro penetration of sodium lauryl sulfate and nickel chloride through human skin. *Acta Derm Venereol* 1993;73:203–7.
256. de Groot AC. Contact allergy to cosmetics: causative ingredients. *Contact Dermatitis* 1987;17:26–34.
257. Schnuch A, Uter W, Geier J, Frosch PJ, Rustemeyer T. Contact allergies in healthcare workers—results from the IVDK. *Acta Derm Venereol* 1998;78:358–63.
258. Rastogi SC, Heydorn S, Johansen JD, Basketter DA. Fragrance chemicals in domestic and occupational products. *Contact Dermatitis* 2001;45:221–5.
259. Uter W, Schnuch A, Geier J, Pfahlberg A, Gefeller O. Association between occupation and contact allergy to the fragrance mix: a multifactorial analysis of national surveillance data. *Occup Environ Med* 2001;58:392–8.
260. Perrenoud D, Bircher A, Hunziker T, et al. Frequency of sensitization to 13 common preservatives in Switzerland. *Contact Dermatitis* 1994;30:276–9.
261. Kiec-Swierczynska M, Krecisz B. Occupational skin diseases among the nurses in the region of Lodz. *Int J Occup Med Environ Health* 2000;13:179–84.
262. Garvey LH, Roed-Petersen J, and Husum B. Anaphylactic reactions in anaesthetised patients—four cases of chlorhexidine allergy. *Acta Anaesthesiol Scand* 2001;45:1290–4.
263. Pham, NH, Weiner JM, Reisner GS, and Baldo BA. Anaphylaxis to chlorhexidine. Case report. Implication of immunoglobulin E antibodies and identification of an allergenic determinant. *Clin Exp Allergy* 2000;30:1001–7.
264. Nishioka K, Seguchi T, Yasuno H, Yamamoto T, Tominaga K. The results of ingredient patch testing in contact dermatitis elicited by povidone-iodine preparations. *Contact Dermatitis* 2000;42:90–4.
265. Wong CSM, Beck MH. Allergic contact dermatitis from triclosan in antibacterial handwashes. *Contact Dermatitis* 2001;45:307.
266. Guin JD, Goodman J. Contact urticaria from benzyl alcohol presenting as intolerance to saline soaks. *Contact Dermatitis* 2001;45:182–3.
267. Podda M, Zollner T, Grundmann-Kollmann M, Kaufmann R, Boehncke WF. Allergic contact dermatitis from benzyl alcohol during topical antimycotic treatment. *Contact Dermatitis* 1999;41:302–3.
268. Yesudian PD, King CM. Allergic contact dermatitis from stearyl alcohol in Efudix[®] cream. *Contact Dermatitis* 2001;45:313–4.

269. Aust LB, Maibach HI. Incidence of human skin sensitization to isostearyl alcohol in two separate groups of panelists. *Contact Dermatitis* 1980;6:269–71.
270. Funk JO, Maibach HI. Propylene glycol dermatitis: re-evaluation of an old problem. *Contact Dermatitis* 1994;31:236–41.
271. Hannuksela M. Moisturizers in the prevention of contact dermatitis. *Curr Probl Dermatol* 1996;25:214–20.
272. Berndt U, Wigger-Alberti W, Gabard B, Elsner P. Efficacy of a barrier cream and its vehicle as protective measures against occupational irritant contact dermatitis. *Contact Dermatitis* 2000;42:77–80.
273. McCormick RD, Buchman TL, Maki DG. Double-blind, randomized trial of scheduled use of a novel barrier cream and an oil-containing lotion for protecting the hands of health care workers. *Am J Infect Control* 2000;28:302–10.
274. Larson E, Killien M. Factors influencing handwashing behavior of patient care personnel. *Am J Infect Control* 1982;10:93–9.
275. Zimakoff J, Kjelsberg AB, Larsen SO, Holstein B. A multicenter questionnaire investigation of attitudes toward hand hygiene, assessed by the staff in fifteen hospitals in Denmark and Norway. *Am J Infect Control* 1992;20:58–64.
276. Mayer JA, Dubbert PM, Miller M, Burkett PA, Chapman SW. Increasing handwashing in an intensive care unit. *Infect Control* 1986;7:259–62.
277. Ojajärvi J. The importance of soap selection for routine hand hygiene in hospital. *J Hyg (Lond)* 1981;86:275–83.
278. Scott D, Barnes A, Lister M, Arkell P. An evaluation of the user acceptability of chlorhexidine handwash formulations. *J Hosp Infect* 1991;18(suppl B):51–5.
279. Taylor LJ. An evaluation of handwashing techniques—2. *Nursing Times* 1978;74:108–10.
280. Preston GA, Larson EL, Stamm WE. The effect of private isolation rooms on patient care practices, colonization and infection in an intensive care unit. *Am J Med* 1981;70:641–5.
281. Kaplan LM, McGuckin M. Increasing handwashing compliance with more accessible sinks. *Infect Control* 1986;7:408–10.
282. Freeman, J. Prevention of nosocomial infections by location of sinks for hand washing adjacent to the bedside [Abstract 60]. In: Program and abstracts of the 33rd Interscience Conference on Antimicrobial Agents and Chemotherapy. Washington, DC: American Society for Microbiology, 1993:130.
283. Bischoff WE, Reynolds TM, Sessler CN, Edmond MB, Wenzel RP. Handwashing compliance by health care workers. The impact of introducing an accessible, alcohol-based hand antiseptic. *Arch Intern Med* 2000;160:1017–21.
284. Pittet D. Compliance with hand disinfection and its impact on hospital-acquired infections. *J Hosp Infect* 2001;48(suppl A):S40–S46.
285. Wurtz R, Moye G, Jovanovic B. Handwashing machines, handwashing compliance, and potential for cross-contamination. *Am J Infect Control* 1994;22:228–30.
286. Kohan C, Ligi C, Dumigan DG, Boyce JM. The importance of evaluating product dispensers when selecting alcohol-based handrubs. *Am J Infect Control* 2002 (in press).
287. Boyce JM. Antiseptic technology: access, affordability, and acceptance. *Emerg Infect Diseases* 2001;7:231–3.
288. Taylor LJ. An evaluation of handwashing techniques—1. *Nursing Times* 1978:54–5.
289. Albert RK, Condie F. Hand-washing patterns in medical intensive-care units. *N Engl J Med* 1981;304:1465–6.
290. Larson E. Compliance with isolation technique. *Am J Infect Control* 1983;11:221–5.
291. Donowitz LG. Handwashing technique in a pediatric intensive care unit. *Am J Dis Child* 1987;141:683–5.
292. Conly JM, Hill S, Ross J, Lertzman J, Loule TJ. Handwashing practices in an intensive care unit: the effects of an educational program and its relationship to infection rates. *Am J Infect Control* 1989;17:330–9.
293. DeCarvalho M, Lopes JMA, Pellitteri M. Frequency and duration of handwashing in a neonatal intensive care unit. *Pediatr Infect Dis J* 1989;8:179–80.
294. Graham M. Frequency and duration of handwashing in an intensive care unit. *Am J Infect Control* 1990;18:77–80.
295. Dubbert PM, Dolce J, Richter W, Miller M, Chapman SW. Increasing ICU staff handwashing: effects of education and group feedback. *Infect Control Hosp Epidemiol* 1990;11:191–3.
296. Simmons B, Bryant J, Neiman K, Spencer L, Arheart K. The role of handwashing in prevention of endemic intensive care unit infections. *Infect Control Hosp Epidemiol* 1990;11:589–94.
297. Pettinger A, Nettleman MD. Epidemiology of isolation precautions. *Infect Control Hosp Epidemiol* 1991;12:303–7.
298. Lohr JA, Ingram DL, Dudley SM, Lawton EL, Donowitz LG. Hand washing in pediatric ambulatory settings: an inconsistent practice. *Am J Dis Child* 1991;145:1198–9.
299. Raju TNK, Kobler C. Improving handwashing habits in the newborn nurseries. *Am J Med.Sci* 1991;302:355–8.
300. Larson EL, McGinley KJ, Foglia A, et al. Handwashing practices and resistance and density of bacterial hand flora on two pediatric units in Lima, Peru. *Am J Infect Control* 1992;20:65–72.
301. Zimakoff J, Stormark M, Larsen SO. Use of gloves and handwashing behaviour among health care workers in intensive care units. A multicentre investigation in four hospitals in Denmark and Norway. *J Hosp Infect* 1993;24:63–7.
302. Pelke S, Ching D, Easa D, Melish ME. Gowning does not affect colonization or infection rates in a neonatal intensive care unit. *Arch Pediatr Adolesc Med* 1994;148:1016–20.
303. Gould D. Nurses' hand decontamination practice: results of a local study. *J Hosp Infect* 1994;28:15–30.
304. Shay DK, Maloney SA, Montecalvo M, et al. Epidemiology and mortality risk of vancomycin-resistant enterococcal bloodstream infections. *J Infect Dis* 1995;172:993–1000.
305. Berg DE, Hershov RC, Ramirez CA. Control of nosocomial infections in an intensive care unit in Guatemala City. *Clin Infect Dis* 1995;21:588–93.
306. Tibballs J. Teaching hospital medical staff to handwash. *Med J Aust* 1996;164:395–8.
307. Slaughter S, Hayden MK, Nathan C, et al. A comparison of the effect of universal use of gloves and gowns with that of glove use alone on acquisition of vancomycin-resistant enterococci in a medical intensive care unit. *Ann Intern Med* 1996;125:448–56.
308. Dorsey ST, Cydulka RK, Emerman CL. Is handwashing teachable?: failure to improve handwashing behavior in an urban emergency department. *Acad Emerg Med* 1996;3:360–5.
309. Watanakunakorn C, Wang C, Hazy J. An observational study of hand washing and infection control practices by healthcare workers. *Infect Control Hosp Epidemiol* 1998;19:858–60.

310. Avila-Agüero ML, UmaZa MA, Jiménez AL, Faingezicht I, París MM. Handwashing practices in a tertiary-care, pediatric hospital and the effect on an educational program. *Clin Perform Qual Health Care* 1998;6:70-2.
311. Kirkland KB, Weinstein JM. Adverse effects of contact isolation. *Lancet* 1999;354:1177-8.
312. Maury E, Alzieu M, Baudel JL, et al. Availability of an alcohol solution can improve hand disinfection compliance in an intensive care unit. *Am J Respir Crit Care Med* 2000;162:324-7.
313. Muto CA, Siström MG, Farr BM. Hand hygiene rates unaffected by installation of dispensers of a rapidly acting hand antiseptic. *Am J Infect Control* 2000;28:273-6.
314. Jarvis WR. Handwashing—the Semmelweis lesson forgotten? *Lancet* 1994;344:1311-2.
315. Larson E, Kretzer EK. Compliance with handwashing and barrier precautions. *J Hosp Infect* 1995;30(suppl):88-106.
316. Sproat LJ, Inglis TJJ. A multicentre survey of hand hygiene practice in intensive care units. *J Hosp Infect* 1994;26:137-48.
317. Kretzer EK, Larson EL. Behavioral interventions to improve infection control practices. *Am J Infect Control* 1998;26:245-53.
318. Voss A, Widmer AF. No time for handwashing!? Handwashing versus alcoholic rub: can we afford 100% compliance? *Infect Control Hosp Epidemiol* 1997;18:205-8.
319. Larson E. Handwashing and skin physiologic and bacteriologic aspects. *Infect Control* 1985;6:14-23.
320. Thompson BL, Dwyer DM, Ussery XT, Denman S, Vacek P, Schwartz B. Handwashing and glove use in a long-term care facility. *Infect Control Hosp Epidemiol* 1997;18:97-103.
321. Doebbeling BN, Pfäller MA, Houston AK, Wenzel RP. Removal of nosocomial pathogens from the contaminated glove. *Ann Intern Med* 1988;109:394-8.
322. McLane C, Chenelly S, Sylwestrak ML, Kirchoff KT. A nursing practice problem: failure to observe aseptic technique. *Am J Infect Control* 1983;11:178-82.
323. Pittet D. Improving compliance with hand hygiene in hospitals. *Infect Control Hosp Epidemiol* 2000;21:381-6.
324. Teare L, Handwashing Liaison Group. Hand washing: a modest measure—with big effects. *Br Med J* 1999;318:686.
325. Teare EL, Cookson B, French GL, et al. UK handwashing initiative. *J Hosp Infect* 1999;43:1-3.
326. Larson EL, Bryan JL, Adler LM, Blane C. A multifaceted approach to changing handwashing behavior. *Am J Infect Control* 1997;25:3-10.
327. Weeks A. Why I don't wash my hands between each patient contact [Letter]. *Br Med J* 1999;319:518.
328. Webster J. Handwashing in a neonatal intensive care nursery: product acceptability and effectiveness of chlorhexidine gluconate 4% and triclosan 1%. *J Hosp Infect* 1992;21:137-41.
329. Kelen GD, Green GB, Hexter DA, et al. Substantial improvement in compliance with universal precautions in an emergency department following institution of policy. *Arch Intern Med* 1991;151:2051-6.
330. Lundberg GD. Changing physician behavior in ordering diagnostic tests [Editorial]. *JAMA* 1998;280:2036.
331. Phillips DF. "New look" reflects changing style of patient safety enhancement. *JAMA* 1999;281:217-9.
332. Harbarth S, Martin Y, Rohner P, Henry N, Auckenthaler R, Pittet D. Effect of delayed infection control measures on a hospital outbreak of methicillin-resistant *Staphylococcus aureus*. *J Hosp Infect* 2000;46:43-9.
333. Early E, Battle K, Cantwell E, English J, Lavin JE, Larson E. Effect of several interventions on the frequency of handwashing among elementary public school children. *Am J Infect Control* 1998;26:263-9.
334. Butz AM, Larson E, Fosarelli P, Yolken R. Occurrence of infectious symptoms in children in day care homes. *Am J Infect Control* 1990;18:347-53.
335. Kimel LS. Handwashing education can decrease illness absenteeism. *J Sch Nurs* 1996;12:14-6, 18.
336. Master D, Hess Longe S, Dickson H. Scheduled hand washing in an elementary school population. *Fam Med* 1997;29:336-9.
337. Roberts L, Smith W, Jorm L, Patel M, Douglas RM, McGilchrist C. Effect of infection control measures on the frequency of upper respiratory infection in child care: a randomized, controlled trial. *Pediatrics* 2000;105:738-42.
338. Roberts L, Jorm L, Patel M, Smith W, Douglas RM, McGilchrist C. Effect of infection control measures on the frequency of diarrheal episodes in child care: a randomized, controlled trial. *Pediatrics* 2000;105:743-6.
339. Khan MU. Interruption of shigellosis by handwashing. *Trans R Soc Trop Med Hyg* 1982;76:164-8.
340. Shahid NS, Greenough WB, Samadi AR, Huq MI, Rahman N. Hand washing with soap reduces diarrhoea and spread of bacterial pathogens in a Bangladesh village. *J Diarrhoeal Dis Res* 1996;14:85-9.
341. Stanton BE, Clemens JD. An educational intervention for altering water-sanitation behaviors to reduce childhood diarrhea in urban Bangladesh. *Am J Epidemiol* 1987;125:292-301.
342. McGinley KJ, Larson EL, Leyden JJ. Composition and density of microflora in the subungual space of the hand. *J Clin Microbiol* 1988;26:950-3.
343. Hedderwick SA, McNeil SA, Lyons MJ, Kauffman CA. Pathogenic organisms associated with artificial fingernails worn by healthcare workers. *Infect Control Hosp Epidemiol* 2000;21:505-9.
344. Baumgardner CA, Maragos CS, Larson EL. Effects of nail polish on microbial growth of fingernails: dispelling sacred cows. *AORN J* 1993;58:84-8.
345. Wynd CA, Samstag DE, Lapp AM. Bacterial carriage on the fingernails of OR nurses. *AORN J* 1994;60:796-805.
346. Gross A, Cutright DE, D'Allessandro SM. Effect of surgical scrub on microbial population under the fingernails. *Am J Surg* 1979;138:463-7.
347. Pottinger J, Burns S, Manske C. Bacterial carriage by artificial versus natural nails. *Am J Infect Control* 1989;17:340-4.
348. McNeil SA, Foster CL, Hedderwick SA, Kauffman CA. Effect of hand cleansing with antimicrobial soap or alcohol-based gel on microbial colonization of artificial fingernails worn by health care workers. *Clin Infect Dis* 2001;32:367-72.
349. Rubin DM. Prosthetic fingernails in the OR. *AORN J* 1988;47:944-5, 948.
350. Moolenaar RL, Crutcher M, San Joaquin VH, et al. A prolonged outbreak of *Pseudomonas aeruginosa* in a neonatal intensive care unit: did staff fingernails play a role in disease transmission? *Infect Control Hosp Epidemiol* 2000;21:80-5.
351. Passaro DJ, Waring L, Armstrong R, et al. Postoperative *Serratia marcescens* wound infections traced to an out-of-hospital source. *J Infect Dis* 1997;175:992-5.
352. Foca M, Jakob K, Whittier S, et al. Endemic *Pseudomonas aeruginosa* infection in a neonatal intensive care unit. *N Engl J Med* 2000;343:695-700.

353. Parry MF, Grant B, Yukna M, et al. *Candida* osteomyelitis and diskitis after spinal surgery: an outbreak that implicates artificial nail use. *Clin Infect Dis* 2001;32:352–7.
354. Garner JS, Simmons BP. Guideline for isolation precautions in hospitals. *Infect Control* 1983;4(suppl 4):245–325.
355. CDC. Recommendations for prevention of HIV transmission in health-care settings. *MMWR* 1987;36(suppl 2S):3S–18S.
356. Occupational Safety and Health Administration. 29 CFR Part 1910.1030. Occupational exposure to bloodborne pathogens: final rule. *Federal Register* 1991;29CFR Part 1910:1030.
357. Hartstein AI, Denny MA, Morthland VH, LeMonte AM, Pfaller MA. Control of methicillin-resistant *Staphylococcus aureus* in a hospital and an intensive care unit. *Infect Control Hosp Epidemiol* 1995;16:405–11.
358. Maki DG, McCormick RD, Zilz MA, Stolz SM, Alvarado CJ. An MRSA outbreak in a SICU during universal precautions: new epidemiology for nosocomial MRSA: downside for universal precautions [Abstract 473]. In: Program and abstracts of the 30th Interscience Conference on Antimicrobial Agents and Chemotherapy. Washington, DC: American Society for Microbiology, 1990.
359. Kotilainen HR, Brinker JP, Avato JL, Gantz NM. Latex and vinyl examination gloves: quality control procedures and implications for health care workers. *Arch Intern Med* 1989;149:2749–53.
360. Reingold AL, Kane MA, Hightower AW. Failure of gloves and other protective devices to prevent transmission of hepatitis B virus to oral surgeons. *JAMA* 1988;259:2558–60.
361. Korniewicz DM, Laughon BE, Butz A, Larson E. Integrity of vinyl and latex procedures gloves. *Nurs Res* 1989;38:144–6.
362. DeGroot-Kosolcharoen J, Jones JM. Permeability of latex and vinyl gloves to water and blood. *Am J Infect Control* 1989;17:196–201.
363. Korniewicz DM, Kirwin M, Cresci K, Markut C, Larson E. In-use comparison of latex gloves in two high-risk units: surgical intensive care and acquired immunodeficiency syndrome. *Heart Lung* 1992;21:81–4.
364. Korniewicz DM, Kirwin M, Cresci K, et al. Barrier protection with examination gloves: double versus single. *Am J Infect Control* 1994;22:12–5.
365. Sistrom MG, Muto CA, Neal J, Strain BA, Farr BM. Glove leakage rates as a function of latex content and brand: caveat emptor [Abstract 24]. In: Program and abstracts of the 10th Annual Meeting of Society of Healthcare Epidemiology of America, Orlando, Florida, 1998.
366. Flanagan H, Farr B. Continued evaluation of glove leakage rates at the University of Virginia. Presented at the 11th Annual Meeting of the Society for Healthcare Epidemiology of America, Toronto, Canada, April 1, 2001.
367. Korniewicz DM, Laughon BE, Cyr WH, Lytle CD, Larson E. Leakage of virus through used vinyl and latex examination gloves. *J Clin Microbiol* 1990;28:787–8.
368. Rego A, Roley L. In-use barrier integrity of gloves: latex and nitrile superior to vinyl. *Am J Infect Control* 1999;27:405–10.
369. Fisher MD, Reddy VR, Williams FM, Lin KY, Thacker JG, Edlich RF. Biomechanical performance of powder-free examination gloves. *J Emerg Med* 1999;17:1011–8.
370. Edlich RF, Suber F, Neal JG, Jackson EM, Williams FM. Integrity of powder-free examination gloves to bacteriophage penetration. *J Biomed Mater Res* 1999;48:755–8.
371. Murray CA, Burke FJT, McHugh S. An assessment of the incidence of punctures in latex and non-latex dental examination gloves in routine clinical practice. *Br Dental Journal* 2001;190:377–80.
372. Jones RD, Jampani H, Mulberry G, Rizer RL. Moisturizing alcohol hand gels for surgical hand preparation. *AORN J* 2000;71:584–99.
373. Patterson JE, Vecchio J, Pantelick EL, et al. Association of contaminated gloves with transmission of *Acinetobacter calcoaceticus* var. *antitratius* in an intensive care unit. *Am J Med* 1991;91:479–83.
374. Lowbury EJJ. Aseptic methods in the operating suite. *Lancet* 1968;1:705–9.
375. Hoffman PN, Cooke EM, McCarville MR, Emmerson AM. Microorganisms isolated from skin under wedding rings worn by hospital staff. *Br Med J* 1985;290:206–7.
376. Jacobson G, Thiele JE, McCune JH, Farrell LD. Handwashing: ring-wearing and number of microorganisms. *Nurs Res* 1985;34:186–8.
377. Hayes RA, Trick WE, Vernon MO, et al. Ring use as a risk factor (RF) for hand colonization in a surgical intensive care unit (SICU) [Abstract K-1333]. In: Program and abstracts of the 41st Interscience Conference on Antimicrobial Agents and Chemotherapy. Washington, DC: American Society for Microbiology, 2001.
378. Salisbury DM, Hutfilz P, Treen LM, Bollin GE, Gautam S. The effect of rings on microbial load of health care workers' hands. *Am J Infect Control* 1997;25:24–7.
379. Spire B, Barré-Sinoussi F, Montagnier L, Chermann JC. Inactivation of lymphadenopathy associated virus by chemical disinfectants. *Lancet* 1984;2:899–901.
380. Martin LS, McDougal JS, Loskoski SL. Disinfection and inactivation of the human T lymphotropic virus type III/lymphadenopathy-associated virus. *J Infect Dis* 1985;152:400–3.
381. Resnick L, Veren K, Salahuddin SZ, Tondreau S, Markham PD. Stability and inactivation of HTLV-III/LAV under clinical and laboratory environments. *JAMA* 1986;255:1887–91.
382. van Bueren J, Larkin DP, Simpson RA. Inactivation of human immunodeficiency virus type 1 by alcohols. *J Hosp Infect* 1994;28:137–48.
383. Montefiori DC, Robinson WE Jr, Modliszewski A, Mitchell WM. Effective inactivation of human immunodeficiency virus with chlorhexidine antiseptics containing detergents and alcohol. *J Hosp Infect* 1990;15:279–82.
384. Wood A, Payne D. The action of three antiseptics/disinfectants against enveloped and non-enveloped viruses. *J Hosp Infect* 1998;38:283–95.
385. Harbison MA, Hammer SM. Inactivation of human immunodeficiency virus by Betadine products and chlorhexidine. *J Acquir Immune Defic Syndr* 1989;2:16–20.
386. Lavelle GC, Gubbe SL, Neveaux JL, Bowden BJ. Evaluation of an antimicrobial soap formula for virucidal efficacy in vitro against human immunodeficiency virus in a blood-virus mixture. *Antimicrob Agents Chemother* 1989;33:2034–6.
387. Bond WW, Favero MS, Petersen NJ, Ebert JW. Inactivation of hepatitis B virus by intermediate-to-high level disinfectant chemicals. *J Clin Microbiol* 1983;18:535–8.
388. Kobayashi H, Tsuzuki M, Koshimizu K, et al. Susceptibility of hepatitis B virus to disinfectants or heat. *J Clin Microbiol* 1984;20:214–6.
389. Kurtz JB. Virucidal effect of alcohols against echovirus 11 [Letter]. *Lancet* 1979;1:496–7.
390. Sattar SA, Raphael RA, Lochnan H, Springthorpe VS. Rotavirus inactivation by chemical disinfectants and antiseptics used in hospitals. *Can J Microbiol* 1983;29:1464–9.
391. Larson E, Silberger M, Jakob K, et al. Assessment of alternative hand hygiene regimens to improve skin health among neonatal intensive care unit nurses. *Heart Lung* 2000;29:136–42.

392. Gould D, Chamberlain A. The use of a ward-based educational teaching package to enhance nurses' compliance with infection control procedures. *J Clin Nursing* 1997;6:55-67.
393. Aspöck C, Koller W. A simple hand hygiene exercise. *Am J Infect Control* 1999;27:370-2.
394. McGuckin M, Waterman R, Porten L, et al. Patient education model for increasing handwashing compliance [Practice forum]. *Am J Infect Control* 1999;27:309-14.
395. Khatib M, Jamaledine G, Abdallah A, Ibrahim Y. Hand washing and use of gloves while managing patients receiving mechanical ventilation in the ICU. *Chest* 1999;116:172-5.
396. Haley RW, Bregman DA. The role of understaffing and overcrowding in recurrent outbreaks of staphylococcal infection in a neonatal special-care unit. *J Infect Dis* 1982;145:875-85.
397. Pittet D, Boyce JM. Hand hygiene and patient care: pursuing the Semmelweis legacy. *Lancet Infectious Diseases* 2001;April:9-20.
398. Boyce JM. Scientific basis for handwashing with alcohol and other waterless antiseptic agents. In: Rutala WA, ed. *Disinfection, sterilization and antiseptics: principles and practices in healthcare facilities*. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc, 2001.
399. O'Boyle CA, Henly SJ, Duckett LJ. Nurses' motivation to wash their hands: a standardized measurement approach. *Applied Nursing Research* 2001;14:136-45.
400. Semmelweis IP. *Die aetiologie, der begriff und die prophylaxis des kindbettfiebers*. Pest, Wien und Leipzig: CA Hartleben's Verlags-Expedition 1861.
401. Eggmann P, Harbarth S, Constantin MN, Touveneau S, Chevrolet JC, Pittet D. Impact of a prevention strategy targeted at vascular-access care on incidence of infections acquired in intensive care. *Lancet* 2000;355:1864-8.
402. Bull DA, Neumayer LA, Hunter GC, et al. Improved sterile technique diminishes the incidence of positive line cultures in cardiovascular patients. *J Surgical Research* 1992;52:106-10.
403. Hirschmann H, Fux L, Podusel J, et al. The influence of hand hygiene prior to insertion of peripheral venous catheters on the frequency of complications. *J Hosp Infect* 2001;49:199-203.
404. Drusin LM, Sohmer M, Groshen SL, Spiritos MD, Senterfit LB, Christenson WN. Nosocomial hepatitis A infection in a paediatric intensive care unit. *Arch Dis Child* 1987;62:690-5.
405. Doebbeling BN, Li N, Wenzel RP. An outbreak of hepatitis A among health care workers: risk factors for transmission. *Am J Public Health* 1993;83:1679-84.
406. Standaert SM, Hutcheson RH, Schaffner W. Nosocomial transmission of *Salmonella gastroenteritis* to laundry workers in a nursing home. *Infect Control Hosp Epidemiol* 1994;15:22-6.
407. Rodriguez EM, Parrott C, Rolka H, Monroe SS, Dwyer DM. An outbreak of viral gastroenteritis in a nursing home: importance of excluding ill employees. *Infect Control Hosp Epidemiol* 1996;17:587-92.
408. Schaffner W, Lefkowitz LB Jr, Goodman JS, Koenig MG. Hospital outbreak of infections with group A streptococci traced to an asymptomatic anal carrier. *N Engl J Med* 1969;280:1224-5.
409. Viglionese A, Nottebart VF, Bodman HA, Platt R. Recurrent group A streptococcal carriage in a health care worker associated with widely separated nosocomial outbreaks. *Am J Med* 1991;91(suppl 3B):329S-33S.
410. Ojajarvi J. An evaluation of antiseptics used for hand disinfection in wards. *J Hyg (Lond)* 1976;76:75-82.
411. Mermel LA, Josephson SL, Dempsey J, Parenteau S, Perry C, Magill N. Outbreak of *Shigella sonnei* in a clinical microbiology laboratory. *J Clin Microbiol* 1997;35:3163-5.
412. McBride ME. Microbial flora of in-use soap products. *Appl Environ Microbiol* 1984;48:338-41.
413. Kabara JJ, Brady MB. Contamination of bar soaps under "in use" condition. *J Environ Pathol Toxicol Oncol* 1984;5:1-14.
414. Heinze JE, Yackovich F. Washing with contaminated bar soap is unlikely to transfer bacteria. *Epidem Inf* 1988;101:135-42.
415. Bannan EA, Judge LF. Bacteriological studies relating to handwashing: 1. the inability of soap bars to transmit bacteria. *Am J Public Health* 1965;55:915-21.
416. Field EA, McGowan P, Pearce PK, Martin MV. Rings and watches: should they be removed prior to operative dental procedures? *J Dent* 1996;24:65-9.
417. Lowbury EJJ, Lilly HA. Gloved hand as applicator of antiseptic to operation sites. *Lancet* 1975;2:153-6.
418. AORN Recommended Practices Committee. Recommended practices for surgical hand scrubs. *AORN J* 1999;69:842-50.
419. Grohskopf LA, Roth VR, Feikin DR, et al. *Serratia liquefaciens* bloodstream infections from contamination of epoetin alfa at a hemodialysis center. *N Engl J Med* 2001;344:1491-7.
420. Dharan S, Hugonnet S, Sax H, Pittet D. Evaluation of interference of a hand care cream with alcohol-based hand disinfection. *Occup Environ Dermatol* 2001;49:81-4.
421. Heeg P. Does hand care ruin hand disinfection? *J Hosp Infect* 2001;48(suppl A):S37-S39.
422. McGuckin M, Waterman R, Storr J, et al. Evaluation of a patient-empowering hand hygiene programme in the U.K. *J Hosp Infect* 2001;48:222-7.
423. Girou E, Oppein F. Handwashing compliance in a French university hospital: new perspective with the introduction of hand-rubbing with a waterless alcohol-based solution. *J Hosp Infect* 2001;48(suppl A):S55-S57.

Appendix

Antimicrobial Spectrum and Characteristics of Hand-Hygiene Antiseptic Agents*

Group	Gram-positive bacteria	Gram-negative bacteria	Mycobacteria	Fungi	Viruses	Speed of action	Comments
Alcohols	+++	+++	+++	+++	+++	Fast	Optimum concentration 60%–95%; no persistent activity
Chlorhexidine (2% and 4% aqueous)	+++	++	+	+	+++	Intermediate	Persistent activity; rare allergic reactions
Iodine compounds	+++	+++	+++	++	+++	Intermediate	Causes skin burns; usually too irritating for hand hygiene
Iodophors	+++	+++	+	++	++	Intermediate	Less irritating than iodine; acceptance varies
Phenol derivatives	+++	+	+	+	+	Intermediate	Activity neutralized by nonionic surfactants
Tricolsan	+++	++	+	—	+++	Intermediate	Acceptability on hands varies
Quaternary ammonium compounds	+	++	—	—	+	Slow	Used only in combination with alcohols; ecologic concerns

Note: +++ = excellent; ++ = good, but does not include the entire bacterial spectrum; + = fair; — = no activity or not sufficient.

*Hexachlorophene is not included because it is no longer an accepted ingredient of hand disinfectants.



MMWR™

Morbidity and Mortality Weekly Report

Recommendations and Reports

October 25, 2002 / Vol. 51 / No. RR-16

Continuing Education Activity Sponsored by CDC Guideline for Hand Hygiene in Health-Care Settings

Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force

EXPIRATION — October 25, 2004

You must complete and return the response form electronically or by mail by **October 25, 2004**, to receive continuing education credit. If you answer all of the questions, you will receive an award letter for 1.75 hours Continuing Medical Education (CME) credit; 0.15 Continuing Education Units (CEUs); 1.5 hours Certified Health Education Specialist (CHES) credit; or 1.9 contact

hours Continuing Nursing Education (CNE) credit. If you return the form electronically, you will receive educational credit immediately. If you mail the form, you will receive educational credit in approximately 30 days. No fees are charged for participating in this continuing education activity.

INSTRUCTIONS

By Internet

1. Read this *MMWR* (Vol. 51, RR-16), which contains the correct answers to the questions beginning on the next page.
2. Go to the *MMWR* Continuing Education Internet site at <<http://www.cdc.gov/mmwr/cme/conted.html>>.
3. Select which exam you want to take and select whether you want to register for CME, CEU, CHES, or CNE credit.
4. Fill out and submit the registration form.
5. Select exam questions. To receive continuing education credit, you must answer all of the questions. Questions with more than one correct answer will instruct you to "Indicate all that apply."
6. Submit your answers no later than **October 25, 2004**.
7. Immediately print your Certificate of Completion for your records.

By Mail or Fax

1. Read this *MMWR* (Vol. 50, RR-16), which contains the correct answers to the questions beginning on the next page.
2. Complete all registration information on the response form, including your name, mailing address, phone number, and e-mail address, if available.
3. Indicate whether you are registering for CME, CEU, CHES, or CNE credit.
4. Select your answers to the questions, and mark the corresponding letters on the response form. To receive continuing education credit, you must answer all of the questions. Questions with more than one correct answer will instruct you to "Indicate all that apply."
5. Sign and date the response form or a photocopy of the form and send no later than **October 25, 2004**, to
Fax: 404-639-4198 Mail: MMWR CE Credit
Office of Scientific and Health Communications
Epidemiology Program Office, MS C-08
Centers for Disease Control and Prevention
1600 Clifton Rd, N.E.
Atlanta, GA 30333
6. Your Certificate of Completion will be mailed to you within 30 days.

ACCREDITATION

Continuing Medical Education (CME). CDC is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. CDC designates this educational activity for a maximum of 1.75 hours in category 1 credit toward the AMA Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

Continuing Education Unit (CEU). CDC has been approved as an authorized provider of continuing education and training programs by the International Association for Continuing Education and Training and awards 0.15 Continuing Education Units (CEUs).

Certified Health Education Specialist (CHES). CDC is a designated provider of continuing education contact hours in health education by the National Commission for Health Education Credentialing, Inc. This program is a designated event for CHES to receive 1.5 hours in category 1 credit in health education. CDC provider number GA0082.

Continuing Nursing Education (CNE). This activity for 1.9 contact hours is provided by CDC, which is accredited as a provider of continuing education in nursing by the American Nurses Credentialing Center's Commission on Accreditation.

CENTERS FOR DISEASE CONTROL AND PREVENTION

SAFER • HEALTHIER • PEOPLE™

Goal and Objectives

This *MMWR* provides evidence-based recommendations for hand hygiene in health-care settings. These recommendations were developed by the Healthcare Infection Control Practices Advisory Committee (HICPAC), the Society for Healthcare Epidemiology of America, the Association for Professionals in Infection Control and Epidemiology, and the Infectious Diseases Society of America Hand Hygiene Task Force. The goal of this report is to provide guidance for clinicians and other health-care practitioners regarding strategies to improve hand-hygiene practices and reduce transmission of microorganisms in health-care settings. Upon completion of this educational activity, the reader should be able to 1) describe the indications for hand hygiene in health-care settings; 2) list the advantages of alcohol-based hand rubs; and 3) describe the barriers to hand hygiene in health-care settings.

To receive continuing education credit, please answer all of the following questions.

1. **Hand hygiene refers to . . .**
 - A. handwashing using plain soap and water.
 - B. using an antiseptic hand rub (e.g alcohol, chlorhexidine, iodine).
 - C. handwashing using antimicrobial soap and water.
 - D. all of the above.
2. **Hand hygiene adherence in health-care facilities might be improved by . . .**
 - A. providing personnel with individual containers of alcohol-based hand rubs.
 - B. providing personnel with hand lotions or creams.
 - C. providing personnel with feedback regarding hand-hygiene adherence/performance.
 - D. all of the above.
3. **Alcohol-based hand rubs have good or excellent antimicrobial activity against all of the following except . . .**
 - A. viruses.
 - B. fungi.
 - C. mycobacteria.
 - D. bacterial spores.
 - E. gram-positive and gram-negative bacteria.
4. **Alcohol-based hand rubs are indicated for all of the following clinical situations except . . .**
 - A. when the hands are visibly soiled.
 - B. preoperative cleaning of hands by surgical personnel.
 - C. before inserting urinary catheters, intravascular catheters, or other invasive devices.
 - D. after removing gloves.
5. **Each of the following statements regarding alcohol-based hand rubs is true except . . .**
 - A. alcohol-based hand rubs reduce bacterial counts on the hands of health-care personnel more effectively than plain soaps.
 - B. alcohol-based hand rubs can be made more accessible than sinks or other handwashing facilities.
 - C. alcohol-based hand rubs require less time to use than traditional handwashing.
 - D. alcohol-based hand rubs have been demonstrated to cause less skin irritation and dryness than handwashing using soap and water.
 - E. alcohol-based hand rubs are only effective if they are applied for ≥ 60 seconds.
6. **Which of the following statements regarding preoperative surgical hand antisepsis is true?**
 - A. Antimicrobial counts on hands are reduced as effectively with a 5-minute scrub as with a 10-minute scrub.
 - B. A brush or sponge must be used when applying the antiseptic agent to adequately reduce bacterial counts on hands.
 - C. Alcohol-based hand rubs for preoperative surgical scrub have been associated with increased surgical site infection rates.
 - D. A and B are true.
 - E. A and C are true.
7. **Antimicrobial-impregnated wipes (i.e., towelettes) . . .**
 - A. might be considered as an alternative to handwashing with plain soap and water.
 - B. are as effective as alcohol-based hands rubs.
 - C. are as effective as washing hands with antimicrobial soap and water.
 - D. A and C.
8. **The following statements regarding hand hygiene in health-care settings are true except . . .**
 - A. Overall adherence among health-care personnel is approximately 40%.
 - B. Poor adherence to hand-hygiene practice is a primary contributor to health-care-associated infection and transmission of antimicrobial-resistant pathogens.
 - C. Personnel wearing artificial nails or extenders have been linked to nosocomial outbreaks.
 - D. Hand hygiene is not necessary if gloves are worn.
9. **Indicate your work setting.**
 - A. State/local health department.
 - B. Other public health setting.
 - C. Hospital clinic/private practice.
 - D. Managed care organization.
 - E. Academic institution.
 - F. Other.
10. **Which best describes your professional activities?**
 - A. Patient care — emergency/urgent care department.
 - B. Patient care — inpatient.
 - C. Patient care — primary-care clinic or office.
 - D. Laboratory/pharmacy.
 - E. Public health.
 - F. Other.
11. **I plan to use these recommendations as the basis for . . . (Indicate all that apply.)**
 - A. health education materials.
 - B. insurance reimbursement policies.
 - C. local practice guidelines.
 - D. public policy.
 - E. other.
12. **Each month, approximately how many patients do you examine?**
 - A. None.
 - B. 1–5.
 - C. 6–20.
 - D. 21–50.
 - E. 51–100.
 - F. >100.
13. **How much time did you spend reading this report and completing the exam?**
 - A. 1–1.5 hours.
 - B. More than 1.5 hours but fewer than 2 hours.
 - C. 2–2.5 hours.
 - D. More than 2.5 hours.

- 14. After reading this report, I am confident I can describe the guidance for clinicians and other health-care practitioners regarding strategies to improve hand-hygiene practices and reduce transmission of microorganisms in health-care settings.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.
- 15. After reading this report, I am confident I can describe the indications for hand hygiene in health-care settings.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.
- 16. After reading this report, I am confident I can list the advantages of alcohol-based hand rubs.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.

- 17. After reading this report, I am confident I can describe the barriers to hand hygiene in health-care settings.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.
- 18. The objectives are relevant to the goal of this report.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.
- 19. The tables and text boxes are useful.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.
- 20. Overall, the presentation of the report enhanced my ability to understand the material.
 - A. Strongly agree.
 - B. Agree.
 - C. Neither agree nor disagree.
 - D. Disagree.
 - E. Strongly disagree.

**MMWR Response Form for Continuing Education Credit
October 25, 2002/Vol. 51/No. RR-16
Guideline for Hand Hygiene in Health-Care Settings**

Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force

To receive continuing education credit, you must

1. provide your contact information;
2. indicate your choice of CME, CEU, CHES, or CNE credit;
3. answer all of the test questions;
4. sign and date this form or a photocopy;
5. submit your answer form by October 25, 2004.

Failure to complete these items can result in a delay or rejection of your application for continuing education credit.

Detach or photocopy.

- Check One*
- CME Credit
 - CEU Credit
 - CHES Credit
 - CNE Credit

Last Name _____ First Name _____

Street Address or P.O. Box _____

Apartment _____ or _____ Suite _____

City _____ State _____ ZIP Code _____

Phone Number _____ Fax Number _____

E-Mail Address _____

Fill in the appropriate blocks to indicate your answers. Remember, you must answer all of the questions to receive continuing education credit!

1. [] A [] B [] C [] D	14. [] A [] B [] C [] D [] E
2. [] A [] B [] C [] D	15. [] A [] B [] C [] D [] E
3. [] A [] B [] C [] D [] E	16. [] A [] B [] C [] D [] E
4. [] A [] B [] C [] D	17. [] A [] B [] C [] D [] E
5. [] A [] B [] C [] D [] E	18. [] A [] B [] C [] D [] E
6. [] A [] B [] C [] D [] E	19. [] A [] B [] C [] D [] E
7. [] A [] B [] C [] D	20. [] A [] B [] C [] D [] E
8. [] A [] B [] C [] D	21. [] A [] B [] C [] D [] E
9. [] A [] B [] C [] D [] E [] F	22. [] A [] B [] C [] D [] E
10. [] A [] B [] C [] D [] E [] F	23. [] A [] B [] C [] D [] E [] F
11. [] A [] B [] C [] D [] E	
12. [] A [] B [] C [] D [] E [] F	
13. [] A [] B [] C [] D	

Signature _____ Date I Completed Exam _____

21. These recommendations will affect my practice.

- A. Strongly agree.
- B. Agree.
- C. Neither agree nor disagree.
- D. Disagree.
- E. Strongly disagree.

22. The availability of continuing education credit influenced my decision to read this report.

- A. Strongly agree.
- B. Agree.
- C. Neither agree nor disagree.
- D. Disagree.
- E. Strongly disagree.

23. How did you learn about this continuing education activity?

- A. Internet.
- B. Advertisement (e.g., fact sheet, *MMWR* cover, newsletter, or journal).
- C. Coworker/supervisor.
- D. Conference presentation.
- E. *MMWR* subscription.
- F. Other.

Correct answers for questions 1–8:
1. D; 2. D; 3. D; 4. A; 5. E; 6. A; 7. A; 8. D.

Hospital Infection Control Practices Advisory Committee Members

Chair: Robert A. Weinstein, M.D., Cook County Hospital, Chicago, Illinois.

Co-Chair: Jane D. Siegel, M.D., University of Texas Southwestern Medical Center, Dallas, Texas.

Executive Secretary: Michele L. Pearson, M.D. CDC, Atlanta, Georgia.

Members: Raymond Y.W. Chinn, M.D., Sharp Memorial Hospital, San Diego, California; Alfred DeMaria, Jr, M.D., Massachusetts Department of Public Health, Jamaica Plain, Massachusetts; Elaine L. Larson, Ph.D., Columbia University School of Nursing, New York, New York; James T. Lee, M.D., Ph.D., University of Minnesota, Minneapolis, Minnesota; Ramon E. Moncada, M.D., Coronado Physician's Medical Center, Coronado, California; William A. Rutala, Ph.D., University of North Carolina, Chapel Hill, North Carolina; William E. Scheckler, M.D., University of Wisconsin, Madison, Wisconsin; and Marjorie A. Underwood, Mt. Diablo Medical Center, Concord, California.

Hand Hygiene Task Force Members

Chair: John M. Boyce, M.D., Hospital of Saint Raphael, New Haven, Connecticut.

Members: Barry M. Farr, M.D., University of Virginia, Charlottesville, Virginia; William R. Jarvis, M.D., CDC, Atlanta, Georgia; Elaine L. Larson, Ph.D., Columbia School of Nursing, New York, New York; Maryanne McGuckin, DrScEd, University of Pennsylvania, Philadelphia, Pennsylvania; Carol O'Boyle, Ph.D., Minnesota Department of Health, Minneapolis, Minnesota; Didier Pittet, M.D., University of Geneva, Geneva, Switzerland; Jane D. Siegel, M.D., University of Texas Southwestern Medical Center, Dallas, Texas; Marjorie A. Underwood, Mt. Diablo Medical Center, Concord, California; Andreas F. Widmer, M.D., University Hospitals, Basel, Switzerland; Jolynn Zeller, Avera St. Lukes Hospital, Aberdeen, South Dakota; and Beth H. Stover, Kosair Children's Hospital, Louisville, Kentucky.

Disclosure of Financial Interests and Relationships

John Boyce: Research and educational grants from Gojo Industries; honorarium from Procter and Gamble; and consultant to Bode Chemical.

Barry Farr: Research support from Gojo Industries.

Elaine Larson: Received products for testing in research studies from Procter and Gamble, 3M Corporation, and Steris.

Carol O'Boyle: Honorarium from 3M Corporation.

All *MMWR* references are available on the Internet at <http://www.cdc.gov/mmwr>. Use the search function to find specific articles.

Use of trade names and commercial sources is for identification only and does not imply endorsement by the U.S. Department of Health and Human Services.

References to non-CDC sites on the Internet are provided as a service to *MMWR* readers and do not constitute or imply endorsement of these organizations or their programs by CDC or the U.S. Department of Health and Human Services. CDC is not responsible for the content of these sites. URL addresses listed in *MMWR* were current as of the date of publication.

The *Morbidity and Mortality Weekly Report (MMWR)* series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format and on a paid subscription basis for paper copy. To receive an electronic copy each week, send an e-mail message to listserv@listserv.cdc.gov. The body content should read *SUBscribe mmwr-toc*. Electronic copy also is available from CDC's Internet server at <http://www.cdc.gov/mmwr> or from CDC's file transfer protocol server at <ftp://ftp.cdc.gov/pub/publications/mmwr>. To subscribe for paper copy, contact Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone 202-512-1800.

Data in the weekly *MMWR* are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the following Friday. Address inquiries about the *MMWR* series, including material to be considered for publication, to Editor, *MMWR* Series, Mailstop C-08, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30333; telephone 888-232-3228.

All material in the *MMWR* series is in the public domain and may be used and reprinted without permission; however, citation of the source is appreciated.



Surgical Site Infection (SSI) Toolkit

Activity C: ELC Prevention Collaboratives

S.I. Berríos-Torres, MD

Division of Healthcare Quality Promotion
Centers for Disease Control and Prevention

Draft - 12/21/09 --- Disclaimer: The findings and conclusions in this presentation are those of the authors and do not necessarily represent the views of the Centers for Disease Control and Prevention.





Outline



- **Background**
 - Impact
 - HHS Prevention Targets
 - Pathogenesis
 - Epidemiology
- **Prevention Strategies**
 - Core
 - Supplemental
- **Measurement**
 - Process
 - Outcome
- **Tools for Implementation/Resources/References**



Background: Impact

Burden-US

- ~300,000 SSIs/yr (17% of all HAI; second to UTI)
- 2%-5% of patients undergoing inpatient surgery

Mortality

- 3 % mortality
- 2-11 times higher risk of death
- 75% of deaths among patients with SSI are directly attributable to SSI

Morbidity

- long-term disabilities

Anderson DJ, et al. Strategies to prevent surgical site infections in acute care hospitals. Infect Control Hosp Epidemiol 2008;29:S51-S61 for individual references





Background: Impact

Length of Hospital Stay

- ~7-10 additional postoperative hospital days

Cost

- \$3000-\$29,000/SSI depending on procedure & pathogen
- Up to \$10 billion annually
- Most estimates are based on inpatient costs at time of index operation and do not account for the additional costs of rehospitalization, post-discharge outpatient expenses, and long term disabilities

Anderson DJ, et al. Strategies to prevent surgical site infections in acute care hospitals. Infect Control Hosp Epidemiol 2008;29:S51-S61 for individual references



Background: HHS Prevention Targets

- **Reduce the admission and readmission SSI Standardized Incidence Ratio (SIR) by at least 25% from baseline**
 - Outcome – SSI SIR
- **95% adherence rates to each SCIP/NQF infection process measure**
 - Process - Adherence to SCIP/NQF infection process measures

<http://www.hhs.gov/ophs/initiatives/hai/prevtargets.html>
Appendix G



Background: Pathogenesis
Pathogen Sources



Endogenous

- Patient flora
 - skin
 - mucous membranes
 - GI tract
- Seeding from a distant focus of infection



Background: Pathogenesis

Pathogen Sources



Exogenous

- Surgical Personnel (surgeon and team)
 - Soiled attire
 - Breaks in aseptic technique
 - Inadequate hand hygiene
- OR physical environment and ventilation
- Tools, equipment, materials brought to the operative field



Background: Pathogenesis Organisms Causing SSI January 2006-October 2007

<i>Staphylococcus aureus</i>	30.0%
Coagulase-negative staphylococci	13.7%
Enterococcus spp.	11.2%
<i>Escherichia coli</i>	9.6%
<i>Pseudomonas aeruginosa</i>	5.6%
Enterobacter spp	4.2%
<i>Klebsiella pneumoniae</i>	3.0%
Candida spp.	2.0%
<i>Klebsiella oxytoca</i>	0.7%
<i>Acinetobacter baumannii</i>	0.6%

N=7,025

Hidron AI, et.al., Infect Control Hosp Epidemiol 2008;29:996-1011
Hidron AI et.al., Infect Control Hosp Epidemiol 2009;30:107–107(ERRATUM)



Background: Epidemiology Emerging Challenges

Challenges in detecting SSIs

- Lack of standardized methods for post-discharge/outpatient surveillance
 - Increased number of outpatient surgeries
 - Shorter postoperative inpatient stays

Antimicrobial Prophylaxis

- Increasing trend toward resistant organisms may undermine the effectiveness of existing recommendations for antimicrobial prophylaxis



Background: Epidemiology

Important Modifiable Risk Factors

- Antimicrobial prophylaxis
 - Inappropriate choice (procedure specific)
 - Improper timing (pre-incision dose)
 - Inadequate dose based on body mass index, procedures >3h, or increased blood loss
- Skin or site preparation ineffective
 - Removal of hair with razors
- Colorectal procedures
 - Inadequate bowel prep/antibiotics
 - Improper intraoperative temperature regulation



Background: Epidemiology

Additional Modifiable Risk Factors

- Excessive OR traffic
- Inadequate wound dressing protocol
- Improper glucose control
- Colonization with preexisting microorganisms
- Inadequate intraoperative oxygen levels



Prevention Strategies

- **Core Strategies**
 - High levels of scientific evidence
 - Demonstrated feasibility

- **Supplemental Strategies**
 - Some scientific evidence
 - Variable levels of feasibility

The Collaborative should at a minimum include core prevention strategies. Supplemental prevention strategies also may be used. Most core and supplemental strategies are based on HICPAC guidelines. Strategies that are not included in HICPAC guidelines will be noted by an asterisk () after the strategy. HICPAC guidelines may be found at www.cdc.gov/hicpac



Prevention Strategies: Core Preoperative Measures

Administer antimicrobial prophylaxis in accordance with evidence based standards and guidelines

- Administer within 1 hour prior to incision*
 - 2hr for vancomycin and fluoroquinolones
- Select appropriate agents on basis of
 - Surgical procedure
 - Most common SSI pathogens for the procedure
 - Published recommendations

*Fry DE. Surgical Site Infections and the Surgical Care Improvement Project (SCIP): Evolution of National Quality Measures. Surg Infect 2008;9(6):579-84.



Prevention Strategies: Core Preoperative Measures

- **Remote infections-whenever possible:**
 - Identify and treat before elective operation
 - Postpone operation until infection has resolved
- **Do not remove hair at the operative site unless it will interfere with the operation; do not use razors**
 - If necessary, remove by clipping or by use of a depilatory agent



Prevention Strategies: Core



Preoperative Measures (continued)

- **Skin Prep**
 - Use appropriate antiseptic agent and technique for skin preparation
- **Maintain immediate postoperative normothermia***
- **Colorectal surgery patients**
 - Mechanically prepare the colon (Enemas, cathartic agents)
 - Administer non-absorbable oral antimicrobial agents in divided doses on the day before the operation

*Fry DE. Surgical Site Infections and the Surgical Care Improvement Project (SCIP): Evolution of National Quality Measures. Surg Infect 2008;9(6):579-84.



Prevention Strategies: Core Intraoperative Measures



- **Operating Room (OR) Traffic**
 - Keep OR doors closed during surgery except as needed for passage of equipment, personnel, and the patient



Prevention Strategies: Core Postoperative Measures



- **Surgical Wound Dressing**
 - Protect primary closure incisions with sterile dressing for 24-48 hrs post-op
- **Control blood glucose level during the immediate post-operative period (cardiac)***
 - Measure blood glucose level at 6AM on POD#1 and #2 with procedure day = POD#0
 - Maintain post-op blood glucose level at <200mg/dL
- **Discontinue antibiotics within 24hrs after surgery end time (48hrs for cardiac)***

*Fry DE. Surgical Site Infections and the Surgical Care Improvement Project (SCIP): Evolution of National Quality Measures. Surg Infect 2008;9(6):579-84.





Prevention Strategies: Supplemental Preoperative



- Nasal screen and decolonize only *Staphylococcus aureus* carriers undergoing elective cardiac and other procedures (i.e., orthopaedic, neurosurgery procedures with implants) with preoperative mupirocin therapy*
Bode LGM, et al. Preventing SSI in nasal carriers of Staph aureus. NEJM 2010;362:9-17
- Screen preoperative blood glucose levels and maintain tight glucose control POD#1 and POD#2 in patients undergoing select elective procedures (e.g., arthroplasties, spinal fusions)*

NOTE: These supplemental strategies are not part of the 1999 HICPAC Guideline for Prevention of Surgical Site Infections



Prevention Strategies: Supplemental Perioperative



- Redose antibiotic at the 3 hr interval in procedures with duration >3hrs (* See exceptions to this recommendation in*Engelman R, et al. The Society of Thoracic Surgeons Practice Guideline Series:Antibiotic Prophylaxis in Cardiac Surgery, Part II:Antibiotic Choice. Ann Thor Surg 2007;83:1569-76
- Adjust antimicrobial prophylaxis dose for obese patients (body mass index >30)* Anderson DJ, Kaye KS, Classen D, et al. Strategies to prevent surgical site infections in acute care hospitals. Infect Control Hosp Epidemiol 2008;29 (Suppl 1):S51-S61
- Use at least 50% fraction of inspired oxygen intraoperatively and immediately postoperatively in select procedure(s)* Maragakis LL, Cosgrove SE, Martinez EA, et al. Intraoperative fraction of inspired oxygen is a modifiable risk factor for surgical site infection after spinal surgery. Anesthesiology 2009;110:556-562. and Meyhoff CS, Wetterslev J, Jorgensen LN, et al. Effect of high perioperative oxygen fraction on surgical site infection and pulmonary complications after abdominal surgery: The PROXI randomized clinical trial. JAMA 2009;302:1543-1550.

NOTE: These supplemental strategies are not part of the 1999 HICPAC Guideline for Prevention of Surgical Site Infections





Prevention Strategies: Supplemental Postoperative



- Feedback of surgeon specific infection rates.



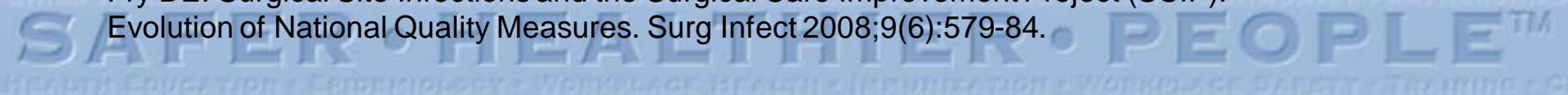
Measurement: Surgical Care Improvement Project (SCIP)



Process Measures

Quality Indicator	Numerator	Denominator
Appropriate antibiotic choice	Number of patients who received the appropriate prophylactic antibiotic	All patients for whom prophylactic antibiotics are indicated
Appropriate timing of prophylactic antibiotics	Number of patients who received the prophylactic antibiotic within 1hr prior to incision (2hr: Vancomycin or Fluoroquinolones)	All patients for whom prophylactic antibiotics are indicated
Appropriate discontinuation of antibiotics	Number of patients who received prophylactic antibiotics and had them discontinued in 24 h (48h cardiac)	All patients who received prophylactic antibiotics

Fry DE. Surgical Site Infections and the Surgical Care Improvement Project (SCIP): Evolution of National Quality Measures. Surg Infect 2008;9(6):579-84.





Measurement: Surgical Care Improvement Project (SCIP)



Process Measures (continued)

Quality Indicator	Numerator	Denominator
Appropriate hair removal	Number of patients who did not have hair removed or who had hair removed with clippers	All surgical patients
Normothermia	Number of patients with postoperative temperature $\geq 36.0^{\circ}\text{C}$	All surgical patients
Glucose control	Number of cardiac surgery patients with glucose control at 6AM POD1 and POD2 (operation = POD0)	Patients undergoing cardiac surgery

Fry DE. Surgical Site Infections and the Surgical Care Improvement Project (SCIP): Evolution of National Quality Measures. Surg Infect 2008;9(6):579-84.



Measurement: Outcome Measures

SSI Rate



Patients with SSI after selected operations X100
Total # of selected operations performed

- Crude, unadjusted rate
- Can lead to erroneous conclusions regarding SSI risk by institution and/or surgeon
- NOT for reporting or inter-hospital comparisons



Measurement: Outcome Measures

Risk Adjustment (1)

NNIS Risk Index

Score to predict risk of acquiring SSI

- Widely used-targeted at surveillance
- Operation-specific
- Allows monitoring of trends
- Facilitates comparison
 - facility vs. national

Culver DH, Horan TC, Gaines RP. Surgical infection rates by wound class, operative procedure, patient risk index. Am J Med;1991:152S-157S.



Measurement: Outcome Measures

Risk Adjustment (2)

NNIS Risk Index



- Focus on high volume operations
- Employs Risk Stratification
 - American Society of Anesthesiologists (ASA) score (3, 4, or 5)
 - Wound Classification (contaminated or dirty)
 - Duration of Procedure (over T [proc specific] hours)
- Does not include many patient & perioperative related SSI risk factors
- Increased NNIS Risk index = Increased risk of SSI

Culver DH, Horan TC, Gaines RP. Surgical infection rates by wound class, operative procedure, patient risk index. Am J Med;1991:152S-157S.



Measurement: Outcome Measures

Risk Adjustment (2)

Standardized Incidence Ratio - SIR

$$\text{SIR} = \frac{\text{Observed \# SSI}}{\text{Expected \# SSI}}$$

Expected # SSI =
operations* in each proc risk category X NNIS rate
100

- Value >1.0 = more SSIs than expected
- Helps better identify outliers
- Will be used for comparison within NHSN in 2010

*Performed by a surgeon, a surgical subspecialty service or a hospital
Detailed explanation and examples in: Edwards JR, Horan TC. Risk-adjusted Comparisons.
In: Carrico R, ed. APIC Text of Infection Control and Epidemiology, 3rd ed. Washington DC
APIC 2009. Chapter 7, p.1-7.



Evaluation Considerations

- **Assess baseline policies and procedures**
- **Areas to consider**
 - **Surveillance**
 - **Prevention strategies**
 - **Measurement**
- **Coordinator should track new policies/practices implemented during collaboration**



References

- Casey AL, Elliott TSJ. Progress in the prevention of surgical site infection. *Curr Opin Infect Dis* 2009;22:370-375
- Chong T, Sawyer R. Update on the epidemiology and prevention of surgical site infections. *Curr Infect Dis Rep* 2002;4:484-490)
- Department of Health and Human Services. Action Plan to Prevent Healthcare-Associated Infections. <http://www.hhs.gov/ophs/initiatives/hai/infection.html> Accessed 17 February 2010
- Fry DE. A systems approach to the prevention of surgical infections. *Surg Clin N Am* 2009;89:521-537.
- Haynes AB, Weiser TG, Berry WR, et al,. A surgical safety checklist to reduce morbidity and mortality in a global population. *N Eng J Med* 2009;360(5):491-499.



References

- Horan TC, Andrus M, Dudeck MA. CDC/NHSN surveillance definition of health care-associated infection and criteria for specific types of infections in the acute care setting. *Am J Infect Control* 2008;36:309-32
- Kirby JP, Mazuski JE. Prevention of surgical site infection. *Surg Clin N Am* 2009;89:365-389.
- Mangram AJ, Horan TC, Pearson ML, et al. Guideline for the prevention of surgical site infection, 1999. Hospital Infection Control Practices Advisory Committee. *Infect Control Hosp Epidemiol* 1999; 20:250-278.
- McKibben L, Horan T, Tokars JI, et al. Guidance on Public Reporting of Healthcare-Associated Infections: Recommendations of the Healthcare Infection Control Practices Advisory Committee. *Am J Infect Control* 2005;33:217-26.



References

- Nichols RL. Preventing surgical site infections. Clin Med Res 2004;2(2):115-118.
- Travis J, Carr JB, Saylor D, et.al., Coronary Artery Bypass Graft Surgery: Surgical Site Infection Prevention. J Healthcare Quality 2009;31:16-23
- Trussell J, Impact of a patient care pathway protocol on surgical site infection rates in cardiothoracic surgery patients. Am J Surg 2008;196:883-889.
- World Alliance for Patient Safety. WHO guidelines for safe surgery. Geneva: World Health Organization, 2008.
- Yokoe DS, Mermel LA, Anderson DJ, et.al. A compendium of strategies to prevent healthcare-associated infections in acute care hospital. Infect Control Hosp Epidemiol 2008;29:S12-S21.



References

SSI Bundles



- Canadian Getting Started Kit:
<http://www.saferhealthcarenow.ca/EN/Interventions/SSI/Pages/ask.aspx> (Select SSI Getting Started Kit)
- IHI:
<http://www.ihl.org/IHI/Programs/Campaign/SSI.htm>
(Select “Power Point Presentation with Facilitator Notes)
<http://www.100liveswashington.org/resources/SSI-summary.pdf>



References

SSI Bundles



- Australian:
http://www.health.vic.gov.au/sss1/downloads/prev_surgical.pdf
- Scottish:
<http://www.hps.scot.nhs.uk/haic/ic/SSIPreventionBundle.aspx>



Resources for Implementation

WHO Surgical Safety Checklist



Surgical Safety Checklist



World Health Organization

Patient Safety

A World Alliance for Safer Health Care

Before induction of anaesthesia

(with at least nurse and anaesthetist)

Has the patient confirmed his/her identity, site, procedure, and consent?

Yes

Is the site marked?

Yes

Not applicable

Is the anaesthesia machine and medication check complete?

Yes

Is the pulse oximeter on the patient and functioning?

Yes

Does the patient have a:

Known allergy?

No

Yes

Difficult airway or aspiration risk?

No

Yes, and equipment/assistance available

Risk of >500ml blood loss (7ml/kg in children)?

No

Yes, and two IVs/central access and fluids planned

Before skin incision

(with nurse, anaesthetist and surgeon)

Confirm all team members have introduced themselves by name and role.

Confirm the patient's name, procedure, and where the incision will be made.

Has antibiotic prophylaxis been given within the last 60 minutes?

Yes

Not applicable

Anticipated Critical Events

To Surgeon:

What are the critical or non-routine steps?

How long will the case take?

What is the anticipated blood loss?

To Anaesthetist:

Are there any patient-specific concerns?

To Nursing Team:

Has sterility (including indicator results) been confirmed?

Are there equipment issues or any concerns?

Is essential imaging displayed?

Yes

Not applicable

Before patient leaves operating room

(with nurse, anaesthetist and surgeon)

Nurse Verbally Confirms:

The name of the procedure

Completion of instrument, sponge and needle counts

Specimen labelling (read specimen labels aloud, including patient name)

Whether there are any equipment problems to be addressed

To Surgeon, Anaesthetist and Nurse:

What are the key concerns for recovery and management of this patient?

This checklist is not intended to be comprehensive. Additions and modifications to fit local practice are encouraged.

Revised 1 / 2009

© WHO, 2009



Cost-estimates of antibiotics in nursing homes range from

\$38 million to
\$137 million
per year.¹



Residents in nursing homes with higher antibiotic use have a

24%
increased risk
of antibiotic-related harm.²



In nursing homes with higher antibiotic use,
even residents who do not receive antibiotics are at increased risk

of indirect antibiotic-related harms due to the spread of resistant bacteria or *C. difficile* germs from other patients.²



Core Elements for Antibiotic Stewardship in Nursing Homes

Creating a Culture to Improve Antibiotic Use in Nursing Homes

Why is Antibiotic Stewardship Important for Nursing Homes?

- ▶ Antibiotics are some of the most commonly prescribed medications in nursing homes.
 - Over the course of a year, up to 70% of nursing home residents get an antibiotic.
- ▶ Roughly 40% to 75% of antibiotics are prescribed incorrectly.
 - In nursing homes, high rates of antibiotics are prescribed to prevent urinary tract infection (UTI) and respiratory tract infection (RTI). Prescribing antibiotics before there is an infection often contributes to misuse.
 - Often residents are given antibiotics just because they are colonized with (carrying) bacteria that are not making the person sick. Prescribing antibiotics for colonization contributes to antibiotic overuse.
- ▶ When patients are transferred between facilities, for example from a nursing home to a hospital, poor communication between facilities about prescribed antibiotics (e.g., rationale, number of days) plus insufficient infection control practices can result in antibiotic misuse and the spread of antibiotic resistance.
- ▶ Antibiotic-related harms, such as diarrhea from *C. difficile*, can be severe, difficult to treat, and lead to hospitalizations and deaths, especially among people over age 65.
- ▶ Current nursing home regulations (e.g., F-tag 441, F-tag 329, F-tag 428) already include a requirement to review and monitor antibiotic use.

What Can I Do as a Leader to Improve Antibiotic Use?

- ▶ Share formal statements in support of improving antibiotic use with staff, residents and families.
- ▶ Commit resources for monitoring antibiotic use and providing feedback to staff.
- ▶ Identify and empower the medical director, director of nursing, and/or consultant pharmacist to lead stewardship activities.
- ▶ Have clear policies to improve prescribing practices for staff to ensure patients are not started on antibiotics unless needed.
 - Establish minimum criteria for prescribing antibiotics,
 - Develop facility-specific standards for empiric antibiotic use, based on data from the facility; and
 - Review antibiotic appropriateness and resistance patterns on a regular basis.
- ▶ Print and distribute materials to educate staff, residents and families.
- ▶ Provide access to individuals with antibiotic expertise for support staff accountable for implementing antibiotic stewardship activities.
- ▶ Partner with antibiotic stewardship program leaders at hospitals and infectious diseases consultants in the community.

¹ Strausbaugh LJ, Joseph CL. Burden of Infections in Long-Term Care. *Infect Control Hosp Epidemiol* 2000;21:674-679.

² Daneman, N et al. Variability in Antibiotic Use Across Nursing Homes and the Risk of Antibiotic-Related Adverse Outcomes for Individual Residents. *JAMA Intern Med.* 2015; E1-E9.



Core Elements for Antibiotic Stewardship in Nursing Homes

Leading Antibiotic Stewardship in Nursing Homes



In nursing homes, approximately

20% of healthcare providers

account for about

80% of antibiotics prescribed.¹



Roughly

40–75%

of antibiotics are prescribed incorrectly.

Nearly

50%

of antibiotics prescribed in nursing homes may be given longer than necessary.¹



Current nursing home regulations (e.g., F-tag 441, F-tag 329, F-tag 428)

already include requirements

to review and monitor antibiotic use.

Who are the Antibiotic Stewardship Leaders in Nursing Homes?

- ▶ **Medical Director**
- ▶ **Director of Nursing**
- ▶ **Consultant Pharmacist**

What are their Roles?



Medical Directors can:

- ▶ Set standards for antibiotic prescribing practices for all healthcare providers prescribing antibiotics.
- ▶ Oversee adherence to antibiotic prescribing practices.
- ▶ Review antibiotic use data and ensure best practices (e.g., the right drug at the right dose for the right amount of time) are followed.



Directors of Nursing can:

- ▶ Establish standards for nursing staff to assess, monitor and communicate changes in a resident's condition that could impact the need for antibiotics.
- ▶ Use their influence as nurse leaders to help ensure antibiotics are prescribed only when appropriate.
- ▶ Educate front line nursing staff about the importance of antibiotic stewardship and explain policies in place to improve antibiotic use.



Consultant Pharmacists can:

- ▶ Provide education to staff about the different types of antibiotics and their uses.
- ▶ Review antibiotic prescriptions as part of the drug regimen review for new medications and ensure they are ordered appropriately.
- ▶ Establish laboratory testing protocols to monitor for adverse events and drug interactions related to use of antibiotics and other high risk medications.
- ▶ Review microbiology culture results and provide feedback to prescribers on initial antibiotic selection to let them know if it is the right drug to treat the infection or if the bacteria may be resistant to the antibiotic.

¹ Daneman, N et.al. Prolonged Antibiotic Treatment in Long-term Care. JAMA Intern Med. 2013; E1-E10.



CAMBRIDGE
UNIVERSITY PRESS

Policy Statement on Antimicrobial Stewardship by the Society for Healthcare Epidemiology of America (SHEA), the Infectious Diseases Society of America (IDSA), and the Pediatric Infectious Diseases Society (PIDS)

Author(s): Society for Healthcare Epidemiology of America, Infectious Diseases Society of America and Pediatric Infectious Diseases Society

Source: *Infection Control and Hospital Epidemiology*, Vol. 33, No. 4, Special Topic Issue: Antimicrobial Stewardship (April 2012), pp. 322-327

Published by: Cambridge University Press on behalf of The Society for Healthcare Epidemiology of America

Stable URL: <http://www.jstor.org/stable/10.1086/665010>

Accessed: 20-04-2016 05:18 UTC

Your use of the JSTOR archive indicates your acceptance of the Terms & Conditions of Use, available at <http://about.jstor.org/terms>

JSTOR is a not-for-profit service that helps scholars, researchers, and students discover, use, and build upon a wide range of content in a trusted digital archive. We use information technology and tools to increase productivity and facilitate new forms of scholarship. For more information about JSTOR, please contact support@jstor.org.



The Society for Healthcare Epidemiology of America, Cambridge University Press are collaborating with JSTOR to digitize, preserve and extend access to *Infection Control and Hospital Epidemiology*

Policy Statement on Antimicrobial Stewardship by the Society for Healthcare Epidemiology of America (SHEA), the Infectious Diseases Society of America (IDSA), and the Pediatric Infectious Diseases Society (PIDS)

Society for Healthcare Epidemiology of America; Infectious Diseases Society of America;
Pediatric Infectious Diseases Society

Antimicrobial resistance has emerged as a significant healthcare quality and patient safety issue in the twenty-first century that, combined with a rapidly dwindling antimicrobial armamentarium, has resulted in a critical threat to the public health of the United States. Antimicrobial stewardship programs optimize antimicrobial use to achieve the best clinical outcomes while minimizing adverse events and limiting selective pressures that drive the emergence of resistance and may also reduce excessive costs attributable to suboptimal antimicrobial use. Therefore, antimicrobial stewardship must be a fiduciary responsibility for all healthcare institutions across the continuum of care. This position statement of the Society for Healthcare Epidemiology of America, the Infectious Diseases Society of America, and the Pediatric Infectious Diseases Society of America outlines recommendations for the mandatory implementation of antimicrobial stewardship throughout health care, suggests process and outcome measures to monitor these interventions, and addresses deficiencies in education and research in this field as well as the lack of accurate data on antimicrobial use in the United States.

Infect Control Hosp Epidemiol 2012;33(4):322-327

It is widely acknowledged that the availability of effective antimicrobial therapy is one of the most important developments in clinical medicine. The harnessing of antibacterial agents for clinical use began during the 1930s–1940s, when sulfonamides, penicillin, and streptomycin became available. It was recognized early that bacteria exposed to antimicrobial agents evolved strategies to survive them, raising the concern that these agents should be used carefully in order to preserve their effectiveness. Sir Alexander Fleming made the following cautionary statements on June 26, 1945, in a *New York Times* article “... the microbes are educated to resist penicillin and a host of penicillin-fast organisms is bred out....In such cases the thoughtless person playing with penicillin is morally responsible for the death of the man who finally succumbs to infection with the penicillin-resistant organism. I hope this evil can be averted.”¹

In the latter half of the twentieth century, a large number of antimicrobial products, including synthetic compounds, became available for clinical use. The ability to control infections through the use of antimicrobial agents has had a major impact in all clinical areas, but particularly in surgery, transplantation medicine, oncology, and intensive care medicine. Penicillin resistance in *Staphylococcus aureus* was initially detected in clinical specimens in 1945, and resistance

to methicillin emerged in 1961.^{2,3} By 1999, methicillin resistance in *S. aureus* was observed in over 53% of *S. aureus* isolates obtained from patients in intensive care units in a US surveillance system.⁴ Strains of methicillin-resistant *S. aureus* (MRSA) emerged in the 1990s as causes of infections in community-residing patients and became common in most geographic areas in the United States in 2000.⁵⁻⁷

The past 30 years have brought multidrug-resistant pneumococci, gonococci, and *Salmonella* spp. and extremely drug-resistant tuberculosis to patients in the community.⁸⁻¹¹ Vancomycin-resistant enterococci and vancomycin-resistant *S. aureus* have also emerged.¹²⁻¹⁴ Extremely drug-resistant gram-negative bacteria, such as carbapenemase-producing *Klebsiella pneumoniae* and other carbapenem-resistant *Enterobacteriaceae* spp., extended-spectrum beta-lactamase-producing *Enterobacteriaceae*, *Pseudomonas aeruginosa*, and *Acinetobacter baumannii* have spread widely among patients in healthcare settings; in some cases these pathogens have been panresistant, that is, resistant to all available antibiotics.¹⁵⁻²²

Unfortunately, during the last decade there has also been a dramatic drop in the development and approval of new antibacterial agents.²³ The antimicrobial armamentarium has been depleted and our ability to treat infectious diseases has been severely compromised. Resistant infections not only re-

Received February 1, 2012; accepted February 1, 2012; electronically published March 15, 2012.

© 2012 by The Society for Healthcare Epidemiology of America. All rights reserved. 0899-823X/2012/3304-0002\$15.00. DOI: 10.1086/665010

sult in increased morbidity and mortality but also dramatically increase healthcare costs.²⁴⁻²⁸ It is ironic that in the twenty-first century we are encountering bacterial infections for which we have no treatment. A multifaceted approach is necessary to prevent, detect, and control the emergence of antimicrobial-resistant organisms. This includes ensuring the availability of adequate and appropriate therapeutic agents, the existence of diagnostic capacity to rapidly and reliably detect specific pathogens and their antimicrobial susceptibilities, and the promotion of robust infection prevention, control, and antimicrobial stewardship programs. This document focuses on issues relating to antimicrobial stewardship. Other issues important to the emergence, transmission, and management of antimicrobial resistance are addressed elsewhere.^{29,30,34}

The Society for Healthcare Epidemiology of America (SHEA) and the Infectious Diseases Society of America (IDSA) recognized these needs in 1997 with the publication of "Guidelines for the Prevention of Antimicrobial Resistance in Hospitals."^{31,32} In 2007, these societies promoted the concept of antimicrobial stewardship when they issued "Guidelines for Developing an Institutional Program to Enhance Antimicrobial Stewardship,"³³ which discusses the development of multidisciplinary teams in acute care settings to review and improve antimicrobial use and improve patient care. A recent IDSA policy paper titled "Combating Antimicrobial Resistance: Policy Recommendations to Save Lives" has been issued.³⁴ It urges a strengthening of US efforts to improve prevention and control efforts, including the adoption of antimicrobial stewardship programs in all US healthcare facilities. Other recommendations include research to define optimal elements and goals of antimicrobial stewardship programs in different healthcare settings, expanded educational efforts on antimicrobial stewardship, novel mechanisms to prevent the overprescription of newly approved antibacterial agents, and the development of new antibacterial therapies, vaccines, and rapid, point-of-care diagnostic tests that would enable appropriate care, including the avoidance of antibacterial agents for viral etiologies.

In recognizing the importance of antimicrobial stewardship as it relates to children, the Pediatric Infectious Diseases Society (PIDS) has developed an annual meeting to address the importance of antimicrobial stewardship for children. PIDS and SHEA have partnered to form a joint antimicrobial stewardship committee to address inpatient antibiotic use, outpatient antibiotic use, antimicrobial stewardship in special populations, education involving antibiotic use, and research on antibiotic use and stewardship. In this joint SHEA-IDSA-PIDS position paper, we focus on the need for public policy around the issue of antimicrobial stewardship.

DEFINITION

Antimicrobial stewardship refers to coordinated interventions designed to improve and measure the appropriate use of anti-

microbial agents by promoting the selection of the optimal antimicrobial drug regimen including dosing, duration of therapy, and route of administration. The major objectives of antimicrobial stewardship are to achieve best clinical outcomes related to antimicrobial use while minimizing toxicity and other adverse events, thereby limiting the selective pressure on bacterial populations that drives the emergence of antimicrobial-resistant strains. Antimicrobial stewardship may also reduce excessive costs attributable to suboptimal antimicrobial use.

RECOMMENDATIONS

1. Antimicrobial Stewardship Programs Should Be Required through Regulatory Mechanisms

At present there are no national or coordinated legislative or regulatory mandates designed to optimize the use of antimicrobial therapy through antimicrobial stewardship. Legislation is also limited at the state level.

California Senate Bill 739 mandated that by January 1, 2008, the California Department of Public Health (CDPH) require that all general acute care hospitals develop a process for evaluating the judicious use of antibiotics, the results of which shall be monitored jointly by appropriate representatives and committees involved in quality improvement activities. While this is the first legislative mandate of its kind, it does not specify that hospitals must intervene to improve antimicrobial use, that is, to have an antimicrobial stewardship program. Thus, the CDPH is learning that given the nonspecific wording used in the mandate, many hospitals are able to meet this requirement without having an antimicrobial stewardship program that meets the objectives as defined above. On the other hand, successful antimicrobial stewardship programs in California are varied, utilizing different combinations of staff, strategies, and criteria; therefore, changing the regulation to be too specific may prevent resource-limited hospitals from developing robust antimicrobial stewardship programs on the basis of facility-specific attributes.

In a preliminary assessment of acute care hospitals in California, 23% of hospitals reported being influenced to start an antimicrobial stewardship program because of Senate Bill 739. Lessons learned from statutory requirements in California include that regulatory mandates are important in convincing hospital administration to fund and staff antimicrobial stewardship programs. It is important to use the wording "antimicrobial stewardship program" in the regulation, as defined above, but it is also important to allow hospitals the flexibility to define how their facility can best meet the objectives of an antimicrobial stewardship program. Inasmuch as current legislation is limited to a single state and focuses only on institutional evaluation of antimicrobial use in hospitals, we support broad implementation of comprehensive antimicrobial stewardship programs across all healthcare settings. Antimicrobial resistance is a critical issue that signifi-

cantly impacts healthcare quality, patient safety, and public health. As such, antimicrobial stewardship and other efforts to limit the emergence and transmission of antimicrobial resistance must be viewed as the fiduciary responsibility of all healthcare institutions across the continuum of care.

SHEA, IDSA, and PIDS recommend that the Centers for Medicare and Medicaid Services (CMS) require participating healthcare institutions to develop and implement antimicrobial stewardship programs. This can be achieved by incorporating the requirement into existing regulations via expansion of interpretive guidelines of the relevant regulation(s). All healthcare facilities, including hospitals, long-term care facilities, long-term acute care facilities, ambulatory surgical centers, and dialysis centers should develop and implement an antimicrobial stewardship plan that is modeled after the IDSA and SHEA "Guidelines for Developing an Institutional Program to Enhance Antimicrobial Stewardship."³³ Minimum requirements for the program should include:

- A. Creation of a multidisciplinary interprofessional antimicrobial stewardship team that is physician directed or supervised. At a minimum, 1 or more members of the team should have training in antimicrobial stewardship. The number of team members may vary on the basis of the size and complexity of the facility. Team members should include but are not limited to:
 - A physician.
 - A pharmacist.
 - A clinical microbiologist.
 - An infection preventionist.
- B. A formulary limited to nonduplicative antibiotics with demonstrated clinical need.
- C. Institutional guidelines for the management of common infection syndromes.
- D. Additional interventions to improve the use of antimicrobials, including those designed to detect and eliminate:
 - Multidrug regimens with unnecessarily redundant antimicrobial spectra.
 - Antibiotic therapy for the management of nonbacterial syndromes or cultures that represent contamination or routine colonization.
 - Empiric regimens that are either inadequately or excessively broad spectrum for infection syndromes.
 - Regimens that do not adequately treat infections caused by culture-confirmed pathogens.
- E. Processes to measure and monitor antimicrobial use at the institutional level for internal benchmarking.
- F. Periodic distribution of a facility-specific antibiogram indicating the rates of relevant antibiotic susceptibilities to key pathogens.

CMS should seek to improve the development, imple-

mentation, and monitoring of antimicrobial stewardship plans and programs over time by requiring additional activities. Such measures may include:

- A. Reporting to the Antimicrobial Use and Resistance option of the Medication-Associated Module of the Centers for Disease Control and Prevention's (CDC's) National Healthcare Safety Network (NHSN).
- B. Prospective surveillance and concurrent intervention for the inappropriate use of antimicrobial agents.
- C. National benchmarking of antimicrobial use at the institutional level based on acuity of care and patient mix.
- D. Relevant future outcome measures, which may include:
 - Prevalence and incidence of drug-resistant phenotypes among common clinical pathogens (eg, carbapenem-resistant *Enterobacteriaceae*, carbapenem-resistant *Acinetobacter*, extensively drug-resistant *Pseudomonas*, MRSA).
 - Incidence of diarrhea caused by *Clostridium difficile*.
 - Rates of adverse antimicrobial drug reactions and interactions.

2. Antimicrobial Stewardship Should Be Monitored in Ambulatory Healthcare Settings

Effective mechanisms do not currently exist to optimize antimicrobial use in ambulatory healthcare settings. Ambulatory settings include but are not limited to outpatient clinical practices, ambulatory surgical centers, and dialysis centers. Inasmuch as these settings account for a significant portion of the antimicrobial use in the United States and there is ample evidence that antimicrobial resistance is emerging as a problem in the community, effective and efficient antimicrobial stewardship initiatives must be developed for these settings. Additionally, such a focus coincides with and complements the implementation of tier 2 of the Department of Health and Human Services' Action Plan to Prevent Healthcare-Associated Infections.³⁵ Therefore, SHEA, IDSA, and PIDS believe that federal agencies such as the Agency for Healthcare Research and Quality (AHRQ), the Office of the National Coordinator for Health Information Technology, CMS, the National Institutes of Health (NIH), and CDC should fund pilot projects designed to develop and implement antimicrobial stewardship in ambulatory settings. We believe that expanded utilization of electronic health records (EHRs) offers great potential in this regard. Areas of study may include:

- Integration of clinical decision support technology into EHRs.
- Integration of clinical decision support technology into e-prescribing mechanisms.

If these interventions are validated in these pilot project

programs, then we support the subsequent integration in the CMS requirement for meaningful use of EHRs.

3. Education about Antimicrobial Resistance and Antimicrobial Stewardship Must Be Accomplished

SHEA, IDSA, and PIDS believe that significant knowledge deficits in the areas of antimicrobial resistance and antimicrobial stewardship are prevalent among healthcare providers in the United States. Educational programs should be developed for those in training programs as well as for all prescribing clinicians that teach about the science behind, the principles of, and the tools essential for the practice of effective antimicrobial stewardship. Education about antimicrobial resistance and stewardship should be incorporated into curriculum requirements for medical students and post-graduate residents and fellows. It is crucial that currently practicing clinicians become proficient in these areas. In addition to ensuring that these areas are included in curricula and programs for those in training, there are a number of ways in which proficiency may be accomplished for practicing clinicians, including partnering with specialty societies and the Food and Drug Administration (FDA) to provide educational resources. Moreover, as a part of the drug-review process, pharmaceutical sponsors should include a plan to educate healthcare providers about both the optimal use of the drug and precautions that reduce the emergence of antimicrobial resistance.

Individual facilities should be responsible for supporting the education of the members of the antimicrobial stewardship team. Antimicrobial stewardship is a patient safety issue and a public health issue and must be taken seriously in all aspects of the continuum of patient care. Additionally, because of the gravity of the problems with antimicrobial resistance that confront society and the paucity of readily available clinical solutions, SHEA, IDSA, and PIDS support appropriations to fund these education initiatives.

4. Antimicrobial Use Data Should Be Collected and Readily Available for Both Inpatient and Outpatient Settings

Accurate and readily available data to track and benchmark antimicrobial use is currently lacking in the United States. The United States is unique among developed countries in that there is no access to these data. We believe that these data are critical to being able to monitor antimicrobial use and its relationship to antimicrobial resistance, and therefore we advocate for a reliable and accurate national system for collecting data on antimicrobial use. When this system is developed, validated, and operationalized, antimicrobial use can be benchmarked, and these data should be utilized as a component of an incentive-based payment system. Reporting to the Antimicrobial Use and Resistance option of the Med-

ication-Associated Module of the CDC's NHSN may accomplish this goal.

5. Research on Antimicrobial Stewardship Is Needed

Significant knowledge gaps exist in our understanding of antimicrobial resistance and interventions to limit both the emergence and the transmission of resistance, as well as in our ability to measure associated impacts and clinical outcomes in these areas. SHEA, IDSA, and PIDS believe that we must refocus translational research efforts in order to answer these questions that are critical to our future ability to effectively treat and manage infectious diseases in the United States. All areas of the translational research paradigm must be addressed, ranging from basic bench science and epidemiologic investigations (T0) to implementation science (T4). Two primary issues of equal importance must be considered in this regard: (1) the benchmarking of antimicrobial use within and between institutions, and the most effective and efficient interventions to optimize these measures; and (2) the development of clear, well-defined, and validated process and outcome measures that may be utilized to assess the clinical impact of stewardship efforts. Initial research proposals should focus on but not necessarily be limited to the following critical issues:

- A. Research is needed to develop a standardized definition of both appropriate and inappropriate antimicrobial use, clear and unambiguous measures of such use, and the risk factors that promote the unnecessary overuse and abuse of antimicrobial therapy. Standardized data collection tools should also be developed to facilitate measurement and interpretation of antimicrobial use data by both government and professional agencies. Furthermore, delineation of the primary drivers of inappropriate antimicrobial use and the relative contribution of individual risk factors that contribute to this outcome are essential to the development of the most effective interventions to prevent these prescriptions.
- B. Patient-centered outcomes research is needed to determine the most effective and cost-efficient deployment of antimicrobial stewardship interventions in different healthcare settings. To date, research in these areas has been plagued by poor study design issues and an absence of standardized definitions. Specifically, current research efforts demonstrate selection biases, insufficient power to answer proposed questions, varying duration of interventions, failure to deal with confounding variables, failure to measure compliance with the intervention processes, and a lack of generalizability. Therefore, SHEA, IDSA, and PIDS recommend using robust study designs that include multicenter randomized-cluster-designed studies that compare stewardship interventions across various healthcare settings as well as the impact of these interventions

on epidemic and endemic antimicrobial resistance within single and across multiple institutions.

- C. Research is needed to develop and validate clear and well-defined process and outcome measures that may be utilized to assess the impact of antimicrobial stewardship interventions both within and across various healthcare settings. While it is critical to understand the impact of antimicrobial stewardship on epidemic and endemic resistance rates both within and between healthcare institutions, we must also develop and validate additional surrogate markers of success. Such measures may include but are not limited to rates of *C. difficile* infection, time to administration of appropriate therapy, adverse drug reactions or interactions related to antimicrobial therapy, drugs administered to patients with documented allergies, multidrug regimens with redundant antimicrobial spectra, regimens that are either inadequate or excessive, and duration of intensive care and overall hospitalization for patients treated with antimicrobials.
- D. SHEA, IDSA, and PIDS believe that it is critical that the United States develop accurate measures of antimicrobial use such as those available in most other developed countries. Such measures can be used to track antimicrobial utilization and correlate such use with emerging antimicrobial resistance patterns. Therefore, an accurate understanding of antimicrobial use data may be used to develop and implement regional targeted interventions to limit the transmission of emerging multidrug-resistant organisms. As noted above, these data may be obtained through annual national point-prevalence surveys of antimicrobial use and/or by reporting to the Antimicrobial Use and Resistance option of the Medication-Associated Module of CDC's NHSN. However, research is needed to determine the validity of both data sets across the continuum of care. For instance, one may prove to be a more accurate representation of antimicrobial use in hospitalized patients whereas the other may more precisely reflect antimicrobial use in the community.
- E. Research is required to understand the impact of the use of generic versus branded antimicrobial agents on how antibiotics are used.
- F. Research is needed to develop and evaluate accurate, easy-to-use, rapid point-of-care diagnostic tests so that antibacterial therapy can be avoided when a viral etiology is identified and used appropriately as indicated by specific bacterial etiologies. The scientific issues surrounding the development and use of such rapid diagnostics are discussed in 2 other IDSA position papers.^{34,36} In addition, further research into the use of biomarkers (such as procalcitonin) that can help to distinguish bacterial from viral disease would be useful in optimizing the use of antibacterial agents, including determining the appropriate duration of therapy.³⁷

Finally, it is imperative that the appropriate federal agen-

cies, such as CDC, AHRQ, FDA, and NIH, receive adequate appropriations to fund these research efforts.

ACKNOWLEDGMENTS

The boards of directors of SHEA, IDSA, and PIDS wish to recognize the many experts who contributed to the development of this joint statement, including, for SHEA: Drs. Neil Fishman, Jan Patterson, Lisa Saiman, Arjun Srinivasan, Kavita K. Trivedi, Trevor van Schooneveld; for IDSA: Drs. Ruth Lynfield, Dale Gerding, Edward Septimus, David Schwartz, and Robert Daum; and for PIDS: Drs. Janet A. Englund, Christopher J. Harrison, John S. Bradley, and Jason Newland. In particular, the SHEA, IDSA, and PIDS boards would like to extend their deep appreciation to Drs. Fishman, Lynfield, and Harrison for shepherding the development of the joint policy statement.

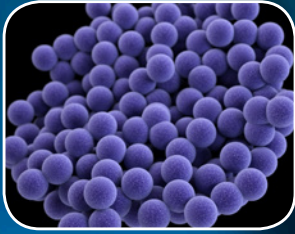
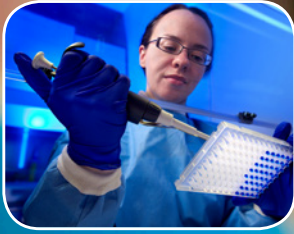
Potential conflicts of interest: Societal policies regarding disclosure of potential conflicts of interest can be obtained by contacting each society listed as an author.

Address correspondence to Neil Fishman, MD, University of Pennsylvania Health System, 3400 Spruce Street, Penn Tower, Suite 101, Philadelphia, PA 19104 (neil.fishman@uphs.upenn.edu).

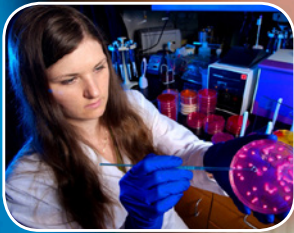
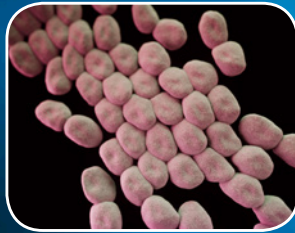
REFERENCES

1. Penicillin finder assays its future. *New York Times*. 26 June 1945: 21.
2. Spink WW, Ferris V. Quantitative action of penicillin inhibitor from penicillin-resistant strain of *Staphylococcus*. *Science* 1945; 102:221–223.
3. Barrett FF, McGehee RF, Finland M. Methicillin-resistant *Staphylococcus aureus* at Boston City Hospital. *N Engl J Med* 1968; 279:441–448.
4. Centers for Disease Control and Prevention NNIS System. National Nosocomial Infections Surveillance (NNIS) system report, data summary from January 1992–April 2000; issued June 2000. *Am J Infect Control* 2000;28:429–448.
5. Herold BC, Immergluck LC, Maranan MC, et al. Community-acquired methicillin-resistant *Staphylococcus aureus* in children with no identified predisposing risk. *JAMA* 1998;279:593–598.
6. Naimi TS, LeDell KH, Como-Sabetti K, et al. Comparison of community- and health care-associated methicillin-resistant *Staphylococcus aureus* infection. *JAMA* 2003;290:2976–2984.
7. Deleo FR, Otto M, Kreisworth BN, Chambers HF. Community-associated methicillin-resistant *Staphylococcus aureus*. *Lancet* 2010;375:1557–1568.
8. Lynch JP 3rd, Zhanell GG. *Streptococcus pneumoniae*: epidemiology and risk factors, evolution of antimicrobial resistance, and impact of vaccines. *Curr Opin Pulm Med* 2010;16:217–225.
9. Deguchi T, Nakane K, Yasuda M, Maeda S. Emergence and spread of drug resistant *Neisseria gonorrhoeae*. *J Urol* 2010;184: 851–858.
10. Parry CM, Threlfall EJ. Antimicrobial resistance in typhoidal and nontyphoidal salmonellae. *Curr Opin Infect Dis* 2008;21: 531–538.
11. Jacobson KR, Tierney DB, Jeon CY, Mitnick CD, Murray MB. Treatment outcomes among patients with extensively drug-resistant tuberculosis: systematic review and meta-analysis. *Clin Infect Dis* 2010;51:6–14.
12. Bonten MJ, Willems R, Weinstein RA. Vancomycin-resistant en-

- terococci: why are they here, and where do they come from? *Lancet Infect Dis* 2001;1:314–325.
13. Chavers LS, Moser SA, Benjamin WH, et al. Vancomycin-resistant enterococci: 15 years and counting. *J Hosp Infect* 2003; 53:159–171.
 14. Hiramatsu K. Vancomycin-resistant *Staphylococcus aureus*: a new model of antibiotic resistance. *Lancet Infect Dis* 2001;1:147–155.
 15. Nordmann P, Cuzon G, Naas T. The real threat of *Klebsiella pneumoniae* carbapenemase-producing bacteria. *Lancet Infect Dis* 2009;9:228–236.
 16. Ho J, Tambyah PA, Paterson DL. Multiresistant gram-negative infections: a global perspective. *Curr Opin Infect Dis* 2010;23: 546–553.
 17. Oteo J, Pérez-Vázquez M, Campos J. Extended-spectrum beta-lactamase producing *Escherichia coli*: changing epidemiology and clinical impact. *Curr Opin Infect Dis* 2010;23:320–326.
 18. Garnacho-Montero J, Amaya-Villar R. Multiresistant *Acinetobacter baumannii* infections: epidemiology and management. *Curr Opin Infect Dis* 2010;23:332–339.
 19. Zavascki AP, Carvalhaes CG, Picão RC, Gales AC. Multidrug-resistant *Pseudomonas aeruginosa* and *Acinetobacter baumannii*: resistance mechanisms and implications for therapy. *Expert Rev Anti Infect Ther* 2010;8:71–93.
 20. Falagas ME, Rafailidis PI, Matthaïou DK, Vrtzili S, Nikita D, Michalopoulos A. Pandrug-resistant *Klebsiella pneumoniae*, *Pseudomonas aeruginosa* and *Acinetobacter baumannii* infections: characteristics and outcome in a series of 28 patients. *Int J Antimicrob Agents* 2008;32:450–454.
 21. Chen LF, Chopra T, Kaye KS. Pathogens resistant to antibacterial agents. *Infect Dis Clin N Am* 2009;23:817–845.
 22. Rice LB. The clinical consequences of antimicrobial resistance. *Curr Opin Microbiol* 2009;12:476–481.
 23. Boucher HW, Talbot GH, Bradley JS, et al. Bad bugs, no drugs: no ESKAPE! an update from the Infectious Diseases Society of America. *Clin Infect Dis* 2009;48:1–12.
 24. Lautenbach E, Weiner MG, Nachamkin I, Bilker WB, Sheridan A, Fishman NO. Imipenem resistance among *Pseudomonas aeruginosa* isolates: risk factors for infection and impact of resistance on clinical and economic outcomes. *Infect Control Hosp Epidemiol* 2006;27:893–900.
 25. Roberts RR, Hota B, Ahmad I, et al. Hospital and societal costs of antimicrobial-resistant infections in a Chicago teaching hospital: implications for antibiotic stewardship. *Clin Infect Dis* 2009;49:1175–1184.
 26. Patel G, Huprikar S, Factor SH, Jenkins SG, Calfee DP. Outcomes of carbapenem-resistant *Klebsiella pneumoniae* infection and the impact of antimicrobial and adjunctive therapies. *Infect Control Hosp Epidemiol* 2008;29:1099–1106.
 27. Filice GA, Nyman JA, Lexau C, et al. Excess costs and utilization associated with methicillin resistance for patients with *Staphylococcus aureus* infection. *Infect Control Hosp Epidemiol* 2010;31: 365–373.
 28. Mauldin PD, Salgado CD, Hansen IS, Durup DT, Bosso JA. Attributable hospital cost and length of stay associated with health care-associated infections caused by antibiotic-resistant gram-negative bacteria. *Antimicrob Agents Chemother* 2010;54: 109–115.
 29. Infectious Diseases Society of America. The 10 × 20 Initiative: pursuing a global commitment to develop 10 new antibacterial drugs by 2020. *Clin Infect Dis* 2010;50:1081–1083.
 30. Infectious Diseases Society of America. *Antimicrobial resistance*. http://www.idsociety.org/AR_Policy/. Accessed October 27, 2010.
 31. Shlaes DM, Gerding DN, John JF Jr, et al. Society for Healthcare Epidemiology of America and Infectious Diseases Society of America Joint Committee on the Prevention of Antimicrobial Resistance: guidelines for the prevention of antimicrobial resistance in hospitals. *Infect Control Hosp Epidemiol* 1997;18: 275–291.
 32. Shlaes DM, Gerding DN, John JF Jr, et al. Society for Healthcare Epidemiology of America and Infectious Diseases Society of America Joint Committee on the Prevention of Antimicrobial Resistance: guidelines for the prevention of antimicrobial resistance in hospitals. *Clin Infect Dis* 1997;25:584–599.
 33. Dellit TH, Owens RC, McGowan JE Jr, et al. Infectious Diseases Society of America and the Society for Healthcare Epidemiology of America guidelines for developing an institutional program to enhance antimicrobial stewardship. *Clin Infect Dis* 2007;44: 159–177.
 34. Spellberg B, Blaser M, Guidos RJ, et al for the Infectious Diseases Society of America. Combating antimicrobial resistance: policy recommendations to save lives. *Clin Infect Dis* 2011;52(suppl 5): S397–S428.
 35. Department of Health and Human Services. *HHS action plan to prevent healthcare-associated infections*. <http://www.hhs.gov/ash/initiatives/hai/actionplan/index.html>. Accessed October 27, 2010.
 36. Gilbert DN, Spellberg B, Bartlett JG for the Infectious Diseases Society of America. Position paper: an unmet medical need: rapid molecular diagnostic tests for respiratory tract infections. *Clin Infect Dis* 2011;52(suppl 4):S384–S395.
 37. Gilbert DN. Use of plasma procalcitonin levels as an adjunct to clinical microbiology. *J Clin Microbiol* 2010;48:2325–2329.



The Core Elements of
Antibiotic Stewardship
for Nursing Homes
APPENDIX A





Appendix A: Policy and practice actions to improve antibiotic use

This document contains more detailed explanations of policy and practice actions which can be taken by nursing homes as part of their antibiotic stewardship activities.

Antibiotic prescribing and use policies

Documentation of dose, duration, and indication.

Specify the dose (including route), duration (i.e., start date, end date, and planned days of therapy), and indication, which includes both rationale (i.e., prophylaxis vs. therapeutic) and treatment site (i.e., urinary tract, respiratory tract), for every course of antibiotics. This bundle of antibiotic prescribing elements should be documented for both nursing home-initiated antibiotic courses as well as courses continued in the nursing home which were initiated by a transferring facility or emergency department. Documenting and making this information accessible (e.g., verifying indication and planned duration is documented on transfer paperwork) helps ensure that antibiotics can be modified as needed based on additional laboratory and clinical data and/or discontinued in a timely manner.¹

Establish best practices for use of microbiology testing.

Inappropriate use of microbiology tests in nursing homes may drive unnecessary antibiotic treatment.² For example, submitting urine cultures or *C. difficile* stool tests to demonstrate “test of cure” following clinical resolution after an appropriate treatment course may uncover asymptomatic colonization and drive additional unnecessary antibiotic exposure. Review the current protocols and laboratory testing practices to ensure that laboratory tests are used correctly in your facility (e.g., your facility should not require one or more negative *C. difficile* stool studies following completion of therapy for *C. difficile* infection). Identifying and reducing inappropriate use of laboratory testing may be a high-yield effort for improving antibiotic use and reducing other management costs.

Develop facility-specific treatment recommendations.

Facility-specific treatment recommendations, based on national guidelines^{3,4} and local susceptibilities can optimize antibiotic selection and duration, particularly for common indications for antibiotic use like pneumonia, urinary tract infection, and skin and soft tissue infections.

Review the antibiotic agents available in the facility

including an inventory of drugs accessible during off hours (e.g., emergency kit or overnight box) to ensure availability is not a barrier to use of preferred agents.

Broad interventions to improve antibiotic use

Develop and implement algorithms for the assessment of residents

suspected of having an infection using evidence-based guidance.^{4,5}

Utilize a communication tool for residents suspected of having an infection.

Since attending physicians, nurse practitioners and/or physician assistants are not always available on-site in nursing homes, a significant amount of management of nursing home residents is mediated via phone interactions. Clinical providers must rely on the assessment and information conveyed to them by the front-line nursing staff to make diagnostic and treatment decisions. Barriers to effective telephone interactions between physicians and nurses, such as inadequate preparation or feeling rushed on the phone,

likely impact the quality of information exchange.⁶ Implementing structured communication tools to guide nursing-physician interactions (e.g., situation, background, assessment, recommendation, or SBAR protocol) may improve the quality of communication and the subsequent management process^{7,8} when an infection is suspected. Communication tools used to facilitate information when a resident is suspected of having an infection should include key pieces of the clinical history including new symptoms and complaints, physical exam findings (e.g., vital signs, pulse oximetry, localizing pain, etc.) and other relevant information (e.g., previous antibiotic exposure, previous culture and susceptibility results, current medications, and medication allergy history). Forms used for this information exchange could not only include information about the resident from nursing staff, but also options for how the off-site provider may want to manage the resident based on the information provided (e.g., hydrate and monitor, send further diagnostic tests, initiate treatment). In addition, any tools or forms utilized to improve communication should become part of the resident's medical record to improve documentation of decision making.

Develop and disseminate a facility-specific report of antibiotic susceptibility to clinical providers.

Nursing homes should work with consultant laboratories to create a facility-specific summary of antibiotic susceptibility patterns from the organisms commonly isolated in microbiology cultures. One example of a susceptibility summary is called an antibiogram. Antibiograms are tables developed by the microbiology laboratory showing the percent susceptibility for a panel of common bacteria tested against a panel of common antibiotics.⁹ Nursing home laboratories may have to tailor the antibiogram based on the facility's diagnostic testing practices. For example, a nursing home antibiogram may only include organisms causing urinary tract infection if urine cultures are the most frequent test sent to the laboratory.¹⁰ Antibiograms may be updated every 12 to 24 months, based on the number of cultures submitted by a facility. Summaries of susceptibility patterns should be disseminated to front-line nursing staff, clinical providers and consultant pharmacists as an educational tool and to guide management decisions.

Perform antibiotic “time outs.” Antibiotics are often started empirically in nursing home residents when the resident has a change in

physical or mental status while diagnostic information is being obtained. However, providers often do not revisit the selection of the antibiotic after more clinical and laboratory data (including culture results) become available.^{11,12} An antibiotic “time out” is a formal process designed to prompt a reassessment of the ongoing need for and choice of an antibiotic once more data is available including: the clinical response, additional diagnostic information, and alternate explanations for the status change which prompted the antibiotic start. Nursing homes should have a process in place for a review of antibiotics by the clinical team two to three days after antibiotics are initiated to answer these key questions:

- Does this resident have a bacterial infection that will respond to antibiotics?
- If so, is the resident on the most appropriate antibiotic(s), dose, and route of administration?
- Can the spectrum of the antibiotic be narrowed or the duration of therapy shortened (i.e., de-escalation)?
- Would the resident benefit from additional infectious disease/antibiotic expertise to ensure optimal treatment of the suspected or confirmed infection?

Reduce prolonged antibiotic treatment courses for common infections. A large study of antibiotic prescribing practices in nursing homes demonstrated that over 50% of antibiotic treatment courses extended beyond a week with no correlation with resident characteristics or type of infection being treated.¹³ Given the growing body of evidence that short courses of antibiotics are effective for common infections,¹⁴⁻¹⁶ interventions designed to decrease antibiotic duration among nursing home residents may reduce the complications and adverse events associated with antibiotic exposure.

Pharmacy interventions to improve antibiotic use

Review of antibiotic prescriptions as part of the drug regimen review (F-tag 428) for new medications is an existing practice for the

consultant pharmacist.¹⁷ Elements of the antibiotic review should include dosing and administration data, to ensure prescribers are making appropriate adjustments for renal function and potential drug interactions. Consultant pharmacists can also review indication and justification of use to verify that antibiotics are used in accordance with facility-specific treatment guidelines.

Establish standards on laboratory testing to monitor for adverse drug events related to use of antibiotics and other high risk medications such as warfarin.^{18,19}

Review of microbiology culture results by the consultant pharmacist can add an additional level of feedback to prescribing clinicians on initial antibiotic selection and subsequent modifications of therapy once data is available. Consultant pharmacists can be given a predefined set of criteria and/or guidance developed in collaboration with physician support^{20,21} to help optimize antibiotic use.

Infection specific interventions to improve antibiotic use

Reduce antibiotic use in asymptomatic bacteriuria (ASB). The prevalence of ASB, bacteriuria without localizing signs or symptoms of infection, ranges from 25% to 50% in non-catheterized nursing home residents and up to 100% among those with long-term urinary catheters.²² Antibiotic use for treatment of ASB in nursing home residents does not confer any long-term benefits in preventing symptomatic urinary tract infections (UTI) or improving mortality, and may actually increase the incidence of adverse drug events and result in subsequent infections with antibiotic-resistant pathogens.²³ The unreliable clinical assessment for infections in nursing home residents coupled with the diagnostic uncertainties in differentiating ASB from infection contributes greatly to inappropriate antibiotic use and its related complications. Suspected UTIs account for 30% to 60% of antibiotic prescriptions in nursing homes.²⁴ Implementing a set of diagnostic testing and management algorithms to help providers differentiate ASB from symptomatic UTI has been shown to reduce inappropriate antibiotic use for ASB.^{25,26}

Reduce antibiotic prophylaxis for prevention of UTI.

Surveys of antibiotic use have shown that UTI prophylaxis accounts for a significant proportion of antibiotic prescriptions.²⁷ Very few studies support antibiotic use for UTI prophylaxis, especially in older adults, and many studies have shown this antibiotic exposure increases risk of side effects and resistant organisms.²³ Therefore, efforts to educate providers on the potential harm of antibiotics for UTI prophylaxis could reduce unnecessary antibiotic exposure and improve resident outcomes.

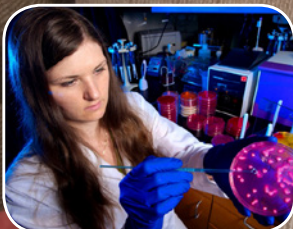
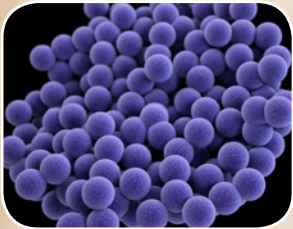
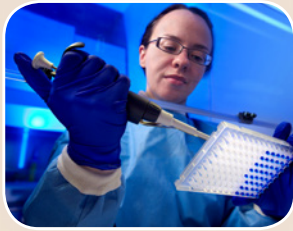
Optimize management of nursing home-associated pneumonia. Limited access to high-quality diagnostic testing makes the differentiation of viral and bacterial causes of lower respiratory tract infections very difficult in nursing home residents.²⁸ Implementation of algorithms for diagnosis and management of nursing home-associated pneumonia may be valuable in helping guide decision-making about use of antibiotics and need for hospital transfer.²⁹⁻³¹

Optimize use of superficial cultures for management of chronic wounds. Although obtaining specimens for wound culture can help guide antimicrobial treatment, reliance on superficial swab cultures alone may drive inappropriate or unnecessary antibiotic use. Superficial wound swabs cannot differentiate bacterial colonization from infection and there may be a lack of correlation between organisms identified by superficial swab cultures compared with deep tissue cultures.³² Reviewing the indications for obtaining cultures in residents with chronic wounds (e.g., presence of purulent drainage) and assessing the type of specimen submitted for culture (e.g., superficial swab vs. tissue specimen from debrided wound base) may identify opportunities for improving antibiotic use in residents with chronic wounds.³³

References

1. Centers for Disease Control and Prevention. Core Elements of Hospital Antibiotic Stewardship Programs. Atlanta, GA: US Department of Health and Human Services, CDC; 2014
2. Phillips CD, Adepoju O, Stone N Asymptomatic bacteriuria, antibiotic use, and suspected urinary tract infections in four nursing homes *BMC Geriatr.* 2012 Nov 23;12:73
3. Nicolle LE, Bentley D, Garibaldi R, et al. Antimicrobial use in long-term care facilities. *Infect Control Hosp Epidemiol* 2000; 21:537–45.
4. High K, Bradley SF, Gravenstein S et al. Clinical practice guideline for the evaluation of fever and infection in older adult residents of long term care facilities. *Clin Infect Dis.* 2009; 48: 149-171
5. Loeb M, Bentley DW, Bradley S, et al. Development of minimum criteria for the initiation of antibiotics in residents of long-term care facilities: Results of a consensus conference. *Infect Control Hosp Epidemiol* 2001; 22: 120-4.
6. Tjia J, Mazor KM, Field T, Meterko V, Spenard A, Gurwitz JH Nurse-physician communication in the long-term care setting: perceived barriers and impact on patient safety *J Patient Saf.* 2009;5(3):145-52
7. Ouslander JG, Bonner A, Herndon L, Shutes J. The interventions to reduce acute care transfers (INTERACT) quality improvement program: An overview for medical directors and primary care clinicians in long term care. *J Am Med Dir Assoc.* 2014;15(3):162-70
8. Renz SM, Boltz MP, Wagner LM, Capezuti EA, Lawrence TE. Examining the feasibility and utility of an SBAR protocol in long-term care. *Geriatr Nurs.* 2013;34(4):295-301
9. Furuno JP, Comer AC, Johnson JK, et al. Using antibiograms to improve antibiotic prescribing in skilled nursing facilities. *Infect Control Hosp Epidemiol.* 2014;35 (Suppl 3):S56-61.
10. Drinka P, Podzorski RP, Griffin V, Crnich CJ. Antibiogram of urinary isolates. *J Am Med Dir Assoc.* 2013;14(6):443
11. Katz PR, Beam TR Jr., Brand F, Boyce K. Antibiotic use in the nursing home: Physician practice patterns. *Arch Int Med.* 1990; 150: 1465-1468
12. Shirts BH, Perera S, Hanlon JT, et al. Provider management of and satisfaction with laboratory testing in the nursing home setting: Results of a national internet-based survey. *J Am Med Dir Assoc.* 2009;10: 161–166.
13. Daneman N, Gruneir A, Bronskill SE et al. Prolonged antibiotic treatment in long-term care: role of the prescriber. *JAMA Intern Med.* 2013; 173 (8): 673-682
14. Lutters M, Vogt-Ferrier NB. Antibiotic duration for treating uncomplicated, symptomatic lower urinary tract infections in elderly women. *Cochrane Database Syst Rev.* 2008;(3):CD001535.
15. Hepburn MJ, Dooley DP, Skidmore PJ, Ellis MW, Starnes WF, Hasewinkle WC. Comparison of short-course (5 days) and standard (10 days) treatment for uncomplicated cellulitis. *Arch Intern Med.* 2004;164(15):1669-1674.
16. El Moussaoui R, de Borgie CA, van den Broek P, et al. Effectiveness of discontinuing antibiotic treatment after three days versus eight days in mild to moderate severe community-acquired pneumonia: randomised, double blind study. *BMJ.*2006;332(7554):1355.

17. Centers for Medicare and Medicaid Services. Summary of Requirements for Drug Regimen Review- F428 State Operations Manual (SOM); Appendix PP; Rev 107, 04-04-2014 Pages 539 – 548: https://cms.gov/manuals/Downloads/som107ap_pp_guidelines_itcf.pdf Accessed 9/30/2014.
18. Baillargeon J1, Holmes HM, Lin YL. Concurrent use of warfarin and antibiotics and the risk of bleeding in older adults. *Am J Med.* 2012 Feb;125(2):183-9
19. Hines LE, Murphy JE. Potentially harmful drug-drug interactions in the elderly: a review. *Am J Geriatr Pharmacother.* 2011; 9(6): 364-377
20. Faulks JT, Drinka P, Gauerke C, Miller J. Pharmacy review of culture and sensitivity with prompting of physicians to reduce antibiotic pressure. *J Am Geriatr Soc.* 2001 Sep;49(9):1259-60
21. Gugkaeva Z, Franson M. Pharmacist-led model of antibiotic stewardship in a long-term care facility. *Annals of Long Term Care.* 2012; 20(10). <http://www.annalsoflongtermcare.com/article/pharmacist-led-model-antibiotic-stewardship-long-term-care-facility> Accessed 12/30/2014.
22. Nicolle LE. Urinary tract infections in long-term-care facilities. *Infect Control Hosp Epidemiol* 2001; 22:167–75.
23. Nicolle LE. Asymptomatic bacteriuria: Review and discussion of the IDSA guidelines *Int J Antimicrob Agents* 2006; 28S:S42–S48.
24. Benoit SR, Nsa W, Richards CL et al. Factors associated with antimicrobial use in nursing homes: A multilevel model. *J Am Geriatr Soc.* 2008; 56:2039–2044
25. Loeb M, Brazil K, Lohfeld L, et al. Effect of a multifaceted intervention on number of antimicrobial prescriptions for suspected urinary tract infections in residents of nursing homes: cluster randomised controlled trial. *British Med J* 2005; 331: 669
26. Zarbarsky TF, Sethi AK, Donskey CJ. Sustained reduction in inappropriate treatment of asymptomatic bacteriuria in a long-term care facility through an educational intervention. *Am J Infect Contr* 2008; 36: 476-480
27. Latour K, Catry B, Broex E et al. Indications for antimicrobial prescribing in European nursing homes: results from a point prevalence survey. *Pharmacoepidem and drug safety.* 2012; 21: 937–944
28. Mubareka S, Duckworth H, Cheang M, et al. Use of diagnostic tests for presumed lower respiratory tract infection in long-term care facilities. *J Am Geriatr Soc.* 2007; 55:1365–1370
29. Carusone SC, Loeb M, Lohfeld L. A clinical pathway for treating pneumonia in the nursing home: part I: the nursing perspective. *J Am Med Dir Assoc.* 2006;7(5):271-8.
30. Carusone SC, Loeb M, Lohfeld L. A clinical pathway for treating pneumonia in the nursing home: part II: the administrators' perspective and how it differs from nurses' views. *J Am Med Dir Assoc.* 2006;7(5):279-286.
31. Loeb M, Carusone SC, Goeree R et al. Effect of a clinical pathway to reduce hospitalizations in nursing home residents with pneumonia: a randomized controlled trial. *JAMA.* 2006;295(21):2503-10
32. Mutluoglu M, Uzun G, Turhan V et al. How reliable are cultures of specimens from superficial swabs compared with those of deep tissue in patients with diabetic foot ulcers? *J Diabetes Comp.* 2012; 26: 225-229
33. Drinka P, Bonham P, Crnich CJ. Swab culture of purulent skin infection to detect infection or colonization with antibiotic-resistant bacteria. *J Am Med Dir Assoc.* 2012; 13: 75-79



The Core Elements of
Antibiotic Stewardship
 for Nursing Homes

APPENDIX B



Appendix B: Measures of antibiotic prescribing, use and outcomes

This document contains more detailed explanations of antibiotic use process and outcome measures which can be tracked by nursing homes to monitor the impact of their antibiotic stewardship activities.

Process measures for tracking antibiotic stewardship activities

Completeness of clinical assessment documentation at the time of the antibiotic prescription. Incomplete assessment and documentation of a resident's clinical status, physical exam or laboratory findings at the time a resident is evaluated for infection can lead to uncertainty about the rationale and/or appropriateness of an antibiotic. If a facility has developed algorithms or protocols for evaluating a resident suspected of having an infection, then perform audits of the quality of the assessment to ensure that algorithm was followed.

Completeness of antibiotic prescribing

documentation. Ongoing audits of antibiotic prescriptions for completeness of documentation, regardless of whether the antibiotic was initiated in the nursing home or at a transferring facility, should verify that the antibiotic prescribing elements have been addressed and recorded. These elements include: dose, (including route), duration (i.e., start date, end date and planned days of therapy), and indication (i.e., rationale and treatment site) for every course of antibiotics.

Antibiotic selection is consistent with recommended agents for specific indications.

If a facility has developed and implemented facility-specific treatment guidelines for one or more infections, then an intermittent review of antibiotic selection is warranted to ensure practices are consistent with facility policies.

Measures of antibiotic use

Point prevalence of antibiotic use. Point prevalence surveys of antibiotic use track the proportion of residents receiving antibiotics during a given time period (i.e., a single-day, a week, or a month). Because the data collection is time-limited, point prevalence surveys are an easier way to capture antibiotic use data. In addition to providing a snap-shot of the burden of antibiotic use in a facility, point-prevalence surveys can capture specific information about the residents receiving antibiotics and indications for antibiotic therapy.¹ Unlike other antibiotic use measures which focus only on the prescriptions initiated in the nursing home, prevalence surveys could also include data on residents admitted to the facility already receiving an antibiotic to track the total burden of individuals at risk for complications from antibiotic use (e.g., *C. difficile* infection).

- Percent of residents receiving antibiotics: (Number of residents on antibiotic/total residents in the facility) X 100
 - Prevalence data can be stratified by specific resident characteristics, for example percent of residents receiving antibiotics among short-stay versus long-stay residents
- Percent of new admissions receiving antibiotics: (Number of residents admitted to nursing home receiving antibiotics/total number of new admissions) X 100

Because prevalence surveys are often conducted for a brief window of time, this data may not portray the magnitude of antibiotic use over time. While a single-day prevalence survey may show 5% to 13% of residents are receiving an antibiotic, studies which follow a group of residents over long periods of time (e.g., 12 months) show that as many as 50% to 75% of residents receive one or more courses of antibiotics.²

Antibiotic starts. Most nursing home infection prevention and control programs already track new antibiotic starts occurring in the facility as part of their infection surveillance activity. Generally, rates of antibiotic starts are based on the prescriptions written after the resident has been admitted to the facility. Data on antibiotic starts can be calculated and reported in the following ways:

- Rate of new antibiotic starts initiated in nursing home (per 1,000 resident-days): (Number of new antibiotic prescriptions/total number of resident-days) X 1,000
 - Rate of antibiotic starts can be calculated by indication, for example: (Number of new antibiotic starts for urinary tract infection/total number of resident-days) X 1,000
- Rates of antibiotic starts could also be calculated for individual prescribers in the nursing home to compare

prescribing patterns among different providers practicing in the facility. However, prescriber-specific rates must take into account differences in the total number of residents cared for by each provider.

Tracking and reporting antibiotic start data could assess the impact of antibiotic stewardship initiatives designed to educate and guide providers on situations when antibiotics are not appropriate. However, interventions focused on shortening the number of days of therapy may not demonstrate significant changes in antibiotic starts.

Antibiotic days of therapy (DOT). Tracking antibiotic DOTs requires more effort than tracking antibiotic starts, but may provide a better measure to monitor changes in antibiotic use over time. The ratio of antibiotic DOT to total resident-days has been referred to as the antibiotic utilization ratio (AUR).³ Below are the steps for calculating monthly rates of antibiotic DOT and AUR.

- An antibiotic day: each day that a resident receives a single antibiotic
 - For example, if a resident is prescribed a 7-day course of amoxicillin, that course equals 7 antibiotic days. However, if a resident is prescribed a 7-day course of ceftriaxone plus azithromycin, then that course equals 14 antibiotic days.
- Antibiotic DOT: the sum of all antibiotic days for all residents in the facility during a given time frame (e.g., 1 month or 1 quarter)
 - Rate of antibiotic DOT (per 1,000 resident-days):
(Total monthly DOT/total monthly resident-days) X 1,000
 - Antibiotic utilization ratio: Total monthly DOT/total monthly resident-days

Antibiotic outcome measures

Track *C. difficile* and antibiotic resistance.

The National Healthcare Safety Network (NHSN) is a CDC-operated web-based system for tracking and reporting targeted infections and antibiotic-resistant organisms from healthcare facilities. In 2012, NHSN launched a reporting component specifically designed for use by nursing homes and other long-term care facilities. The Laboratory-identified event module in NHSN (<http://www.cdc.gov/nhsn/ltc/cdiff-mrsa/index.html>) allows facilities to track rates of *C. difficile* and selected multidrug-resistant organisms such as methicillin-resistant *Staphylococcus aureus* (MRSA) and antibiotic resistant gram-negative bacteria like *E.coli* using laboratory based surveillance as a proxy for infections.⁴

Track adverse drug events related to antibiotic use.

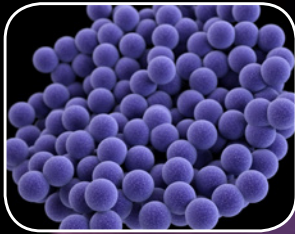
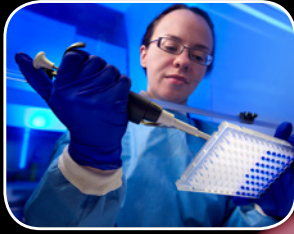
Adverse events due to use of medications in skilled nursing homes accounted for nearly 40% of harms identified in a recent report.⁵ Antibiotics are among the most frequently prescribed medications in LTCFs and have a high rate of adverse drug events.^{6,7}

Track costs related to antibiotic use.

Very few, if any, studies on antibiotic use in nursing homes have calculated the financial costs of antibiotic use.^{8,9} However, in acute care settings, antibiotic stewardship has been shown to reduce hospital pharmacy costs in addition to improving antibiotic use.¹⁰ This metric can be useful in justifying support of staff time and external consultant support for ASP activities.

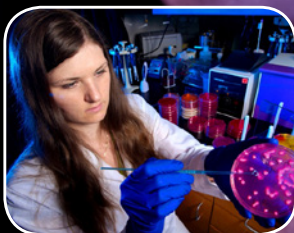
References

1. Zarbarsky TF, Sethi AK, Donskey CJ. Sustained reduction in inappropriate treatment of asymptomatic bacteriuria in a long-term care facility through an educational intervention. *Am J Infect Contr.* 2008; 36: 476-480
2. Lim CJ, Kong DCM, Stuart RL. Reducing inappropriate antibiotic prescribing in the residential care setting: current perspectives. *Clin Interv Aging.* 2014; 9: 165-177
3. Mylotte JM. Antimicrobial prescribing in long-term care facilities: Prospective evaluation of potential antimicrobial use and cost indicators. *Am J Infect Control.* 1999; 27(1): 10-19.
4. Centers for Disease Control and Prevention. Laboratory-identified Event Module for Long-term care facilities. http://www.cdc.gov/nhsn/PDFs/LTC/LTCF-LabID-Event-Protocol_FINAL_8-24-12.pdf Accessed 12/30/14
5. Office of the Inspector General. Adverse Events in Skilled Nursing Facilities: National Incidence Among Medicare Beneficiaries (OEI-06-11-00370), February 2014.
6. Nicolle LE, Bentley D, Garibaldi R, et al. Antimicrobial use in long-term care facilities. *Infect Control Hosp Epidemiol* 2000; 21:537-45.
7. Gurwitz JH, Field TS, Avorn J et al. Incidence and preventability of adverse drug events in nursing homes. *Am J Med.* 2000;109:87-94.
8. Mylotte JM. Antimicrobial prescribing in long-term care facilities: Prospective evaluation of potential antimicrobial use and cost indicators. *Am J Infect Control.* 1999; 27(1): 10-19.
9. Mylotte JM, Keagle J. Benchmarks for antibiotic use and cost in long-term care. *J Am Geriatr Soc* 2005; 53:1117-1122.
10. Beardsley JR, Williamson JC, Johnson JW, Luther VP, Wrenn RH, Ohl CC. Show me the money: long-term financial impact of an antimicrobial stewardship program. *Infect Control and Hosp Epidemiol.* 2012;33(4):398-400



The Core Elements of
Antibiotic Stewardship
 for Nursing Homes

CHECKLIST





Checklist for Core Elements of Antibiotic Stewardship in Nursing Homes

The following checklist is a companion to the Core Elements of Antibiotic Stewardship in Nursing Homes. The CDC recommends that all nursing homes take steps to implement antibiotic stewardship activities. Before getting started, use this checklist as a baseline assessment of policies and practices which are in place. Then use the checklist to review progress in expanding stewardship activities on a regular basis (e.g., annually). Over time, implement activities for each element in a step-wise fashion.

LEADERSHIP SUPPORT

ESTABLISHED
AT FACILITY

1. Can your facility demonstrate leadership support for antibiotic stewardship through one or more of the following actions? Yes No
- If yes, indicate which of the following are in place (select all that apply)
- Written statement of leadership support to improve antibiotic use
 - Antibiotic stewardship duties included in medical director position description
 - Antibiotic stewardship duties included in director of nursing position description
 - Leadership monitors whether antibiotic stewardship policies are followed
 - Antibiotic use and resistance data is reviewed in quality assurance meetings

ACCOUNTABILITY

2. Has your facility identified a lead(s) for antibiotic stewardship activities? Yes No
- If yes, indicate who is accountable for stewardship activities (select all that apply)
- Medical director
 - Director or assistant director of nursing services
 - Consultant pharmacist
 - Other: _____

DRUG EXPERTISE

3. Does your facility have access to individual(s) with antibiotic stewardship expertise? Yes No
- If yes, indicate who is accountable for stewardship activities (select all that apply)
- Consultant pharmacy has staff trained/is experienced in antibiotic stewardship
 - Partnering with stewardship team at referral hospital
 - External infectious disease/stewardship consultant
 - Other: _____

ACTIONS TO IMPROVE USE

4. Does your facility have policies to improve antibiotic prescribing/use? Yes No
- If yes, indicate which policies are in place (select all that apply)
- Requires prescribers to document a dose, duration, and indication for all antibiotic prescriptions
 - Developed facility-specific algorithm for assessing residents
 - Developed facility-specific algorithms for appropriate diagnostic testing (e.g., obtaining cultures) for specific infections
 - Developed facility-specific treatment recommendations for infections
 - Reviews antibiotic agents listed on the medication formulary
 - Other: _____

5. Has your facility implemented practices to improve antibiotic use? Yes No

If yes, indicate which practices are in place (select all that apply)

- Utilizes a standard assessment and communication tool for residents suspected of having an infection
- Implemented process for communicating or receiving antibiotic use information when residents are transferred to/from other healthcare facilities
- Developed reports summarizing the antibiotic susceptibility patterns (e.g., facility antibiogram)
- Implemented an antibiotic review process/"antibiotic time out"
- Implemented an infection specific intervention to improve antibiotic use
Indicate for which condition(s): _____

6. Does your consultant pharmacist support antibiotic stewardship activities? Yes No

If yes, indicate activities performed by the consultant pharmacist (select all that apply)

- Reviews antibiotic courses for appropriateness of administration and/or indication
- Establishes standards for clinical/laboratory monitoring for adverse drug events from antibiotic use
- Reviews microbiology culture data to assess and guide antibiotic selection

TRACKING: MONITORING ANTIBIOTIC PRESCRIBING, USE, AND RESISTANCE

7. Does your facility monitor one or more measures of antibiotic use? Yes No

If yes, indicate which of the following are being tracked (select all that apply)

- Adherence to clinical assessment documentation (signs/symptoms, vital signs, physical exam findings)
- Adherence to prescribing documentation (dose, duration, indication)
- Adherence to facility-specific treatment recommendations
- Performs point prevalence surveys of antibiotic use
- Monitors rates of new antibiotic starts/1,000 resident-days
- Monitors antibiotic days of therapy/1,000 resident-days
- Other: _____

8. Does your facility monitor one or more outcomes of antibiotic use? Yes No

If yes, indicate which of the following are being tracked (select all that apply)

- Monitors rates of *C. difficile* infection
- Monitors rates of antibiotic-resistant organisms
- Monitors rates of adverse drug events due to antibiotics
- Other: _____

REPORTING INFORMATION TO STAFF ON IMPROVING ANTIBIOTIC USE AND RESISTANCE

9. Does your facility provide facility-specific reports on antibiotic use and outcomes with clinical providers and nursing staff? Yes No

If yes, indicate which of the following are being tracked (select all that apply)

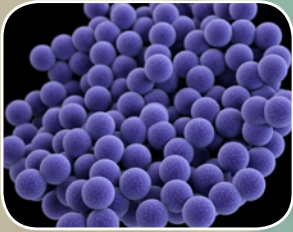
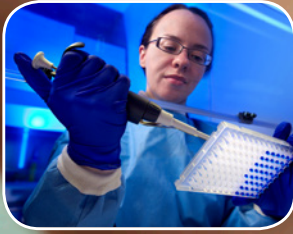
- Measures of antibiotic use at the facility
- Measures of outcomes related to antibiotic use (i.e., *C. difficile* rates)
- Report of facility antibiotic susceptibility patterns (within last 18 months)
- Personalized feedback on antibiotic prescribing practices (to clinical providers)
- Other: _____

EDUCATION

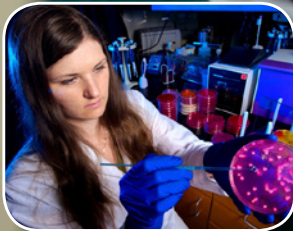
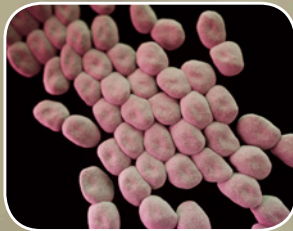
10. Does your facility provide educational resources and materials about antibiotic resistance and opportunity for improving antibiotic use? Yes No

If yes, indicate which of the following are being tracked (select all that apply)

- Clinical providers (e.g., MDs, NPs, PAs, PharmDs)
- Nursing staff (e.g., RNs, LPNs, CNAs)
- Residents and families
- Other: _____



The Core Elements of **Antibiotic Stewardship** for Nursing Homes



National Center for Emerging and Zoonotic Infectious Diseases
Division of Healthcare Quality Promotion



The Core Elements of Antibiotic Stewardship for Nursing Homes is a publication of The National Center for Emerging and Zoonotic Infectious Diseases within the Centers for Disease Control and Prevention.

Centers for Disease Control and Prevention
Thomas R. Frieden, MD, MPH, Director

National Center for Emerging and Zoonotic Infectious Diseases
Beth P. Bell, MD, MPH, Director

Suggested citation:

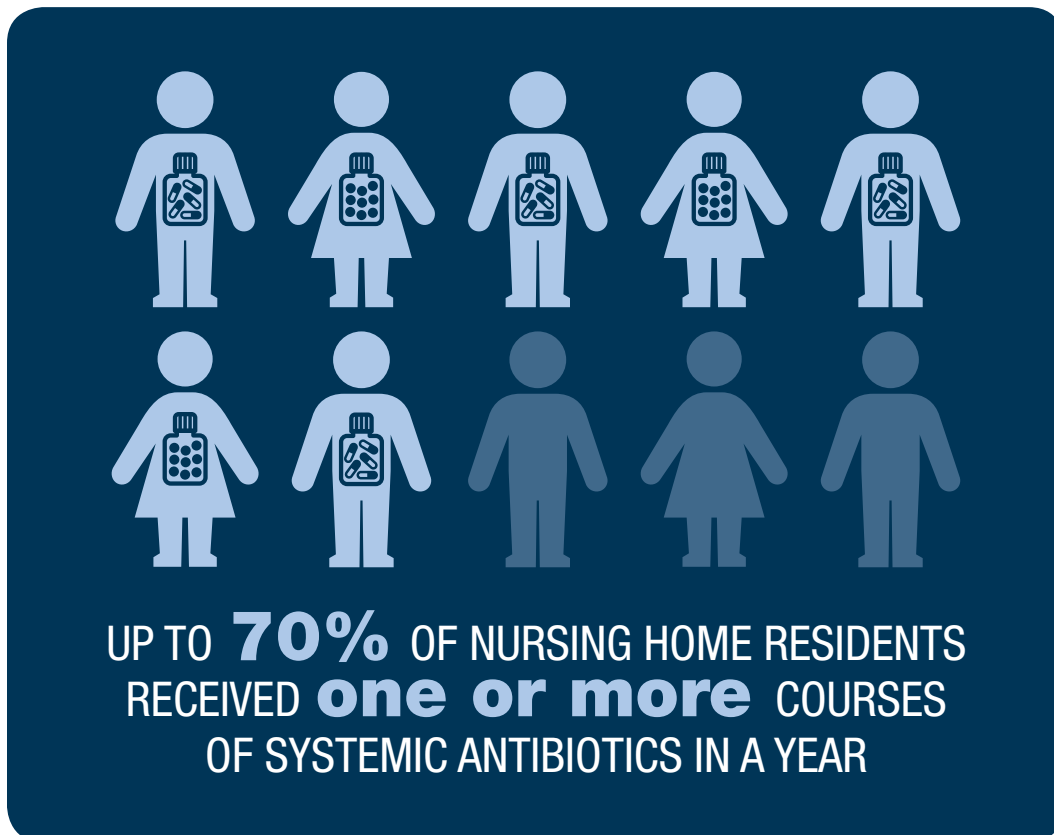
CDC. *The Core Elements of Antibiotic Stewardship for Nursing Homes*. Atlanta, GA: US Department of Health and Human Services, CDC; 2015. Available at: <http://www.cdc.gov/longtermcare/index.html>



Introduction

Improving the use of antibiotics in healthcare to protect patients and reduce the threat of antibiotic resistance is a national priority.¹ Antibiotic stewardship refers to a set of commitments and actions designed to “optimize the treatment of infections while reducing the adverse events associated with antibiotic use.”² The Centers for Disease Control and Prevention (CDC) recommends that all acute care hospitals implement an antibiotic stewardship program (ASP) and outlined the seven core elements which are necessary for implementing successful ASPs.² CDC also recommends that all nursing homes take steps to improve antibiotic prescribing practices and reduce inappropriate use.

Antibiotics are among the most frequently prescribed medications in nursing homes, with up to 70% of residents in a nursing home receiving one or more courses of systemic antibiotics when followed over a year.^{3,4} Similar to the findings in hospitals,^{5,6} studies have shown that 40–75% of antibiotics prescribed in nursing homes may be unnecessary or inappropriate.^{3,4} Harms from antibiotic overuse are significant for the frail and older adults receiving care in nursing homes. These harms include risk of serious diarrheal infections from *Clostridium difficile*, increased adverse drug events and drug interactions, and colonization and/or infection with antibiotic-resistant organisms.



This document adapts the [CDC Core Elements of Hospital Antibiotic Stewardship](#) into practical ways to initiate or expand antibiotic stewardship activities in nursing homes. While the elements are the same for both hospitals and nursing homes, the implementation of these elements may vary based on facility staffing and resources. Nursing homes are encouraged to work in a step-wise fashion, implementing one or two activities to start and gradually adding new strategies from each element over time. Any action taken to improve antibiotic use is expected to reduce adverse events, prevent emergence of resistance, and lead to better outcomes for residents in this setting.



Antibiotic stewardship refers to a set of commitments and activities designed to *“optimize the treatment of infections while reducing the adverse events associated with antibiotic use.”*

Summary of Core Elements for Antibiotic Stewardship in Nursing Homes



Leadership commitment

Demonstrate support and commitment to safe and appropriate antibiotic use in your facility



Accountability

Identify physician, nursing and pharmacy leads responsible for promoting and overseeing antibiotic stewardship activities in your facility



Drug expertise

Establish access to consultant pharmacists or other individuals with experience or training in antibiotic stewardship for your facility



Action

Implement **at least one** policy or practice to improve antibiotic use



Tracking

Monitor **at least one process** measure of antibiotic use and **at least one outcome** from antibiotic use in your facility



Reporting

Provide regular feedback on antibiotic use and resistance to prescribing clinicians, nursing staff and other relevant staff



Education

Provide resources to clinicians, nursing staff, residents and families about antibiotic resistance and opportunities for improving antibiotic use



Leadership Commitment

Nursing home leaders commit to improving antibiotic use. Facility leadership, both owners and administrators, as well as regional and national leaders if the facility is part of a larger corporation, can demonstrate their support in the following ways:

Write statements in support of improving antibiotic use to be shared with staff, residents and families

Include stewardship-related duties in position descriptions for the medical director, clinical nurse leads, and consultant pharmacists in the facility

Communicate with nursing staff and prescribing clinicians the facility's expectations about use of antibiotics and the monitoring and enforcement of stewardship policies

Create a culture, through messaging, education, and celebrating improvement, which promotes antibiotic stewardship



Accountability

Nursing homes identify individuals accountable for the antibiotic stewardship activities who have the support of facility leadership:

Empower the medical director to set standards for antibiotic prescribing practices for all clinical providers credentialed to deliver care in a nursing home and be accountable for overseeing adherence. To be effective in this role, the medical director should review antibiotic use data (see Tracking and Reporting section) and ensure best practices are followed in the medical care of residents in the facility.¹⁰

Empower the director of nursing to set the practice standards for assessing, monitoring and communicating changes in a resident's condition by front-line nursing staff. Nurses and nurse aides play a key role in the decision-making process for starting an antibiotic. The knowledge, perceptions and attitudes among nursing staff of the role of antibiotics in the care of nursing home residents can significantly influence how information is communicated to clinicians who are deciding whether to initiate antibiotic therapy. Therefore the importance of antibiotic stewardship is conveyed by the expectations set by nursing leadership in the facility.

Engage the consultant pharmacist in supporting antibiotic stewardship oversight through quality assurance activities such as medication regimen review and reporting of antibiotic use data.

Nursing home antibiotic stewardship leads utilize existing resources to support antibiotic stewards' efforts by working with the following partners:

Infection prevention program coordinator

Infection prevention coordinators have key expertise and data to inform strategies to improve antibiotic use. This includes tracking of antibiotic starts, monitoring adherence to evidence-based published criteria^{12,13} during the evaluation and management of treated infections, and reviewing antibiotic resistance patterns in the facility to understand which infections are caused by resistant organisms. When infection prevention coordinators have training, dedicated time, and resources to collect and analyze infection surveillance data, this information can be used to monitor and support antibiotic stewardship activities.

Consultant laboratory

Nursing homes contracting laboratory services can request reports and services to support antibiotic stewardship activities. Examples of laboratory support for antibiotic stewardship include developing a process for alerting the facility if certain antibiotic-resistant organisms are identified, providing education for nursing home staff on the differences in diagnostic tests available for detecting various infectious pathogens (e.g., EIA toxin test vs. nucleic amplification tests for *C. difficile*), and creating a summary report of antibiotic susceptibility patterns from organisms isolated in cultures. These reports, also known as antibiograms, help inform empiric antibiotic selection (i.e., before culture results are available) and monitor for new or worsening antibiotic resistance.¹⁴

State and local health departments

Nursing homes benefit from the educational support and resources on antibiotic stewardship and infection prevention which are provided by the Healthcare-Associated Infection (HAI) Prevention programs at state and local health departments.



Drug Expertise

Nursing homes establish access to individuals with antibiotic expertise to implement antibiotic stewardship activities. Receiving support from infectious disease consultants and consultant pharmacists with training in antibiotic stewardship can help a nursing home reduce antibiotic use and experience lower rates of positive *C. difficile* tests.¹¹ Examples of establishing antibiotic expertise include:

Work with a consultant pharmacist who has received specialized infectious diseases or antibiotic stewardship training. Example training courses include the Making a Difference in Infectious Diseases (MAD-ID) antibiotic stewardship course (<http://mad-id.org/antimicrobial-stewardship-programs/>), and the Society for Infectious Diseases Pharmacists antibiotic stewardship certificate program (<http://www.sidp.org/page-1442823>).

Partner with antibiotic stewardship program leads at the hospitals within your referral network.

Develop relationships with infectious disease consultants in your community interested in supporting your facility's stewardship efforts.



Take Action through Policy and Practice Change to Improve Antibiotic Use

Nursing homes implement prescribing policies and change practices to improve antibiotic use. The introduction of new policies and procedures which address antibiotic use should be done in a step-wise fashion so staff become familiar with and not overwhelmed by new changes in practice. Prioritize interventions based on the needs of your facility and share outcomes from successful interventions with nursing staff and clinical providers. Below are brief descriptions of policy and practice changes. For more details, see *Appendix A: Policy and practice actions to improve antibiotic use*.

Policies that support optimal antibiotic use

Ensure that current medication safety policies, including medication regimen review, developed to address Centers for Medicare and Medicaid Services (CMS) regulations¹⁵⁻¹⁷ are being applied to antibiotic prescribing and use.

Broad interventions to improve antibiotic use

Standardize the practices which should be applied during the care of any resident suspected of an infection or started on

an antibiotic. These practices include improving the evaluation and communication of clinical signs and symptoms when a resident is first suspected of having an infection, optimizing the use of diagnostic testing, and implementing an antibiotic review process, also known as an “antibiotic time-out,” for all antibiotics prescribed in your facility. Antibiotic reviews provide clinicians with an opportunity to reassess the ongoing need for and choice of an antibiotic when the clinical picture is clearer and more information is available.

Pharmacy interventions to improve antibiotic use

Integrate the dispensing and consultant pharmacists into the clinical care team as key partners in supporting antibiotic stewardship in nursing homes. Pharmacists can provide assistance in ensuring antibiotics are ordered appropriately, reviewing culture data, and developing antibiotic monitoring and infection management guidance in collaboration with nursing and clinical leaders.

Infection and syndrome specific interventions to improve antibiotic use

Identify clinical situations which may be driving inappropriate courses of antibiotics such as asymptomatic bacteriuria or urinary tract infection prophylaxis^{18,19} and implement specific interventions to improve use.



Tracking and Reporting Antibiotic Use and Outcomes

Nursing homes monitor both antibiotic use practices and outcomes related to antibiotics in order to guide practice changes and track the impact of new interventions. Data on adherence to antibiotic prescribing policies and antibiotic use are shared with clinicians and nurses to maintain awareness about the progress being made in antibiotic stewardship. Clinician response to antibiotic use feedback (e.g., acceptance) may help determine whether feedback is effective in changing prescribing behaviors. Below are examples of antibiotic use and outcome measures. For more details, see *Appendix B: Measures of antibiotic prescribing, use and outcomes*.

Process measures: Tracking how and why antibiotics are prescribed

Perform reviews on resident medical records for new antibiotic starts to determine whether the clinical assessment, prescription documentation and antibiotic selection were in accordance with facility antibiotic use policies and practices. When conducted over time, monitoring process measures can assess whether antibiotic prescribing policies are being followed by staff and clinicians.

Antibiotic use measures: Tracking how often and how many antibiotics are prescribed

Track the amount of antibiotic used in your nursing home to review patterns of use and determine the impact of new stewardship interventions. Some antibiotic use measures (e.g., prevalence surveys) provide a snap-shot of information; while others, like

nursing home initiated antibiotic starts and days of therapy (DOT) are calculated and tracked on an ongoing basis.^{20,21} Selecting which antibiotic use measure to track should be based on the type of practice intervention being implemented. Interventions designed to shorten the duration of antibiotic courses, or discontinue antibiotics based on post-prescription review (i.e., “antibiotic time-out”), may not necessarily change the rate of antibiotic starts, but would decrease the antibiotic DOT.

Antibiotic use data from nursing homes to improve antibiotic stewardship efforts is important both for individual facility improvements and for public health action. Expansion of electronic health records in nursing homes will allow for facilities to obtain systems which integrate pharmacy and laboratory data and make antibiotic use and resistance data to inform stewardship efforts more accessible to facility staff and leadership. CDC is working closely with many nursing home partners including providers, long-term care pharmacies, and professional organizations, to develop an Antibiotic Use (AU) reporting option for nursing homes within the CDC’s National Healthcare Safety Network (NHSN). The NHSN AU option allows for standardized antibiotic use data, submitted electronically, to be aggregated and summarized for developing facility-adjusted national benchmarks.

Antibiotic outcome measures: Tracking the adverse outcomes and costs from antibiotics

Monitor clinical outcomes such as rates of *C. difficile* infections, antibiotic-resistant organisms or adverse drug events to demonstrate that antibiotic stewardship activities are successful in improving patient outcomes. Nursing homes already tracking these clinical outcomes for their infection prevention program can submit data on *C. difficile* and selected antibiotic-resistant bacteria, such as methicillin-resistant *Staphylococcus aureus* (MRSA) and carbapenem-resistant *Enterobacteriaceae* (CRE) into the CDC’s NHSN Laboratory-identified event reporting module for long-term care facilities.



Education

Nursing homes provide antibiotic stewardship education to clinicians, nursing staff, residents and families. Effective educational programs address both nursing staff and clinical providers on the goal of an antibiotic stewardship intervention, and the responsibility of each group for ensuring its implementation.^{3,22} There are a variety of mechanisms for disseminating antibiotic education to nursing home staff including flyers, pocket-guides, newsletters or electronic communications; however, interactive academic detailing (e.g., face-to-face interactive workshops) has the strongest evidence for improving medication prescribing practices.²³

Nursing homes sustain improvements by incorporating both education and feedback to providers. One nursing home antibiotic stewardship intervention demonstrated a sustained reduction in antibiotic use for two years after the intervention by linking education with feedback on physician prescribing practices.²⁴ Another study showed a 64% reduction in inappropriate antibiotic use (i.e., prescriptions which did not adhere to guidelines), by providing feedback on individual physician prescribing practices and adherence to the guidelines over 12 months.²⁵

Nursing homes engage residents and their family members in antibiotic use and stewardship educational efforts to ensure clinicians have their support to make appropriate antibiotic use decisions. Working with residents and families will reduce the perception that their expectations may be a barrier to improving antibiotic use in nursing homes.^{26,27}



Conclusion

The core elements of antibiotic stewardship are the same for both hospitals and nursing homes. This guide provides examples of how these elements can be applied by nursing home leadership, clinicians and staff to monitor and improve antibiotic use. Nursing homes are encouraged to select one or two activities to start with and over time, as improvements are implemented, expand efforts to add new strategies to continue improving antibiotic use. Commit now to ensure antibiotic stewardship policies and practices are in place to protect patients and improve clinical care in nursing homes.



References

1. The White House. NATIONAL STRATEGY FOR COMBATING ANTIBIOTICRESISTANT BACTERIA. 2014; http://www.whitehouse.gov/sites/default/files/docs/carb_national_strategy.pdf Accessed 9/30/2014.
2. Centers for Disease Control and Prevention. Core Elements of Hospital Antibiotic Stewardship Programs. Atlanta, GA: US Department of Health and Human Services, CDC; 2014 <http://www.cdc.gov/getsmart/healthcare/implementation/core-elements.html> Accessed 9/30/2014
3. Lim CJ, Kong DCM, Stuart RL. Reducing inappropriate antibiotic prescribing in the residential care setting: current perspectives. Clin Interv Aging. 2014; 9: 165-177
4. Nicolle LE, Bentley D, Garibaldi R, et al. Antimicrobial use in long-term care facilities. Infect Control Hosp Epidemiol 2000; 21:537–45.
5. Dellit TH, Owens RC, McGowan JE, Jr., et al. Infectious Diseases Society of America and the Society for Healthcare Epidemiology of America guidelines for developing an institutional program to enhance antimicrobial stewardship. Clinical infectious diseases. 2007;44(2):159-177.
6. Fridkin SK, Baggs J, Fagan R, et al. Vital Signs: Improving antibiotic use among hospitalized patients. MMWR. Morbidity and mortality weekly report. 2014;63.
7. Gerwitz JH, Field TS, Harrold LR. Incidence and preventability of adverse drug events among older persons in the ambulatory setting. JAMA 2003;289: 1107–11.
8. Loeb MB, Craven S, McGeer A, et al. Risk factors for resistance to antimicrobial agents among nursing home residents. Am J Epidemiol 2003;157:40–7.
9. Centers for Disease Control and Prevention. Antibiotic resistance threats in the United States, 2013 Atlanta, GA: CDC; 2013.
10. Centers for Medicare and Medicaid Services. Summary of Requirements for Medical Director- 501 State Operations Manual (SOM); Appendix PP; Rev 107, 04-04-2014 Pages 626 – 633: https://cms.gov/manuals/Downloads/som107ap_pp_guidelines_itcf.pdf Accessed 9/30/2014.
11. Jump RLP, Olds DM, Seifi N et al. Effective antimicrobial stewardship in a long-term care facility through an infectious disease consultation service: Keeping a LID on antibiotic use. Infect Control Hosp Epidemiol 2012;33(12):1185-1192

12. Stone ND, Ashraf MS, Calder J, Crnich CJ, et al. Surveillance definitions of infections in long-term care facilities: revisiting the McGeer criteria. *Infect Control Hosp Epidemiol* 2012; 33: 965-977.
13. Loeb M, Bentley DW, Bradley S, et al. Development of minimum criteria for the initiation of antibiotics in residents of long-term care facilities: Results of a consensus conference. *Infect Control Hosp Epidemiol* 2001; 22: 120-4.
14. Furuno JP, Comer AC, Johnson JK, et al. Using antibiograms to improve antibiotic prescribing in skilled nursing facilities. *Infect Control Hosp Epidemiol*. 2014;35 (Suppl 3):S56-61.
15. Centers for Medicare and Medicaid Services. Summary of Requirements for Unnecessary Drugs - F329 State Operations Manual (SOM); Appendix PP; Rev 107, 04-04-2014 Pages 563–588: https://cms.gov/manuals/Downloads/som107ap_pp_guidelines_itcf.pdf Accessed 9/30/2014.
16. Centers for Medicare and Medicaid Services. Summary of Requirements for Medication Errors- F332 and 333 State Operations Manual (SOM); Appendix PP; Rev 107, 04-04-2014 Pages 430–439: https://cms.gov/manuals/Downloads/som107ap_pp_guidelines_itcf.pdf Accessed 9/30/2014.
17. Centers for Medicare and Medicaid Services. Summary of Requirements for Drug Regimen Review- F428 State Operations Manual (SOM); Appendix PP; Rev 107, 04-04-2014 Pages 539–548: https://cms.gov/manuals/Downloads/som107ap_pp_guidelines_itcf.pdf Accessed 9/30/2014.
18. Benoit SR, Nsa W, Richards CL et al. Factors associated with antimicrobial use in nursing homes: A multilevel model. *J Am Geriatr Soc* 2008; 56:2039–2044
19. Latour K, Catry B, Broex E et al. Indications for antimicrobial prescribing in European nursing homes: results from a point prevalence survey. *Pharmacoepidem and drug safety*. 2012; 21: 937–944
20. Mylotte JM. Antimicrobial prescribing in long-term care facilities: Prospective evaluation of potential antimicrobial use and cost indicators. *Am J Infect Control*. 1999; 27(1): 10-19.
21. Mylotte JM, Keagle J. Benchmarks for antibiotic use and cost in long-term care. *J Am Geriatr Soc* 2005; 53:1117-1122.
22. Nicolle LE. Antimicrobial stewardship in long-term care facilities: what is effective?. *Antimicrob Resist Infect Contr* 2014; 3:6. <http://www.aricjournal.com/content/3/1/6> Accessed 12/3/14
23. Loganathan M, Singh S, Franklin BD, Bottle A, Majeed A. Interventions to optimise prescribing in care homes: systematic review. *Age Aging* 2011; 40: 150-162
24. Schwartz DN, Abiad H, DeMarais PL. et al. An educational intervention to improve antimicrobial use in a hospital-based long-term care facility. *J Am Geriatr Soc*. 2007; 55(8), 1236-1242.
25. Monette J, Miller MA, Monette M, et al. Effect of an educational intervention on optimizing antibiotic prescribing in long-term care facilities. *J Am Geriatr Soc*. 2007; 55(8), 1231-1235
26. Lim CJ, Kwong MW, Stuart RL Antibiotic prescribing practice in residential aged care facilities--health care providers' perspectives. *Med J Aust*. 2014; 201(2):98-102.
27. Lohfeld L, Loeb M, Brazil K. Evidence-based clinical pathways to manage urinary tract infections in long-term care facilities: a qualitative case study describing administrator and nursing staff views. *J Am Med Dir Assoc*. 2007; 8(7):477-84.



Checklist for Core Elements of Antibiotic Stewardship in Nursing Homes

The following checklist is a companion to the Core Elements of Antibiotic Stewardship in Nursing Homes. The CDC recommends that all nursing homes take steps to implement antibiotic stewardship activities. Before getting started, use this checklist as a baseline assessment of policies and practices which are in place. Then use the checklist to review progress in expanding stewardship activities on a regular basis (e.g., annually). Over time, implement activities for each element in a step-wise fashion.

LEADERSHIP SUPPORT

ESTABLISHED
AT FACILITY

1. Can your facility demonstrate leadership support for antibiotic stewardship through one or more of the following actions? Yes No

If yes, indicate which of the following are in place (select all that apply)

- Written statement of leadership support to improve antibiotic use
- Antibiotic stewardship duties included in medical director position description
- Antibiotic stewardship duties included in director of nursing position description
- Leadership monitors whether antibiotic stewardship policies are followed
- Antibiotic use and resistance data is reviewed in quality assurance meetings

ACCOUNTABILITY

2. Has your facility identified a lead(s) for antibiotic stewardship activities? Yes No

If yes, indicate who is accountable for stewardship activities (select all that apply)

- Medical director
- Director or assistant director of nursing services
- Consultant pharmacist
- Other: _____

DRUG EXPERTISE

3. Does your facility have access to individual(s) with antibiotic stewardship expertise? Yes No

If yes, indicate who is accountable for stewardship activities (select all that apply)

- Consultant pharmacy has staff trained/is experienced in antibiotic stewardship
- Partnering with stewardship team at referral hospital
- External infectious disease/stewardship consultant
- Other: _____

ACTIONS TO IMPROVE USE

4. Does your facility have policies to improve antibiotic prescribing/use? Yes No

If yes, indicate which policies are in place (select all that apply)

- Requires prescribers to document a dose, duration, and indication for all antibiotic prescriptions
- Developed facility-specific algorithm for assessing residents
- Developed facility-specific algorithms for appropriate diagnostic testing (e.g., obtaining cultures) for specific infections
- Developed facility-specific treatment recommendations for infections
- Reviews antibiotic agents listed on the medication formulary
- Other: _____

5. Has your facility implemented practices to improve antibiotic use? Yes No

If yes, indicate which practices are in place (select all that apply)

- Utilizes a standard assessment and communication tool for residents suspected of having an infection
- Implemented process for communicating or receiving antibiotic use information when residents are transferred to/from other healthcare facilities
- Developed reports summarizing the antibiotic susceptibility patterns (e.g., facility antibiogram)
- Implemented an antibiotic review process/"antibiotic time out"
- Implemented an infection specific intervention to improve antibiotic use

Indicate for which condition(s): _____

6. Does your consultant pharmacist support antibiotic stewardship activities? Yes No
- If yes, indicate activities performed by the consultant pharmacist (select all that apply)
- Reviews antibiotic courses for appropriateness of administration and/or indication
 - Establishes standards for clinical/laboratory monitoring for adverse drug events from antibiotic use
 - Reviews microbiology culture data to assess and guide antibiotic selection

TRACKING: MONITORING ANTIBIOTIC PRESCRIBING, USE, AND RESISTANCE

7. Does your facility monitor one or more measures of antibiotic use? Yes No
- If yes, indicate which of the following are being tracked (select all that apply)
- Adherence to clinical assessment documentation (signs/symptoms, vital signs, physical exam findings)
 - Adherence to prescribing documentation (dose, duration, indication)
 - Adherence to facility-specific treatment recommendations
 - Performs point prevalence surveys of antibiotic use
 - Monitors rates of new antibiotic starts/1,000 resident-days
 - Monitors antibiotic days of therapy/1,000 resident-days
 - Other: _____

8. Does your facility monitor one or more outcomes of antibiotic use? Yes No
- If yes, indicate which of the following are being tracked (select all that apply)
- Monitors rates of *C. difficile* infection
 - Monitors rates of antibiotic-resistant organisms
 - Monitors rates of adverse drug events due to antibiotics
 - Other: _____

REPORTING INFORMATION TO STAFF ON IMPROVING ANTIBIOTIC USE AND RESISTANCE

9. Does your facility provide facility-specific reports on antibiotic use and outcomes with clinical providers and nursing staff? Yes No
- If yes, indicate which of the following are being tracked (select all that apply)
- Measures of antibiotic use at the facility
 - Measures of outcomes related to antibiotic use (i.e., *C. difficile* rates)
 - Report of facility antibiotic susceptibility patterns (within last 18 months)
 - Personalized feedback on antibiotic prescribing practices (to clinical providers)
 - Other: _____

EDUCATION

10. Does your facility provide educational resources and materials about antibiotic resistance and opportunity for improving antibiotic use? Yes No
- If yes, indicate which of the following are being tracked (select all that apply)
- Clinical providers (e.g., MDs, NPs, PAs, PharmDs)
 - Nursing staff (e.g., RNs, LPNs, CNAs)
 - Residents and families
 - Other: _____